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Illness Representations, Cognitive Deficits & Mood In Stroke

Patients

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University of St. Andrews

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Declaration


(i) I, Martin P. Morthland, hereby certify that this thesis, which is approximately 51,000 words in length has been written by me, that it is the record of work carried out by me and that it has not been submitted in any previous application for a higher degree.

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(ii) I was admitted as a research student in September, 1997 and as a candidate for the degree of Ph.D. in October 1998; the higher study for which this is a record was carried out in the University of St. Andrews between 1997 and 2001.

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Abstract

The studies contained in this thesis sought to replicate findings in the health psychology and neuropsychology literature regarding predictors of stroke recovery (perceived control and attention control). Following from the premises that perceived control may be thought of as an illness representation and, that attention control is a cognitive skill, this thesis also studied the relationships between cognitive impairment and illness representations. A third construct that has also been shown in the literature to have significant relationships with cognitive deficit, illness representations and, stroke is mood. The relationship between cognitive impairment and mood was therefore, also examined. Results from the first study (N=56) replicated the finding that attention control predicts stroke recovery. Significant differences were also found between patients with and without cognitive deficit regarding illness representations and mood at 1yr. These results, however, were mixed and gave no clear support for cognitive deficit resulting in greater illness impact. The second study was designed to improve on the first as it included a larger sample and, was conducted longitudinally at three time-points (initial, 6mo. and 1yr. time-points). Results from the second study (N=65) concluded that initial perceived control predicted stroke recovery at a trend level and that 1yr. attention control significantly predicted stroke recovery. These two constructs could not be said, however, to account for independent amounts of explained variance for stroke recovery. The majority of results regarding cognitive deficit and illness representations were non-significant. Results regarding cognitive deficit and mood were non-significant. There was some evidence, however, to suggest that a curvilinear relationship may exist between cognitive status and mood. Taking the findings in past literature and those of studies 1 and 2, the overall results were inconclusive. Suggestions for improvements in future studies are posed in the discussion chapter.

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CHAPTER 1

Introduction

1.1 Stroke

Cerebrovascular Accident (CVA), or Stroke, is the major cause of severe disability in Scotland (Robertson, Baddeley, Ridgeway, Greenfield, Parr & Tongue 1997). As such, it remains an area of science that warrants much attention. The incidence of stroke in Britain is approximately 114/100,000. The prevalence of stroke in Britain is 518/100,000. Stroke morbidity in Britain leaves 24% of the population with severe disability (MacWalter, R., 1999).

Stroke is defined as, "A rapidly developing episode of neurologic dysfunctioning lasting more than 24 hours or leading to death and which is presumed to be due to a disturbance in the vascular supply to the brain, either cerebral infarction or intracranial hemorrhage" (Warlow, 1984).

There are primarily three types of strokes: obstructive (ischemic), transient ischemic attacks (TIAs) and hemorrhage. Ischemic and TIAs are caused by the same process of a build-up of fat deposits. Hemorrhage has a different cause which is the weakening and rupture of blood vessels and arteries.

Ischemic strokes account for approximately 85% of all stroke cases (Anderson, 1985). This type of stroke occurs when a build-up of fat deposits occlude an artery or vein to the point that blood flow is stopped. An embolus occurs when the fat deposit of the clot, or thrombus, breaks off and travels through the artery until it lodges into a smaller blood vessel of the brain. Since blood vessels get

progressively smaller from the arteries, the size of the embolus determines the location of obstruction and degree of damage.

Transient ischemic attacks are temporary obstructions of blood vessels that last less than 24 hours (Warlow, 1984). During this time recovery from symptoms occurs. The cause is the same for ischemic strokes but, the deposits are passed through the cerebral vasculature before extensive damage can occur. During the period of occlusion, however, the symptoms are the same as with a normal stroke.

The most dangerous form of stroke is hemorrhage due to the extent of damage which can result. There are two common types of cerebral hemorrhage: intracranial and subarachnoid. The most common sites for intracranial hemorrhage are the basal ganglia, internal capsule, pons and the cerebellum (Anderson, 1985). The site of subarachnoid hemorrhage is within the subarachnoid space. The cause is usually due to a berry aneurysm, usually at the bifurcation of the middle cerebral artery (Anderson, 1985).

The effects of stroke

Once a stroke occurs it may affect one or more domains for the patient. Some of these domains have been studied more recently than others. It may affect them physically, their beliefs about their stroke and themselves (an area of more recent study), cognitively (i.e., intellectually) and, emotionally. The patients' physical disability is concerned with how they are limited in what they can achieve in their daily lives after having a stroke. Their beliefs concern their perceptions about their stroke. Their cognitive status is concerned with their cognitive skills

which may have been affected by stroke. And, their emotional status is concerned with how their stroke may have affected their mood. Each of these effects of stroke will be discussed in following sections.

Physical symptoms may be one of the first signs that a stroke has occurred. Ischemia in the carotid area, for example, may cause loss of strength and/or dexterity and/or numbness in the opposite hand, arm, leg or face in isolation or various combinations (Warlow, 1984).

Stroke patients may also have particular beliefs or perceptions regarding their stroke and themselves, e.g., what caused it, how long it will last, what the consequences are, how to cope with their symptoms, how to avoid having another and, if they perceive themselves as having control over their recovery.

Cognitive deficits often follow a stroke. Such cognitive deficits may affect a patient's general intellectual ability, perceptual skills, executive functioning, constructional skills, memory, visuospatial skills or any combination of the above (Crawford et al., 1992).

Emotional impairment has also been demonstrated to accompany stroke. Patients may experience depression or anxiety. Depression may be less severe in the patient presenting with apathy or dysthymia (Lezak, 1995).

1.2 Control

As stated earlier, following a stroke patients may have unique perceptions regarding aspects about their stroke and themselves. Such perceptions may even

differ for individuals who have similar strokes. One such perception is that of *control*. Research has shown the importance of control in psychological functioning, and that a sense of control is predictive of mental and physical health (Johnston, M., Morrison, V., Pollard, B. & MacWalter, R., 2000; Lachman & Burack, 1993; Fiske & Taylor, 1991; Thompson & Spacapan, 1991; Bandura, 1989; Strickland, 1989; Rodin, 1986).

Explaining the mechanism of control, however, is achieved in different ways in the literature. Researchers have taken different approaches in defining what constitutes control and therefore, what is important when studying it as a psychological construct. Such examples of the diversity of constructs used in defining and researching control are: personal control, sense of control, locus of control, vicarious control, illusory control, primary control, secondary control, self-efficacy, causal attributions and outcome expectancy (Skinner, 1996). One major domain of control is termed, *perceived control*. As defined by Wallston et al. (1989) perceived control is, "The belief that one can determine one's own internal states and behavior, influence one's environment, and/or bring about desired outcomes" (p.5). Under the domain of perceived control, influential behavioural models have been created which incorporate similar, yet distinguishable, constructs of control. Two main examples are: Social Cognitive Theory (self-efficacy; Bandura, 1989) and, Theory of Planned Behaviour (perceived behavioural control, Ajzen, 1986). The terms self-efficacy, perceived behavioural control and perceived control are often substituted for one another in the literature; leading to confusion amongst researchers when trying to explain the relationship between control and

behaviour. These terms, however, are distinct in their definitions. In a paper by Terry & O'Leary (1995) the differences between these constructs are highlighted. As for the difference between self-efficacy and perceived behavioural control they state,

Bandura proposes that there are two types of expectancies that will influence people's decisions not to engage in a particular behaviour. In the first instance, people may be discouraged from performing a behaviour, because they doubt their ability to perform it (efficacy expectancies). Second, even if people are confident that they will be able to perform a behaviour, they may be reluctant to do so, if they perceive that the behaviour will not lead to the desired outcome. The latter type of expectancy is referred to by Bandura as an outcome expectancy and can be regarded as being similar to the notion of perceived behavioural control (unconfounded with efficacy expectancies) (p. 202).

There is also a distinction between self-efficacy and perceived behavioural control with regard to the outcome. In a paper by Abraham, Sheeran & Johnston (1998) they state, 'Self-efficacy has been typically defined in terms of perceived personal competence or confidence (e.g., 'I believe I can do X successfully'.) while perceived behavioural control also includes measures of perceived barriers and difficulties (e.g., 'Doing X would be difficult'.). In other words, self-efficacy is one's belief that one is able to perform a behaviour. And, perceived behavioural control is one's belief of engaging in the behaviour based on its value to the outcome; the distinction being made between ability alone and ability plus the valuation to outcome. The

term perceived control, as defined by Wallston et al., is a culmination of these two distinctions by saying that one believes ones' self to be able to perform a behaviour and by doing so, the desired outcome (which they can control) will result.

Perceived Control

Perceived control has been investigated in the literature regarding different diseases/conditions, e.g., stroke, multiple sclerosis, motor neuron disease, joint fractures, arthritis (Fisher & Johnston, 1996a,b; Fisher & Johnston, 1998; Johnston, Morrison et al., 1999; Johnston et al., 1992; Marteau & Johnston, 1987; Partridge & Johnston, 1989) . It also has been used to help explain variance in the resultant disability of diseases/conditions such as stroke (Fisher & Johnston, 1996a,b; Fisher & Johnston, 1998; Johnston, Morrison et al., 1999; Johnston et al., 1992; Partridge & Johnston, 1989).

Perceived Control Predicts Recovery

Johnston, Morrison et al. (1999) showed in their study how psychological variables (perceived control) predicted functional recovery in stroke patients. Stroke patients were recruited (N = 101) and studied at three time points: 10-20 days after admission, 1mo. after hospital discharge and, 6mo. after hospital discharge (N = 71). The measures used to assess disability/recovery were the Barthel Index of activities of daily living (Mahoney & Barthel, 1965) and an observer-assessed measure (OAD) containing 13 movements or activities (Partridge, Johnston & Edwards, 1987). Initial levels of disability were accounted for in relation to final

disability by measuring a standardized regression residual (recovery). The measure used to assess perceived control was the Recovery Locus Control Scale (Partridge & Johnston; 1989). The bivariate relationships (Pearson's correlations) between 1 and 6 mo. perceived control and 1 and 6mo. Barthel were significant (1mo. RLOC & 1mo. Barthel: $r = .31, p < .05$; 1mo. RLOC & 6mo. Barthel: $r = .27, p < .05$; 6mo. RLOC & 6mo. Barthel: $r = .25, p < .05$). One-month perceived control did not significantly predict recovery. Six month perceived control, however, did significantly predict recovery ($r = .29, p < .05$). There were also significant correlations between 1 and 6mo. perceived control and 1 and 6mo. observer-assessed disability: 1mo RLOC & 1mo. OAD: $r = .36, p < .01$; 1mo. RLOC & 6mo. OAD: $r = .32, p < .05$; 6mo. RLOC & 6mo. OAD: $r = .33, p < .01$). These results supported the findings of Partridge & Johnston (1989), Johnston et al. (1992) and, Johnston, Morrison et al. (1999) where perceived control was shown to predict recovery.

Manipulating Perceived Control

Following the finding that perceived control predicted recovery, Fisher & Johnston (1996a) experimentally studied the possibility that perceived control moderated the relationship between impairment and disability. A cohort of 50 chronic low back pain patients were used to examine the relationship between manipulating perceived control and resultant disability. The patients' mean duration of reported pain was 7.6 years ($sd = 7.0$). The patients were randomly allocated to either the increase or decrease perceived control groups. Perceived control was

increased by instructing the patients to tell the examiner times in which they, the patients, felt 'in control and achieving things well'. The patients were asked to give examples of such times. Conversely, the other group (decrease perceived control) were instructed to report times in which they have felt 'out of control and unable to achieve something you set out to do'. Patients again, reported a minimum of three such examples.

The disability assessment task was to hold a weight until uncomfortable. Weight and time were recorded for each patient.

There were four areas of measurement: disability, perceived control, pain and, emotional distress. Disability was measured using a behavioural measure of disability (bmd), a visual analogue rating and, the Oswestry Low Back Pain Disability Questionnaire (ODQ; Fairbank, Couper, Davies & O'Brien, 1980). The behavioural measure was a task that required the patients to hold a plastic bag which was then filled with packets of rice until the weight was as great as the patients felt comfortable holding. The visual analogue scale for disability ranged from low 'Not at all disabled' to high 'As disabled as it's possible to be'. The ODQ was given primarily as a measure of baseline disability since it is not sensitive to changes over a short period of time. It also does not incorporate any behavioural assessment. Within the questionnaire there are ten areas that assess different activities. The third section deals with lifting and, therefore, was most appropriate as it lends itself to being particularly applicable against the measure of lifting/holding weight.

For perceived control, two measures were used: a visual analogue scale and a version of the Modified Multidimensional Health Locus of Control Scale

(MMHLC; Wallston, Wallston & DeVellis, 1978). The visual analogue scale was in the same format as with disability. Statements ranged from low 'Not at all in control' to high 'As in control as it's possible to be'. Using the MMHLC allowed the examiners to ascertain the degree to which patients felt that their pain was due to external or internal factors (locus of control).

Pain was measured in two ways. The first was a visual analogue rating. The scale ranged from low 'no pain' to high 'worst pain'. The second measure was the McGill Pain Questionnaire (MPQ; Melzack, 1975). Present Pain intensity scores and Total Number of Words Chosen from the MPQ were used in measuring the levels of pain.

Emotional distress was measured also in two ways: a visual analogue rating scale and the General Health Questionnaire (GHQ 28; Goldberg, 1978). The rating scale ranged from low 'not at all anxious' to high 'as anxious as it's possible to be'. The GHQ 28 is a measure of emotional disorder. It gives an overall score on four factors: anxiety, depression, somatic symptoms and social dysfunction.

The above measures were administered to all of the patients at recruitment, establishing baseline measures. With exception to age, there were no significant differences between the patients of the two experimental groups: $F(1, 48) = 5.5$, $p < .05$.

Significant differences were found for perceived control as a result of cognitive manipulation. Group one's (increase perceived control) measure of perceived control increased from 51.0 to 69.0: $F(1, 24) = 21.0$, $p < .01$. Group two's (decrease perceived control) measure of perceived control decreased from 49.5 to

32.2: $F(1, 24) = 9.33, p < .01$. Taking all subjects into account, there were no significant correlations between disability, perceived control and trait measures of pain, emotional distress nor locus of control. However, baseline perceived control was highly correlated with change in perceived control ($r = .60, p < .001$).

The key finding was that the cognitive manipulation was found to significantly affect perceived control and the time of the behavioural task (i.e., disability). However, perceived control was not shown to moderate the relationship between impairment and disability (as defined by Baron & Kenny, 1986).

The importance of the above findings are two-fold. Firstly, that patients' amount of recovery was predicted by their level of perceived control. And, secondly, levels of perceived control were experimentally manipulated in predicted directions which, affected disability in the predicted directions.

1.3 Attention Control

As stated earlier, many patients are affected with cognitive deficits following a stroke. Patients may present with one or more cognitive deficits, e.g., memory, verbal skills, visuomotor skills, abstract reasoning skills, etc. One cognitive ability, central to this thesis, is *attention control*. As defined by Robertson, Manly, Andrade, Baddeley & Yiend (1997) attention control is, 'The ability to self-sustain mindful, conscious processing of stimuli whose repetitive, non-arousing qualities would otherwise lead to habituation and distraction to other stimuli' (p.747). In short, it is the ability to attend to a given task for a prolonged

length of time without being distracted.

Attention Control Predicts Recovery

An important aspect of attention control research is that it, like perceived control, has been shown in the literature to predict stroke recovery using similar designs and methodologies (Robertson, Ridgeway, Greenfield & Parr, 1997). As with perceived control, it has also been shown that attention control can be experimentally manipulated (Robertson, Baddeley, Ridgeway et al., 1997).

In a study by Robertson et al. (1997), stroke patients were recruited to examine the relationship between attention control and functional recovery over 2yrs. (N = 47) Patients were examined at 2mo. and at 2yrs. Functional independence (recovery) was assessed using: the Barthel Index, the Nottingham Extended Activities of Daily Living (Lincoln & Gladman, 1992), the Nine Hole Peg Test (Mathiowetz, Volland, Kashman & Weber; 1985) and, the Rivermead Mobility Scale (Collen, Wade, Robb & Bradshaw; 1991). The measure used to assess attention control was the elevator counting subtest of the Test of Everyday Attention (Robertson, Ward, Ridgeway & Nimmo-Smith, 1994). This test consists of a series of tones presented at different intervals which the patient must keep count of silently. At the end of each presentation the patient reports the total number of tones counted. A more complete description will be provided in the next chapter. Two-month performance on elevator counting was significantly correlated with 2yr. Nine Hole Peg Test ($r = -.42, p < .003$) and 2yr. elevator counting was significantly correlated with 2yr. Nottingham ADL ($r = .32, p < .04$), Rivermead Mobility ($r = .33,$

$p < .03$) and, Nine Hole Peg Test ($r = -.41$, $p < .004$). Predictively, multiple regression analysis showed that attention control (2mo. elevator counting) significantly predicted functional recovery at 2yrs. (Nine Hole Peg Test) ($F(2, 42) = 38.5$, $p < .0001$).

Manipulating Attention Control

As with the perceived control research, following the finding that attention control predicted recovery, research was carried out to examine if patients' levels of attention control could be experimentally manipulated. Robertson et al. (1997) examined the effectiveness of a stroke rehabilitation regime using three experimental groups: no treatment, physical exercise and, attention control. The study used 42 acute stroke patients (mean age = 63.7, $sd = 11.4$). Groups did not differ from one another on any of the inclusion criteria except for age. Patients were randomly allocated to the three conditions. Assessments occurred at 1mo. and at 6mo.

Both treatment groups were visited for treatment five times for forty-five minutes each time for one month. The attention control group received training for attention improvements. This entailed listening to audio tapes which instructed the patients to monitor their attention and relax their breathing.

The exercise group received training in a similar manner. They also listened to audio tapes. The tapes instructed the patients in a non-specialist physical exercises recommended by the Stroke Association of the U.K.

Functional status was measured by the Barthel Index and the Nottingham Extended Activities of Daily Living Scale (EADL; Lincoln & Gladman, 1992). Ratings of attentional functioning, by self and informants, was measured by the Cognitive Failures Questionnaire (CFQ; Broadbent et al.; 1982). These measures were given upon recruitment to provide baseline levels of disability and attention.

Emotional function was measured by the Beck Depression Inventory (BDI; Beck et al.; 1961) and the Hospital Anxiety and Depression Questionnaire. Actual attentional performance was measured by the Test of Everyday Attention. The Health Locus of Control Scale (HLCS; Wallston, Kaplan & Maides; 1976) was given at six months as, '... a measure of perceived control over recovery after stroke' (p.5).

One month results showed a significant main effect for EADL: $F(2,36) = 11.4, p < .0001$, with post hoc tests showing significance for all comparisons of treatment: attention control training (ACT) vs. no treatment (NT) ($p < .0001$), physical exercise (PE) vs. NT ($p < .0001$) and, ACT vs. PE ($p < .014$). The PE group showed the highest score on EADL (18 out of 20). The ACT group showed the next highest score on EADL (16 out of 20). And, the NT group showed the lowest EADL score (12 out of 20). Significant main effects were also found for the Barthel Index: $F(2,36) = 3.7, p < .03$. Post hoc significance was found for all comparisons of treatment except for attention control vs. PE: ACT vs. NT ($p < .0001$), PE vs. NT ($p < .0001$). There were no significant effects between groups for anxiety or depression.

Six month follow-up showed a main effect for EADL: $F(2,36) = 6.9$, $p < .003$. Post hoc tests showed significant differences between all comparisons except for attention control and physical exercise: ACT vs. NT ($p < .0003$), PE vs. NT ($p < .004$). The ACT group showed the highest EADL score (17.5 out of 20). The PE group showed the next highest EADL score (16.5 out of 20). And, the NT group showed the lowest EADL score (12.5 out of 20). A main effect was also found for the Barthel Index: $F(2,34) = 11.4$, $p < .0002$. Post hoc tests showed significant differences between all comparisons except for attention control and physical exercise: ACT vs. NT ($p < .0002$), PE vs. NT ($p < .0003$). Again, there were no significant differences between groups on anxiety or depression. Results from the HLCS showed no significant differences between groups.

It was determined that improvements in EADL and Barthel Index were in fact associated with changes in sustained attention performance. Six-month follow-up sustained attention performance was covaried and eliminated differences in functional status. No differences were found between groups on 6mo. Barthel after a one-way ANCOVA on 6mo. Barthel, covarying 6mo. Elevator Counting (the primary measure of sustained attention) scores, was performed: $F(2,34) = .02$, ns. The results confirmed their hypothesis of a mediating relationship between attention control and functional status.

As shown previously, perceived control predicts recovery and levels of perceived control can be manipulated in the desired direction. Similarly, attention control predicts recovery and levels of it can also be manipulated in the desired

direction. The relationships between perceived control, attention control and recovery and, perceived control and attention control are therefore of interest to this thesis and will be discussed in greater detail in later chapters.

1.4 Illness Representations

Returning to the fact that stroke patients have been shown to maintain unique beliefs or perceptions (of which perceived control was one example) following stroke, studies have examined these *illness representations* in greater detail (Lau & Hartman, 1983; Leventhal et al., 1983; Lau, R., Bernard, T. & Hartman, K., 1989; Weinman et al., 1996). Leventhal, Meyer & Nerenz (1980) introduced the utility of studying illness representations by showing that patients develop a theoretical framework when faced with illness which allows them to understand and cope more effectively. Within the last decade there has been increasing attention to the notions of how patients' health beliefs affect health behaviours and outcomes. Skelton & Croyle (1991) provide an overview of this quickly-developing area of health psychology. They state, 'An important new line of theory and research can be traced, a line of work concerning basic questions of how the individual thinks about health and illness. This is the study of health and illness representation (p.1). Leventhal & Crouch (1997) explain that our history (medical and psychosocial) forms a basis of memory from which we draw upon to maintain our health and, avoid/control disease. These memories form a knowledge base from which, 'in combination with new somatic sensations and information

about illness in other persons generate illness representations whose attributes define the *cause*, *identity* (symptoms and label), potential *consequences*, possibility for *control* and *time-lines* associated with each of these attributes (time for development: for cure, disability and/or death)' (p.77 In *Perceptions of Health and Illness*, Petrie & Weinman, 1997). These five illness representations are based on work by Leventhal et al. (1983) and Lau & Hartman (1983). Leventhal et al. developed a model to predict behaviour called the Self-Regulatory model (Figure 1.1).

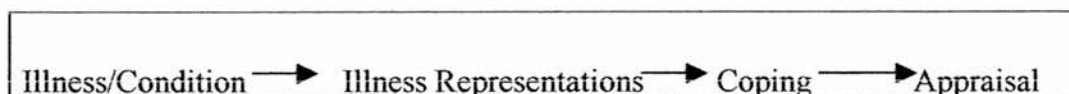


Figure 1.1 Self-regulatory model

This model suggests that when one is faced with an illness/condition specific illness representations are formed which lead to a coping response ending in appraisal (outcome behaviour). The five illness representation components: identity, cause, timeline, consequences & cure/control were identified through content analysis on data gathered from hypertension and cancer patients (Leventhal, 1983).

Our *identity* of an illness concerns what label is attributed to it and its symptoms. For an elderly person who exhibits symptoms of hemiparesis and slurred speech a diagnosis, or identity, of 'stroke' might be attributed. Identity also goes beyond the label itself. For example, abstractions such as 'malignant' may

accompany the defining label of 'cancer'. Though the five components within the illness representation stage of the Self Regulation model do not occur in any particular order, it can be argued that identity must either precede the other components or, that it is simultaneous to the first component to be ascribed. The function of identity provides a basis from which other attributes can be made. That is, one must give a label to their symptoms before (or at the same time) *cause*, *control/cure*, *timeline* & *consequences* can be given meaning.

The attribution of *cause* concerns beliefs about that which brought the illness/condition into being. As with stroke, for example, the determined cause may be anything from diet, stress, genetics, etc.

The *timeline* of an illness/condition is imputed based on how long the illness/condition is thought to typically last and in what manner. The timeline can be thought in terms of: chronic, acute, cyclic, etc. It is determined based on some idea of how long and in what manner the symptoms will occur.

The *consequences* of an illness or condition is ascribed based on the known severity of the illness/condition. As the name implies, it is based upon what result may be expected due to the illness/condition. It reflects how the person believes their illness or condition will impact their physical, social and psychological functioning (Weinman et al., 1996).

The above four components were created from the work of Leventhal and his colleagues based on research with seriously/chronically ill patients. Lau &

Hartman (1983) were responsible for identifying the final component, *cure/control*, by applying Leventhal et al.'s procedures with less severe patients. The five components were later confirmed in a study by Lau, Bernard & Hartman (1989). The *cure/control* component reflects the individual's belief about the degree and manner their illness/condition may be amenable to cure or management. Depending upon how the individual represents their illness/condition, this component can either be cure (the individual perceives their illness/condition as having a permanent, single-occasion, amelioration of symptoms) or control (the individual perceives their illness as being chronic and incurable).

A study conducted to examine patients' illness representations was conducted by Heijmans (1998). This study tested Leventhal's self regulation model using ninety-eight chronic fatigue syndrome (CFS) patients. It examined the relationship between illness representations, coping behaviour and adaptive outcome. Illness representations were measured using the Illness Perception Questionnaire (IPQ; Weinman et al., 1996). The IPQ was used to measure: identity, cause, timeline and control/cure. For the cause component, a score was extracted by partitioning it into three causal areas: biological, psychological and environmental. Coping was measured using the shortened version of the Utrecht Coping Questionnaire (Schreurs et al., 1993). Three coping strategies were measured: problem-focused coping, behavioural-avoidant coping and, cognitive-avoidant coping. Adaptive outcome was measured using four scales from the 36-Item Short-Form Health Survey (Ware & Sherbourne, 1992). These four scales were: Physical

Functioning Scale (a measure of physical functioning), Social Functioning Scale (a measure of social functioning), Mental Health Scale (a measure of psychological adjustment) and, the Vitality Scale (a measure of subjective well-being).

Correlational data showed significant relationships between illness representations, coping strategies and adaptive outcome. Problem-focused coping was found to significantly correlate with: timeline ($r = -.23, p < .001$); control/cure ($r = .25, p < .001$) and environmental cause ($r = .27, p < .0001$). Behavioural-avoidant coping significantly correlated with: identity ($r = .22, p < .01$) and, consequences ($r = .27, p < .001$). Cognitive-avoidant coping significantly correlated with: identity ($r = .37, p < .001$); timeline ($r = .34, p < .001$) and control/cure ($r = -.30, p < .001$). The Physical Functioning Scale significantly correlated with: identity ($r = -.36, p < .001$); timeline ($r = -.24, p < .001$) and, consequences ($r = -.38, p < .001$). The Mental Health Scale significantly correlated with: identity ($r = -.39, p < .001$); timeline ($r = -.32, p < .001$); psychological cause ($r = -.28, p < .001$) and, consequences ($r = -.22, p < .01$). The Vitality Scale significantly correlated with: identity ($r = -.41, p < .001$); timeline ($r = -.25, p < .001$); control/cure ($r = .24, p < .01$); biological cause ($r = -.23, p < .01$) and, consequences ($r = -.37, p < .001$).

Correlations between coping strategies and adaptive functioning were, in general, not significant. However, cognitive-avoidant coping was significantly related to: social functioning ($r = -.26, p < .01$); mental health ($r = -.63, p < .001$) and, vitality ($r = -.30, p < .001$). Mental health was also significantly related to: problem focused coping ($r = .25, p < .01$) and seeking of social support ($r = .32, p < .001$).

In determining whether coping strategies and illness representations predict different aspects of adaptive outcome, step-wise regression analysis was performed. The first step entered into the analysis included gender, age and duration of illness. These three variables were not significantly related to adaptive outcome. For step two, coping strategies were entered. Cognitive-avoidant coping was found to negatively predict: social functioning ($\beta = -.25, p < .01$); mental health ($\beta = -.51, p < .001$) and, vitality ($\beta = -.30, p < .001$). The third step entered was illness representations. Identity was found to negatively predict: physical functioning ($\beta = -.30, p < .001$); mental health ($\beta = -.25, p < .01$) and, vitality ($\beta = -.32, p < .001$). Biological cause was found to negatively predict vitality ($\beta = -.27, p < .01$). Psychological cause was found to negatively predict mental health ($\beta = -.24, p < .01$). Consequences was found to negatively predict: physical functioning ($\beta = -.54, p < .001$); social functioning ($\beta = -.38, p < .001$) and, vitality ($\beta = -.25, p < .05$).

The results suggested specific relationships between coping strategies, illness representations and adaptive outcome. The use of cognitive-avoidant coping, as suggested by the data, explains poorer social functioning, mental health and vitality. Regarding illness representations, identifying with illness (identity) explains poorer physical functioning, mental health and vitality. Consideration of illness as having a biological cause is related to poorer vitality. Illness that is believed to have a psychological cause is related to poorer mental health. Illness that is considered to have more serious consequences is related to poorer mental health. Illness that is considered to have more serious consequences is related to

poorer physical functioning, social functioning and vitality.

The above studies point to the importance that illness representations have on disability and recovery. They also exemplify how those differences influence coping and adaptive outcome. With the exception of perceived control, the study of illness representations for stroke patients has been seemingly neglected in the literature. Such examinations will be discussed in later chapters.

1.5 Mood

Another area of investigation following stroke is mood. Several researchers have investigated the effect of stroke on mood (Fisher & Johnston, 1996a; Fisher & Johnston, 1998; Herrmann, Black, Lawrence et al., 1998; Johnston, Earll et al., 1999; Johnston, Morrison et al., 1999; Kotila et al., 1998; Wade & Langton-Hewer, 1987). As stated by Coffey & Cummings, 'The mood complications of stroke include depression, emotional incontinence, irritability, anxiety, mania, and mood lability' (p.249). An example condition, common after a stroke, is post-stroke depression (Anderson et al., 1995; Johnson, Burvill, Starkstein, Fedoroff, Price et al., 1993; Tiller, 1992). Mood not only has been found to be affected by stroke but, like perceived control and attention control, it has also been shown in the literature to predict stroke recovery (Johnston, Morrison et al., 1999) as well as being significantly correlated with functional outcome and disability (Herrman et al., 1997; Morrison, Johnston, & Mac Walter, 2000). As such, understanding the

constructs that affect mood following stroke is an important endeavor to this thesis.

In a study by Johnston, Earll et al. (1999), mood was studied as a predictor of survival, disease progression and, disability and for motor neuron disease (MND) patients. Thirty-eight patients were recruited and studied at 3 time point: initial, 6 weeks and 6mo. Disability was measured using the Office of Population Census and Surveys (OPCS) assessment (Martin, Meltzer & Elliot, 1988). Overall mood was measured by creating an a mood index 'developed in the same manner as McDonald et al. (1994) by summing standard scores' (p.3). Anxiety and depression were measured using the HADS. Results showed that overall mood at 6 weeks predicted survival at 6mo. ($t(27) = 2.08, p < .05$). Overall mood also predicted rate of disease progression as patients who had poorer mood at the time of diagnosis had faster disease progression than those with higher mood ($t(32) = 2.64, p < .007$). At 6 weeks the difference was also significant ($t(27) = 2.93, p < .004$). Finally, overall mood at diagnosis and at 6 weeks significantly predicted disability at 6 mo. Overall mood at diagnosis predicted 6mo. disability ($r = -.43, p < .02$) and overall mood at 6 weeks predicted disability at 6mo. ($r = -.51, p < .006$). Six weeks total HADS also predicted 6mo. disability ($r = .38, p < .06$). Also, 6 weeks HADS depression significantly predicted 6mo. disability ($r = .37, p < .05$).

The relationship between cognitive impairment and mood is also of interest to this thesis and, has been studied in the literature (Iezzi, T., Archibald, Y., Barnett, P., et al., 1999; Herrman et al., 1997; Robertson et al., 1997; Starkstein et al., 1993).

In a study by Robinson et al. (1985) support was found for a significant

relationship between cognitive impairment and mood. The study sought to examine prospective relationships between different variables from the point of admission to 3mo. and, from admission to 6mo. Patients were recruited from a stroke data bank (N=103). All patients were psychiatrically examined using the Hamilton Depression Scale (HDS; Hamilton, M., 1960), the Zung Depression Scale (Zung, W., 1965) and, the Present State Examination (PSE; Wing, J., Cooper, J. & Sartorius, N., 1974). All patients met DSM-III criteria for either minor or major depression. These were also the measures used to measure depression in their study. Cognitive performance was measured using the Mini Mental State Examination (MMSE; Folstein, M., Folstein, S. & McHugh, P., 1975). Results showed that there was a significant relationship (Pearson correlation) between initial mood and cognitive impairment at 3mo. (PSE & MMSE: $r = -.36$, $p < .05$; Zung & MMSE: $r = -.33$, $p < .05$). There was also a significant relationship between initial cognitive impairment and mood at 6mo. (MMSE & HDS: $r = -.33$, $p < .05$). And, there was a significant relationship between initial mood and 6mo. cognitive impairment (PSE & MMSE: $r = -.28$, $p < .05$). The authors of the study felt that the prospective data suggested that there was stronger evidence to suggest that initial cognitive impairment predicted later mood better than initial mood predicting later cognitive impairment. Though regression analyses were not performed to verify this, the significant relationships were of interest and warranted future research into this area.

1.6 WHO Model of Disability

As stated earlier, one or more domains may be affected for the patient after suffering a stroke. When studying these domains the nomenclature is usually in terms of impairment or disability. Impairment and disability have been operationalised by the World Health Organisation (WHO). The WHO defines *impairment* as, "Any loss or abnormality of psychological, physiological or anatomical structure or function". As such, a stroke can cause impairment at any or all of these levels.

The physiological and anatomical impairment caused by stroke is determined by its location and type. Such impairment concerns how the physiology and anatomy of the brain is affected in its structure or function. The psychological impairment is determined by what psychological function(s) was lost or affected due to a stroke, e.g., a cognitive or emotional function. The loss or abnormality in the function leads to a restriction or lack of ability in performance. This restriction or lack of ability to perform an activity is what defines *disability*. In other words, as defined by the WHO, disability stems from impairment (Figure 1.2). The WHO defines *disability* as, "Any restriction or lack (resulting from impairment) of ability to perform an activity in the manner or within the range considered normal for a human being".

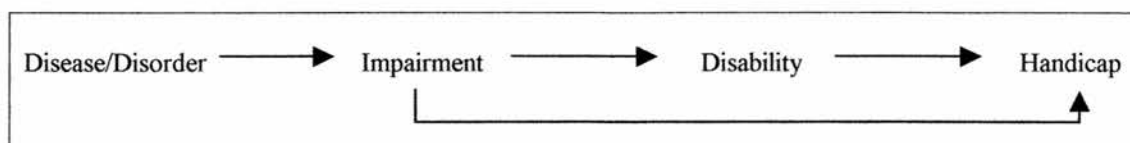


Figure 1.2 WHO model of disability

1.7 Statement of the Problem

As shown in figure 1. the WHO model (1980) proposes that disability results from impairment. The first component being *disease/disorder* and the last being *handicap*. Research has shown, however, that this model is too simplistic in that the level of disability is not directly related to the level of impairment. In a study by Williams, Johnston, Willis & Bennett (1976), looking for patterns of disability with different diseases, results did not correspond to a pattern which would validate the WHO model. Older community residents were asked about their abilities. Abilities were scaled from 1-10 using Guttman scaling techniques and item reproducibility for each disability was established. The results showed that in this sample of residents, with different levels of disability, the order of difficulty for items representative of different levels of disability was found to be cumulative. That is, regardless of different impairments within the sample, overall there was a cumulative order for disability. It could not be said that different diseases which lead to different impairments showed different patterns of disability. Regarding this, Johnston M. (1996) states,

If impairment determined disability, and impairment was in turn determined

by the underlying condition, then one should expect a different pattern of disability with each disease. But this is not what is found. When we examined older people living in the community who had disabilities and looked at the pattern of these disabilities, we could find no evidence of different patterns with different diseases (p.261).

Following the finding that there is not a 1:1 relationship between impairment and disability, future research should focus on possible mediating psychological factors. For this thesis it is suggested that the relationships between a.) perceived control and recovery, b.) attention control and recovery and, c.) perceived control and attention control be investigated further. It is also suggested that other illness representations and cognitive impairments and, mood be investigated. In particular, the relationships that cognitive deficit has with illness representation and mood should be further examined.

The structure for this thesis is therefore two fold (Figure 1.3). Firstly, the relationships between perceived control and recovery and, attention control and recovery will be examined. The relationship between perceived control and attention control will also be studied. Secondly, the relationships between cognitive deficit and illness representations and, cognitive deficit and mood will be examined.

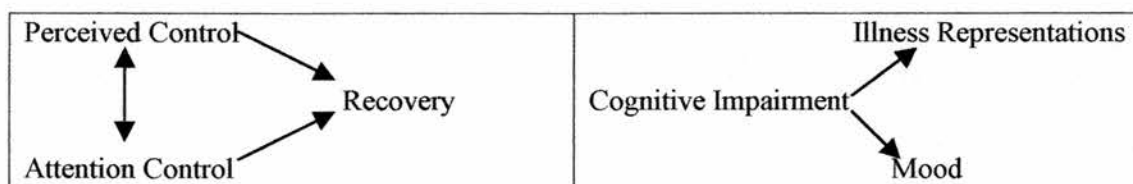


Figure 1.3 Relationships of interest to this thesis

The research questions for this thesis therefore, follow the proposed relationships of interest. The research questions are: 1.) Do perceived control (PC) and attention control (AC) explain some of the same variance in stroke recovery? and, 2.) In what way are cognitive deficits, caused by stroke, related to illness representations and mood?

The relationships to be studied in this thesis will be examined using methodologies which incorporated several measures. The specificity of these measures will be discussed in the next chapter.

CHAPTER 2

Measures & Measurement Issues

Summary

The measures administered in this thesis were chosen based on their specific uses, psychometric strengths and, the practicalities for the population being studied. Another reason for choosing the measures used in this thesis, other than their appropriateness, is the examiner's familiarity of administering and scoring each. This familiarity facilitated the measures being administered and assessed as intended. It also allowed the administration times to be manageable, lessening any fatigue for the patients. Each measure was chosen to assess a particular cognitive skill, illness representation and mood. In some instances, these measures were part of a well standardised battery (WAIS-R) from which the examiner could administer subtests. Administering such measures from standardised batteries, compared with tests developed singularly, was often a preferred choice in terms of validity and reliability. The measures being used in this thesis are listed below under the areas being examined. The aim of this chapter is to discuss the various measures used in this thesis, relevant to their specific domains, in a comparative fashion with other similar measures.

Disability

Disability is a central outcome measure in this thesis as well as other studies of stroke. Disability refers to the inability of a person to perform a given task. This limitation is typically in terms of practical activities, e.g., dressing, walking, performing daily tasks, etc. A commonly used measure for assessing

such disabilities is the Barthel Index of Activities of Daily Living (Mahoney & Barthel, 1965). The Barthel has been referred to as the 'universal standard' of activities of daily living scales (Gompertz, Pound & Ebrahim, 1993). The Barthel Index has been used as a measure of disability in several stroke studies (Johnston, Morrison et al., 1999; Morrison, Johnston & MacWalter, 2000; Lincoln, Gladman, Berman, Noad & Challen, 2000; Wade & Langton-Hewer, 1987). The Barthel Index rates functional independence on ten activities of daily living.

In a study by Gompertz et al. (1993) test-retest reliability was examined for various measures often administered to stroke patients. In their study, 191 stroke patients were recruited, through the mail, from the NE Thames stroke registry and asked to complete various questionnaires (the Barthel being one) at 6mo. after admission to hospital. Of those patients physically able to respond, and who had responded via mail without prompting, a second identical letter/questionnaire was sent ($N=31$, mean age = 69) two-weeks later. The authors tested reliability by plotting the difference between the test-retest scores against the mean of the two scores (method suggested by Bland & Altman, 1986). Reliability coefficients were reported in terms of kappa as suggested by Brennan & Silman (1992). The analysis of individual items was chosen as a preferred method of judging reliability as, 'measures of correlation are inappropriate for testing reliability because (a) they are not measures of agreement, (b) correlation depends on the range of the scale and (c) data with quite poor agreement can show high levels of correlation' (p.293). The Barthel reliability scores were as follows: poor ($\kappa < .20$) = 0, fair ($\kappa = .20-.40$) = 0, moderate ($\kappa = .41-.60$) = 2, good ($\kappa = .61-.80$) = 7 and, very good ($\kappa \geq .81$) = 1.

The category of 'good' had the highest number of ratings. Unsurprisingly, the Barthel also had a test-retest Spearman correlation of .92 ($p < .05$).

Validity for the Barthel was examined in a study using 713 acute stroke patients (Wade & Langton-Hewer, 1987). Predictive validity was demonstrated showing that people recovered 100% independence at a rate of 12% for the first week, 31% for the three weeks and 47% for six months; showing increased independence as a function of time. Factor analysis showed that all items measured the same dimension, i.e., functional independence.

The primary advantage of the Barthel is its ease of administration. As stated by Mahoney & Barthel (1965), 'The advantage of the BI is its simplicity... It can be easily understood by all who work with a patient and can accurately and quickly be scored by anyone who adheres to the definitions of items listed above (p.59). Also, as discussed in Johnston, Morrison et al. (1999), disability is either patient-assessed or observer-assessed; both of which the Barthel Index allows for.

One weakness of the Barthel is that the items were chosen based on their face validity from being used in different chronic disease hospitals rather than on empirical data. Normative studies were performed after the Barthel had been in use for several years. Another weakness is that the items assessing activities of daily living are not weighted using hierarchical methods, i.e., Guttman scaling. This makes the assumption that inability to perform one activity (e.g., walking) is the same as the inability to perform another (e.g., bathing oneself). As an example, this would make the measure less valid for someone who has been in a wheelchair all of their life, suffered a stroke and afterwards cannot bathe themselves. A final weakness is that as a measure of disability, it is limited to

physical activities. Disabilities which concern cognitive skills, for example, are not taken into account.

A second measure of disability is the Sickness Impact Profile (SIP; Gilson, Gilson, Bergner et al., 1975). The SIP was constructed using a top-down procedure where statements were obtained, 'describing behavioral dysfunction from patients, health care professionals, individuals caring for the patients, and the apparently healthy' (Gilson et al., 1975, p.1307). Other 'functional assessment instruments' were also used to generate statements. Extensive piloting and field-testing (1973, 1974, 1976) has been performed on the SIP. Preliminary assessments of the reliability and validity of the SIP was conducted in 1973. In 1974, a field trial was conducted to further assess and refine the SIP. In 1976 a second field-trial was conducted to determine the final content, format and scoring. The current SIP contains two dimensions (Physical and Psychosocial) and, one category that contains items that do not relate to either physical nor psychosocial (Independent Categories), i.e., sleep and rest, eating, work, home management, recreation and pastimes. These two dimensions and 'independent categories' are separated into twelve categories. The twelve categories are then subdivided into a further 136 items.

Psychometric properties of the SIP being reported are based on the 1976 field-trial. This field-trial was conducted using 'a large stratified random sample (N = 696) of members of a prepaid group practice' (Bergner, Bobbitt, Carter & Gilson, 1981). To further assess 'adequate frequency of response' a further 199 subjects (who considered themselves to be ill) were interviewed. Along with the SIP, selected questions from the National Health Interview Survey (NHIS; U.S. Dept. of Commerce, Bureau of the Census, 1973) were administered.

Internal consistency (Cronbach's α) of the SIP was high: $r = .94$, p-value not reported. Test-retest reliability was also good ($r = .97$, $p < .01$).

Convergent validity was shown as the SIP correlated with self-assessment of: dysfunction ($r = .64$, p-value not reported) and sickness ($r = .55$, p-value not reported). Congruent validity was also shown as the SIP correlated with the NHIS ($r = .57$, p-value not reported).

A strength of the SIP, which many other measures of health dysfunction and disability suffer from, is that it was designed to be sensitive to low levels of disability; covered by the 136 items. This measure can also be either practitioner-administered or self-administered. A third strength, as considered by the developers, is that the SIP items are weighted. This is to emphasise the differences in severity that the individual items have on overall disability. Though these are intuitive strengths, there have been a number of limitations associated with its design.

In a recent study by Pollard & Johnston (2001), several limitations of the Functional Limitations Profile (FLP; Patrick & Peach, 1978) (the British version of the SIP) have been discussed. The limitations of the FLP and the SIP are analogous. As Pollard & Johnston note, they and others (Williams, 1996; De Bruin, Diederiks, de Witte, Stevens & Philipson; 1994; Jenkinson, 1994) have identified several limitations with the SIP/FLP. These limitations include: illogical scoring, the nature and meanings of overall scores, ambiguity of items, the order of items, the length of the questionnaire and, the work category. The primary limitation of the SIP/FLP, as discussed by Pollard & Johnston, is the consideration of illogical scoring/weighting of items, categories and overall score. As an example, they consider a scenario proposed by Williams (1996). It

is possible that, due to the Thurstone and Guttman scaling and the number items answered affirmative for the SIP/FLP, one who is paraplegic may score as having less disability than one who is arthritic. This is possible by the person who is paraplegic having a total ambulation score which is lower than the person with arthritis. Pollard & Johnston have identified items on the FLP where different scoring methods are used: mutually exclusive Thurstone, mutually exclusive not Thurstone, inclusive (Guttman), independent, ambiguous and, combination of items (items which are likely to include a combination of scaling methods). It was proposed that a more valid way to determine the *category* score for the SIP/FLP is to use the highest individual weightings for the items thus being, 'consistent with the items and results in each respondent being defined by their most severe limitation for each category' (p.931). To calculate a more valid *overall* score, all limitations in all categories should be taken into account. They proposed that the mean of the maximum category scores should be used in the calculation of overall limitation. The total SIP/FLP score would therefore be calculated by taking the mean of the maximum category score and dividing it by the number of categories. These changes to the SIP/FLP would resolve such scoring anomalies as mentioned before between highly disabled persons vs. mildly disabled ones.

A third popular measure of disability is the Short Form-36 (SF-36; Ware & Sherbourne, 1992). This measure has been normed on both general and clinical populations for several countries: U.S., U.K., Denmark, Sweden, Germany, Australia, France, Italy and the Netherlands. The SF-36 is a 36-item questionnaire designed to assess eight health domains: limitations in physical activities due to health problems, limitations in social activities due to physical or

emotional problems, limitations in usual role activities due to physical health problems, bodily pain, general mental health (psychological distress and well-being), limitations in usual role activities due to emotional problems, vitality and, general health perceptions (Ware & Sherbourne, 1992).

Roberts, Hemingway & Marmot (1997) conducted a study to assess the psychometric properties of the SF-36. The population used in this study was part of a larger study using British Civil Servants (The Whitehall II Study). The Whitehall II study examined the relationship of occupation, life-style and biological factors on health (of which the SF-36 was an outcome measure). The data presented by Roberts et al. (1997) was based on 8,375 participants (5,786 males and 2,589 females) of the original 10,308. Test-retest data were based on a subsample (N=289) of the 8,375 participants. Internal-reliability (Cronbach's α , N = 8,213) for the eight scales were good ranging from .75 - .85 (all p-values < .0001). Test-retest data were based on retesting after one month. All confidence intervals (95%) for the eight scales were significant (mean reliability coefficient = .67). The validity of the SF-36 was measured using principal component factor analysis (N = 8213). The scales were analysed in terms of their validity as measures of physical health and psychological well-being. Orthogonal (Varimax) rotation showed that the physical scales: physical functioning, role limitations (physical), bodily pain and, general health perceptions all significantly correlated with the component 'physical health' (range = .56 - .79). The mental health scales: general mental health, role limitations (emotional), social functioning and, vitality all significantly correlated with the component 'mental health' (range = .62 - .87). Oblique factor analysis (promax) also supported the two components: physical and mental. The

physical scales significantly correlated with the component of 'physical health' (range = .64 - .80). And the mental health scales significantly correlated with the component 'mental health' (range = .71 - .87).

In a study by Brazier, Harper, Jones et al. (1992) the psychometrics of the SF-36 were studied using a clinical population (a random sample of G.P. patients, N = 1,980, age = 16-74). They found that the internal reliability (Cronbach's α) for the SF-36 was also good (range = .73 - .96). Convergent validity was satisfied by correlating similar scales of the SF-36 and the Nottingham Health Profile (NHP). Significant correlations were as follows: SF-36 (physical functioning) vs. NHP (physical morbidity) = -.52 ($p < .05$), SF-36 (social functioning) vs. NHP (social isolation) = -.41 ($p < .05$), SF-36 (pain) vs. NHP (pain) = -.55 ($p < .05$), SF-36 (mental health) vs. NHP (emotional reactions) = -.67 ($p < .05$) and, SF-36 (vitality) vs. NHP (energy) = -.68 ($p < .05$).

Strengths of the SF-36 include its psychometric reliability and validity for both general and clinical populations, its normative value for different nationalities, its division of health status in relation to physical and mental well-being and, its brevity of administration (5-10 minutes; Ware, 2001). A possible weakness that may affect the use of the SF-36 for stroke populations concerns its validity for older participants (Yip, Wilber, Myrtle & Grazman, 2001). This concern mostly stems from the opinion that the SF-36 scales are not sensitive enough for health status issues that may be applicable to the elderly. And, that significant others often provide the information for the questionnaire. These issues, however, are endemic to any measure which has been designed with consideration to administration time. Ware, responds to this point by stating, 'How useful is the SF-36 for purposes of comparing general and specific

population groups, relative to longer surveys? Some of the SF-36 scales have been shown to have 10-20% less precision than the long form MOS measures they were constructed to reproduce (Ware, 2001). This disadvantage of the SF-36 should be weighed against the fact that some of these long-form measures require 5-10 times greater response burden.

A second limitation for the SF-36 concerns its use as a measure of disability. The 'limitations in physical activities due to health problems' and 'limitations in usual role activities due to physical health problems' domains are the most relevant categories specific to disability. The other health domains are more specific to handicap, pain, mental health/QoL, vitality and perceptions of health. When the use of the measure is intended to be an assessment of disability, the SF-36 is limited in the areas it was constructed to evaluate.

The Barthel was used in this thesis to measure disability primarily because, results from other studies were being attempted to be found to replicate. These other studies used the Barthel as their measure of disability. Also, the attempts to replicate results concerning recovery from stroke used the Barthel in their calculations of recovery residuals. Compared with other measures of disability, the Barthel was also more appropriate for research settings as it has faster administration times.

Table 2.1 Summary of Disability Measures

	reliability	validity	pros	cons
Barthel	test-retest: majority of κ values in the 'good' range	predictive: independence as a function of time; factor analysis: all factors load on 'functional independence'	ease of administration: short time, may be patient or observer-assessed	Items chosen for their face- validity; items not hierarchically weighted; only assesses physical activities
SIP	Cronbach's α , test-retest	convergent, congruent	sensitive to low levels of disability, patient or observer- assessed	illogical scoring, nature and meanings of the overall score, ambiguity of items, time to administer
SF-36	internal: Cronbach's α ; test-retest: significant CIs	factor analysis: physical items loaded on 'physical health', mental health items loaded on 'mental health'; convergent: significant similar item correlations for NHP	many normative populations; range of health status: physical and mental items; ease of administration: short-time	questionable validity for elderly populations; limited assessment of pure disability

Perceived Control

For this thesis, perceived control was measured in the manner that Partridge & Johnston (1989) used in their development of the Recovery Locus of Control scale (RLOC). Partridge & Johnston developed this measure, using a bottom-up method to illicit items, in order to better assess locus of control in elderly populations for which such measures generally are not normed. Subjects used in this study included 20 cerebrovascular accident patients (mean age=70) and 20 wrist fracture patients (mean age=69). It was hypothesised that patients with higher internal locus of control would recover more than patients with low internal locus of control. The results supported their hypothesis. RLOC scores

for both stroke patients and wrist fracture patients significantly correlated with recovery ($r = .39-.40$, $p < .05$; $r = .48-.54$, $p < .05$, $.01$ respectively). The RLOC has been found to be both a valid and reliable measure of perceived control. In their original study, Partridge & Johnston found that the mean scores for 'internal' locus of control items significantly correlated with the 'internal' items (range = $.54-.75$). 'External' mean scores significantly correlated with 'external' items (range = $.64-.80$). Thus, validity for the RLOC as a measure of perceived control was established. Reliability of the RLOC has since then been established. The RLOC was used as a measure of perceived control in the study by Johnston, Morrison et al. (1999) referred to in chapter 1 of this thesis; where perceived control was shown to predict recovery from stroke. Cronbach's α levels were shown to be adequate at each of the three time-points (3 weeks post-stroke, 1 month post-discharge & 6 months post-discharge). Reported alpha-levels were: $.64$, $.77$ & $.53$ accordingly.

The RLOC has been established as a measure for perceived control in elderly and stroke populations. It is easy and quick to administer and has suitable psychometric properties. Furthermore, the RLOC was needed to examine the first research question as it was seeking to replicate previous research.

A possible weakness of the RLOC is that the construct(s) it measures may or may not be considered perceived control depending on how the examiner defines it. As discussed in the previous chapter, perceived control is not conceptualised on an agreed set of parameters. Indeed, as the items which were chosen for the RLOC were derived by interrater agreement as to the item's internality/externality, it may be argued that the RLOC is largely measuring

locus of control and not perceived control as specifically defined in chapter 1 of this thesis by Wallston et al. (1989). Therefore, using the RLOC as a pure measure of perceived control may be debatable.

Another measure of control that has been used in the literature is the Multidimensional Health Locus of Control Scale (MHLC; Wallston, Wallston & De Vellis, 1978). This measure has been used in other studies where control cognitions were measured (Fisher & Johnston, 1996a,b, 1998). This 18 item test measures the patients' level of control with regard to managing their health. It measures control on three indices: internal, external and chance, i.e., they have control, others have control and/or, control is determined by chance. Internal reliability (Cronbach's alpha) is also moderate to high for the three subscales of the MHLC: .71 (internal), .68 (powerful others) and, .57 (chance) (Wallston et al., 1978). Factor analysis has shown that the MHLC has good factor validity as 41% of the variance was explained by three factors. Convergent validity was shown in a study by Bonetti (1999) where the MHLC, GSES and the Perceived Health Competence Scale (PHCS; Smith, Wallston & Smith, 1995) were administered to a group of university students. The internal MHLC scale was found to significantly correlate (Pearson's coefficient) with the PHCS ($r = .40$, $p < .01$). The MHLC is a widely used measure of control that covers different domains of locus of control. Its strength is that it provides the examiner with data which is 'multidimensional'. A weakness, however, is that as such, it provides little information on specifically perceived control. It is better used as a measure for general beliefs of health outcome and behaviour.

A third measure of control is the Perceived Health Competence Scale. The PHCS was developed to assess a person's efficacy or competency health

beliefs at an intermediate level of domain-specificity. Whereas Bandura (1997) has advocated the measurement of self-efficacy at specific levels corresponding to the behaviour and, Schwarzer (1992) has advocated the measurement of perceived competence at a more general levels, Smith et al. (1995) developed the PHCS to measure 'efficacy/competence beliefs concerning one's health' at an *intermediate* level. The PHCS has eight items which assess both outcome expectancies and behavioural expectancies.

In the original validation paper of the PHCS (Smith et al., 1995) the authors used the results of five (3 used university populations (unpublished) and 2 used clinical populations) studies where the PHCS had been given as part of a battery of measures in each to report psychometric properties of the PHCS.

The first study (Smith & Wallston, 1992) used patients who had been diagnosed with rheumatoid arthritis within the last eleven years ($N = 238$; 176 females, 62 males; mean age = 56; $SD=13.6$). These patients were administered the PHCS at 4 years after the start of this longitudinal study and again 30 mo. later.

The second study used 100 middle management employees of a university (68 females, 32 males; mean age = 43; range = 26-65). They were administered the PHCS at the beginning of a health promotion program.

The third study (Smith, 1988) used 186 psychology undergraduates (93 females, 93 males; mean age = 19.5; range = 17-23). The subjects were 'participating in one of two experimental studies of the relationship of susceptibility beliefs, values and perceived control beliefs to intentions to engage in preventative health behavior (see Smith, 1988)' (p.55).

The fourth study also used psychology undergraduates of a different university (N = 54; 29 females, 25 males; mean age = 19.3; range = 18-23). The format for this study was the same as in the previous one.

The final study incorporated West Point cadets (N = 528; 53 females, 475 males; age range = 17-21). Measures were given at the beginning of their first year and at 4mo. later (N = 520).

The internal reliability (Cronbach's α) for the PHCS was good in all studies: rheumatoid arthritis (RA) time 1 = .82, time 2 = .83; middle management (MM) = .85; first undergraduate (U1) = .89; second undergraduate (U2) = .90 and West Point (WP) time 1 = .85, time 2 = .84.

Construct and discriminant validity was shown by comparing the PHCS scores of the RA group against the combined scores of the other four groups. The scores of the RA group were significantly different suggesting that the RA group reliably reported lower levels of perceived health ($t(1101) = 36.87$, $p < .001$). Convergent validity was shown by its correlations to Ware's general measure of health (1976)¹ administered to the undergraduate populations (U1: $r = .50$, $p < .001$; U2: $r = .50$, $p < .001$). The PHCS was also significantly correlated with locus of control and susceptibility beliefs. Chance health locus of control was negatively correlated to the PHCS (U1: $r = -.20$, $p < .01$; U2: $r = -.38$, $p < .01$). Susceptibility to illness was negatively correlated to PHCS (U1: $r = -.18$, $p < .05$; U2: $r = -.56$, $p < .001$).

A particular strength of the PHCS is that it measures health beliefs at an intermediate level of domain-specificity. In essence, capturing data relevant to

¹ A 9-item health-status scale developed by Ware (1976). Items range on a 5-point Likert-scale from 'definitely true' to 'definitely false'.

specific and general health beliefs. Another strength of the PHCS is its short administration time. Having only eight items it can be administered in under 5 minutes. It can be participant or observer-administered. And, it can be used with either general or clinical populations.

A weakness inherent in the theoretical design of the PHCS is that while it measures health beliefs at an intermediate level, it may not fully represent persons' specific or general beliefs about their health to the degree as a measure which is designed to do so.

The RLOC was chosen as the measure of use in this thesis primarily because the examiner was attempting to replicate specific findings of past research (Johnston, Morrison et al., 1999; Fisher & Johnston, 1998; Fisher & Johnston, 1996a,b; Johnston et al., 1992; Partridge & Johnston, 1989). As perceived control was the specific area of control being examined and, other studies have used it for this, the RLOC was chosen as the measure of perceived control. With the added benefit of it being a quick scale to administer, the RLOC was also considered the best measure of perceived control for elderly stroke patients.

Table 2.2 Summary of Control Measures

	reliability	validity	pros	cons
RLOC	Cronbach's α	construct: internal & external mean/item correlations	normed for elderly populations; ease of administration: 5-10 minutes	debatable as a measure of perceived control
MHLC	Cronbach's α	factor analysis; convergent	'multidimensional' nature	provides little information on perceived control
PHCS	Cronbach's α	construct, discriminant & convergent	normed for general and clinical populations; patient or observer- administered; ease of administration: <5min.; measures health beliefs at intermediate levels of specificity	limited in its specificity of either specific or general beliefs

Attention Control

Attention control is another variable central to the first research question. It is also a cognitive variable and thus is also central to the second research question. Attention control is the ability to maintain focus (attention) on a given task for a set amount of time. A popular measure of attention control is the Test of Every Day Attention (TEA; Robertson et al., 1994). The TEA was developed to improve existing methods of assessing attentional disorders.

In a study undertaken to develop a measure for clinicians to assess attention, Robertson et al. (1994) developed the Test of Everyday Attention (TEA). Within the TEA are two subtests of sustained attention, Elevator Counting and Elevator Counting with Distraction. Elevator counting has been used in previous research in the assessment of sustained attention (Crawford,

Sommerville & Robertson, 1997; Robertson, Baddeley et al., 1997; Robertson, Manly, Beschin et al., 1997; Robertson, Ridgeway et al., 1997).

Psychometric analysis for the TEA was reported in the normative studies cited in the TEA manual (Robertson et al., 1994). Equivalent forms reliability was tested between three versions of the TEA (A, B & C) using two samples of general control subjects (sample 1: N = 118; sample 2: N = 39) and a sample of unilateral stroke patients recruited 2mo. post-stroke (N = 74). In the first instance version A and B were compared with the first sample of controls. The correlation (Pearson's) between the two versions for elevator counting was not reported as it reached a 'ceiling effect'. The correlation for elevator counting with distraction was reported to be .71 (p-value not reported). The comparison between versions B & C were made with the second control group. The correlation for elevator counting was again not reported due to a 'ceiling effect'. The correlation for elevator counting with distraction was .68 (p-value not reported). Correlations for the stroke group were made between versions A & B. The correlation for elevator counting was .88 (p-value not reported) and for elevator counting with distraction, .83 (p-value not reported).

Differential validity was shown by comparing performance of the TEA in two similar age brackets. In the first age bracket (50-64) groups of controls (N = 26) were compared against a group of stroke patients (N = 39). The stroke patients did not score significantly poorer on elevator counting than the control group. Stroke patients did, however, score significantly poorer on elevator counting with distraction than the controls ($t = 3.45, p < .001$). In the second age bracket (65-80) the stroke patients scored significantly poorer on both the

elevator counting ($t = -3.4, p < .001$) and on elevator counting with distraction ($t = -4.68, p < .000$).

Factorial validity using 154 normal subjects was also established for the elevator counting subtest as a measure of attention control. Varimax rotation yielded four factors which accounted for 62.4% of the variance. Elevator counting showed a factor loading of .56 for the factor 'sustained attention'.

A main strength of the elevator counting and elevator counting with distraction subtests, and in fact the TEA, is that they were developed based on measuring attention on 'real-life' practical skills. The elevator counting and elevator counting subtests are administered via an audio-tape presentation under the instruction for the patient to imagine him/herself in a lift where the floor indicator light is inoperable. They are to direct their attention to a tone which represents the floor and count the number of tones (floors). Attentional deficits as measured by the TEA can be said to mimic those which might exist in 'real-life' circumstances. Another strength is that it has been normed on a clinical population, i.e., stroke.

A possible weakness of the elevator counting subtest concerns its sensitivity. As mentioned above, ceiling effects occurred for normal controls. These may be countered by administering more than one version of this subtest but, this would make testing times lengthier; any errors on which might be due to fatigue rather than attentional deficits.

A second 'vigilance' test of attention is the Sustained Attention Response Task (SART; Robertson, Manly, Andrade, Baddeley & Yiend, 1997). The SART procedure is administered via computer console. Two-hundred and twenty-five single digits are visually presented over 4.3 minutes. Following each

digit, a mask (900-msec) was shown. Subjects respond to the digit by pressing a key, except for when the number '3' was shown. This target was presented in a prefixed quasi-random order. To enhance the numeric processing, five fonts were randomly allocated.

This measure was developed from the premise that tests of attention which are sensitive to traumatic brain injury (TBI) involve several cognitive operations and, therefore, cannot be said to be specifically measuring sustained attention. As Robertson, Manly, Andrade et al. (1997) state, 'In fact, most authors in this area interpret impaired clinical performance on such tests as being due to reduced speed of processing, rather than in terms of any more specific attentional processes' (p.748). The paradigms used in tests of continuous attention, 'require participants to monitor long sequences of stimuli and respond on detecting infrequent targets... Certainly such tasks have problems with ceiling effects, which have led researchers to perceptually degrade targets or load working memory in order to reduce high levels of performance' (p.748). The authors argue that deficits in sustained attention are exaggerations of 'action slips' that the normal population experiences in everyday occurrences. An example of such everyday action slips may be throwing away vegetables while keeping the peelings. The authors suggest that determining such action slips in TBI is, 'one significant factor... indicative of faulty sustained attention' (P.748). The SART was developed to require, 'a high level of continuous attention to respond and be sensitive to transitory reduction in attention or lapses, while keeping to a minimum demands on other cognitive processes such as memory, planning and general intellectual effort' (p.748).

Psychometric data on the SART was gathered in an experiment (Robertson, Manly, Andrade et al., 1997) attentional deficits were compared between normal participants and TBI patients. The SART was administered to a normal sample of 75 participants (23 male, 52 female; age range = 18-65; mean age = 34.0, SD = 11.0).

Test-retest reliability was determined from a subsample of the controls (N = 25; 10 males, 15 females; mean age = 36.0; SD = 8.0). The SART was administered on two occasions over 1wk. Pearson correlation was good ($r = .76$, p-value not reported).

Validity for the SART was determined using a matched subgroup of group of 17 normals (11 males, 6 females; mean age = 39.8; SD = 11.9) and 22 TBI patients (16 males, 6 females; mean age = 34.2; SD = 12.1) who were between 9-18 months post injury. Patients and controls were administered the SART, Glasgow Coma Scale (GCS; disability), Cognitive Failures Questionnaire (CFQ; Broadbent, Cooper, Fitzgerald & Parkes, 1982; self-report questionnaire measuring slips of action and memory in everyday life; also given to significant others), Lottery and Telephone Search subtests of the TEA (being used as other measures of sustained attention), the Modified Wisconsin Card Sorting Test (Nelson, 1976) and Visual Elevator subtest of the TEA (attentional switching), the Stroop Test (Trenerry, Crosson, DeBoe & Leber, 1989) and Telephone Search subtest of the TEA (selective attention) and, the PASAT ('one of the best established measures of attentional deficit following TBI', p.751).

Discriminant validity was shown as patients and controls differed significantly on the SART. Patients had a mean error score of 7.6 (4.8) whereas controls had a mean error score of 4.0 (3.2): $F = 7.0$, $p < .01$.

Convergent validity was also shown as the SART score for the patients significantly correlated with the GCS ($r = .58, p < .001$). Convergent/discriminant validity was again shown through correlation between the CFQ and the attentional measures. There were no significant differences between the patients' self-report scores of attentional failure on the CFQ and any of the attentional deficits. However, the significant others-assessed reports of attentional deficits on the CFQ for the patients were significantly correlated with: the visual elevator test ($r = -.49, p < .05$), the Stroop test ($r = .47, p < .05$), the SART (.44, $p < .05$), the PASAT (-.73, $p < .01$) and the GCS (-.51, $p < .05$).

The main strength of the SART is that it was developed as a specific measure of sustained attention which is not subject to biases from other cognitive processes, e.g., memory. A particular weakness is that the SART is computer-administered; making testing impractical for anywhere other than the laboratory.

A third measure of attention is the Paced Auditory Serial-Addition Task (PASAT; Gronwall, 1977). The PASAT has been used in studies to determine attentional deficits among different populations (Leininger et al., 1990; Robertson, Manly, Andrade et al., 1997; Stuss et al, 1989). The presentation of the PASAT is given via an audio-recording of a series of digits. There are a total of 61 digit presented for four trials, one digit every 2.4, 2.0, 1.6 and 1.2 sec. intervals. The participant is to add the first two digits, take the total and add it to the next digit which is added to the next digit, etc.

The psychometric properties of the PASAT were discussed in Gronwall's original paper (1977). In this study, PASAT performance of concussion patients were compared against normal controls. The patient sample consisted of 320 patients who met the criteria of being concussed (e.g., loss of

consciousness) and were between the ages of 14-55. Sixty control subjects were used who had had accidents without head-injury and were between the same age bracket. Demographics were not discussed.

Test-retest data were based on a weekly interval for the controls.

Reliability was reported in terms of a cut-off score of 1SD below the mean. All controls for each of the four presentations scored above the cut-off: 2.4 sec. (mean = 46, SD = 6), 2.0 sec. (mean = 40, SD = 7), 1.6 sec. (mean = 32, SD = 8), 1.2 sec. (mean = 22, SD = 5). On retest, scores were similar: 2.4 sec. (mean = 50, SD = 5), 2.0 sec. (mean = 45, SD = 5), 1.6 sec. (mean = 39, SD = 6), 1.2 sec. (mean = 31, SD = 4). Though not presented, the author stated that 99% of the patients' mean scores fell significantly below that of the controls'. Split-half reliability for post-concussion patients has been reported to be high ($r = .90$, $p < .01$) in other studies as well (Egen, 1988).

Criterion-validity was suggested by charting PASAT performance (time per correct response) on number of days post-injury. This procedure was performed for individual patients ($N=6$). As was shown by a line graph of the six patients, time per correct response decreased as a function of days. Data for patient 6 exemplified this as their time score for day 1 was '7' and their time score for day 60 (last day measured) was '2.5'.

As was shown with the study by Robertson, Manly, Andrade et al. (1997), discriminant-validity was also shown for the PASAT as it negatively correlated with the CFQ ($r = -.73$, $p < .01$). Convergent-validity was also shown as the PASAT significantly correlated with the GCS ($r = .61$, $p < .001$).

The straight-forward presentation and objective of this test are the strengths of the PASAT. It is easily understood by the patients and the

administration is easily transferable to different settings. The major disadvantage of the PASAT is the stress it often induces in patients. The digit presentation is 1:2.4 seconds. As Lezak (1995) states, 'Unfortunately, patients experience this sensitive test as very stressful: most persons-whether cognitively intact or impaired-feel under great pressure and that they are failing even when doing well' (p.373).

In comparison, elevator counting is much easier to administer, with a similar format. The brevity and simplicity make it suitable for elderly stroke patients. The psychometric strengths of elevator counting, while not as high as the PASAT's, are respectable making it an adequate test of attention without the addition problem of being too stressful or demanding for the patients. Though the PASAT is a sensitive measure of attentional deficit, the elevator counting subtest of the TEA was chosen for use in this thesis based upon the comparisons.

As with measuring perceived control, replication of results, using the same measure as previous studies (Robertson, Baddeley et al., 1997; Robertson, Manly, Beschin et al., 1997; Robertson, Ridgeway et al., 1997), were being attempted. Therefore, the elevator counting and, elevator counting subtests of the TEA were used in this thesis as measures of attention control.

Table 2.3 Summary of Attention Measures

	reliability	validity	pros	cons
TEA	test-retest	differential: stroke pts. performed poorer than controls; factorial: elevator counting loaded significantly on 'sustained attention'	developed to measure 'everyday' attention; clinical norms	possible sensitivity problems: ceiling effects
SART	test-retest	discriminant: patients & controls differed significantly; convergent: significant correlation with the GCS, significant correlation with the CFQ	developed as a specific measure of sustained attention	impractical for field-use
PASAT	test-retest: patients perform below the cut-off; split-half: reported by other sources	criterion: patients performance increases over time; discriminant: negatively correlates with CFQ; convergent: significantly correlates with GCS	easy to comprehend and administer	stress-inducing

Illness Representations

Due to the variability of how health beliefs may be conceptualised, there are several different measures which examine patients' illness representations (e.g., Multidimensional Health Locus of Control Scale, Perceived Health Competence Scale, Outcome Expectancy Scale, Implicit Models of Illness Questionnaire). These measures are usually theoretically-based ranging from non-validated questionnaires to valid and reliable measures.

The first measure to be discussed is the Illness Perception Questionnaire (IPQ; Weinman, Petrie, Moss-Morris & Horne, 1996). The IPQ is a recent, theoretically-derived measure that tests the five components of the *theory of self-regulation* (Leventhal, Meyer & Nerenz, 1980). The identity (symptom) list was based on 12 'common symptoms from other checklists (e.g., Bowling, 1991)' (p.433). The remaining lists (cause: 10 items; timeline: 3 items; consequences: 7 items; control/cure: 6 items) were constructed by the authors to fit with Leventhal's model or, were taken from patients in preliminary interviews. The subscale 'control/cure' was also consistent with work done by Lau & Hartman (1983). This scale provides qualitative data should the research warrant it.

In Weinman et al. (1996), the IPQ was validated on a variety of clinical samples. The samples included: hospitalised MI patients (N = 143; 1% female; mean age = 53.0; SD = 8.5; mean length of illness = 2-5 days), discharged MI patients (N = 91; 13% female; mean age = 53.8; SD = 8.2; mean length of illness = 3mo.), discharged MI patients (N = 91; 12% female; mean age = 53.5; SD = 8.1; mean length of illness = 6mo.), chronic fatigue syndrome (N = 115; 73% female; mean age = 48.2; SD = 12.6; mean length of illness = 11.8 yrs.), rheumatoid arthritis (N = 22; 81% female; mean age = 62.2; SD = 16.6; mean length of illness = 14.5 yrs.), diabetes (N = 88, 48% female; mean age = 45.6; SD = 15.9; mean length of illness = 15.2 yrs.), pain (N = 60; 40% female; mean age = 42.2; SD = 13.9; mean length of illness = 3.8 yrs.), renal (N = 32; 41% female; mean age = 48.3; SD = 15.6; mean length of illness = 9.3 yrs.) and, asthma (N = 193; 66% female; mean age = 37.5; SD = 13.3; mean length of illness = 6.8 yrs.).

Psychometrics of the IPQ were reported for the scales deriving quantitative data (cause excluded). Internal consistency (Cronbach's α) was reported for the four scales (MI & renal sample): identity ($r = .82, p < .05$), timeline ($r = .73, p < .05$), consequences ($r = .82, p < .05$), control/cure ($r = .73, p < .05$). Test-retest data were gathered at 3 times (MI sample): 1mo., 3mo. and 6mo. One-month test-retest for the scales are as follows: identity ($r = .84, p < .001$), timeline ($r = .49, p < .01$), consequences ($r = .68, p < .001$) and control/cure ($r = .68, p < .001$). Test-retest data at 3mo. was as follows: identity ($r = .34, p < .001$), timeline ($r = .51, p < .001$), consequences ($r = .55, p < .001$) and control/cure ($r = .54, p < .001$). Six-month test-retest data were as follows: identity ($r = .06, ns$), timeline ($r = .36, p < .001$), consequences ($r = .55, p < .001$) and control/cure ($r = .46, p < .001$).

Concurrent validity was shown by significant correlations within the MI sample's data. Identity significantly correlated with: the SIP ($r = .54, p < .001$), recent doctor visits ($r = .31, p < .01$), the Health Distress Scale ($r = .32, p < .01$), self-rated health ($r = -.55, p < .001$), likelihood of future MI ($r = .24, p < .05$) and control over heart problems ($r = -.30, p < .001$). Timeline significantly correlated with: SIP ($r = .25, p < .05$), the Health Distress Scale ($r = .23, p < .05$), RLOC ($r = -.18, p < .05$), self-rated health ($r = -.29, p < .01$), likelihood of future MI ($r = .42, p < .001$) and control over heart problems ($r = -.38, p < .001$). Consequences significantly correlated with: SIP ($r = .34, p < .001$), recent doctor visits ($r = .21, p < .05$), the Health Distress Scale ($r = .53, p < .001$), self-rated health ($r = -.52, p < .01$), likelihood of future MI ($r = .36, p < .001$) and control over heart problems ($r = -.39, p < .001$). Control/cure significantly correlated with: RLOC ($r = .38,$

$p < .001$), likelihood of future MI ($r = -.28, p < .01$) and control over heart problems ($r = .42, p < .001$).

Discriminant validity was shown by a series of one-way ANOVA tests followed by post-hoc Scheffé to determine differences between clinical groups on the subscales of the IPQ. Significant differences were shown for the groups regarding their illness representations: identity ($F = 37.53, p < .001$) and, consequences ($F = 13.34, p < .001$), timeline ($F = 18.84, p < .001$). Control/cure differences were not significant. It was also shown that the different patient groups attributed significantly different causes to their illnesses.

Predictive validity was also tested using the MI sample's admission data, their data at 3mo. and their data at 6mo. There were significant correlations between the IPQ subscales and other data. At 3mo., identity significantly correlated with self-rated health ($r = -.24, p < .05$). Timeline significantly correlated with likelihood of future MI ($r = .30, p < .05$). Consequences significantly correlated with self-rated health ($r = -.28, p < .05$). And, control/cure significantly correlated with likelihood of future MI ($r = -.27, p < .01$) and control over heart problems ($r = .35, p < .001$). At 6mo., timeline significantly correlated with likelihood of future MI ($r = .26, p < .01$). Consequences significantly correlated with likelihood of future MI ($r = .23, p < .05$). And, control/cure significantly correlated with likelihood of future MI ($r = .20, p < .05$) and, control over heart problems ($r = .25, p < .01$).

A strength of the IPQ is that its use is appropriate for a large number of clinical populations, four of which were discussed above. Its development is also based on a strong theoretical model (Self-Regulation) which allows it to be used in research that incorporates/tests Leventhal's model. Another strength of

the IPQ, as stated by Weinman et al. is that researchers may adapt the questionnaire to be specific for a particular population, e.g., stroke.

A possible weakness of the IPQ is that though the items were generated from valid sources, there was not any information to suggest the appropriateness of the items to the constructs they are purported to measure, e.g., factor analyses. Also, the distances between the items of each subscale are assumed to be equal along a numerical scale.

As the study of illness representation and its effect on health-beliefs grows, so will the number of valid and reliable measures. As the IPQ is theory-based and has been shown to validly and reliably measure illness representations in a variety of populations, it has been chosen to measure illness representations in this thesis.

A second measure of illness representation is the Implicit Models of Illness Questionnaire (IMIQ; Turk, Rudy & Salovey, 1986). The IMIQ is a 45-item questionnaire, which like the IPQ, was developed to, 'operationalize constructs such as identity (Label), timeline, consequences and cause described by Leventhal et al., and cure as described by Lau & Hartman. In addition we included items related to personal responsibility (e.g., Jenkins, 1966; Taylor et al., 1984) and disruptiveness (Jones & Weise, 1982)' (Turk et al., 1986, p.456). As in the study by Weinman et al., Schiaffino & Cea (1995) conducted a study to examine illness representations in different clinical populations and, normal controls. The clinical patient populations consisted of an RA sample (N = 63, 90% female), an MS sample (N = 101, 90% female) and, an undergraduate sample (N = 71). The patient populations were administered the IMIQ (for both

MS and RA) and measures of functional and psychological status. The students completed the IMIQ for the two conditions as well as a third: MS, RA and HIV.

Internal reliability (Cronbach's α) for the five subscales (cause included) between patients and students ranged from poor to good. In general, only the cure and consequences subscales showed adequate internal reliability ranging from .56-.87. The label, timeline and cause subscales ranged from .06-.70.

Factorial validity was shown through an exploratory factor analysis which suggested that the IMIQ subscales supported the Leventhal model. A four-factor solution was shown to explain 46% of the variance. The four factors were labelled: curability, personal responsibility, symptom variability and, serious consequences. The curability factor consisted of items combining causes of illness and possible cures. The personal responsibility factor included cause of illness items as well but, related to what the patient was responsible for. The symptom variability factor included items which pertained to illness controllability, changeable symptoms across time, symptoms related to stress, weather and rest, and symptoms affecting different body parts. The serious consequences factor included items pertaining to the severity of different illnesses.

The internal reliabilities (Cronbach's α) of the four factors was much higher overall than was for the subscales. The range was smaller at a higher level: .62 (serious consequences) to .91 (curability).

Two MANOVA calculations were undertaken to examine differences of illness representations across illnesses and, differences between patients and students on the same illness (differential-validity). In the first MANOVA, significant differences were shown for students' representations across the

illnesses: curability [$F(2,209) = 14.41, p < .001$]; responsibility [$F(2,209) = 35.0, p < .001$]; variability [$F(2,209) = 5.19, p < .01$] and consequences [$F(2,209) = 3.34, p < .05$]. Post-hoc tests showed that students felt that RA was more curable and more variable than either MS or HIV. Students felt that individuals were more responsible for HIV and least so for RA. The students also considered MS a more serious disease than RA and, MS was deemed more serious than HIV.

The second MANOVA showed that there were significant interactions between illness and status for both responsibility [$F(1,300) = 24.43, p < .001$] and seriousness [$F(1,300) = 24.89, p < .001$]. The only main effect was found for symptom variability and curability. RA was seen as more variable than MS [$F(1,300) = 10.72, p < .001$]. Patients rated these illnesses as being more variable than did the students [$F(1,300) = 83.25, p < .001$]. RA was considered to be more curable than MS [$F(1,300) = 33.95, p < .001$].

Like the IPQ, the IMIQ strengths lie in its development based on a well-validated theory of illness representation. As a measure of illness representation, it seems weaker than the IPQ. It appears to be less reliable and its items seem to be less specific to the five components of illness representations in Leventhal's model.

The IPQ was chosen in this thesis as the measure of illness representations primarily because, of its design being theoretically-based on a widely accepted model of illness representation. A second reason for choosing the IPQ over another measure of illness representation is that the IPQ lends itself to be modified specifically for stroke by replacing the word 'illness' with 'stroke', as suggested by its authors.

Table 2.4 Summary of Illness Representation Measures

	reliability	validity	pros	cons
IPQ	Cronbach's α ; test-retest: significant at 3 time-points	concurrent: with other like- measures; discriminant: different representations for different populations; predictive: with other like- measures	may be used for several different populations; based on a well- tested model; may be adapted to fit a specific population	no evidence to support the internal validity of the items; distances between items are assumed to be equal
IMIQ	Cronbach's α : higher for factors than for subscales	factorial: support for the Leventhal model; differential: illness representation differ for illnesses and between students and patients	based on a well- validated model of illness representation	questionable reliability and less specific to Leventhal's model

Issues of Defining Cognitive Deficit

Unlike the other areas being assessed in this thesis, cognitive deficit was defined in a dichotomous division of either its presence or absence for each patient. This was done in order to address the second research question. The question of how to define cognitive deficit then became apparent. There is no single test or methodology that defines cognitive deficit in the literature. This is due to the fact that cognitive deficit may be measured in different ways, using different criteria. The usual method therefore, is to compare a patient's cognitive ability against normative data.

Cognitive deficits have been measured in several ways. Generally, it is agreed that one's cognitive ability is comprised of several different skills and therefore, cannot be assessed using simply one test. However, there are several batteries which have been developed in order to tap specific cognitive skills for

which the cumulative score can be said to represent general ability. That said, in a clinical setting it is important to recognise the differences within the battery to determine a person's strengths and weaknesses, i.e., the subtest scatter. For research purposes however, specific subtests from a battery or test may be employed to measure certain skills/deficits. As stated in Lezak (1995), 'For research purposes, the prime consideration in selecting examination techniques is whether they will effectively test the hypothesis...Just as the basic battery can be modified for individuals in the clinical examination, so too tests can be added or subtracted depending upon research needs' (p.123).

As discussed in the previous chapter, the second research question concerns the relationship between cognitive deficit and illness representations and, cognitive deficit and mood. The means of defining 'deficit' are discussed in the following sections for both general and specific cognitive abilities.

General Cognitive Deficit:

To assess general cognitive deficit in brain injured patients (in this case, stroke), the examiner should either have a previous measure of the patient's cognitive ability or, create an estimation of their premorbid ability. Though many measures (for both general and specific) of cognitive ability provide normative data which single scores may be compared against, this is not as accurate as either having or creating a premorbid score to make such a comparison against. This premorbid assessment is then compared with the patient's postmorbid ability. The difference(s) between the two levels may then be attributed to the brain injury (holding all other variables constant).

Premorbid General Intelligence

In determining a patient's current cognitive abilities, or to what extent a brain injury has had upon their cognitive performance, some idea of their premorbid cognitive functioning must be determined. This can be either through direct measurement taken against pre-existing data, i.e., such as a premorbid WAIS-R score. Or, more commonly premorbid cognitive functioning is determined through indirect measurement, i.e., estimating premorbid ability using current measures. As Lezak (1995) explains the deficit measurement paradigm, 'A statistically significant discrepancy between expected and observed performance levels for any cognitive function or activity represents a cognitive deficit' (p.109). The method used in this thesis was an indirect measure widely used for the purpose of determining premorbid ability (Crawford et al., 1989; Korten et al., 1997; Sharp & O'Carroll, 1991; Starr et al., 1992): *the National Adult Reading Test* (NART; Nelson, 1991). Since verbal skills are known to remain fairly consistent in spite of aging (Flicker et al., 1987; Bayles, Tomoeda & Boone, 1985), the NART tends to withstand the effects of intellectual deterioration (Korten et al., 1997).

As Nelson (1991) states, 'The NART was specifically designed to provide a means of estimating the premorbid intelligence levels of adult patients suspected of suffering from intellectual deterioration' (p.1). The NART contains a list of 50 phonologically irregular short words from which the patient reads. Scores are based on the number of errors (mispronounced words). Using regression tables provided in the manual, WAIS or WAIS-R FSIQ, VIQ and PIQ scores are then obtained.

The psychometrics of the NART have been shown in the literature to be strong. Split-half reliability (Cronbach's α) was reported in the NART manual as being .93. Other authors have also demonstrated good reliability for the NART. Crawford et al. (1989) used 61 normal subjects who did not have any neurological, psychiatric nor sensory disabilities (mean age = 37.1; SD = 12.2). The subjects were administered the NART and retested 10 days later. Test-retest reliability (Pearson's coefficient) was high ($r = .98, p < .01$). In the same study, inter-rater reliability was also examined. The NART performance of 40 (demographics not reported) subjects was audio-taped and scored by 10 clinical psychologists. Pearson correlations between all clinicians were high and ranged from .96-.98. Their results of the inter-rater reliability for the NART replicated similar findings in the literature (O'Carroll, 1987).

Convergent validity for the NART was demonstrated in a cross-validation study by Willshire, Kinsella & Prior (1991). Their study examined the predictive relationships between the NART + education level, age and, occupation on WAIS-R FSIQ. In their sample, 104 normal subjects (recruited from the general public) were administered the NART and the WAIS-R (42 males, 63 females; mean age = 42.2; SD = 14.90). The NART significantly correlated with WAIS-R FSIQ ($r = -.51, p < .001$); as did occupation ($r = -.32, p < .001$) and education ($r = .56, p < .001$). The NART + education level significantly predicted WAIS-R FSIQ ($R^2 = .46, F = 43.37, df = 2, 101, p < .000$). Predictiveness increased as the age of the sample increased: ≤ 55 ($N = 76; R^2 = .34, F = 19, df = 2, 73, p < .000$); ≥ 55 ($N = 28; R^2 = .67, F = 25.69, df = 2, 25,$

$p < .000$). Results suggested a significant relationship between the NART and the WAIS-R, as its design intended.

Administering the NART takes 5-10 minutes thus making it an appropriate measure of premorbid cognitive ability for elderly stroke patients. As the NART was developed to estimate intelligence in terms of WAIS-R scores, the examiner was confident in understanding the patients' premorbid abilities.

Another indirect method of measuring premorbid ability is through the use of demographic variables. Studies using regression analysis have shown that demographic variables can predict significant amounts of WAIS full scale IQ (FSIQ), verbal IQ (VIQ) and, performance IQ (PIQ) (Barona, Reynolds & Chastain, 1984; Matarazzo, 1972; Vanderploeg & Schinka, 1995). In a study by Wilson et al. (1978), the original standardisation sample of the WAIS was taken and step-wise regression equations for FSIQ, VIQ and PIQ were performed based on demographic variables. These equations predicted 54%, 53% and 42% of the variance respectively. By simply using demographic variables, often obtained in 5 minutes, premorbid cognitive functioning may be estimated.

Following the method of using demographic variables to predict cognitive ability, several authors have combined existing cognitive scores with demographic variables to further regress premorbid cognitive ability. This was the method used in a study by Vanderploeg & Schinka (1995) who used the original standardisation sample of the WAIS-R. Subtest scores were combined with demographic variables to further predict FSIQ, VIQ and PIQ. As much as 82% of the variance for FSIQ, 87% for VIQ and 80% for PIQ was predicted using this method.

In a review of assessment methods used to estimate premorbid IQ, O'Carroll (1995) noted that the NART has also been used in the literature in conjunction with demographic variables to better predict premorbid cognitive ability. O'Carroll cited a study by Crawford et al. (1989) in which this method was used. Using a sample of 151 normal subjects, Crawford found that: the NART predicted 66% of WAIS FSIQ, demographic variables predicted 50% and the NART combined with demographics predicted 73%. In terms of a balance between psychometric strength and testing practicalities (length of administration time and level of difficulty to administer), this method of using demographic variables and the NART seems ideal.

Unfortunately, there were not enough demographic variables in the patients' records to incorporate them with the NART scores for enhanced predictions of premorbid IQ. The specific demographic variables needed were: class, age, education and, gender. Patients' records did not include social class nor, education. Using the NART alone, however, was a valid and reliable method of estimating premorbid ability. As well as being a sound measure, its ease and time of administration made the NART a desirable measure to use with elderly stroke patients.

The NART is a measure which provides a valid estimation of premorbid IQ in a format which is easy and quick to administer. This is a particular strength as compared with the alternative of administering a full version intelligence scale. The weakness is that as it provides such an estimation, its validity is somewhat compromised. This is particularly in light of research which has shown the NART to be susceptible to the influences of education,

occupation, gender and social status (Beardsall & Brayne, 1990; Crawford et al., 1990).

Postmorbid General Intelligence

In assessing postmorbid general cognitive ability, different methods may be used. Either administering a full neuropsychological battery of tests or, testing the patient's full-scale intelligence. A full neuropsychological battery, however, is inappropriate for most research studies due to the administration time. Testing the patient's full-scale intelligence (e.g., WAIS-R) is often used in research as a faster method of assessing cognitive abilities. However, depending upon the study, this too can be overly time consuming. Another method, particularly suited to research, is to estimate the patient's full-scale intelligence (in essence, equating it with postmorbid cognitive ability).

One method of estimating full-scale IQ is to administer selected subtests for the WAIS-R and prorate the scores into an estimated full-scale IQ score. One method of WAIS-R FSIQ proration is called the Short-Form II, or 'the dyad'. As stated in Spreen & Strauss (1998), the dyad is, 'a popular short form that has good psychometric properties (mean reliability = .94, mean validity = .91) (Sattler, 1992)' (p.93). The Short-Form II consists of prorating the vocabulary and block design subtests of the WAIS-R. A pioneer of this technique, Jerome Sattler (1974, 1982, 1992, 1998), established proration scales for the Short-Form II. Using his calculations, an estimation of postmorbid full-scale IQ can be obtained.

As stated before, this estimated postmorbidity level of cognitive ability should then be compared with some form of premorbidity cognitive ability in order to assess cognitive deficit.

Method Used in This Thesis

The method used for measuring general cognitive deficit in this thesis incorporated a comparison of the patients' estimated premorbidity FSIQ (NART) with an estimation of their WAIS-R FSIQ, as discussed above. If the difference between the premorbidity FSIQ score and the postmorbidity FSIQ score was >13 (as determined from the NART estimated FSIQ table), the patient was considered to have general cognitive deficit at the .05 level of significance. This consideration was determined as 5% or less of a normal population would have a discrepancy of 13 points or more based on the normative study of the NART. Specific cognitive deficits were similarly determined on WAIS-R subscales which are discussed in the next section.

An advantage of using the Short-Form II as an overall measure of cognitive deficit is that it is much less time consuming to determine. This is a particular advantage in research, particularly with elderly stroke patients. Another strength of the dyad proration is that it is based 50% on a 'hold' test (vocabulary) which itself has been shown to significantly correlate with overall IQ ($r = .87$, p-value not reported; Wechsler, 1981).

A weakness of using prorated scores to determine overall IQ, i.e., cognitive deficit, is based on the skills being assessed by the subtests being used. Using the dyad cognitive deficit is being assumed if the patient performs poorly on a verbal test and, a test of construction. Actual cognitive deficit may be much

lower if other areas are considered. Though the dyad correlates well with overall IQ, an assumption is nevertheless made.

The second method for determining cognitive deficit is simply to administer a measure which assesses different cognitive skills. It is acknowledged, however, that this method is not as precise as a premorbid/postmorbidity comparison. This method is dependent upon the use of a set cut-off score; anything below which is considered a deficit score. One such measure that has been used in this manner is the Mini Mental State Exam (MMSE; Folstein, Folstein & McHugh, 1975). Though originally designed as a screening instrument for dementia the MMSE gives a gross estimate of cognitive functioning. Folstein et al. (1975) formalised this screening as many of the items were previously used by neurologists to screen for mental ability. As stated in Spreen & Strauss (1998) the purpose of the MMSE is to 'screen for mental impairment, particularly in the elderly' (p.65). Further, Folstein et al. state, 'It is "mini" because it concentrates only on the cognitive aspects of mental functions, and excludes questions concerning mood, abnormal mental experiences and the form of thinking. But within the cognitive realm it is thorough' (p.189). The MMSE is partitioned into five sections, each one assessing a different cognitive area: orientation, registration, attention & calculation, recall and, language.

In a study by O'Connor, Hyde, Fellows, et al. (1989) the psychometrics of the MMSE was studied using a clinical, elderly British population. Patients (N = 2302) were recruited from 5 general practices in Cambridge. Patients completed the MMSE and the Cambridge Mental Disorders of the Elderly Examination (CAMDEX; Roth et al., 1986). The CAMDEX is similar to the

MMSE in that it was designed to detect cognitive impairment in the elderly and, covers similar areas, e.g., orientation tasks.

Reliability of MMSE was assessed by test-retest and inter-rater agreement. Test-retest reliability (8 weeks) was calculated for 586 patients ($r = .84$, p -value not reported). Ten tape-recorded initial testings were reviewed by a team of psychiatrists (between 5-9) in session meetings to determine inter-rater agreement. The inter-rater reliability was good with a kappa range of .85-1.00 (mean $\kappa = .97$).

Criterion-validity for the MMSE was established in terms of percentages of the entire patient population who scored between set cut-off scores. Out of the 2302 patients, 2117 (92%) scored the customary cut-off score of over '23'. Sixty percent of the patients who scored '23' or below were subsequently found to have dementia or delirium (true-value ratio).

As further evidence for the MMSE having acceptable psychometrics, Tombaugh & McIntyre (1992) reported results from a literature review. The following results are from their paper.

Concerning internal reliability (Cronbach's α), four studies reported values for the MMSE. Holzer et al. (1984) reported a correlation of .77 ($p < .05$) in a community survey sample ($N = 4917$; age range = 18-85+). Kay et al (1985) reported a correlation of .68 ($p < .05$) in a community survey sample ($N = 274$; age range = 70-80+). Forman (1987) reported a correlation of .96 ($p < .05$) in a survey of medical patients (normal, dementia & delirium) ($N = 66$; mean age = 76). And, Jorm et al. (1988) reported a correlation of .65 ($p < .05$; grades 0-8) and .54 ($p < .05$; > grade 8) for a community survey sample ($N = 269$; age range = 70+).

Tombaugh & McIntyre cite 14 other studies where test-retest values were reported for the MMSE. Correlations range from .38 ($p < .05$) for control subjects ($N = 278$; mean age = 68 mo. test-retest interval; Morris et al., 1989) to .99 ($p < .05$) for dementia patients ($N = 23$; mean age = 74; 28 day test-retest interval; Folstein et al., 1975).

Concerning criterion-validity for the MMSE, Tombaugh & McIntyre cite 24 studies where '23' was used as the cut-off score for the MMSE. The range of sensitivity was 20% for an Alzheimer's dementia population ($N = 162$; age range = 65+; DSM III-R diagnostic criteria; Pfeffer et al., 1987) to 100% for cognitively intact where all cognitively intact subjects were identified as such ($N = 63$; mean age = 74; diagnostic criteria = psychiatric diagnosis; Folstein et al., 1975) and dementia patients populations ($N = 17$; mean age = 75; diagnostic criteria = psychiatric diagnosis; Folstein et al., 1975). Congruent-validity for the MMSE was also shown by Folstein et al. The MMSE was found to significantly correlate with the WAIS verbal IQ ($r = .78$, $p < .0001$) and WAIS performance IQ ($r = .66$, $p < .001$).

Deficit for the MMSE was defined in this thesis as a score of '23' or less as it was the most common cut-off point in the literature. Patients who had a total score of ≤ 23 were considered to have general cognitive deficit and were compared against those who scored ≥ 24 .

One advantage of using the MMSE is the brevity of its administration (5-10 minutes). This makes it suitable to use on populations where fatigue and/or maintaining attention may be an issue, i.e., elderly stroke patients. A second advantage of using the MMSE as a measure of general cognitive functioning is the generally agreed cut-off score of '23' and below being indicative of cognitive

impairment. This is particularly useful in research where subjects may be divided into dichotomous groups of cognitive deficit and no cognitive deficit.

A weakness of the MMSE is that while it gives an overall score of cognitive performance, the cognitive skills being assessed are limited. For research and screening purposes, it may be helpful to use such a quick measure but, in more clinical settings any deficit seen on the MMSE should be further assessed using more extensive measures. While the cut-off score of '23' is again useful for screening and research purposes, definite distinctions between patients who are truly demented or cognitively impaired and normals are easier made when the critical score is lower.

The Mental Status Questionnaire (MSQ; Kahn & Miller, 1978) was also used in this study as a general measure of cognitive deficit. The MSQ is a popular screening tool, particularly in clinical settings where a quick and easily administered measure may be used to assess cognitive status. The MSQ consists of 10 questions: age, month of birth, year of birth, current date, current month, current year, town, present location, prime minister and, previous prime minister. Kahn & Miller partitioned MSQ scores into three brackets: 0-2 (severe deficit), 3-8 (moderate deficit) and, 9-10 (mild deficit). Total possible score is out of 10.

In Lezak (1995), test-retest reliability for the MSQ is reported to be good ($r = .80$, p -value not reported) by different authors (Cohen et al., 1984; Leshner & Whelihan, 1986). The validity of the MSQ was examined in a study by MacKenzie, Copp, Shaw & Goodwin (1996). In this study, the MSQ was compared with the NART, MMSE, the CAMDEX and the Abbreviated Mental Test (AMT; Hodkinson, 1972). One hundred and forty-five elderly participants

(49 male, 96 female) were recruited from two group practices (Lothian area) at their annual 'over 75s' general health check.

Criterion-validity for the MSQ was shown as sensitivity, specificity, false positives and, false negatives were calculated against the CAMDEX as diagnostic criteria (score of <70). Using a cut-off score of 8-9, the MSQ had a sensitivity of 70%, a specificity of 89%, a false positive score of 38% and, a false negative score of 8%.

For the purposes of this thesis, general cognitive deficit on the MSQ was defined as a score of ≤ 8 . This division seemed logical as it excluded any score that might be questionable, i.e., mild deficit.

Taking approximately 3-5 minutes to administer makes the MSQ especially appropriate for elderly stroke patients. The administration time makes the MSQ appropriate for screening purposes in both research and clinical practice.

A weakness of the MSQ, as a measure of cognitive status, is due to its brevity. Having only 10 items, 5 of which are orientation-focused, limits its ability to fully assess the cognitive scope of the patient; particularly when the patient is not severely impaired.

The use of the WAIS-R and the MMSE as measures of general cognitive deficit is two-fold. Firstly, both measures have been extensively used in the literature as they were intended for in this thesis. Secondly, the author was familiar with the testing/administration and scoring of both of these measures at the beginning of this research; thus ensuring the measures were administered in the intended standardised fashion. The MSQ was chosen as it was also an initial

measure of cognitive deficit which was included in the information provided for each patient by the hospital.

Table 2.5 Summary of General Cognitive Deficit Measures

	reliability	validity	deficit defined	pros	cons
Prorated WAIS-R	reported as high	reported as high	difference of ≥ 13 points compared with NART FSIQ	faster than full WAIS-R; subtests correlate highly with FSIQ	cognitive ability is assumed
MMSE	test-retest; inter-rater agreement: high κ score; Cronbach's α	criterion: most patients scoring $\leq '23'$ were later diagnosed with dementia or delirium, 100% sensitivity reported; congruent: significant correlation with WAIS-R verbal IQ	cut-off score of 23-24	quick and easy to administer; cut-off score is useful for research	limited cognitive skills are assessed; difficult to make clear diagnoses when score is near '23'
MSQ	test-retest: .80 as reported by different authors	criterion: 70% sensitivity, 89% specificity, 38% false negative, 8% false positive	cut-off score: ≤ 8	quick and easy to administer	risk of type II error due to its brevity

Specific Cognitive Deficit

As with measuring general cognitive deficit, when assessing specific cognitive deficits it is helpful to make conclusions based on individuals' premorbid performance. Again, many tests provide normative data from which comparisons may be made but, it is not as accurate as a comparison against a premorbid level. Where possible, premorbid estimation/postmorbidity comparisons were made in defining specific cognitive deficits.

In measuring specific cognitive deficits one should determine areas that assess the spectrum of patients' abilities. Lezak (1995) lists eight areas that comprise a 'basic battery' of neuropsychological assessment: attention, visuoperception/visual reasoning, memory & learning, verbal functions & academic skills, construction, concept formation, self-regulation & motor ability and, emotional status. This basic battery served as a template for the specific cognitive areas assessed in this thesis (with the exception of academic skills).

Orientation

Orientation is the patient's self awareness of themselves within their environment. Though the concept may seem simplistic, this ability requires constant updating of perceptions which requires attention and the ability to compare and integrate a notion of time. A formal test of orientation is usually only done when, after the mental status examination, there is reason to suspect the patient has a deficit. In research, however, measuring orientation may be needed to insure the subjects meet a basic level of cognitive awareness. Deficits in orientation usually occur in descending order of: person, place and time, where time disorientation is first to occur. The test for 'person' is to simply ask the patient their name. Testing 'place' requires the patient to state the name or location of the testing site. 'Place' may also be assessed by gauging the patient's appreciation of geography and direction to and from the testing site. 'Time' is assessed by asking the patient the date and day of week. 'Time' has gained more attention in the literature as formal tests have been developed to assess this aspect of orientation, e.g., the Discrimination of Recency test (Milner, 1971). Time, however, was only one aspect of orientation being assessed in this thesis.

Unlike other areas of cognitive deficit being discussed in this chapter, the assessment of orientation was not included in any formal analyses. Orientation is, therefore, not discussed in a comparative fashion with other tests. The purpose of assessing orientation in this thesis was to ensure the patients satisfied part of the inclusion criteria, i.e., being oriented x 3 (person, place and, time).

Patients' orientation was measured in this thesis using the orientation section of the MMSE. This section of the MMSE adequately measures the patients' knowledge of the three aspects of orientation, and takes less than a minute to assess. It also allowed a standardised method of assessing orientation to be carried out. Administering another test where the assessment of orientation was measured was unnecessary as the MMSE orientation section satisfied the criteria.

For the purposes of the criteria met in this thesis, orientation is either satisfied or is not. Given its brevity and dichotomous nature, this measure was appropriate for the formal assessment of orientation for elderly stroke patients.

Visual Neglect

Following stroke many patients experience unilateral neglect. This is an unawareness of perception for the half of the patient contralateral to the hemisphere where a unilateral stroke has occurred. Often this imperception is manifest in the form of visual neglect. As stated by Halligan, Marshall & Wade (1989), 'Visual neglect, a frequent consequence of unilateral (usually right hemisphere) stroke, is associated with poor functional recovery and in many patients is resistant to remedial treatment' (p.908). This association with poor

functional recovery warrants its inclusion as an area of specific cognitive deficit being examined in this thesis.

One measure that has been used in research to study visual neglect is a subtest of the Behavioural Inattention Test (BIT, Wilson, Cockburn & Halligan, 1987), star cancellation. Star cancellation consists of several stars (52 large and 54 small), 13 letters and, 10 short words positioned on a page. The small stars are positioned 27 on the left half of the page and 27 on the right. The patient's position relative to the page is important when taking this test. The patient's midsagittal plane must be equal to the center of the page to ensure the visual field is divided.

Normative data for star cancellation is provided in the BIT manual. The BIT was administered to 50 normal control participants (14 males, 36 females; age range = 22-82; mean age = 58.2; SD = 13.5). The controls were 'non-brain damaged' recruited from several sources, e.g., university students, hospital employees. The controls were found to have a mean score of 53.72 (out of a possible 54) (SD = .54; range = 52-54; cut-off score = 51).

Specific psychometrics for the BIT subtests, e.g., star cancellation, were not reported in the manual. The psychometrics of the BIT were assessed using unilateral cerebral lesion patients (N = 80; 52 male, 28 female; mean age = 56.2; SD = 10.5) who had entered a rehabilitation unit over an 18mo. period.

Inter-rater reliability was assessed using a subsample of 13 patients, scored independently by two examiners. Inter-rater reliability was high ($r = .99$, $p < .001$). Parallel form reliability (A & B) was assessed using 10 patients and was also high ($r = .91$, $p < .001$). Test-retest reliability (mean interval = 15 days) was also assessed using 10 patients and was high ($r = .99$, $p < .001$).

Validity for the BII was measured using the 80 patients recruited for the study. The content validity of the BII was assessed by examining the relationship between the 'conventional' tests of unilateral neglect on the BII (line crossing, letter cancellation, figure and shape copying, line bisection and, representational drawing) and its 'behavioural tests (picture scanning, telephone dialling, menu reading, article reading, telling and setting time, coin sorting, address and sentence copying, map navigation and, card sorting). The content validity correlation between the conventional and behavioural tests was high ($r = .92, p < .001$). Criterion validity for the BII was also assessed by correlating patient responses to a short questionnaire with their behavioral subtest scores ($r = .67, p < .001$).

Deficit for star cancellation was defined in this thesis as 3 or more errors on the same half of the page as determined by the protocol in the manual. This cut-off score was determined from the scores of the normative sample discussed above.

Two advantages of star cancellation are its lack of complexity and short administration time. The patient must simply cross-out the small stars on the page which takes approximately 3 minutes to complete. This makes star cancellation an appropriate measure of visual neglect for stroke patients.

A possible weakness of star cancellation is that it is purely a 'cancellation' test. Though a patient's performance is likely to reflect a presence/absence of visual neglect (as discussed below), authors such as Crawford, Parker & McKinlay (1992) suggest the need for an array of visual neglect tests to truly determine visual neglect.

A second measure of visual neglect is another 'conventional' subtest of the BIT which has often been used in research is figure and shape copying. The procedure for this subtest is again to align the patient's midsagittal plane with the center of the page. The format of this test, as given in the BIT, is as follows. The page consists of three figures/shapes (star, cube, daisy) that are arranged on the left side of the page in a column. The patient is to copy the figures/shapes on the left hand side of the page in spaces provided. Next, a page with geometric shapes is presented and the patient is asked to copy the figures that they see. Scoring is based on the number of correct representations drawn, lacking any omissions (maximum score = 4).

The normative data for figure and shape copying was gathered on the same sample as star cancellation. All normal subjects fully performed this subtest (mean score = 4; SD = 0). The cut-off score for this subtest was therefore 3. The psychometrics for figure and shape copying are in relation to the entire BIT, as was stated above with star cancellation.

This is another measure of visual neglect that is easy to administer and takes approximately 5 minutes to complete. A possible weakness of figure and shape copying is as a measure of visual neglect, it relies solely upon copying. The unitary nature of using copying excludes other skills which may be indicative of visual neglect.

A third measure of visual neglect that has been used in the literature is another 'conventional' subtest in the BIT, line bisection. The BIT procedure for line bisection is as follows. The center of the page is presented equally to the patient's midsagittal plane. The page consists of three horizontal lines which are staggered in a staircase fashion. The patient is told to simply divide each by

drawing a line through the middle. The maximum score for each line is 3 which is achieved if each dividing mark is within $\frac{1}{2}$ an inch of the true middle. Other scores are in reference to the line bisection template.

As with the two previous subtests of the BII, normative data were based on the same sample. The normal population performed nearly perfect (mean score = 8.96; SD = .10). The cut-off score for line bisection is 7. As was the case with star cancellation and figure and shape copying, the psychometric properties of line bisection were reported in terms of the full BII (reported above).

Line cancellation is another measure which is quick and easy to administer, taking approximately 5 minutes to complete. Again, as with star cancellation and figure & shape copying, line cancellation is a unitary measure of visual neglect. This possible weakness may be compounded when administered only once. Lezak (1995) discusses three reasons for this: a natural deviation toward the left of center (normals tend to mark the center 1 to 2mms. left of the true center), increased accuracy with shorter lines (Halligan & Marshall, 1988) and, less sensitivity using the ipsilateral hand to the damaged hemisphere (Schenkenberg, Bradford & Ajax, 1980).

In further study of the psychometric properties of the visual neglect measures discussed above, data were examined from a study conducted by Halligan, Wade & Marshall (1989). In their study, six conventional tests of visual neglect were assessed with regard to their homogeneity and their sensitivities for identifying visual neglect. These tests were: line crossing, letter cancellation, star cancellation, figure and shape copying, line bisection and, representational drawing. This study included 80 unilateral stroke patients

(males = 52, females = 28; mean age = 57; SD = 9.6; age range = 19-83).

Patients were administered all tests and their data were analysed.

Factorial construct validity was assessed by a principal component factor analysis using the six measures which significantly correlated with each other at the .0001 level of significance. A single factor was determined which accounted for 72.6% of the total variance thus suggesting that the tests measure the same construct. Factor loading for each of the measures were as follows: line crossing (.81), letter cancellation (.87), star cancellation (.90), figure and shape copying (.92), line bisection (.85) and, representational drawing (.75).

In determining the sensitivities of the measures to identify visual neglect, cut-off scores were determined based on 50 age-matched controls. Visual neglect was determined for each measure as ≥ 1 point below the lowest score for the controls. To be more precise, overall (aggregate performance) visual neglect was determined as a total cut-off score of 130-146. Only 30 patients (male = 16, female = 14; mean age = 58.2; SD = 8.3) met the overall criteria of a cut-off score of 130-146, for which sensitivity was examined. Criterion validity was assessed for each of the measures by determining the number/percentage of the 30 patients who scored below the cut-off for each test. Results were as follows: line crossing (17, 57%), letter cancellation (24, 80%), star cancellation (30, 100%), figure and shape copying (22, 73%), line bisection (16, 53%) and, representational drawing (11, 37%). The one test that showed full sensitivity (100%) was star cancellation. The other two measures discussed previously were less sensitive (figure and shape copying = 73% and line bisection = 53%).

Star cancellation was chosen as the measure for visual neglect primarily based on the previously discussed findings of Halligan et al. where star cancellation was found to be the most sensitive test of visual neglect. Also, at the beginning of this research the author was familiar with the testing/administration and scoring of this measure.

Table 2.6 Summary of Visual Neglect Measures

	reliability	validity	deficit defined	pros	cons
BIT: star cancellation	inter-rater; parallel form; test-retest	content: 'conventional' subtests significantly correlated with 'behavioural' subtests; criterion: 'behavioral' subtests significantly correlated with questionnaire, identified 100% of visual neglect patients	≥ 3 errors on same half of page	quick and easy to administer	unitary measure of visual neglect
BIT: figure & shape copying	same	content; criterion	-	quick and easy to administer	unitary measure of visual neglect; less sensitive than star cancellation
BIT: line bisection	same	content; criterion	-	quick and easy to administer	unitary measure of visual neglect; less sensitive than star cancellation

Memory

The processes by which memory occurs and the conceptualisation of it as a construct is complex. As stated by Baddeley (1990), '...it is not one system but many. The systems range in storage duration from fractions of a second up to a lifetime, and in storage capacity from tiny buffer stores to the long-term memory system that appears to far exceed in capacity and flexibility the largest available computer' (p. 4). Measures which test memory are varied according to the type of memory being assessed, e.g., verbal, nonverbal, visual, tactile, remote, etc.

One widely used measure of memory in the literature is the digit span subtest of the WAIS-R (Johansson & Berg, 1989; Storandt, Botwinick & Danzinger, 1986). Digit span consists of a series of digits (becoming progressively harder) orally presented to the patient. The subjects' task is to repeat as many digits back as possible ('forwards' section). The 'backwards' section is presented the same but the subjects repeat the digits in reverse order.

As a subtest of the WAIS-R, digit span has strong psychometric properties. Test-retest data, as shown in the WAIS-R manual, were gathered on a large sample of 'normal' subjects between 115 testing centres throughout 39 U.S. states over a 4 yr. period (N = 1880; males = 940, females = 940; age range = 16-74). The age-average (across all age groups) test-retest reliability coefficient for digit span, as reported in the WAIS-R manual, is good ($r = .83$, p-value not reported).

Congruent validity for digit span was provided in a study by Heilbronner, Henry, Buck, Adams & Fogle (1991). Their study examined the performance of lateralised brain injury patients (N = 121; mean age = 30.1; SD =

12.2) on specific memory tests; two of which were the Tactual Performance Test (TPT; Halstead, 1947) and digit span. The TPT consists of a board with different shapes cut into it. The patient's view is blocked as they feel what the shapes are and where they are located on the board. The patients then draw from memory the shapes ('memory') and their placements ('location') on the board.

Heilbronner et al. (1991) found that digit span forward significantly correlated with TPT memory ($r = .23, p < .01$). They also found that digit span backwards significantly correlated with TPT memory ($r = .23, p < .01$) and TPT location ($r = .24, p < .001$).

Deficit was defined in this thesis for digit span in relation to the patients' NART (WAIS-R) performance IQ estimation (premorbid estimation). This was done in two steps. First, the digit span subscale score was transformed into a standardised score with the mean being 100 and standard deviation being 5, e.g., a subscale score of 8 would be 90 standardised = $[100 - ((10-8) \times 5)]$. In other words, this equation subtracts the patient's subscale score from the mean scaled score which, is subtracted from the mean standardised score and multiplied by the standard deviation. Secondly, the difference was taken between the patient's NART PIQ score and the digit span standardised score. If the difference was ≥ 13 , then the patient's digit span score fell above the 5th percentile (as indicated by the NART manual) for a normal population, thus indicating a deficit at the .05 level of significance.

As a test of memory, digit span provides a quick (approximately 5 minutes) and easy measure of short-term memory. Its trait-validity is obvious as recall (memory) progressively reaches a plateau at a certain length of presentation. Its use as a memory test, however, is limited as it is based solely on

the recall of digits. This precludes it to measuring other areas of memory which may be affected; and may not be detected by this test alone.

Another popular test of memory is the Rivermead Behavioural Memory Test (RBMT; Wilson, Cockburn & Baddeley; 1989). The RBMT was developed, 'to provide measures that could be directly related to the practical effects of impaired memory and for monitoring change with treatment for memory disorders. It was also designed to have good face validity so that its findings would be acceptable to nonpsychologists' (p.510; Lezak, 1995). The RBMT comprises 12 subtests developed to assess everyday memory, e.g., remembering: a name, a hidden belonging, an appointment, a new route, etc.

The psychometrics of the RBMT, as stated in the manual, are reported in Lezak (1995). Support was given for the reliability of the RBMT: inter-rater reliability was reported to be perfect (100%). Parallel-form reliability for the profile score of the RBMT between forms A, B and C was good, ranging from .83 to .88 (p-values not reported).

Studies have shown the validity of the RBMT in head-injury populations. Baddeley et al. (1987) showed that compared to normal subjects, who were able to pass all of the items on the RBMT, head-injury patients passed on average 47% of the items. Congruent validity for the RBMT was shown in a study by Stewart, Sunderland & Sluman (1996) which examined memory disorders of stroke patients (N = 70, 47 males, 23 females) long after their strokes (12-72 mo. post-stroke). The patients were recruited from a stroke registry and admissions records from a general hospital. Once recruited, the patients were tested in-home. The patients were administered the RBMT and the Everyday Memory Questionnaire (EMQ-20; Sunderland, Harris & Gleave,

1984). The authors performed a Spearman rank correlation between the RBMT and the EMQ-20 and found a significant relationship between the two tests of memory ($r = -.41, p < .01$, one-tailed).

The particular advantage of the RBMT is its thoroughness in measuring different areas of memory which are practically based on everyday skills. Such a thorough measure however, makes the RBMT awkward to administer for research purposes which look at other cognitive areas besides memory due to its somewhat lengthy administration time (approximately 30 minutes).

Another commonly used measure of memory assessment is the WMS-R (Wechsler, 1987). This measure has nine subtests divided into sections which measure general memory, verbal memory, visual memory, logical memory, attention and concentration and, delayed recall.

The psychometric strengths of the WMS-R were thoroughly discussed by Spreen & Strauss (1998). As they stated, the normative study of the WMS-R suggested good test-retest reliability (median reliability = .74). They also reported authors (Mittenberg et al., 1992) who found the WMS-R subtests to have a good range of reliability coefficients: logical memory (Spearman-Brown = .71) and visual reproduction (Cronbach's $\alpha = .87$).

Concerning the validity of the WMS-R, Spreen & Strauss (1998) cited other studies where congruent validity of the WMS-R was suggested. As they state, performances on the WMS-R and the California Verbal Learning Test (CVLT; another comprehensive test of memory; Delis, Kramer, Kaplan & Ober, 1987) have been shown to be high, e.g., between WMS-R delayed memory and CVLT long delay free recall ($r = .93$, p-value not stated; Delis et al., 1988). Spreen & Strauss also state that Randolph et al. (1994) found that the WMS-R

general memory index significantly correlated with the CVLT total score ($r = .79$, p -value not reported).

The main strength of the WMS-R is that it assesses several different aspects of memory while giving a general memory quotient; much in the same way as the WAIS-R provides VIQ, PIQ and FSIQ. The WMS-R has been further validated by numerous authors for different populations and ages.

The largest weakness of the WMS-R, as noted in Lezak (1995), is that only one form of it was developed. This is a particular weakness in its usefulness in research as it makes it susceptible to practice-effects in longitudinal studies. Also, for the purposes of this thesis, the WMS-R suffers from the same impracticality as the RBMT, i.e., administration time (45 minutes).

There are several other tests of memory which are more comprehensive than digit span (Rivermead Behavioural Memory Test, RBMT; Wechsler Memory Scale-Revised, WMS-R; CVLT). However, as memory was but one aspect of interest within this thesis and administration time had to be minimal, digit span was preferred over more complex measures of memory. Again, the researcher was also familiar with the testing/administration and scoring of this test at the beginning of the research.

Table 2.7 Summary of Memory Measures

	reliability	validity	deficit defined	pros	cons
digit span	test-retest	convergent	≥ 13 difference with NART PIQ	quick & easy administration	only measures immediate recall
RBMT	Interrater; parallel-form	congruent	-	thorough measure of memory based on everyday skills	lengthy administration time
WMS-R	test-retest	congruent	-	thorough measure; validated by several authors	only one form; lengthy administration time

Construction

Constructional skills rely on the ability to form a whole given its parts, i.e., gestalt ability. As stated in Lezak (1995), 'Construction performance combines perceptual activity with motor response and always has a spatial component' (p.559). Construction in its entirety is divided into two classes due to the differences in information processing: drawing and building. In this thesis 'building' construction was primarily examined. The 'drawing' aspect of construction was cursorily examined in the language section of the MMSE where one of the tasks is for the patient to copy a polygon.

One popular measure used to examine construction is the block design subtest of the WAIS-R. Block design is widely used in the literature as a measure of construction and within the WAIS-R is 'generally recognised as the best measure of visuospatial organization' (Lezak, 1995). In administering block design, the subject is shown a card with a design on it from which either four or nine blocks (depending on the progressive difficulty of the items) are used to

replicate it. Each block has two sides red, two sides white and two sides half-red/white. Points are assigned as either 0,1 or 2 depending upon the number of trials to complete and the length of time.

Test-retest data, as shown in the WAIS-R manual, were gathered on a large sample of 'normal' subjects between 115 testing centres throughout 39 U.S. states over a 4 yr. period (N = 1880; males= 940, females = 940; age range = 16-74). The age-average (across 9 age groups) split-half reliability for block design, as reported in the WAIS-R manual, is high. The average coefficient for each age group was first computed into Fisher's Z-statistic and then the mean Z-value was recalculated into the age-average correlation ($r = .87, p < .01$).

Criterion validity of block design was shown in a study by Hemmingsen, Mejsholm, Boysen & Engell (1982) where cognitive functions of stroke patients were compared before and after surgery for internal carotid artery stenosis. Such stenosis had caused strokes in 25 patients recruited to this study (13 right-hemisphere, 12 left hemisphere; 17 males, 8 females; mean age = 56). Following surgery to remove the patients' stenoses (and hence blood flow) cognitive functions were retested. Importantly, block design has been considered to be mainly a right-hemisphere orientated test. As the authors state, 'This test requires visuo-motoric organization, and is related to right hemisphere function (Lezak, 1976)' (p.149). At an 8mo. post-operative testing, performance on block design was higher (paired t-test) at the .10 level of significance. Block Design performance was also significantly higher (paired t-test) in patients who were operated on the right side compared to those operated on their left: mean score of left-side operated patients = 8.6; mean score of right-side operated patients = 10.9 (significantly different at the .005 level).

Deficit performance on block design in this thesis was determined in the same manner described above for digit span. The patients' scaled score was first standardised and, then compared with the NART PIQ. Differences of ≥ 13 points was considered 'deficit'.

Block design is a quick and easily administered construction test with good trait/face validity. As a subtest of the WAIS-R, it has been thoroughly validated on different populations and ages, in the original standardisation sample and by numerous other authors.

A possible weakness of block design is that it may be influenced by health conditions, e.g., arthritis, above and beyond what is accounted for in the standardised normative data. Performance on this test is dependent, in part, on speed. It is possible that individuals with certain conditions, such as arthritis, may obtain lower scores and not have significant deficits in constructional ability.

Another measure of construction is the object assembly subtest of the WAIS-R. This test consists of four presentations of pictures (manikin, profile of a head, hand, elephant) which are segmented into a progressive increase of pieces as the difficulty of the items progress. Scoring is based on the number of complete pictures and the times taken to complete each.

The psychometric reliability of object completion, as stated in the WAIS-R manual, is good. Test-retest data, as shown in the WAIS-R manual, were gathered on a large sample of 'normal' subjects between 115 testing centres throughout 39 U.S. states over a 4 yr. period ($N = 1880$; males = 940, females = 940; age range = 16-74). The age-average (across 9 age groups) split-half reliability for object assembly, as reported in the WAIS-R manual, is good.

The average coefficient for each age group was first computed into Fisher's Z -statistic and then the mean Z -value was recalculated into the age-average correlation ($r = .68, p < .01$).

Factorial validity for the previous measure, block design, and object assembly as being measures of construction was shown in a study of the WAIS by Cohen (1957a). In his study, he used the original normative sample ($N = 1152$) in the WAIS manual to create a factorial structure equation. The original intercorrelation matrices were used for the factorial equation (Thurstone's complete centroid method). Five factors were determined, one being 'perceptual organization'. Both block design and object assembly loaded significantly on 'perceptual organization' at each of the four age groups (significance = factor loadings of $\geq .20$).

As with block design, object assembly has been validated on a large normative sample including different populations and ages. Again, a major flaw of object assembly is its dependence upon speed; of which certain conditions can result in an invalid score of constructional skill.

A third measure of construction is the Rey-Osterrieth Complex Figure test (ROCF; Rey, 1941). The ROCF is a complex figure presented to the subject. The subject copies the figure onto another page. The original scoring method assigns points for correctly drawn representations of the figure which, is determined by each of its sections. The ROCF may also be used as a test of memory by asking the subject to draw from memory the complex figure. As discussed here though, the ROCF is being examined as a test of construction.

The reliability of the ROCF has been shown in the literature to be high (Rapport et al., 1997). In their study, the ROCF was administered to 318

veterans in a hospital setting (312 males, 6 females; mean age = 55.01; SD = 14.31). Inter-rater reliability was determined for the ROCF by 'three independent raters' and shown to be high ($r = .98, p < .001$).

Literature which suggests validity for the ROCF as a measure of construction is cited in Spreen & Strauss (1998). They cite a study by Meyers & Meyers (1995) who conducted a principle-components factor analysis of the ROCF. In their study, five factors were determined to comprise the ROCF. One factor called 'visuospatial constructional ability' which, had a high loading of the 'copy' score.

A strength of the ROCF is that its widespread use had lead to it being validated on several populations and ages. As a pure measure of construction, however, performance on the ROCF can be affected by other skills which affect its performance, e.g., planning, problem-solving, motor and memory (Spreen & Straus, 1998).

Block design was chosen in this thesis as the test used to measure constructional ability. This test was chosen primarily because, of its psychometric properties as a measure of constructional ability; already included in a larger measure of which the author was familiar and administering tests from (WAIS-R)

Table 2.8 Summary of Construction Measures

	reliability	validity	deficit defined	pros	cons
block design	test-retest	criterion	≥13 point difference between standardised score and NART PIQ	quick and easy to administer; validated on several populations and ages	certain conditions may lead to invalid scores
object assembly	test-retest	criterion	-	quick and easy to administer; validated on several populations and ages	certain conditions may lead to invalid scores
ROCF	Interrater	factorial	-	validated on several populations and ages	scores are influenced by other skills besides construction

Executive Function

The term 'executive function' encapsulates cognitive abilities that allow one to reason, problem solve and, think conceptually. Lezak (1995) differentiates executive function from other cognitive functions by stating that executive functioning skills, 'asks how or whether a person goes about doing something (e.g., Will you do it and, if so, how?). Spreen & Strauss (1998) describe executive function more thoroughly by saying that it is a, 'shorthand description of a multidimensional construct referring to a variety of loosely related higher-order cognitive processes including initiation, planning, hypothesis generation, cognitive flexibility, decision making, regulation, judgement, feedback utilization, and self-perception that are necessary for effective and contextually appropriate behavior' (p.171). As this cognitive ability is so diverse in its conceptualisation, executive functioning has been

divided into two measurement areas: executive functioning (concept formation/reasoning) and executive functioning (perceptual set shifting).

concept formation/reasoning

One measure of executive functioning is the comprehension subtest of the WAIS-R. This test consists of sixteen open-ended questions. Taking the description given by Lezak, the questions asked by this measure directly relate to asking how or whether a person goes about doing something (good face validity). An example of such a question is, 'What should you do if while in the movies you are the first person to see smoke and fire'? An appropriate answer reflecting proficient executive functioning would be any answer indicating that one should notify a person in authority, e.g., manager. An answer reflecting poor executive functioning would be one which is disjointed or inappropriate, e.g., 'go for water'.

Test-retest data, as shown in the WAIS-R manual, were gathered on a large sample of 'normal' subjects between 115 testing centres throughout 39 U.S. states over a 4 yr. period (N = 1880; males = 940, females = 940; age range = 16-74). The age-average (across 9 age groups) split-half reliability for vocabulary, as reported in the WAIS-R manual, is high. The average coefficient for each age group was first computed into Fisher's Z-statistic and then the mean Z-value was recalculated into the age-average correlation ($r = .84, p < .01$).

In a study of 227 stroke patients, comprehension was used as a measure of cognitive ability against 240 controls. Results suggesting discriminant validity for comprehension showed that stroke patients performed significantly poorer than the controls (Tatemichi et al., 1994).

Factorial validity for comprehension as being measures of executive functioning was shown in the previously mentioned study of the WAIS by Cohen (1957b). In his study, he used the original normative sample (N = 1152) in the WAIS manual to create a factorial structure equation. The original intercorrelation matrices were used for the factorial equation (Thurstone's complete centroid method). Five factors were determined, one being 'verbal comprehension', defined as 'Vocabulary richness and *verbal-symbolic manipulative ability*' (p.451). comprehension loaded significantly on 'verbal comprehension' at each of the four age groups (significance = factor loadings of $\geq .20$).

Deficit performance on comprehension in this thesis was determined in the same manner described above for digit span and block design. The patients' scaled score was first standardised and, then compared with the NART VIQ. Differences of ≥ 13 points was considered 'deficit'.

As with digit span and block design, comprehension has been thoroughly validated on large samples of diverse populations and ages. A particular weakness of comprehension is that its performance can be affected by the education level (Lezak, 1995) of the subject, e.g., knowing the advantage of free press in a democracy. Another weakness is that compared with other subtests of the WAIS-R, scoring may vary among examiners for responses which do not fit the examples given in the manual. This can lead to questionable reliability when given by inexperienced examiners.

perceptual set shifting

The second measure being used in this thesis to measure executive functioning is the Trail Making Test-part B (Armitage, 1946). For research purposes, Trail Making is useful because, there are few tests of executive functioning which, yield a direct score without having a projective scoring element to them while at the same time being fairly quick and easy to administer (Lezak, 1983).

Originally, this test was chosen as a measure of motor function. To this end it still may be thought as such. However, upon examination of the literature Trail Making is considered to be a measure of executive function with visuomotor skill being a component (Hom & Reitan, 1990). Reitan & Wolfson (1995) cite six studies where trail making-B and the category subtest of the Halstead-Reitan Neuropsychological Battery (discussed next) are, '...identified as "frontal lobe tests", i.e., measure of executive functioning (Butters, Kaszniak, Glisky, Eslinger & Schacter, 1994; Grodzinsky & Diamond, 1992; Jarvis & Barth, 1994).

Trail making-B consists of a sheet of paper with 25 scattered circles presented on it. In each of the circles there is either a number or a letter. There are 13 numbers and 12 letters. The subject is asked to place his/her pencil in the center of the circle with the number '1' and draw a line to each of the other circles without lifting the pencil. The order that the lines are drawn alternates from a number to a letter in ascending order of both ending on the number '13'. The subject has a sample trial that precedes the test. To correctly complete trail making, requires skills of *visual planning*, visuomotor dexterity and *shifting of perceptual set* (Spreeen & Strauss, 1998).

Interrater reliability for trail making-B, as reported in Spreen & Strauss (1998) was high ($r = .90$, p-value not reported; Fals-Stewart, 1991). The authors also cited another study where test-retest reliability for trail making-B was good over a one-year period for a group of older subjects ($N = 100$; mean age = 67; $r = .72$; Snow et al., 1988).

In a study seeking to examine the construct validity of Trail Making, it was determined that Trail Making-B was more difficult than part A not simply because of it being a longer test but, 'also because of its increased demands in motor speed and visual search (Gaudino, Geisler & Squires; 1995). It is also a sensitive measure to the presence of cerebral dysfunction (Armitage, 1946; Reitan, 1958). Criterion validity for trail making-B was suggested by Lezak (1995) who cited a study by Segalowitz, Unsal & Dywan (1992) who found significant correlations between performance on trail making-B and electrophysiological measures (Contingent Negative Variation) that appear to be 'associated with frontothalamic functioning'.

Deficit for trail making-B was defined as a total time of completion, ≥ 86 seconds as defined by the authors Reitan & Wolfson (1988).

As stated earlier a particular strength of trail making-B as a measure of executive functioning, is that its scoring does not depend on projective aspects unlike most measures of executive functioning, e.g., comprehension.

A particular limitation of trails-B is that its performance can be affected by other aspects of the subject besides executive functioning skill, e.g., eye sight and motor speed. This would affect the reliability of this test under certain conditions of the subject such as glaucoma, Parkinson's disease, etc.

A third measure commonly used for assessing executive functions is the Category Test (Halstead, 1947). As stated above, several studies have considered the category test as a measure of frontal lobe functioning, i.e., executive functioning (Bigler, 1988; Butters, Kaszniak, Glisky, Eslinger & Schacter, 1994; Jarvis & Barth, 1994; Schute & Huertas, 1990). This test assesses executive functioning by presenting the patient with a stimulus from which the patient's task is to determine which answer stimuli (four choices) best represents the underlying principle being represented by the test stimulus. As the test progresses, the underlying principles change and, where previous responses were once correct, the subject has to then determine what the new response should be.

As cited in Spreen & Strauss (1998), this measure has been reported to have high odd-even split-half reliability for samples of both 'normal and brain-damaged adults' ($r = .95$ and above, p -values not reported; Charter et al., 1987; Moses, 1985; Shaw, 1966). The authors also cite studies where the category test has been found to have high test-retest reliability on 'severely impaired neuropsychological patients' over a 2 year interval ($r > .90$, p -value not reported; Goldstein & Watson, 1989; Matarazzo et al., 1974; Russell, 1992).

In a study by Corrigan, Agresti & Hinkeldey (1987), the psychometric properties of the category test were measured. The authors hypothesised that the category test would correlate more highly with WAIS-R performance IQ over verbal IQ 'to the extent that it (the category test) assesses fluid intellectual abilities' (convergent validity). The subjects included 102 patients (67 males, 35 females; mean age = 44.25) who had been admitted to hospital for either closed head injuries caused by automobile accidents or for stroke. The results showed

that the category test significantly correlated with PIQ ($r = -.64, p < .01$) whereas it did not significantly correlate with VIQ ($r = -.11, p = ns$).

A particular strength of the category test is that its performance does not depend upon expressive speech and, is not heavily dependent upon motor skill (Corrigan et al., 1987). Also, this test has been modified from its original computer-based form to include several different form of presentation, e.g., booklet, intermediate-form and, short-form.

A weakness of the category test which has been shown in the literature is that performance can be affected by education and age, for which normative data can correct for, but, also intellectual level and ethnicity (Arnold et al., 1994, as cited in Spreen & Strauss, 1998) for which are not accounted for. Though this test has been shown to be a valid test of executive skill, its administration time is lengthy (40 minutes to 2 hours) making it unsuitable for this thesis.

The psychometric evidence for comprehension and Trail Making as executive functioning tests made these tests appropriate for this thesis. Also, given that the comprehension subtest was part of the WAIS-R, from which tests for this thesis were already being given, and the ease of administration for the Trail Making Test, accompanied with the examiner's familiarity with both measures, comprehension and Trail Making were chosen as measures in this thesis to assess executive functioning. A final reason for choosing these two measures is that as discussed earlier, the notion of executive ability is diverse and was divided into two sections in this thesis. The two measures chosen were specific to the two sections.

Table 2.9 Summary of Executive Functioning Measures

	reliability	validity	deficit defined	pros	cons
comprehension	test-retest	discriminant; factorial	≥13 point difference between standardised score and NART VIQ	validated on a large diverse sample of different ages	scores can be affected by education; questionable reliability if scored by inexperienced examiners
trail making-B	interrater; test-retest	construct; criterion	≥86 seconds	scoring is unaffected by projective elements	scoring may be affected by certain conditions of the subject
categories	split-half; test-retest	convergent	-	scoring does not depend upon expressive speech or motor speed; different forms	scoring can be effected by age, education, I.Q. and ethnicity

Verbal

Verbal ability consists of different skills of which vocabulary is one. Examples of verbal skills are: speech production and comprehension, naming ability, reading and writing. The verbal ability being assessed in this thesis is vocabulary. A major reason for this choice is its integral relationship with overall cognitive ability.

One popular measure for assessing verbal ability is the WAIS-R vocabulary subtest. This test is based on the quality of a definition for a given word (35 words in total) presented orally to the patient. Scores are assigned by the researcher (2,1 or 0) depending on the accuracy of the definition. An example of a good definition, e.g., score of 2 to the word 'assemble' would be 'To bring together several parts to make a whole'. A score of '0' would be 'A

group of people'. Though this measure is somewhat lengthy in its administration (approximately 15 min.), as a sensitive test of verbal ability its administration time is comparable to other popular tests of verbal skill. As a subtest of the WAIS-R, vocabulary has strong psychometric properties.

Test-retest data, as shown in the WAIS-R manual, were gathered on a large sample of 'normal' subjects between 115 testing centres throughout 39 U.S. states over a 4 yr. period ($N = 1880$; males = 940, females = 940; age range = 16-74). The age-average (across 9 age groups) split-half reliability for vocabulary, as reported in the WAIS-R manual, is high. The average coefficient for each age group was first computed into Fisher's Z -statistic and then the mean Z -value was recalculated into the age-average correlation ($r = .96$, $p < .01$).

Convergent validity was demonstrated for vocabulary using the same population of 1880 subjects. The same transformation using Fisher's Z was used to calculate age-average scores across the 9 age groups. The age-average vocabulary subtest correlated highly with both the verbal IQ score and the full-scale IQ score ($r = .90$ and $.85$ respectively; p -values not reported).

Criterion validity was also demonstrated for vocabulary in a study by Reitan, Hom & Wolfson (1988). In their study, the authors proposed a brain-behaviour relationship whereby left-hemisphere lesion patients would perform poorly on verbal tests as compared to right-hemisphere lesion patients and controls who had no history of brain injury. All subjects were matched for age, education and handedness. The procedure for recruitment was not reported. Each of the three groups were comprised of 24 males and 2 females (mean age = 40.85, $SD = 13.6$). All subjects were administered the vocabulary subtest and the Word Finding Test (Reitan, 1972). Univariate analysis supported the authors

hypothesis and demonstrated criterion-validity for vocabulary as a good measure of verbal ability. Left-hemisphere lesion patients performed significantly worse than the right-hemisphere lesion patients and the controls on vocabulary (L-hem. mean score = 8.81, SD = 3.39; R-hem. mean score = 11.23, SD = 2.39; controls mean score = 11.65, SD = 2.70; L vs. R: $t = -2.98$, $p < .004$; L vs. controls: $t = -3.53$, $p < .001$; R vs. controls: $t = -.58$, $p = ns$). Similar results were found for the Word Finding Test except that the right-hemisphere lesion group scored significantly worse than the controls.

Deficit performance on vocabulary in this thesis was determined in the same manner described above for digit span, block design and comprehension. The patients' scaled score was first standardised and, then compared with the NART VIQ. Differences of ≥ 13 points was considered 'deficit'.

As with the previously mentioned subtests of the WAIS-R, vocabulary has been validated on a large, diverse population. A particular weakness of vocabulary, unsurprisingly, is that its performance has been shown in the literature to be affected by education to a greater degree than age (Malec, Ivnik et al., 1992, as cited in Lezak, 1995). Also, as with comprehension, scoring can vary among examiners with regard to responses which are not typical; thus leading to questionable reliability.

Another measure of verbal ability is the Boston Naming Test (BNT; Kaplan, Goodglass & Weintraub, 1983). This test is a popular test of vocabulary, particularly within aphasia research. The original, full-version, of the BNT contains 85 pictures are presented independently to the patient. The patient tells the examiner the name of each picture, e.g., pencil, tree, trellis. Scoring is based on: immediately correct answers, number of stimulus cues

given, number of correct answers after a cue, number of phonemic cues and, the number of correct and incorrect responses after a cue.

Psychometric properties of the BNT have been shown in the literature to be strong. In a study by Huff, Collins, Corkin & Rosen (1986), reliability of the BNT was examined and found to be high. The authors divided the BNT into two forms to determine its split-half reliability. The order of the words was determined by 'standard word-frequency norms (Carroll, Davies & Richman, 1971)'. Words were assigned to the two forms by a Latin-square procedure. The two forms were presented to three groups: healthy controls (N = 15; 4 males, 11 females; mean age = 72.3; SD = 8.3), Alzheimer's dementia (N = 24; 10 males, 14 females; mean age = 65; SD = 7.6), brain lesion patients (N = 17; 12 males, 5 females; mean age = 58.7; SD = 14.4). The equivalence of the two forms was tested using a split-plot factorial ANOVA. Though the F-values were not reported the two forms were said to be equivalent, 'The BNT Score and Correct Uncued Score served as the dependent variable in separate analyses. Within each analysis, order of administration (Form I first, Form II first) was a between-subjects factor, and test form (I or II) was a within-subjects factor. Neither order nor the forms x order interaction was significant at the .05 level in any analysis' (p.558). The inter-correlations between the two forms for all subjects were significant ($r = .97, p < .0001$). The internal consistency (Cronbach's α) was also measured for the two forms and found to be high (Form I: $r = .96$; Form II: $r = .96$).

Criterion validity was shown for the BNT as Alzheimer's patients performed below the level of the healthy control subjects and, below published norms. The mean BNT scores of forms I and II for the Alzheimer's patients

were 26.1 and 25.2 respectively. The healthy controls mean BNT scores for the two forms were: 36.7 (form I) and 36.3 (form II). In a normative study (N = 176) by Welch, Doineau, Johnson & King (1996) the suggested cut-off for persons in the same age-group as the Alzheimer's patient group (65) was 45; well above the BNT scores of 26.1 and 25.2. The BNT has also been shown in other literature to be a valid measure of verbal ability. In a study by Hawkins, Sledge, Orleans et al. (1993), the BNT significantly correlated with the vocabulary test of the Gates-MacGintie Reading Test ($r = .83, p < .001$).

The BNT is a widely-used test of verbal skill, specifically naming ability. It has been reported to be the 'single most frequently employed naming test' (Welch et al., 1996). Though it is a test of verbal ability which has been shown to be robust as a function of age it is based solely on naming. As such, it is limited more to 'lexical access and word retrieval' (Crucie et al., 2000) and thus, may be affected by age to a greater degree than other measures of verbal ability.

The vocabulary subtest of the WAIS-R was chosen in this thesis as the test used for verbal ability due to its psychometric strengths, as well as being used in past research for the same purposes, i.e., verbal ability. As with the other subtests of the WAIS-R, vocabulary was also chosen as the author was familiar with its testing/administration and scoring at the inception of this research.

Table 2.10 Summary of Verbal Measures

	reliability	validity	deficit defined	pros	cons
vocabulary	test-retest	convergent; criterion	difference between standardised score & NART VIQ \geq 13	validated on a large diverse population	scores are affected by education; questionable reliability when administered by inexperienced examiners
BNT	split-half; Cronbach's α	criterion	-	wide-use as a verbal test	may be affected by age more so than other verbal measures

Mood

Measurements of mood are numerous and varied depending upon the specificity of mood being studied. As stated in the previous chapter, stroke may affect mood in several ways. The two domains being examined in this thesis are anxiety and depression.

One popular measure of assessing anxiety and depression is the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983). This measure was developed out of constraints that the authors felt were endemic to the General Health Questionnaire (GHQ). They felt that the GHQ was lengthy and that the data it provided offered no information as to the nature of the patients' psychiatric disorder. The HADS was also developed to be self-assessed and for use in 'nonpsychiatric hospitals'. The HADS contains 14 items that on a Likert-type scale the patient rates his/her self on either anxiety or depression-based questions. The HADS yields an overall score and totals for both anxiety and depression.

In research done by Johnston, Pollard & Hennessey (2000) the psychometric properties of the HADS were assessed. Three a-priori questions were raised: 'Do HADS items load on a separate factor from items dealing with symptoms of physical disorder?', 'Do HADS items achieve satisfactory internal consistency in populations with different clinical conditions and at different stages of management?' and, 'Is there support for the separation of the anxiety and depression items of the HADS?' (p.580). In answering each question the authors used different populations. For the first question, 99 women (mean age = 55, 1st prediagnosis visit, 9% had prior diagnosis of malignancy) were used to examine the factor structure of the HADS and a quality of life measure, the Rotterdam Symptom Checklist (RSCL; De Haes, van Knippenberg & Neijt, 1990). The HADS and the RSCL were given again to two other groups of outpatient breast clinic attendees (first: N = 104; mean age = 45; post-diagnosis; second: N = 55; mean age = 53; 1st visit to a radiotherapy clinic) in order to examine the factor structures using principle component analysis, specifying a two-factor solution. For the second question, two other populations were recruited: MI patients (N = 108; 70 males, 38 females; mean age = 56; assessed 2mo., 6mo. & 1yr. post-discharge), stroke patients (N = 68; 33 males, 35 females; mean age = 70; assessed 1mo. & 6mo. post-discharge).

The purpose of the first question was to validate the notion that the HADS is a measure of mood, unconfounded by somatic complaints. Cut-off points for items loading on the 'psychological' factor were set at $>.40$. Cut-off points for items loading on the 'somatic' factor were set at $<.30$. Results showed that all items on the HADS, with exception to one on depression, met the requirement for loading on the 'psychological' factor. Three items on the HADS

showed factor loadings of $>.30$ on the 'somatic' factor. On these three items, however, the factor loadings for 'psychological' was higher in each case. Psychological items for the RSCL also loaded as expected but, a few somatic items loaded inconsistently on the 'somatic' factor. Overall, however, the HADS items were shown, as predicted, to be psychological and not somatic.

Cronbach's α levels for the HADS between groups and at different time points were all good. Eighteen of the 24 α levels were $>.80$. The stroke group showed the lowest α levels (HADS depression score; $r = .70$) at 1mo. The highest α levels were shown for the MI group (overall HADS score; $r = .94$) at 1yr.

Confirmatory factor analysis showed that there was satisfactory separation of anxiety and depression constructs. Four indices were used to assess the model's fit: non-significant fit index, non-normed fit index, normed fit index and comparative fit index. Using the Lagrange multiplier, the fit of the model was assessed after parameters (items) were freed. This being the case, item 4 was moved to 'anxiety', 7 was moved to 'depression' for the 1yr. MI sample. Items 5 & 7 were moved to 'depression' for the 1mo. stroke group. And, item 7 was moved to 'depression' and, 6 & 12 were moved to 'anxiety' for the 6mo. stroke group. Following the analyses, there was evidence that three of the four indices supported the separation of the HADS into anxiety and depression.

As a measure of mood (specifically anxiety and depression), one strength of the HADS is that it is not confounded by somatic complaints. The measure provides data relating the person's mood, distinguishable into complaints of

anxiety and/or depression. The HADS may also be either patient or observer-administered in a format easy to understand and complete.

A weakness of the HADS as a measure of mood is that it is limited in its design as being sensitive to anxiety and depression. Other aspects of mood are neglected, e.g., mania. A second weakness of the HADS is that it is a screening implement for anxiety and depression. Extreme scores can only be interpreted as being indicative of mood disorder. The clinical labels of anxiety and depression cannot be given based on high HADS scores, only that such disorders are probable.

Another common measure of mood is the Beck Depression Inventory (BDI; Beck, 1961). This measure contains 21 depression categories for which each is assigned a Likert-type score (0-3) based on how the patients relates to the question.

The BDI was developed from a top-down approach where Beck derived the questions out of psychoanalytic clinical sessions with clients. The items of the BDI, 'were chosen on the basis of their relationship to the overt behavioral manifestations of depression' (p.562). The subjects chosen for recruitment were all from 2 psychiatric hospitals. The second population was recruited for comparative purposes. The first group was recruited from a university hospital (N = 226; 40.7% male, 59.3% female; age range = 15->55; 33.6% inpatient, 66.4% outpatient). The second group was recruited from a metropolitan hospital (N = 183; 37.2% male, 62.8% female; age range = 15->55; 34.4% inpatient, 65.6% outpatient).

In assessing the reliability of the BDI, two methods were employed. First, the BDI total score was compared against the score for each of the 21

categories in 200 cases. Using a non-parametric ANOVA the author states that all categories were significantly related to the overall score ($p < .001$, except for the category 'weight-loss' $< .01$). Ninety-seven cases were also used to demonstrate split-half reliability. Pearson's correlation between odd and even categories was .86 and, after a Spearman-Brown correction rose to .93.

External-criterion-validity was measured by comparing the BDI score and clinician's ratings ($N = 4$) of depth of depression (none, mild, moderate, severe) for both patient groups. In both groups, significant correlations were shown: group1 ($r = .65$, $p < .01$), group2 ($r = .67$, $p < .01$). Though the author did not report tests of significant differences, depth of depression was compared against cut-off scores on the BDI between the two patient groups. The data showed that different cut-off scores of the BDI were consistent with the clinically judged depths of depression.

A strength of the BDI is that its items were developed in concordance with clinical manifestations of depression. The categories range from severe manifestations such as 'self-hate' and 'self-punitive wishes' to more general manifestations such as 'irritability' and 'fatigue'. Such qualitative data as might be provided by answering affirmative to 'self-hate' reduces the probability of making a clinical type II error.

As a mood measure, the BDI is limited to assessing only depression. This restricts its use in research to one domain of mood. Another weakness, unlike the HADS, is that it is subject to somatic complaints. This would affect categories such as 'sleep disturbance', 'fatigability' and certainly, 'somatic preoccupation' increasing the chance of a type I error, especially in elderly

populations. In fact, as stated in Lezak (1995) other authors have made this complaint (Kasznik & Allender, 1985).

A third test often used to assess depression is the Self-Rating Depression Scale (SDS, or Zung; Zung, 1965). Like the BDI, the SDS was developed as a top-down measurement. Items were derived from patient interview material. There were 20 selected depression statements (10 worded symptomatically positive & 10 symptomatically negative) for which the patient rates each on a four point Likert-type scale (0 = a little of the time and 4 = most of the time).

In Zung's original paper there was no mention of tests for consistency. In research formally mentioned in the previous chapter a longitudinal study examining post-stroke mood disorders was conducted by Robinson et al. (1985). In this study 103 stroke patients were administered several measures of mood, one being the SDS, over three time points: admission, 3mo. and 6mo. post-admission. Test-retest data was significant from admission to 3mo. ($r = .73$, $p < .01$) and from admission to 6mo. ($r = .61$, $p < .01$).

Zung does, however, provide data on the conformity of the SDS. The SDS was administered to 56 psychiatric patients who had a primary diagnosis of depression and 100 normal controls who did not have a history of depression. Discriminant-validity was shown by a comparison of patients who had an admitting diagnosis of depression and were treated and discharged with depressive disorder and, patients who were admitted with a diagnosis of depression but were discharged with another disorder (the split was approximately equal). The SDS scores for the first group ranged from .63-.90 (mean score = .74). The SDS scores for the second group were lower, as expected (range = .38-.71; mean score = .53). The SDS scores for the controls

were lower (range = .25-.43; mean score = .33). Both group's mean scores were significantly higher than controls at the .01 level of significance. Further discriminant-validity was shown by comparing the mean of the item scores between the patients and controls. As demonstrated by line-graphs, the means of the 20 items for the patients who were discharged with a diagnosis of depressive disorder, fell between slightly above 'some of the time' and slightly below 'most of the time'. Patients who were discharged with another disorder were lower with a range of mean item scores falling between slightly below 'some of the time' to slightly above 'a good part of the time'. Controls' mean item scores were lowest with a range falling slightly above 'some of the time' to slightly above 'a little of the time'. Criterion-validity was shown as mean item scores changed, it was suggested, as a function of treatment. The mean item scores of the patients who were discharged with a diagnosis of depressive disorder were compared before and after treatment. As demonstrated by another line-graph, before treatment mean item scores ranged from slightly above 'some of the time' to slightly below 'most of the time'. Following treatment, the patients' mean item scores ranged from slightly above 'a little of the time' to slightly above 'some of the time'.

One strength of the SDS, like with the BDI, is that its development was based on clinical interviews where the items were generated from actual depressed patients. Another strength of the SDS is the qualitative nature of the data. The SDS gives scores based on symptom groups: affect, physiological disturbances, psychomotor disturbances and, psychological disturbances.

As a measure of mood, the SDS is as limited as the BDI in only assessing depression. Another weakness, similar to the BDI, is it is subject to

somatic complaint biases, e.g., sleep disturbances, weight loss, tachycardia and diurnal variation.

The major advantage to choosing the HADS as a measure of mood in this thesis is that: 1. it does also examine anxiety, not just depression as many tests do, 2. it was developed to counter type-I errors caused by questions based on somatic complaints and 3. it has been used in several stroke studies, e.g., Johnston et al., 1999 and Johnson et al., 1995.

Table 2.11 Summary of Mood Measures

	reliability	validity	pros	cons
HADS	Cronbach's α	factorial: support for anxiety and depression as different constructs	not confounded by somatic complaints; mood may be assessed in 2 domains; either patient or observer-assessed	limited to anxiety and depression; only indicative of mood disorder
Beck Depression Inventory	split-half	external-criterion: BDI scores matched clinical judgments	top-down development based on clinical manifestations; provides qualitative data on a range of manifestations	only measures depression; subject to somatic complaints
Zung Self-Rating Depression Scale	test-retest	discriminant: higher scores for patients than controls, higher mean item scores for patients than controls; criterion: scores decreased as a supposed function of treatment	top-down development based on patient interviews; provides qualitative data on a range of manifestations	only measures depression; subject to somatic complaints

This chapter reviewed different measures often employed in the assessment of disability, perceived control, attention control, illness representation, general cognitive deficit, premorbid intelligence, visual neglect, memory, construction, executive function, verbal ability and, mood. Methods for defining cognitive deficit in this thesis were also discussed. Comparisons were made between measures used to assess each domain and conclusions were drawn as to which measure to employ in this thesis. Often a major reason for choosing the measures related to the author's familiarity with their administration and scoring which, was considered an advantage in ensuring the reliability. In such circumstances, justification could not be found for choosing other tests measuring similar constructs.

CHAPTER 3

Study 1

Summary

Disability and recovery from stroke have been predicted independently from perceived control (Johnston, Morrison et al., 1999), attention control (Robertson, Ridgeway et al., 1997) and mood (Johnston, Morison et al., 1999). Stroke disability and recovery have thus been shown to be predicted by levels of illness representations (perceived control), cognitive deficit (attention control) and mood. This study was implemented to firstly address the relationships between perceived control, attention control and recovery from stroke. Secondly, this study examined the relationships between cognitive deficit and illness representations and, cognitive deficit and mood. A cohort of stroke patients (N=56), returning for their 1-year follow-up appointment to the Ninewells Hospital Stroke Clinic, Dundee, were recruited for this study. Patients were tested in their homes. Testing time lasted approximately 1.5 hours. Measures included: Disability-Barthel; Illness Representations-RLOC, IPQ; Cognitive Deficit-MMSE, NART, vocabulary, comprehension, digit span, block design, Trail Making-B; Mood-HADS. The data showed that: perceived control did not significantly correlate with recovery from stroke at one year, attention control (elevator counting subtest) did significantly correlate with recovery from stroke at one year and, perceived control and attention control did not significantly correlate with one another. The data also showed that stroke patients with general and specific cognitive deficits scored significantly

different from stroke patients without cognitive deficits on the IPQ and HADS. These significant differences however, were few. The directionality of the differences on the IPQ and HADS was also mixed depending on the cognitive deficit measure. The conclusions of this study suggests that: a) perceptions of control and attention control act independently in predicting recovery from disability and b) patients with cognitive deficit after a stroke have significantly different illness representations and mood. However, given the differences in the results, there is no clear support for the proposal that cognitive deficits result in greater illness impact.

Introduction

The previous chapters have discussed the theoretical rationale for this thesis as well as the measures chosen. This chapter will discuss the first study of this thesis designed to answer the proposed research questions. As chapter one described, stroke recovery has been examined using psychological theories, employing specific psychological constructs. Three of such constructs being: a.) illness representations, b.) cognitive deficit and c.) mood. Outcome similarities between theoretically different constructs using similar populations at similar times, for stroke recovery, have been found in the literature. Namely, these constructs were *perceived control* and *attention control*. It is the commonality of perceived control and attention control results in the literature, which was the impetus for study one. As perceived control and attention control are defined in this thesis to be specific areas of the

broader areas of *illness representation* and *cognitive deficit*, respectively, these broader areas are also being examined in this thesis. In both illness representation literature and the cognitive deficit literature, *mood* was also found to be a significant predictor of stroke recovery. The first aim of this thesis was to examine the relationship between perceived control and attention control with regard to stroke recovery. The variance in recovery from disability explained by perceived control and attention control was examined, as well as the shared variance of perceived control and attention control. The second aim of this thesis was to a.) examine the relationships between cognitive deficit and illness representation and b.) examine the relationship between cognitive deficit and mood. Correlations between cognitive deficit and illness representations were examined. Cognitively impaired patients were also compared against patients without cognitive deficit with regard to illness representation and mood.

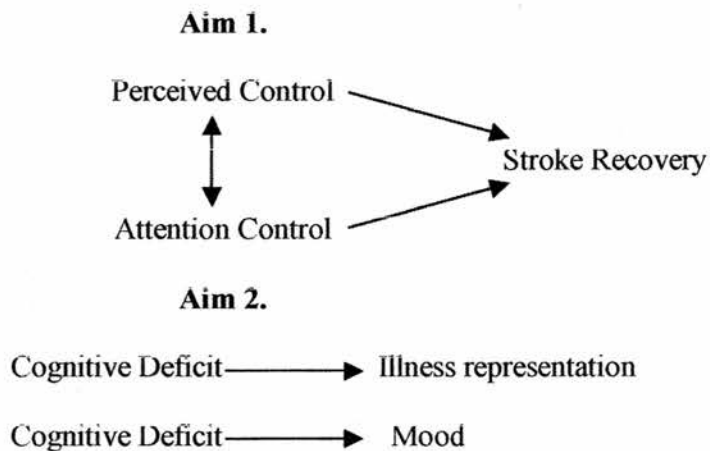


Figure 3.1 Thesis Aims

The aims of this thesis were addressed by two research questions and six hypotheses. The research questions for this thesis were:

Research Question 1: Do perceived control (PC) and attention control (AC) explain some of the same variance in stroke recovery?

Research Question 2: In what way are cognitive deficits, caused by stroke, related to illness representations and mood?

Hypothesis 1a.) Perceived control at one year significantly correlates with recovery from disability in stroke patients at one year.

Hypothesis 1b.) Attention control at one year significantly correlates with recovery from disability in stroke patients at one year.

Hypothesis 1c.) Perceived control and attention control, at one year, significantly correlate with each other.

Hypothesis 1d.) Perceived control and attention control make independent contributions to the explanation of recovery from stroke disability

Hypothesis 2a.) Stroke patients who are cognitively impaired at one year will have significantly different illness representation scores than those patients who do not have cognitive deficit.

Hypothesis 2b.) Stroke patients who are cognitively impaired at one year will have significantly higher depression and higher anxiety those patients who do not have cognitive deficit.

Method

Design

The design of this study is both cross-sectional and longitudinal. Data used in this study was gathered at the time of hospital admission for acute stroke (*initial data*) and, longitudinally, one year following the admission date (*follow-up data*). *Research Question 1* was addressed using this cross-sectional/longitudinal design. The design employed initial and one-year disability data to predict recovery from perceived control and attention control which, were both measured at one year. *Research Question 2* was addressed using a cross-sectional design, analysing psychological variables at one year.

Patients and Setting

Patients examined were recruited at Ninewells Hospital Stroke Centre, Dundee. Patients were returning for their one-year follow-up appointment. This appointment was scheduled by the clinic for the patient to return one year after the date of their stroke to assess their health status. Inclusion criteria were: occurrence of a single stroke 1 yr. previously, orientated to person, place and time, physically able to participate and consent, an initial Barthel Index Score recorded within 1 week of patients' first hospital admission. The exclusion criteria were: history of psychiatric disorders and any language limitations. A cohort of 56 patients were recruited: 36 men with a mean age of 61.39 (s.d.=9.43) and 20 women with a mean age of 60.9 (s.d.=9.20). Data from Computed Tomography (CT) scans were available for thirty-seven

patients and reported brain damage as follows: normal (14), frontal lobe (3), parietal lobe (4), occipital lobe (1), basal ganglia (3), internal capsule (4), middle cerebral artery (8). Clinical notes indicated the following: left hemisphere stroke (23), right hemisphere stroke (24), non-haemorrhagic (19), haemorrhagic (4) and, 'unspecified' (33). Appendix C1 shows demographic data for the patients.

Measures

Table 3.1 shows a summary of each of the measures used in this study. As discussed in chapter two, the measures were chosen to assess specific areas of disability/recovery, illness representations, cognitive deficit and, mood.

Table 3.1 Structure of Thesis Measures

Area of Measure	Disability/Recovery	Illness Representation	Cognitive Deficit	Mood	Physiological
Constructs	Disability	Perceived Control	Attention Control	Anxiety, Depression	Area of Stroke Impairment
Tests	Barthel Index of Disability	Recovery Locus of Control (RLOC), Illness Perception Questionnaire (IPQ)	Test of Everyday Attention: elevator counting (EC), elevator counting with distraction (ECD) MMSE NAKT Vocabulary Comprehension Block Design Digit Span Trail Making-B Star Cancellation MSQ	Hospital Anxiety and Depression Scale (HADS)	CT scan
Time of Data Acquisition	admission & one year	one year	one year (except MSQ-admission)	one year	admission

Procedure

This study was examined and approved by the Tayside Medical Ethics Committee which oversees research performed in, and through, Ninewells Hospital as well as other hospitals in the Tayside area. The patients were recruited when they attended the one-year follow-up stroke clinic in Ninewells Hospital. Attending the stroke clinic was the stroke consultant (Ron MacWalter), his nurse (Hazel Fraser), the researcher (the author), the patient and his/her carer. After each patient was seen by the consultant the researcher asked the patient if they wished to participate in this study. Upon consent, a date and time were scheduled for the researcher to visit the patients' homes and administer the measures. The patients were also given an information sheet at this time that explained the aim and purpose of the study. Letters were mailed to each patient's G.P. to inform them of the study. Administration of the measures, on average, lasted one and half-hours. All psychological measures except the initial Barthel Index and MSQ (administered at admission by either nurse or doctor) were administered, scored and recorded by the researcher. Clinical and demographic data were recorded at admission by either nurse or doctor. Clinical data included: hemisphere of stroke, location of stroke and, stroke type. Demographic data included: age and sex. Psychological variables (measures of disability, illness representation, cognitive deficit and, mood) were measured at one year (with the exception of initial disability).

Statistical Analysis

Each item for each patient was entered into SPSS 8.0 statistical software package. From the database created, all reported statistics were performed.

Research Question 1.

Do perceived control (PC) and attention control (AC) explain some of the same variance in stroke recovery?

The research questions follow the hypotheses for this study in the same order.

1a.) A regression equation was calculated, taking initial disability as the predictor variable and Barthel Index at one year as the dependent variable, in order to provide a regression residual as a measure of recovery.

1b.) Pearson correlations were performed between measures of disability and recovery on the one hand and perceived control and attention control on the other.

1c.) Pearson correlations were performed between disability and recovery and perceived control; and between disability and recovery and attention control to assess explained variance by both PC and AC.

1d.) Pearson correlations were performed between PC and AC.

Research Question 2.

What is the relationship between cognitive deficit and illness representations and mood?

2a.) Pearson correlations were performed between measures of cognitive deficit, illness representation and mood to assess the relationships between cognitive deficit and illness representation and cognitive deficit and mood

2b.) Independent t-tests were performed between patients having cognitive deficit (D) and patients without cognitive deficit (ND), on illness representations and mood.

Results

Patient descriptives

Descriptive statistics for all of the patients and measures administered are reported in Appendices C1-C2.

Internal reliability

Appendix C3 shows Cronbach's alpha coefficients for the patients' initial Barthel Index, one-year Barthel Index, RLOC, IPQ (measures lending themselves to internal reliability analysis) and, HADS. Two measures: IPQ timeline and IPQ control/cure had low internal reliability suggesting possible inadequacy as measures for those two constructs.

Research Question 1.

In order to address hypothesis 1a, recovery from disability was defined by accounting for individual differences between initial disability and final

disability through the calculation of a 'recovery' residual. Figure 3.2 shows the range of recovery for the patients with the residual showing optimum recovery. The patients who were not disabled, as defined by the Barthel Index, show no recovery and are the topmost scores on Figure 3.2. This suggests a ceiling-effect produced by the measure not being sensitive enough to disability for this population.

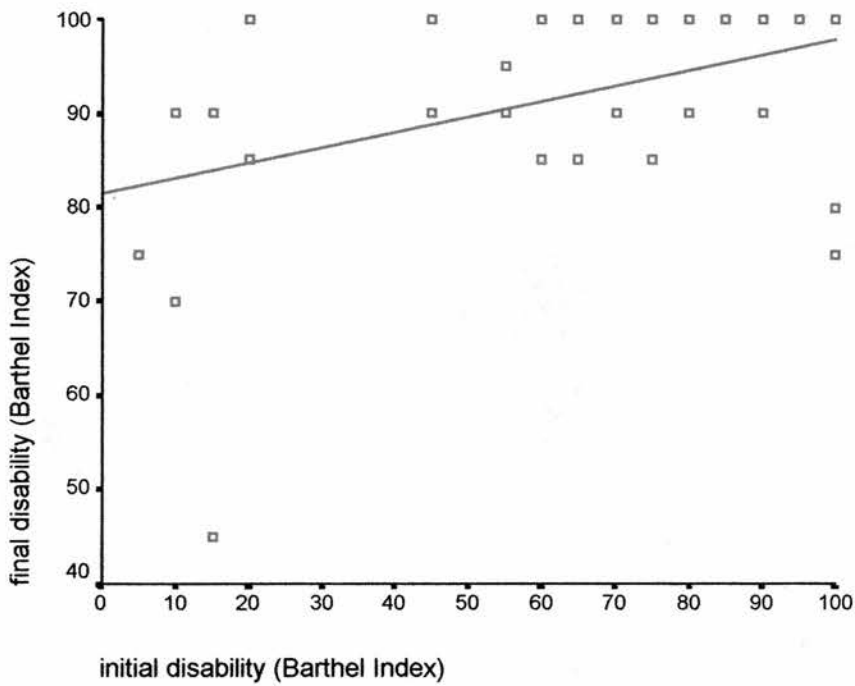


Figure 3.2 Regression residual calculated to create 'recovery'.

H1a: Perceived control at one year significantly correlates with recovery from disability in stroke patients at one year.

Hypothesis 1a. was tested using Pearson correlations. The hypothesis was not supported in that perceived control was found to be significantly correlated only with disability at one year and, not with recovery (Table 3.2).

Table 3.2 Pearson Correlations Between Perceived Control, One-year Barthel & Recovery

	RLOC	Barthel at 1 Year	Recovery
RLOC		.303*	.265

* p<.05

H1b: Attention control at one year significantly correlates with recovery from disability in stroke patients at one year.

Hypothesis 1b. was tested using Pearson correlations. Support for this hypothesis was found. Table 3.3 shows the correlations between attention control (TEA: EC), (TEA: ECD) and disability at one year (Barthel-1yr.), and recovery. Both measures of attention control were significantly correlated with Barthel at 1 year. However, only attention control (EC) was significantly correlated with stroke recovery.

Table 3.3 Pearson Correlations Between Attention Control, Initial Disability & Recovery

	TEA (EC)	TEA (ECD)	Barthel-1yr.	Recovery
TEA (EC)		.478**	-.56**	-.486**
TEA (ECD)			-.34**	-.244

*p<.05; **p<.001

H1c: Perceived control and attention control, at one year, do not significantly correlate.

Hypothesis 1c. was tested using Pearson correlations. Table 3.4 shows the correlation between perceived control & attention control. The results show that these two variables are not significantly related.

Table 3.4 Pearson Correlations Between Perceived Control & Attention Control

	TEA (EC)	TEA (ECD)
RLOC	-.192	-.136

H1d: Perceived control and attention control make independent contributions to the explanation of recovery from disability.

Table 3.4 shows that perceived control and attention control do not significantly correlate and therefore, make independent contributions of explained variance for recovery from stroke.

Research Question 2.

Mean and standard deviation scores for the cognitive deficit, illness representation and mood measures are presented in Appendix C2. Analyses using Star Cancellation were not possible as no patients were considered to have a deficit on this test (a score of ≥ 3 errors on the same side of the page).

A summary of the number of patients with cognitive deficits is presented in Figure 3.3.

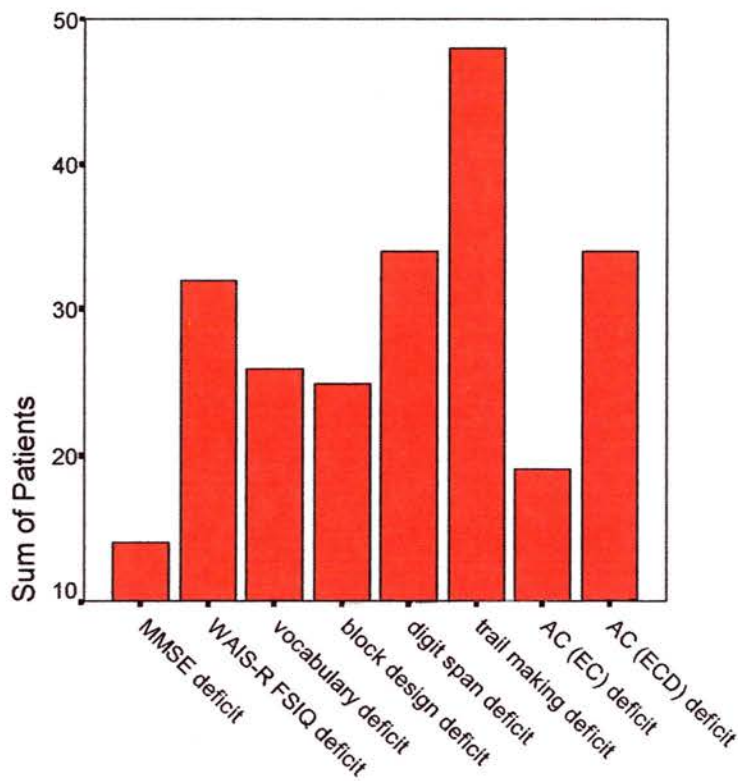


Figure 3.3 Summary graph of patients with cognitive deficits.

H2a: Stroke patients who are cognitively impaired at one year will have significantly different illness representation scores than those patients who do not have cognitive deficit.

Hypothesis 2a. was tested using an independent t-test to examine differences between patients with cognitive deficits and patients without cognitive deficits. Tables 3.5-3.9 shows the complete t-test values, with means and standard deviations, for all cognitive deficit tests between patients with cognitive deficit and those without for each illness representation. Table 3.5 shows that patients with cognitive deficit (MMSE & Trails-B) are not significantly different than patients without cognitive deficit with regard to the illness representation, perceived control.

Table 3.5 T-tests For Cognitive Deficits on RLOC

RLOC							
	N	Mean	sd	t	df	2-tailed sig.	Mean Difference
MMSE							
D	15	33.40	4.72	1.69	54	.09	2.58
ND	41	35.98	5.15				
WAIS-R							
D	32	34.97	5.47	.53	54	.60	.74
ND	24	35.71	4.70				
vocabulary							
D	27	34.26	5.43	1.46	54	.15	1.98
ND	29	36.24	4.71				
comprehension							
D	29	34.31	5.22	1.49	54	.14	2.02
ND	27	36.33	4.90				
block design							
D	25	35.32	5.09	-.05	54	.97	-6.19
ND	31	35.26	5.23				
digit span							
D	34	35.91	5.04	-1.14	54	.26	-1.60
ND	22	34.32	5.21				
Trails-B							
D	49	34.80	4.68	1.94	54	.06	3.92
ND	7	38.71	7.04				
AC (EC)							
D	19	35.47	5.25	-.07	53	.94	-.11
ND	36	35.36	5.09				
AC (ECD)							
D	35	34.97	5.01	.59	54	.56	.83
ND	21	35.81	5.26				

Table 3.6 shows that there are no significant differences between patients with cognitive deficit and those without on the illness representation, IPQ identity.

Table 3.6 T-tests For Cognitive Deficit on IPQ Identity

IPQ identity							
	N	Mean	sd	t	df	2-tailed sig.	Mean Difference
MMSE							
D	15	5.87	2.92	-1.78	54	.08	-1.62
ND	41	4.24	3.06				
WAIS-R							
D	32	4.47	3.24	.59	54	.56	.49
ND	24	4.96	2.90				
vocabulary							
D	27	4.15	2.91	1.25	54	.22	1.02
ND	29	5.17	3.20				
comprehension							
D	29	4.28	3.14	1.01	54	.32	.84
ND	27	5.11	3.02				
block design							
D	25	4.80	3.28	-.26	54	.79	-.22
ND	31	4.58	3.01				
digit span							
D	34	4.18	3.12	1.53	54	.13	1.28
ND	22	5.45	2.92				
Trails-B							
D	49	4.53	2.99	.95	54	.35	1.18
ND	7	5.71	3.73				
AC (EC)							
D	19	4.47	2.20	.37	53	.71	.33
ND	36	4.81	3.52				
AC (ECD)							
D	35	5.09	2.98	-1.28	54	.21	-1.09
ND	21	4.00	3.19				

Table 3.7 shows that there are no significant differences between patients with cognitive deficit and those without on the illness representation, IPQ timeline.

Table 3.7 T-tests For Cognitive Deficit on IPQ Timeline

		IPQ timeline					
	N	Mean	sd	t	df	2-tailed sig.	Mean Difference
MMSE							
D	15	2.98	.97	-.48	54	.63	-.12
ND	41	2.85	.81				
WAIS-R							
D	32	2.73	.76	1.51	54	.14	.34
ND	24	3.08	.94				
vocabulary							
D	27	2.70	.82	1.69	54	.09	.38
ND	29	3.07	.86				
comprehension							
D	29	2.75	.83	1.28	54	.21	.29
ND	27	3.04	.86				
block design							
D				-.47	54	.64	-.11
ND	25	2.94	.83				
	31	2.84	.88				
digit span							
D	34	2.77	.81	1.23	54	.22	.29
ND	22	3.06	.90				
Trails-B							
D	49	2.86	.79	.69	54	.49	.24
ND	7	3.10	1.24				
AC (EC)							
D	19	2.93	.64	-.28	53	.78	.07
ND	36	2.86	.96				
AC (ECD)							
D	35	2.90	.81	-.20	54	.84	.04
ND	21	2.86	.93				

Table 3.8 shows that there is a significant difference between patients with cognitive deficit (WAIS- R FSIQ & block design) and those without on the illness representation, IPQ consequences. Patients with general cognitive deficit (existing IQ minus premorbid IQ), and cognitive deficit as measured by block design score lower on the IPQ consequences subscale suggesting a weaker illness representation for this domain.

Table 3.8 T-tests For Cognitive Deficit on IPQ Consequences

	IPQ consequences						
	N	Mean	sd	t	df	2-tailed sig.	Mean Difference
MMSE							
D	15	3.29	.78	-1.33	54	.19	-.28
ND	41	3.00	.67				
WAIS-R							
D	32	2.90	.65	2.30	54	.03*	.42
ND	24	3.32	.70				
vocabulary							
D	27	3.04	.74	.40	54	.69	.08
ND	29	3.12	.67				
comprehension							
D	29	3.04	.66	.41	54	.68	.08
ND	27	3.12	.75				
block design							
D	25	2.89	.61	2.00	54	.05*	.36
ND	31	3.24	.73				
digit span							
D	34	2.97	.65	1.43	54	.16	.27
ND	22	3.25	.75				
Trails-B							
D	49	3.06	.69	.57	54	.57	.16
ND	7	3.22	.79				
AC (EC)							
D	19	3.00	.31	.50	53	.62	.10
ND	36	3.10	.49				
AC (ECD)							
D	35	3.18	.70	-1.42	54	.16	-.27
ND	21	2.91	.68				

Table 3.9 shows that there is a significant difference between patients with cognitive deficit (Trails- B) and those without on the illness representation, IPQ control/cure. Cognitive deficit patients scored lower on the subscale IPQ control/cure suggesting a weaker illness representation for this domain as compared with patients without cognitive deficit.

Table 3.9 T-tests For Cognitive Deficit on IPQ Control/Cure

IPQ control/cure							
	N	Mean	sd	t	df	2-tailed sig.	Mean Difference
MMSE							
D	15	3.44	.34	.83	54	.41	.10
ND	41	3.55	.46				
WAIS-R							
D	32	3.52	.45	.06	54	.95	.01
ND	24	3.53	.42				
vocabulary							
D	27	3.46	.45	1.12	54	.27	.13
ND	29	3.59	.41				
comprehension							
D	29	3.44	.46	1.58	54	.12	.18
ND	27	3.62	.40				
block design							
D				-.25	54	.81	.03
ND	25	3.54	.44				
	31	3.51	.44				
digit span							
D	34	3.48	.43	.93	54	.36	.11
ND	22	3.59	.45				
Trails-B							
D	49	3.48	.42	2.08	54	.04*	.35
ND	7	3.83	.41				
AC (EC)							
D	19	3.46	.31	.92	53	.36	.11
ND	36	3.57	.49				
AC (ECD)							
D	35	3.47	.45	1.17	54	.25	.14
ND	21	3.61	.41				

H2b: Stroke patients who are cognitively impaired at one year will have significantly higher anxiety and higher depression those patients who do not have cognitive deficit.

Hypothesis 2b. was tested using independent t-tests to determine differences between patients with cognitive deficit and patients without cognitive deficit on mood. Table 3.10 shows that there are significant differences between patients with cognitive deficits (MMSE & digit span) and those without on mood (HADS anxiety). Cognitive deficit patients, as

measured by the MMSE scored higher on anxiety as compared to patients without cognitive deficit. However, the pattern is opposite, i.e., anxiety is lower if using digit span to categorise patients with cognitive deficit.

Table 3.10 T-tests For Cognitive Deficit on HADS Anxiety

HADS anxiety							
	N	Mean	sd	t	df	2-tailed sig.	Mean Difference
MMSE							
D	15	11.27	5.11	-2.07	54	.04*	-3.34
ND	41	7.93	5.44				
WAIS-R				1.09	54	.28	1.63
D	32	8.13	5.23				
ND	24	9.75	5.86				
vocabulary				.99	54	.33	1.44
D	27	8.07	5.72				
ND	29	9.52	5.32				
comprehension				1.16	54	.25	1.70
D	29	8.00	5.59				
ND	27	9.70	5.40				
block design				-1.10	54	.28	-1.62
D	25	9.72	5.75				
ND	31	8.10	5.30				
digit span				2.32	54	.02*	3.36
D	34	7.50	4.41				
ND	22	10.86	6.47				
Trails-B				.90	54	.37	2.00
D	49	8.57	5.08				
ND	7	10.57	8.24				
AC (EC)				.25	53	.81	.38
D	19	8.42	4.78				
ND	36	8.81	5.84				
AC (ECD)				-1.90	54	.06	-2.83
D	35	9.89	5.43				
ND	21	7.05	5.31				

Table 3.11 shows that there is a significant difference between patients with cognitive deficit (MMSE) and those without on mood (HADS depression). Patients with cognitive deficit, as measured by the MMSE, score higher on depression than patients without cognitive deficit.

Table 3.11 T-tests For Cognitive Deficit on HADS Depression

HADS depression							
	N	Mean	sd	t	df	2-tailed sig.	Mean Difference
MMSE							
D	15	9.07	4.89	-2.67	54	.01*	-3.58
ND	41	5.49	4.27				
WAIS-R							
D	32	6.16	4.43	.53	54	.60	.68
ND	24	6.83	5.07				
vocabulary							
D	27	6.30	4.45	.23	54	.82	.29
ND	29	6.59	4.96				
comprehension							
D	29	5.83	4.36	1.03	54	.31	1.28
ND	27	7.11	5.00				
block design							
D				.24	54	.81	.30
ND	25	6.29	4.34				
D	31	6.58	5.01				
digit span							
D	34	5.68	4.18	1.55	54	.13	2.00
ND	22	7.67	5.24				
Trails-B							
D	49	6.29	4.58	.68	54	.50	1.29
ND	7	7.57	5.62				
AC (EC)							
D	19	5.90	3.67	.50	53	.62	.66
ND	36	6.56	5.11				
AC (ECD)							
D	35	7.23	4.81	-1.64	54	.11	-2.09
ND	21	5.14	4.25				

Discussion

The reliability analysis performed on the measures where such analysis was possible suggested that in general, the measures were sufficiently reliable (Appendix C3). The two exceptions being IPQ timeline and IPQ control/cure. These two subscales were also the two lowest internal consistency correlations of Weinman et al.'s original IPQ study (Cronbach's $\alpha=.73$ for both subscales). The alpha levels in this study were low (Cronbach's α for timeline = .39 and .29 for control/cure) and not acceptable. A possible explanation for the low internal consistency on timeline may be that the patients may be conceptualising the statements in an unintended manner. While the statements, 'My stroke is likely to be permanent rather than temporary' and, 'My stroke will last for a long time' seem similar and therefore should highly correlate, the connotations may be different to stroke patients. They may feel that the words, 'permanent' and 'long time' have different meanings. That is to say that while they may have largely recovered from their stroke within a year (not a 'long time'), they may not feel that they will ever recover to their full pre-stroke functioning (that it is 'permanent'). Having only three items for that subscale, a different direction of answer than expected may produce a low internal correlation for that subscale. Similarly, the low internal consistency for control/cure may be explained by the difference between a cure for stroke and control for stroke. Stroke patients may feel that while it may be possible to control the symptoms of a stroke, a cure for stroke may not be possible. Having both possibilities on one scale would produce a low correlation.

In addressing the first research question (Do perceived control and attention control explain some of the same variance in stroke recovery?),

hypothesis 1a was not supported. There was not a significant correlation between perceived control and recovery. Since other studies have also shown that perceived control predicts recovery (Johnston et al., 1999; Partridge & Johnston, 1989) the result in this study that it does not is questionable. In the second study, this relationship will again be investigated.

The hypothesis (1b) that attention control would significantly correlate with recovery from disability was supported by EC (Table 3.3). The fact that attention control (ECD) did not significantly correlate with recovery may be due to the fact that attention control (EC) is considered in the literature (Robertson et al., 1994) to be a purer measure of attention control. The negative correlation directions were as expected, i.e., fewer EC and ECD errors = higher Barthel scores. These results supported the attention control literature where attention control predicted disability (Ben-Yishay et al., 1968; Robertson, Ridgeway et al., 1997).

The hypothesis (1c) that perceived control and attention control significantly correlate was not supported (Table 3.4). Though the author did not have strong evidence for hypothesising that PC and AC would correlate, this direction was chosen over the null due to the commonalities of other findings previously mentioned. As PC and AC do not significantly correlate, yet they each significantly correlate with recovery, it is suggested that these two variables act independently in predicting recovery from disability at one year.

In addressing the second research question, t-tests were performed to assess differences between patients with cognitive deficit and patients without cognitive deficits on illness representations and mood. Support was found for hypothesis 2a. That, stroke patients who are cognitively deficient at one year will have significantly different illness representation scores than those patients who do not have cognitive deficit. Table 3.8 shows that patients with general cognitive deficit (WAIS-R) and, specific cognitive deficit based on construction skills (block design) score significantly lower on IPQ consequences. Patients who have general cognitive deficits or, cognitive deficits specific to construction skills may perceive the consequences of their strokes to be significantly less severe, i.e., less serious than patients without cognitive deficit in this area. Table 3.9 shows that patients with cognitive deficit based on executive skills (Trails-B) score significantly lower on IPQ control/cure. This result must be interpreted in light of the fact that deficit on Trails-B was defined using a non-age corrected cut-off score. As the population in this study was older, other non-executive function issues such as motor-speed may have increased the number of 'deficit' patients. This in turn may have increased the chance of a type I error. However, it may be possible that patients whose cognitive deficit is primarily executive functioning have significantly more pessimistic beliefs regarding the control and/or cure of symptoms for their strokes compared to patients without cognitive deficit.

Support was also found for hypothesis 2b. That, stroke patients who are cognitively deficient at one year will have significantly different mood

scores than those patients who do not have cognitive deficit. Table 3.10 shows that patients with general cognitive deficit (MMSE), and memory-based cognitive deficit (digit span) have significant differences in mood (HADS anxiety). Patients who have general cognitive deficit are significantly more anxious than patients without general cognitive deficit. Patients who have memory-based cognitive deficit (digit span) show significantly less anxiety compared with patients who do not have memory-based cognitive deficit. As mentioned in the previous chapter, the use of digit span as a test of memory is limited in that it is a measure of 'short-term retention capacity' (Lezak, 1995). As memory is not a singular skill, but is comprised of many sub-areas, results regarding digit span can only be relevant to short-term memory processes. Table 3.11 again shows that patients with general cognitive deficit (MMSE) score significantly different than patients without cognitive deficit on mood (HADS depression). Patients with general cognitive deficit score significantly higher on a depression screening measure and, therefore, are possibly more depressed than patients without general cognitive deficit.

The findings of this study suggest that specific cognitive deficits due to stroke, studied at one year, may have links to specific illness representations and mood. In an applied sense, one may look to these relationships to give specific insight into what the patient may be perceiving of their stroke and their rehabilitation. This study does, however, have limitations which were addressed in a second study.

The first limitation of this study was that the number of participants was lower than expected. The intended number of patients was 64 (effect size=.63, α =.05, power=.80). With 56 patients the power of this study was .75. Having had a higher number of patients in this study, the results may have been stronger with regard to what could be concluded from the results. In the second study, the number of patients will be higher since recruitment follows a separate study which the author will use data from.

The second limitation of this study concerns 'recovery'. In this study disability was recorded initially and at one year with a regression residual calculated based on these two times. In the following study, 'recovery' can be plotted for each patient based on three times: initially, 6 months and, at one year. This longitudinal design may allow the researcher to examine a truer index of 'recovery'. Examining disability at three times will also allow the researcher to have a clearer picture of when the greatest amount of recovery happens for most patients. In this study, the Barthel seemed to have a ceiling – effect at one year. Monitoring disability at three times may allow a less sensitive measure such as the Barthel to show when the most recovery takes place.

The third limitation of this study concerns the number of t-tests performed. This study performed sixty-three t-tests. At the .05 level of significance, approximately three t-tests would be significant due to chance. Only eleven t-tests out of the sixty-three were significant however.

Conclusions

Research Question 1.

- 1.) Though perceived control did not significantly predict stroke recovery at one year in this study, enough evidence in other studies suggests that this is an aberrant finding and needs to be further investigated in the following study.
- 2.) Attention control predicts stroke recovery at one year. As a measure of attention control, elevator counting appeared to be a better predictor of stroke recovery at one year.
- 3.) Perceived control and attention control do not have a significant relationship with one another.
- 4.) Perceived control and attention control do not share significant amounts of variance in predicting stroke recovery at one year.

Research Question 2.

- 1.) There were significant differences between stroke patients who are cognitively deficient at one year and those who are not with regard to illness representations and mood in this study. The limitations of this study, however, restrict the conclusions that may be made from the results.
- 2.) A follow-up longitudinal study (addressing the limitations in this study) should be performed which investigates the same research questions.

CHAPTER 4

Study 2

Summary

The study discussed in this chapter was carried out to further investigate the research questions previously addressed in chapter 3. The methods of study 2 were similar to study 1. A sample of 65 stroke patients were recruited one year post-stroke from another stroke study in order to obtain longitudinal data (initial:6mo:1yr). Patients were tested in their homes. Test administration lasted approximately 1.5-2 hours. Measures included: Disability–Barthel; Illness Representations–RLOC, IPQ; Cognitive Deficit–MMSE, NART, vocabulary, comprehension, digit span, block design, Trail Making-B; Mood–HADS. Regarding research question one, the data showed that significant results were found for: One-year attention control correlating with stroke recovery, perceived control and attention control not correlating and, perceived control and attention control making independent contributions to the explanation of recovery from disability. Regarding research question two, significant results were found at 1yr. between patients with cognitive deficit and those without on Trails-B with regard to IPQ timeline and, elevator counting with regard to IPQ control/cure. Finally, there was a significant quadratic relationship, in the predicted direction, suggesting that better mood occurred when cognitive status was high and low; rather than occurring when cognitive status is only high as other authors have suggested. Overall, the results did not replicate the findings from study 1 and directional differences within the results caused the findings to be mixed. Results are discussed in light of possible sample and measure limitations.

Introduction

Study 2 was designed using similar methodologies to study 1 in order to further investigate the same research questions in greater detail as well as look for replication in the results. The primary advantage in this study, as compared to study 1, was that longitudinal data analysis was possible as data was provided for at three time points: admission to hospital (initial), 6 months post-discharge (6mo.) and, 1 year post-stroke (1yr.). A larger group of patients were also recruited into this study as compared with the previous study. As mentioned in the previous chapter, the same constructs were being examined, i.e., disability/recovery, illness representations, cognitive deficit and mood/quality of life. The first aim of this study was the same as the previous study: to examine the relationship between perceived control (PC) and attention control (AC) with regard to stroke recovery. The variance in recovery from disability explained by perceived control and attention control was examined, as well as the shared variance of perceived control and attention control. In this study, the amount of predicted recovery variance was also examined by perceived control and attention control. The second aim of this study was also the same as the previous study: a.) examine the relationships between cognitive deficit and illness representation and b.) examine the relationship between cognitive deficit and mood. Cognitively impaired patients were also compared with patients without cognitive deficit with regard to illness representation and mood. An additional question was later raised in

this study based on the results of the author and those of Kenealy, Beaumont, Lintern & Murrell (2000). Kenealy et al. studied memory (Autobiographical Memory Interview; Kopelman, Wilson & Baddeley, 1990), mood (HADS) and, quality of life (SF-36) on a group of multiple sclerosis patients (N = 30; mean age = 52.4, range = 31-66; mean length of time since diagnosis = 21.4, range = 3-39). The first set of results was based on an independent 2-factor design with 3x2 levels (dependent variable: QoL; independent variables: severity of disability & duration of illness). Results were also based on depression and autobiographical memory as a further two independent variables. Significant results showed that duration of illness and depression were related. Patients with longer duration since time of diagnosis (>22 yrs.) showed better mood (mean HADS depression = 3.69, sd = 2.50) than patients diagnosed more recently (mean HADS depression = 6.00, sd = 4.54; $F(1,24) = 5.50, p < .03$). There was not a significant effect between severity of depression and duration of illness. There was also no significant interaction between extent of disability and duration of illness. Results of a two-factor ANOVA using autobiographical memory and duration of illness as independent variables on QoL showed a significant main effect for autobiographical memory. Patients with impaired memory reported significantly *better* QoL than patients who were not impaired (SF-36: Role Physical, mean = 72.22 and 39.58 respectively; $F(1,26) = 7.32, p < .01$). The main effect of memory and QoL also interacted with duration of illness. Patients who had impaired memory (and had been diagnosed >23 years) reported significantly *better* QoL than patients with more recent diagnoses ($F(1,26) = 5.18, p < .03$). The authors go on

to state that while in the analysis there was no significant interaction between autobiographic memory and depression, poorest QoL was reported by depressed patients with unimpaired memory. The issue most relevant to this thesis was summarised by Kenealy et al. in that, 'Patients with normal autobiographical memory reported the highest levels of depression (HADS) and the lowest levels of QoL (Role Physical)' (p.125). This was contrary to the findings found in the previous study of this thesis which suggested that higher cognitive deficit (MMSE & digit span) accompanied lower mood. This is also contrary to past findings in the literature which have found a relationship between lower cognitive deficit and higher mood (Robinson et al., 1985). It was, therefore, hypothesised that both processes may occur and that there may be a curvilinear relationship between mood and cognitive status, i.e., higher mood may occur when cognitive status is both high and low.

Hypotheses generated for each research question are as follows:

Research Question 1: Do perceived control (PC) and attention control (AC) explain some of the same variance in stroke recovery?

Hypothesis 1.a: Recovery from stroke at 1yr. will significantly correlate with: initial PC, 1yr. PC, 6mo. AC and, 1yr. AC.

Hypothesis 1.b: PC and AC are not significantly correlated initially nor, at 1yr.

Hypothesis 1.c: PC and AC make significantly independent contributions in explaining recovery from stroke at 1yr.

Research Question 2: In what way are cognitive deficits, caused by stroke, related to illness representations and mood?

Hypothesis 2.a: Stroke patients with initial cognitive deficit will have significantly different illness representations than those without cognitive deficit initially, at 6mo. and at 1yr.

Hypothesis 2.b: Stroke patients with cognitive deficit at 6mo. will have significantly different illness representations than those without cognitive deficit at 6mo. and at 1yr.

Hypothesis 2.c: Stroke patients with cognitive deficit at 1yr. will have significantly different illness representations than those without cognitive deficit at 1yr.

Hypothesis 2.d: Stroke patients with initial cognitive deficit will have significantly different mood than those without cognitive deficit initially, at 6mo. and at 1yr.

Hypothesis 2.e: Stroke patients with cognitive deficit at 6mo. will have significantly different mood than those without at 6mo. and at 1yr.

Hypothesis 2.f: Stroke patients with cognitive deficit at 1yr. will have significantly different mood than those without cognitive deficit at 1yr.

Hypothesis 2.g: Better mood is associated with both high cognitive status and low cognitive status.

Method

Design

The design of this study is both cross-sectional and longitudinal. Data was collected at three times for the same patients: initial (at admission to hospital), 6mo. and 1yr. Analyses were done at each of the three times (cross-sectional) and, across the three times (longitudinal).

Patients and Setting

The patients used in this study were recruited from the stroke registry at Ninewells Hospital, Dundee, over a one year period of time. The patients recruited to this study had begun participating in an earlier RCT study (Stroke Workbook Outcome Trial; SWOT) conducted by other researchers from the same institution as the author and of which the author was also involved¹. Patients in the SWOT study were tested at 3 time-points: 2 weeks post-discharge, 6 weeks after and, 6 months post-time-1 interview. As the patients approached their dates of being 1yr. post-stroke, letters (for this study) were sent to the patients asking for their participation in the study at 1yr. From the dates of May 1998 to May 2000, a total of 203 patients had been recruited to the SWOT study (A full demographic summary may be found in the report to the Scottish Executive¹). Recruitment for this study stopped in November 2000. Of the 203 patients in the study, 65 patients had been recruited to this study who had completed testing at admission and 6mo. (SWOT study) and,

¹ Johnston, M., Morrison, V., MacWalter, R & Pollard, B. (in progress). A Randomised Control Trial of a Workbook-based Intervention for Stroke Patients: Effects on disability and distress in patients and careers. Scottish Office: reference # K/CR1/1/7.

wished to participate at the final testing period for this study (33 males, 32 females; mean age = 69.2; SD = 10.89).

Inclusion criteria were: orientated to person, place and time; physically able to participate and consent; an initial Barthel Index Score recorded within 1 week of patients' first hospital admission. Patients who were recruited at 1yr. had been tested at admission and 6mo. The exclusion criteria were: history of psychiatric disorders and any language limitations.

Measures

Table 4.1 shows a summary of each of the measures. Measures used in this study were the same as those used in study 1. Recovery was again calculated by creating a regression residual, using initial disability (Barthel Index score) as the independent variable and 1 year disability (Barthel Index score) as the dependent variable.

Table 4.1 Structure of Thesis Measures

Area of Measure	Disability/Recovery	Illness Representation	Cognitive Deficit	Mood	Physiological
Constructs	Disability	Perceived Control, Illness Representations	Attention Control, General Cognitive Ability, Prémotid Intelligence, Verbal Ability, Executive Functioning, Constructional Ability, Memory	Anxiety, Depression	Area of Stroke, Impairment
Tests	Barthel Index of Disability	Recovery Locus of Control (RLCC), Illness Perception Questionnaire (IPQ)	Test of Everyday Attention: elevator counting (ECA), Elevator Counting with Distraction (ECD) MSQ MMSE NART Vocabulary Comprehension Block Design Digit Span Trail Making-B	Hospital Anxiety and Depression Scale (HADS)	CT scan
Time of Data Acquisition	• Barthel Index - initial, 6 mo., 1 yr.	• RLCC - initial, 1 yr. • IPQ - 6mo., 1yr.	• EC - 6 mo., 1 yr. • ECD - 1 yr. • MSQ - initial • MMSE - 6mo., 1yr. * all other cognitive measures given only at 1 yr.	• HADS - initial, 6 mo., 1 yr.	• CT - admission

Procedure

This study was examined and approved by the Tayside Medical Ethics Committee which oversees research performed in, and through, Ninewells Hospital as well as other hospitals in the Tayside area. These initial recruitments were made by other research staff from the same institution as the author (SWOT study). Patients were recruited from Ninewells Hospital, Dundee. Within two weeks of admission to hospital, patients were visited and asked to participate in this study. Prior to discharge demographic and physiological data were recorded for the patients: hemisphere of stroke, location of stroke and, stroke type (recorded by research staff from the patients' information sheet). Also recorded initially was the patients' MSQ score (administered by the admitting nurse or doctor). At the first interview (2 weeks post-discharge) patients were screened for cognitive and communication disorders using the Clifton Assessment Procedures for the Elderly (CAPE; Pattie & Gillard, 1979). Also at this time-point the Barthel Index (administered by the author and other research staff), the RLOC (administered by the author and other research staff) and, the HADS (administered by the author and other research staff) were administered. At this time a date was scheduled for further testing in the SWOT study at 6 weeks. As 6 weeks was not a time-point of concern in this thesis, this data was not used in this study. At the 6 week interview of the SWOT study, 6 month testing was scheduled. At the 6mo. period, patients were visited in their homes for further testing. The measures administered, of concern to this study at the 6 month time-point were: the Barthel Index (administered by the author and other research staff), MMSE (administered by the author and other research staff), the elevator

counting subtest of the TEA (administered by the author and other research staff) and, the HADS (administered by the author and other research staff). Approximately 1-2 months before the period of 1yr. post-discharge, the patients who had been tested both, initially and at 6months, were sent a letter in the mail (by the author) asking for their participation in a final 1yr. assessment. Patients who wished to participate replied via the post. Upon receiving their replies, the author scheduled a date over the telephone for testing. Patients seen at 1yr. were visited in their homes and administered the 1yr. measures. These measures included: the Barthel Index, the RLOC, elevator counting, elevator counting with distraction, MMSE, the NART, the vocabulary, comprehension, block design and digit span subtests of the WAIS-R, Trail Making-B and, the HADS. All 1yr. measures were administered by the author.

Statistical Analysis

Each item for each patient was entered into SPSS 8.0 statistical software package. From the database created, all reported statistics were performed.

Research Question 1.

Do perceived control (PC) and attention control (AC) explain some of the same variance in stroke recovery?

H1.a.-H1.c.

- 1.) A regression equation was calculated, taking initial disability as the predictor variable and disability at one year as the dependent variable, in order to provide a regression residual as a measure of recovery.
- 2.) Pearson correlations were performed between PC, AC and recovery.

- 3.) Pearson correlations were performed between PC and AC.
- 4.) A linear regression equation was calculated to assess the amount of variance explained by both initial PC and 1yr. AC on recovery from stroke.

Research Question 2.

What is the relationship between cognitive deficit and illness representations; and, cognitive deficit and mood?

H2.a-H2.g

- 1.) Independent t-tests were performed between patients having cognitive deficit (D) and patients without cognitive deficit (ND), on illness representations and mood.
- 2.) Quadratic regressions were performed between cognitive measures which used concurrent data and, mood (both at 6mo. and 1 yr.).

Results

Patient descriptives

Descriptive statistics for all of the patients and measures administered are reported in Appendices D1-D3.

Table Colour Format

To facilitate reading the data for different time-points in this study, information for the three different times are as follows: initial time-point data (blue), 6mo. time-point data (green) and 1yr. time-point data (yellow).

Double-Entry

Double-entry of 1yr. data on approximately 32% of the patients (N = 21) in this study was performed as a reliability check. Double-entry data was recorded by an independent research assistant. Databases were merged and the differences were taken between the two samples. There was one instance where scores were different on a single cases (1 recording for a 1yr. MMSE score). This case did not, however, cause the difference between the two samples to be significantly different with regard to the measure (A one-sample t-test was performed between the MMSE as recorded in this study's data base and as recorded by the research assistant; Appendix D4). Taking all double-entry measures into account, this difference accounted for .21% of a difference between the samples. With regard to the MMSE, it accounted for a 1% difference. There was a 0% difference for all other measures. Reliability of the data entry in this study was shown as there were no significant percentage differences between this study's data base and the double-entry data base.

Internal reliability

Appendix D3 summarises the internal reliabilities (Cronbach's α) on the appropriate measures.

Research Question 1.

In order to address hypotheses 1.a-1.c recovery from disability was defined by the amount of recovery achieved, taking into account individual differences between initial disability and, final disability through the calculation of a 'recovery' residual. Figure 4.1 shows the range of recovery for the patients

(numbered on graph), with the residual showing recovery, taking into account their initial levels of disability. Scores below the residual line represent patients who have worse disability at 1yr. than would be predicted, taking all patients into consideration. Scores above the residual line represent patients who have lower levels of disability at one year than would be predicted.

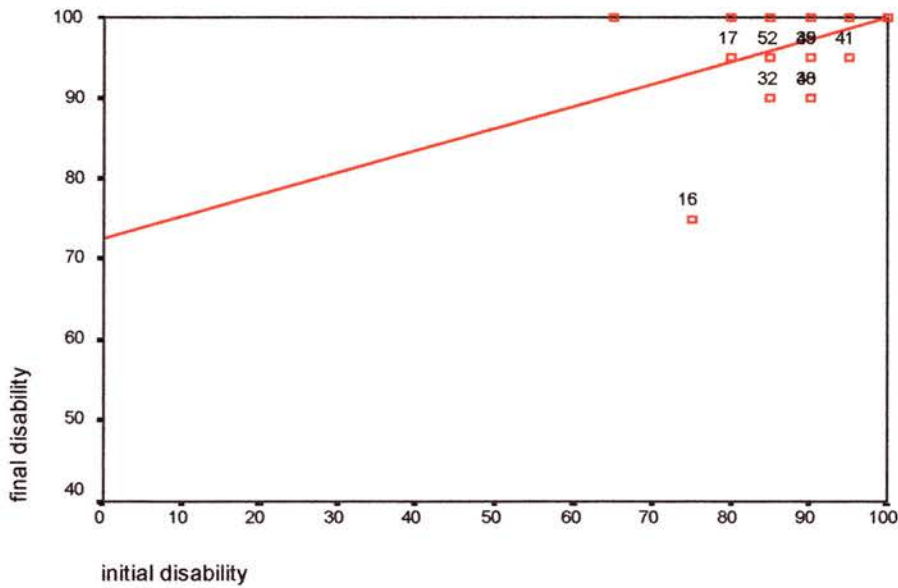


Figure 4.1 Recovery residual calculated from initial disability and 1 yr. disability

Hypothesis 1.a: Recovery from stroke at 1yr. will significantly correlate with: initial PC, 1yr. PC, 6mo. AC and, 1yr. AC.

Hypothesis 1.a. was tested using Pearson correlations. Support for this hypothesis was partially found. Table 4.2 shows the correlation between one year AC was significantly correlated with recovery using the elevator counting subtest but, not with the elevator counting with distraction subtest.

Table 4.2 Pearson Correlations Between initial PC, 1yr. PC, 6mo. AC, 1yr. AC & Recovery

	Recovery
initial RLOC	.24 (p=.06)
1yr. RLOC	.15 (p=.26)
6mo. elevator counting	-.03 (p=.86)
1yr. elevator counting	-.41 (**)
1yr. elevator counting with distraction	.07 (p=.59)
** Correlation is significant at the 0.01 level (2-tailed).	

Hypothesis 1.b: PC and AC are not significantly correlated initially nor, at 1yr.

Hypothesis 1.b was tested using Pearson correlations. Table 4.3 shows the correlations between initial perceived control (initial RLOC) and 1 year perceived control (1yr. RLOC) and, 1 year attention control (1 year elevator counting and 1 year elevator counting with distraction). There were no significant correlations between perceived control and attention control. As expected, there were significant correlations between initial PC and 1yr. PC and, the two AC subtests.

Table 4.3 Pearson Correlations Between Initial and 1yr. Perceived Control and, 1yr. Attention Control

	initial RLOC	1yr. RLOC	1yr. elevator counting	1yr. elevator counting with distraction
initial RLOC	1	.76 (**)	-0.23 (p = .07†)	-.19 (p = .14)
1yr. RLOC		1	-0.22(p = .08†)	-0.12 (p = .36)
1yr. elevator counting			1	.46 (**)
1yr. elevator counting with distraction				1
** Correlation is significant at the 0.01 level (2-tailed).				

Research Question 2: In what way are cognitive deficits, caused by stroke, related to illness representations and mood?

Mean and standard deviation scores for the cognitive deficit, illness representation and mood measures are presented in Appendix D2. A summary of the number of patients with cognitive deficits is presented in Figure 4.2.

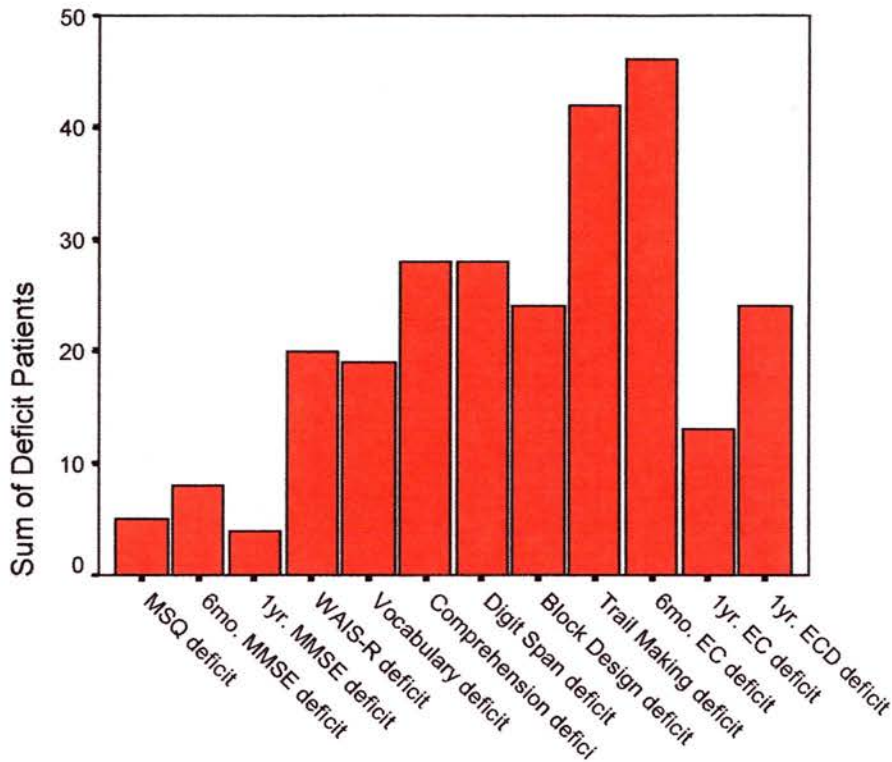


Figure 4.2 Summary graph of patients with cognitive deficits.

Hypothesis 2.a: Stroke patients with initial cognitive deficit will have significantly different illness representations than those without cognitive deficit initially, at 6mo. and at 1yr.

Hypothesis 2.a was tested using an independent t-test to examine differences between patients with cognitive deficits and patients without cognitive deficits, initially, on initial illness representations. The results were not

significant and, therefore, do not support the hypothesis. Table 4.5-4.14 shows the mean differences for initial cognitive deficit patients vs. patients without cognitive deficit on illness representations at the three times.

Table 4.5 T-tests For Initial Cognitive Deficits on Initial Perceived Control

initial RLOC									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ									
D	8	37.38	5.68	2.01					
ND	52	36.23	4.28	.59	-.67	58	.50	-1.14	1.70

Table 4.6 T-tests For Initial Cognitive Deficits on 6mo. IPQ Identity

6mo. IPQ identity									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ									
D	9	2.44	2.65	.88					
ND	51	2.90	2.43	.34	.52	58	.61	.46	.89

Table 4.7 T-tests For Initial Cognitive Deficits on 6mo. IPQ Timeline

6mo. IPQ timeline									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ									
D	9	2.81	1.11	.34					
ND	51	2.88	.94	.13	.18	58	.86	.06	.35

Table 4.8 T-tests For Initial Cognitive Deficits on 6mo. IPQ Consequences

6mo. IPQ consequences									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ					1.06	58	.29	.21	.20
D	9	2.89	.86	.29					
ND	51	3.10	.48	.06					

Table 4.9 T-tests For Initial Cognitive Deficits on 6mo. IPQ Control/Cure

6mo. IPQ control/cure									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ					.64	58	.53	.09	.15
D	9	3.28	.61	.20					
ND	51	3.37	.37	.05					

Table 4.10 T-tests For Initial Cognitive Deficits on 1yr. Perceived Control

1yr. RLOC									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ					-.32	59	.75	-.45	1.39
D	9	39.88	3.89	1.30					
ND	52	39.44	3.86	.53					

Table 4.11 T-tests For Initial Cognitive Deficits on 1yr. IPQ identity

1yr. IPQ identity									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ					.27	59	.78	.19	.71
D	9	2.00	2.55	.85					
ND	52	2.19	1.86	.26					

Table 4.12 T-tests For Initial Cognitive Deficits on 1yr. IPQ timeline

1yr. IPQ timeline									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ					-.01	59	.98	-.004	.31
D	9	2.88	.97	.32					
ND	52	2.88	.83	.12					

Table 4.13 T-tests For Initial Cognitive Deficits on 1yr. IPQ consequences

1yr. IPQ consequences									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ					1.39	59	.17	.27	.20
D	9	2.52	.60	.20					
ND	52	2.80	.54	.07					

Table 4.14 T-tests For Initial Cognitive Deficits on 1yr. IPQ control/cure

1yr. IPQ control/cure									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ					.05	59	.96	.06	.15
D	9	3.43	.45	.15					
ND	52	3.43	.40	.05					

Hypothesis 2.b: Stroke patients with cognitive deficit at 6mo. will have significantly different illness representations than those without cognitive deficit at 6mo. and at 1yr.

Hypothesis 2.b was tested using an independent t-test to examine differences between patients with cognitive deficits and patients without cognitive deficits, at 6 months, on 6 month and 1yr. illness representations. This hypothesis was not supported as there were no significant differences between

patients with 6mo. cognitive deficit and those without regarding illness

representations at 6mo. and 1yr.

Table 4.15 T-tests For 6mo. Cognitive Deficits on 6mo. IPQ Identity

6mo. IPQ identity									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
6mo. MMSE									
D	13	3.23	2.89	.80					
ND	50	2.62	2.32	.33					
					-1.80	61	.43	-.61	.76
6mo. EC									
D	49	2.67	2.38	.34					
ND	6	2.17	2.64	1.08					
					-.49	53	.63	-.51	1.04

Table 4.16 T-tests For 6mo. Cognitive Deficits on 6mo. IPQ Timeline

6mo. IPQ timeline									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
6mo. MMSE									
D	13	3.10	1.06	.29					
ND	50	2.77	.91	.13					
					-1.13	61	.26	-.33	.29
6mo. EC									
D	49	2.83	.95	.14					
ND	6	2.50	.86	.35					
					-.81	53	.42	-.33	.41

Table 4.17 T-tests For 6mo. Cognitive Deficits on 6mo. IPQ Consequences

6mo. IPQ consequences									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
6mo. MMSE									
D	13	3.22	.48	.13					
ND	50	3.02	.56	.07					
					-1.19	61	.24	-.20	.17
6mo. EC									
D	49	3.09	.55	.07					
ND	6	3.10	.27	.11					
					.02	53	.98	.04	.23

Table 4.18 T-tests For 6mo. Cognitive Deficits on 6mo. IPQ Control/Cure

6mo. IPQ control/cure									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
6mo. MMSE					1.91	61	.06	.23	.12
D	13	3.19	.43	.12					
ND	50	3.43	.38	.05					
6mo. EC					.20	53	.84	.03	.17
D	49	3.35	.40	.05					
ND	6	3.39	.33	.13					

Table 4.19 T-tests For 6mo. Cognitive Deficits on 1yr. Perceived Control

1yr. RLOC									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
6mo. MMSE					-.69	61	.50	-.81	1.19
D	13	40.15	3.00	.83					
ND	50	39.34	3.98	.56					
6mo. EC					.01	53	.99	.01	1.65
D	49	39.65	3.86	.55					
ND	6	39.67	3.27	1.33					

Table 4.20 T-tests For 6mo. Cognitive Deficits on 1yr. IPQ Identity

1yr. IPQ identity									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
6mo. MMSE					-.21	61	.83	-.13	.62
D	15	2.23	2.31	.64					
ND	30	2.10	1.90	.27					
6mo. EC					.00	53	1.00	.00	.83
D	49	2.00	1.87	.27					
ND	6	2.00	2.28	.93					

Table 4.21 T-tests For 6mo. Cognitive Deficits on 1yr. IPQ Timeline

1yr. IPQ timeline									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
6mo. MMSE					-92	61	.36	-.24	.26
D	13	3.05	.99	.28					
ND	50	2.81	.82	.16					
6mo. EC					-.73	53	.47	-.27	.37
D	49	2.82	.85	.12					
ND	6	2.56	.81	.33					

Table 4.22 T-tests For 6mo. Cognitive Deficits on 1yr. IPQ Consequences

1yr. IPQ consequences									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
6mo. MMSE					-.94	61	.35	-.16	.17
D	13	2.89	.60	.17					
ND	50	2.73	.51	.07					
6mo. EC					.61	53	.54	.14	.22
D	49	2.77	.53	.07					
ND	6	2.90	.43	.18					

Table 4.23 T-tests For 6mo. Cognitive Deficits on 1yr. IPQ Control/Cure

1yr. IPQ control/cure									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
6mo. MMSE					1.48	61	.14	.18	.12
D	13	3.29	.34	.09					
ND	50	3.48	.41	.05					
6mo. EC					-.38	53	.70	-.06	.18
D	49	3.46	.41	.05					
ND	6	3.39	.33	.13					

Hypothesis 2.c: Stroke patients with cognitive deficit at 1yr. will have significantly different illness representations than those without cognitive deficit at 1yr.

Hypothesis 2.c was tested using independent t-tests to examine differences between patients with cognitive deficits and patients without cognitive deficits, at one year, on illness representations at one year. Some results support the hypothesis, however, overall the results were inconclusive. Tables 4.24 - 4.28 show the significance of the mean differences. Results showed that significant differences were found between patients with cognitive deficit and those without on Trails-B with regard to IPQ timeline and, elevator counting with regard to IPQ control/cure.

Table 4.24 T-tests For 1yr. Cognitive Deficits on 1yr. Perceived Control

1yr. RLOC									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sd
1yr. MMSE					-4.25	63	.23	-2.34	1.93
D	4	41.75	.50	.25					
ND	61	39.41	3.84	.49					
WAIS-R					.87	62	.39	.83	.96
D	26	39.12	4.23	.83					
ND	38	39.95	3.42	.56					
vocabulary					.29	63	.77	.32	1.07
D	25	39.36	4.82	.96					
ND	40	39.68	2.97	.47					

comprehension					.46	63	.65	.43	.94
D	36	39.79	3.91	.65					
ND	29	39.36	3.62	.67					
block design					.56	63	.58	.53	.95
D	28	39.25	4.17	.79					
ND	37	39.78	3.46	.57					
digit span					-.87	63	.39	-.81	.94
D	29	39.92	3.74	.62					
ND	36	39.10	3.80	.71					
Trails-B					1.62	63	.11	2.06	1.28
D	55	39.24	3.82	.52					
ND	10	41.30	2.95	.93					
1yr. EC					1.18	63	.24	1.23	1.04
D	18	38.67	4.41	1.04					
ND	47	39.89	3.47	.51					
1yr. ECD					-.03	63	.98	-.03	.94
D	30	39.57	4.01	.73					
ND	35	39.54	3.59	.61					

Table 4.25 T-tests For 1yr. Cognitive Deficits on 1yr. IPQ Identity

1yr. IPQ identity									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
1yr. MMSE					.11	63	.91	.11	1.01
D	4	2.00	1.41	.71					
ND	61	2.11	1.98	.25					
WAIS-R					.16	62	.87	.08	.50
D	26	2.08	2.08	.41					
ND	38	2.16	1.90	.31					
vocabulary					-1.77	63	.08	-.87	.49
D	25	2.64	2.18	.43					
ND	40	1.78	1.75	.28					

comprehension					-1.44	63	.16	-.69	.48
D	36	2.42	2.26	.38					
ND	29	1.72	1.41	.26					
block design						63	.70	.19	.49
D	28	2.00	2.05	.39	.39				
ND	37	2.19	1.88	.31					
digit span					-.53	63	.60	-.26	.49
D	36	2.22	2.11	.35					
ND	29	1.97	1.74	.32					
Trails-B					-.72	63	.48	-.48	.67
D	55	2.18	2.03	.27					
ND	10	1.70	1.42	.45					
1yr. EC					.99	63	.33	.53	.54
D	18	1.72	1.71	.40					
ND	47	2.26	2.03	.30					
1yr. ECD					-.35	63	.73	-.17	.49
D	30	2.20	1.83	.33					
ND	35	2.03	2.06	.35					

Table 4.26 T-tests For 1yr. Cognitive Deficits on 1yr. IPQ Timeline

1yr. IPQ timeline									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
1yr. MMSE					-.17	63	.87	-.07	.44
D	4	2.92	.83	.42					
ND	61	2.84	.86	.11					
WAIS-R					.14	62	.89	.03	.22
D	26	2.82	.83	.16					
ND	38	2.85	.88	.14					
vocabulary					-.25	63	.80	-.05	.22
D	25	2.88	.95	.19					
ND	40	2.83	.79	.13					
comprehension					.62	63	.54	.13	.21
D	36	2.79	.89	.15					
ND	29	2.92	.80	.15					

block design									
D	28	2.92	.88	.17					
ND	37	2.79	.83	.14					
digit span									
D	36	2.96	.83	.14					
ND	29	2.70	.86	.16					
Trails-B									
D	55	2.94	.82	.11					
ND	10	2.33	.85	.27					
1yr. EC									
D	18	2.96	.80	.19					
ND	47	2.80	.87	.13					
1yr. ECD									
D	30	2.98	.88	.16					
ND	35	2.73	.81	.14					

Table 4.27 T-tests For 1yr. Cognitive Deficits on 1yr. IPQ Consequences

1yr. IPQ consequences									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
1yr. MMSE									
D	4	2.64	.86	.43					
ND	61	2.75	.53	.06					
WAIS-R									
D	26	2.66	.55	.11					
ND	38	2.80	.55	.08					
vocabulary									
D	25	2.77	.61	.12					
ND	40	2.73	.51	.08					
comprehension									
D	36	2.69	.54	.08					
ND	29	2.81	.55	.10					

block design					.26	63	.79	.03	.14
D	28	2.72	.53	.09					
ND	37	2.76	.56	.09					
digit span					1.24	63	.22	.17	.14
D	36	2.67	.59	.09					
ND	29	2.84	.48	.08					
Trails-B					-.10	63	.92	-.01	.19
D	55	2.75	.54	.07					
ND	10	2.73	.59	.19					
1yr. EC					.35	63	.73	.05	.15
D	18	2.71	.59	.14					
ND	47	2.76	.53	.07					
1yr. ECD					-1.56	63	.12	-.21	.13
D	30	2.86	.52	.09					
ND	35	2.65	.55	.09					

Table 4.28 T-tests For 1yr. Cognitive Deficits on 1yr. IPQ Control/Cure

1yr. IPQ control/cure									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
1yr. MMSE					-.09	63	.93	-.01	.20
D	4	3.46	.28	.14					
ND	61	3.44	.40	.05					
WAIS-R					.45	62	.65	.04	.10
D	26	3.41	.44	.08					
ND	38	3.46	.37	.05					
vocabulary					.34	63	.74	.03	.10
D	25	3.42	.45	.08					
ND	40	3.45	.36	.05					
comprehension					.45	63	.66	.04	.09
D	36	3.42	.43	.07					
ND	29	3.47	.34	.06					
block design					-1.27	63	.21	-.12	.09
D	28	3.51	.42	.07					
ND	37	3.39	.37	.06					

digit span					.87	63	.39	.08	.09
D	36	3.40	.42	.06					
ND	29	3.49	.36	.06					
Trails-B					1.25	63	.22	.17	.13
D	55	3.42	.38	.05					
ND	10	3.58	.44	.14					
1yr. EC					-.28	63	.78	-.03	.11
D	18	3.46	.39	.09					
ND	47	3.43	.40	.05					
1yr. ECD					2.83	63	.01 (*)	.26	.09
D	30	3.30	.39	.07					
ND	35	3.56	.35	.05					

Hypothesis 2.d: Stroke patients with initial cognitive deficit will have significantly different mood than those without initial cognitive deficit, initially, at 6mo. and at 1yr.

Hypothesis 2.d was tested using independent t-tests to examine differences between patients with cognitive deficits and patients without cognitive deficits, initially, on initial mood. Results do not support the hypothesis. Table 4.29-4.34 shows the mean differences which, were not significant at any of the time-points.

Table 4.29 T-tests For Initial Cognitive Deficits on Initial Anxiety

initial HADS anxiety									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ					-1.31	59	.20	-2.15	1.64
D	9	7.67	4.56	1.52					
ND	52	5.52	4.54	.63					

Table 4.30 T-tests For Initial Cognitive Deficits on Initial Depression

initial HADS depression									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ					-.39	59	.70	-.58	1.49
D	9	6.44	5.03	1.68					
ND	52	5.87	3.96	.55					

Table 4.31 T-tests For Initial Cognitive Deficits on 6mo. Anxiety

6mo. HADS anxiety									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ					.52	59	.61	.93	1.80
D	9	4.22	5.04	1.68					
ND	52	5.15	4.97	.69					

Table 4.32 T-tests For Initial Cognitive Deficits on 6mo. Depression

6mo. HADS depression									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ					.24	58	.81	.33	1.37
D	9	4.67	4.50	1.50					
ND	51	5.00	3.68	.51					

Table 4.323 T-test For Initial Cognitive Deficit on 1yr. Anxiety

1yr. HADS anxiety									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ					-.14	59	.89	-.10	.77
D	9	1.78	2.49	.83					
ND	52	1.70	2.06	.29					

Table 4.34 T-test For Initial Cognitive Deficit on 1yr. Depression

1yr. HADS depression									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
MSQ					-56	59	.58	-.45	.81
D	9	3.22	2.91	.97					
ND	52	2.77	2.12	.29					

Hypothesis 2.e: Stroke patients with cognitive deficit at 6mo. will have significantly different mood than those without at 6mo. and at 1yr.

Hypothesis 2.e was tested using independent t-tests to examine differences between patients with cognitive deficits and patients without cognitive deficits, at 6 months, on 6 month mood. Results do not support the hypothesis. Tables 4.35 – 4.38 show the mean differences which, were not significant at either 6mo. nor 1yr.

Table 4.35 T-tests For 6mo. Cognitive Deficits on 6mo. Anxiety

6moHADS anxiety									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
6mo. MMSE					-1.40	61	.17	-2.11	1.51
D	13	6.77	5.10	1.41					
ND	50	4.66	4.79	.68					
6mo. EC					.16	53	.87	.34	2.08
D	49	5.16	4.76	.68					
ND	6	5.50	5.17	2.11					

Table 4.36 T-tests For 6mo. Cognitive Deficits on 6mo. Depression

6mo. HADS depression									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
6mo. MMSE					-1.19	61	.24	-1.38	1.16
D	13	6.00	3.89	1.08					
ND	50	4.62	3.70	.52					
6mo. EC					-.25	53	.80	-.40	1.58
D	49	4.90	3.60	.51					
ND	6	4.50	4.14	1.69					

Table 4.37 T-tests For 6mo. Cognitive Deficits on 1yr. Anxiety

1yr. HADS anxiety									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
6mo. MMSE					-1.34	63	.18	-1.43	1.06
D	4	3	3.83	1.91					
ND	61	1.57	1.93	.25					
6mo. EC					.51	53	.61	.47	.93
D	49	1.69	2.17	.31					
ND	6	2.17	1.94	.79					

Table 4.38 T-tests For 6mo. Cognitive Deficits on 1yr. Depression

1yr. HADS depression									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
6mo. MMSE					-1.02	61	.31	-.76	.74
D	13	3.54	2.57	.71					
ND	50	2.78	2.33	.33					
6mo. EC					.56	53	.58	.58	1.04
D	49	2.92	2.23	.32					
ND	6	3.50	3.73	1.52					

Hypothesis 2.f: Stroke patients with cognitive deficit at 1yr. will have significantly different mood than those without cognitive deficit at 1yr.

Hypothesis 2.f was tested using independent t-tests to examine differences between patients with cognitive deficits and patients without cognitive deficits, at one year, on mood, at one year. Results do not support the hypothesis. Tables 4.39 – 4.40 show the mean differences which, were not significant.

Table 4.39 T-tests For 1yr. Cognitive Deficits on 1yr. Anxiety

1yr. HADS anxiety									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
1yr. MMSE					-1.34	63	.18	-1.43	1.06
D	4	3.00	3.83	1.91					
ND	61	1.57	1.92	.25					
WAIS-R					-.02	62	.99	-.08	.53
D	26	1.69	1.85	.36					
ND	38	1.68	2.24	.36					
vocabulary					-.79	63	.43	-.42	.53
D	25	1.92	2.45	.49					
ND	40	1.50	1.81	.29					
comprehension					.48	63	.65	.24	.52
D	36	1.56	2.14	.36					
ND	29	1.79	2.01	.37					
block design					-.54	63	.59	-.28	.52
D	28	1.82	1.81	.34					
ND	37	1.54	2.27	.37					
digit span					-.99	63	.33	-.51	.52
D	36	1.89	2.50	.42					
ND	29	1.38	1.35	.25					

Trails-B									
D	55	1.78	2.19	.30	-1.10	63	.28	-.78	.71
ND	10	1.00	1.05	.33					
1yr. EC					.79	63	.43	.45	.58
D	18	1.33	1.91	.45					
ND	47	1.79	2.14	.31					
1yr. ECD					-.38	63	.71	-.20	.52
D	30	1.77	2.13	.39					
ND	35	1.57	2.05	.35					

Table 4.40 T-tests For 1yr. Cognitive Deficits on 1yr. Depression

1yr. HADS depression									
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference	sed
1yr. MMSE					-.24	63	.81	-.30	1.23
D	4	3.25	2.22	1.11					
ND	61	2.95	2.38	.31					
WAIS-R					-1.16	62	.25	-.70	.60
D	26	3.38	2.53	.50					
ND	38	2.68	2.26	.37					
vocabulary					-.08	63	.94	-.05	.61
D	25	3.00	2.45	.49					
ND	40	2.95	2.33	.37					
comprehension					1.05	63	.30	.62	.59
D	36	2.69	2.39	.40					
ND	29	3.31	2.32	.43					
block design					-.62	63	.54	-.37	.59
D	28	3.18	2.47	.47					
ND	37	2.81	2.30	.38					
digit span					-.12	63	.91	-.06	.59
D	36	3.00	2.41	.40					
ND	29	2.93	2.33	.43					

Trails-B									
D	55	3.10	2.49	.34	-.98	63	.33	-.79	.81
ND	10	2.30	1.34	.42					
1yr. EC					.99	63	.33	.65	.65
D	18	2.50	2.28	.54					
ND	47	3.15	2.39	.35					
1yr. ECD					-.41	63	.68	-.24	.59
D	30	3.10	2.23	.41					
ND	35	2.86	2.49	.42					

Hypothesis 2.g: Better mood is associated with both high cognitive status and low cognitive status.

Hypothesis 2.g was tested using a quadratic regression equation to examine if better mood occurs when cognitive status is both high and low. The relationships between sixteen cognitive measures and mood were assessed where such relationships could be examined concurrently (6mo. on 6mo. or, 1yr. on 1yr.) and predictively (6mo. on 1yr.); two exceptions being ECD and FSIQ. As EC was a predictive measure, ECD was also included as it too measured attention control. Similarly, though 1yr. full scale WAIS-R (FSIQ) was not a predictive measure, it was included due to its consideration by the author as being the best general cognitive measure. Among the two significant quadratic relationships one fitted the hypothesised ‘inverted U’ shape whereby the highest mood was represented when cognitive status was high and when it was low (Figure 4.3). As the graph depicts, better mood (lower HADS depression score) occurs when cognitive deficit is low (lower elevator counting score) and also

when it is high (higher elevator counting score). As can be seen on Table 4.41, there was also a significant relationship in an unpredicted direction suggesting the opposite relationship. The predicted significant relationship, however, was the stronger and could not be explained by chance alone.

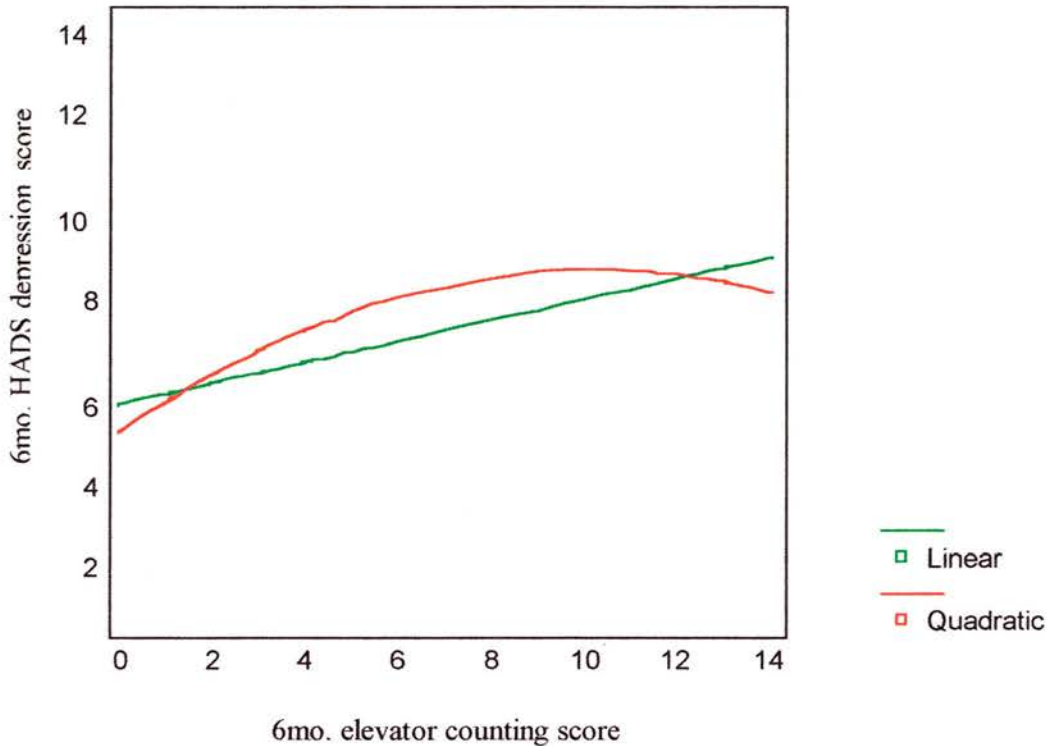


Figure 4.3 Significant Quadratic Relationship Between 6mo. Elevator Counting & 6mo. Depression.

Discussion

Research Question 1.

This study was designed to replicate and extend the previous study by asking the same questions in a longitudinal fashion as well as increasing the number of patients studied. This allowed the author to explore more fully the relationships between PC, AC and recovery as well as those of cognitive deficit with regard to illness representations and mood.

In addressing the first research question, hypothesis 1.a (Recovery from stroke at 1yr. will significantly correlate with: initial PC, 1yr. PC, 6mo. AC and 1yr. AC) was partially supported . Overall, the results were inconclusive. However, the finding that 1yr. PC did not significantly correlate with recovery but, that 1yr. AC (elevator counting) did replicated the findings from the previous study in this thesis. The results of initial PC and recovery also replicated findings in the literature (Johnston, Morrison et al., 1999) where initial PC did not significantly predict recovery (though in their study 6mo. PC did and, in other studies PC has been shown to predict recovery, i.e., Partridge & Johnston; 1989, Johnston et al., 2000). In fact, the amount of variance for recovery that initial PC explained in this study (approximately 6%) was similar to that found by Johnston, Morrison et al. (1999) (approximately 8%). These results also support the findings of the first study as well as those in the literature regarding AC. These results account for similar, though somewhat smaller, amounts of explained variance for recovery by 1yr. PC (approximately 17%) as compared to the results in the first study (approximately 23%). The result of this second study, that 1yr. AC (elevator counting) significantly correlated with 1yr.

recovery, supported the results of Robertson, Ridgeway et al. (1997) where 2yr. AC (elevator counting) significantly correlated with functional outcome at 2yrs. Again, similar explained variance for recovery in this study (approximately 17%) reflected that found by Robertson, Ridgeway et al. (1997) (approximately 17%). Both of these findings, however, occur after recovery has taken place, i.e., 2yr. measure correlating with 2yr. outcome. The result of this study where 6mo. AC did not significantly predict recovery at 1yr. did not replicate the finding by Robertson et al. that 2mo. AC predicted functional outcome at 2yrs.

The direction of the second hypothesis (1.2.b: PC and AC are not significantly correlated initially nor, at 1yr.), addressing research question one, was as hypothesised. In that a null hypothesis cannot be proven, the relationship between PC and AC were as posed in that PC and AC were not significantly related at the .05 level. The non-significance of this result supports a similar finding in the literature by Robertson, Baddeley et al. (1997), where in their study, 6mo. PC, initial AC (2wks. post-stroke) and 6mo. AC were not significantly related. The strength of this relationship by Robertson, Baddeley et al. cannot be commented on as the statistical strength was not reported other than its non-significance.

The third hypothesis of research question (H1.c: PC and AC make significantly independent contributions in explaining recovery from stroke at 1yr.) was not supported as the regression model showed. As shown by the small amount of additional explained variance contributed by PC to the regression equation, the amount of explained variance for initial PC could not be said to be independent from that of the larger explained variance of 1yr. AC.

The first hypothesis of research question 2 (H2.a: Stroke patients with initial cognitive deficit will have significantly different illness representations than those without cognitive deficit initially, at 6mo. and at 1yr.) was not supported. A possible reason for this is that the initial cognitive deficit measure (MSQ) was not sensitive enough to detect significant cognitive impairment. The majority of the patients (N=34) scored 10 out of 10, i.e., the lowest deficit score possible. The next highest score (9) was scored by the next largest group (N=18). One patient scored '6' and eight patients scored the lowest score '0'. The overall mean for the MSQ was 8.33 (SD = 3.33). Whereas deficit was defined by a score of ' ≤ 8 ', most patients were in the 'no deficit' group. There may not have been enough variance among the patients' scores to truly detect initial cognitive deficit. It is possible that had a more sensitive measure been used, significant results might have been seen. It may also be that the high MSQ scores reflected the possibility that the most cognitively impaired patients were excluded from this study in the initial screening process.

The second hypothesis of research question 2 (H2.b: Stroke patients with cognitive deficit at 6mo. will have significantly different illness representations than those without cognitive deficit at 6mo. and at 1yr) was not supported. There were no significant differences between 6mo. cognitive deficit patients and those without cognitive deficit with regard to 6mo. nor 1yr. illness representations.

Some support was found for the third hypothesis of research question 2 (H2.c: Stroke patients with cognitive deficit at 1yr. will have significantly different illness representations than those without cognitive deficit at 1yr.). However, overall results did not strongly support the hypothesis. Patients who

had a deficit on trails-B reported significantly higher IPQ timeline scores. As stated in chapter 3, this result must be in light of the fact that trails-B deficit was defined using a non-age corrected cut-off (possibly increasing the chance of a type I error). Patients with deficits on elevator counting with distraction reported significantly lower IPQ control/cure scores. Two of these three results reflected those found in study 1 (cognitive deficit patients showed higher identity with symptoms of their condition and, also lower perceptions of control/cure). While cognitive deficit patients in study one reported significantly lower perceptions of timeline, the opposite was found in the second study. Though there were similar findings between the two studies with regard to specific illness representations, the fact that they were not found on the same measures of cognitive deficit cause them to be inconclusive. This is particularly true since of the 45 relationships examined, one would expect 2 to be significant by chance alone; possibly explaining the significant results of hypothesis H2c.

The fourth hypothesis of research question 2 (H2.d: Stroke patients with initial cognitive deficit will have significantly different mood than those without initial cognitive deficit, at 6mo. and at 1yr.) was not supported as there were no significant differences between patients with initial cognitive deficit and those without on 6mo. and 1yr. mood. These results do not support previous findings in the literature where initial cognitive deficit was found to be significantly related to 6mo. depression (Robinson et al.; 1985). As with H2.a, the MSQ was possibly too insensitive to detect initial cognitive deficit or, the sample was not representative.

Results did not support the fifth hypothesis of research question 2 (H2.e: Stroke patients with cognitive deficit at 6mo. will have significantly different mood than those without at 6mo. and at 1yr.) as there were no significant differences between patients with cognitive deficit at 6mo. and those without on the HADS at 6mo. and 1yr.

The sixth hypothesis of research question 2 (H2.f: Stroke patients with cognitive deficit at 1yr. will have significantly different mood than those without cognitive deficit at 1yr.) was not supported as there were no significant differences between cognitive deficit patients at 1yr. and those without on 1yr. mood. These finding also did not replicate the results found in study 1 where differences were found on four 1yr. cognitive measures with regard to anxiety and depression at 1yr.

Results from the seventh hypothesis of research question 2 (H2.g: Better mood is associated with both high cognitive status and low cognitive status) were found to be mixed. One of the two significant curvilinear relationships suggested that better mood occurs when cognitive status is neither high nor low but, in-between (unpredicted 'U' shape; Table 4.41). This finding did not support the second significant relationship (between 6mo. elevator counting and 6mo. HADS depression) which was as suggested, supporting the hypothesis. The relationship between 6mo. elevator counting and 6mo. depression significantly suggested that better mood occurs when cognitive status is both high and low. When this curvilinear relationship is plotted it shows an 'inverted-U' shape with HADS depression on the 'Y' axis and 6mo. elevator counting on the 'X' axis (Figure 4.3). Though the differences in

direction between the two significant results yield mixed results, the one significant hypothesised relationship cannot be explained by chance alone. This is because, out of the 16 curvilinear relationships tested, .8 (less than the 2 statistically significant relationships) would be significant at the .05 level. It is also important to note, that as there was a significant curvilinear relationship in the hypothesised direction, this occurrence may explain why there were no significant differences between patients with cognitive deficit and those without on mood. If curvilinear relationships were occurring, it would weaken any linear relationship differences between the groups.

One limitation of this study was the possibility that the population examined was not representative of all stroke patients. The patients who agreed to participate were possibly the patients with the least amount of disability (both physically and cognitively). It is unclear as to whether this is reflected by the high Barthel Index scores or, that the Barthel Index was not sensitive enough for this population. Sample representation may have been compromised by using a too restrictive inclusion criteria. Significant differences may have been shown to exist had the population been more diverse. The issue of the patients used in this study not being a representative sample of stroke patients as a whole, might affect all results in this study. This limits the extent to which the results from this study can be generalised to the entire population of stroke patients.

Another possible limitation was the methods used to assess initial cognitive deficit. The MSQ was possibly too insensitive to detect initial cognitive deficit. Had a more rigorous measure been used, the initial cognitive deficit group may have been larger. Another possible limitation concerning

initial cognitive deficit was that the most cognitively impaired patients were screened by the SWOT study, which may have left an unrepresentative sample of less cognitively impaired stroke patients.

A third possible limitation of this study is that while concurrent administrations allowed the author to gather data at three time points, the administrators were different depending on the time of testing. Administrator differences (administration biases) may have been a factor in the results obtained.

Conclusions

Research Question 1.

- 1.) One-year AC significantly correlates with recovery from stroke at 1yr.
- 2.) PC and AC do not significantly correlate with each other at any time-points.
- 3.) PC and AC do not make significantly independent contributions to explaining recovery.

Research Question 2.

- 1.) Stroke patients with initial cognitive deficit do not have significantly different illness representations than stroke patients without cognitive deficit initially, at 6mo. or at 1yr.
- 2.) Stroke patients with cognitive deficit at 6mo. do not have significantly different illness representations than stroke patients without cognitive deficit at 6mo. or at 1yr.

- 3.) Overall, stroke patients with cognitive deficit at 1yr. do not consistently have significantly different illness representations than stroke patients without cognitive deficit at 1yr.
- 4.) Stroke patients with initial cognitive deficit do not have significantly different mood than stroke patients without cognitive deficit at 6mo. or at 1yr.
- 5.) Stroke patients with cognitive deficit at 6mo. do not have significantly different mood than stroke patients without cognitive deficit at 6mo. or at 1yr.
- 6.) Stroke patients with cognitive deficit at 1yr. do not have significantly different mood than stroke patients without cognitive deficit at 1yr.
- 7.) A curvilinear relationship exists between 6mo. cognitive status and 6mo. mood.
- 8.) Non-significant results regarding initial cognitive deficit may be due to the insensitivity of the measure used.
- 9.) Non-significant results regarding recovery may be due to a sample bias toward healthier, less initially disabled patients as compared with a larger population.
- 10.) Non-significant results regarding mood may be due to a sample bias towards healthier, less disabled, patients with higher initial moods.
- 11.) Non-significant results regarding mood are suggested to be due, in part, to a curvilinear relationship between cognitive deficit and mood which decreases the strength of any linear relationship that may exist.

CHAPTER 5

Discussion of Thesis Results

Summary

This thesis examined relationships in health psychology important for furthering the field of stroke research. It sought to replicate findings in the literature concerned with predicting stroke recovery; as well as introduce original areas of consideration, i.e., the relationships between both general and specific cognitive deficit and illness representations and, cognitive deficit and mood possibly having a curvilinear relationship. This chapter restates the aims of this thesis, the findings and the supporting evidence. Limitations are then discussed, ending with clinical and theoretical implications.

AIMS

1. To examine the relationship between perceived control and attention control with regard to stroke recovery.
2. To: a.) examine the relationships between cognitive deficit and illness representation and b.) examine the relationship between cognitive deficit and mood.

FINDINGS AND EVIDENCE

Finding 1: The relationship between attention control and recovery from stroke is significant at 1yr.

Evidence: H1.b (study 1), H1.a (study 2): The relationship between patients' measure of attention control at 6mo. and recovery was not significant (study 2). It may be that levels of attention control at 6 months would predict recovery at 2 years and thus be similar to the findings by Robertson, Ridgeway et al. (1997) but, the current results do not suggest so. However, patients' measures of attention control at 1yr. was found to significantly correlate with recovery from stroke. This finding also supported results in the first study. This result supported findings by Robertson, Ridgeway et al. (1997) where attention control at 2yrs. predicted recovery at 2yrs. The amount of explained variance for recovery by 1yr. attention control in the larger longitudinal study was similar to that found by Robertson, Ridgeway et al. (1997). The replicated finding that 1yr. attention control predicted recovery at 1yr. may not be meaningful as both measures were taken at the same time-point. It is possible that recovery may have already taken place.

Finding 2: As a measure of attention control, elevator counting was found to be a better predictor of recovery from stroke than elevator counting with distraction.

Evidence: H1.b (study 1), H1.a (study 2): Patients' measure of attention control at 1yr. using elevator counting was found to significantly correlate with recovery from stroke, while 1yr. elevator counting with distraction was not. This was the case for both the first and second study. A possible reason for this is that while patients physically recover from stroke, they also recover their attention control to the degree that elevator counting measures it. Whereas elevator counting with distraction also assesses attention control it is a more taxing measure and, the

degree of attention control it assesses may not recover with physical disability at one year.

Finding 3: Perceived control and attention control do not make independent contributions of explained variance to recovery from stroke.

Evidence: H1.d (study 1), H1.c (study 2): Though it appeared that initial perceived control and 1yr. attention control may have accounted for independent amounts of explained variance for recovery, a regression equation was calculated to determine exact amounts. Noting the larger beta-weight for attention control and that its amount of variance explaining recovery was significant, whereas perceived control was not, suggested that the amount of explained variance by perceived control could not be said to be independent of the amount being explained by attention control. It was also noted that the explained variance of recovery by both initial perceived control and 1yr. attention control (when included as independent variables in a regression equation) explained an additional small amount of variance of approximately 1% more than attention control alone.

Finding 4: Patients who have cognitive deficit, due to stroke, do not have significantly different illness representations than patients without cognitive deficit due to stroke.

Evidence: H2.a (study 2), H2.b (study 2), H2.a (study 1), H2.c (study 2): Patients with initial cognitive deficit, did not report having significantly different illness representations from patients without cognitive deficit initially, at 6mo., nor at 1yr (study 1). This result may have been affected by the possible insensitivity of

the measure used to determine initial cognitive deficit. A second possibility is that patients may have been initially screened from the study who would have been included in the 'deficit' group. Patients with cognitive deficit at 6mo. also did not report having significantly different illness representations at 6mo. than patients without cognitive deficit (study 2). Patients with 6mo. cognitive deficit also did not report having significantly different illness representations at 1yr., compared with patients without 6mo. cognitive deficit (study 2). The design of the second study was limited in what testing was possible initially and at 6mo. given that this data was gathered concurrently with another ongoing study. Had the inclusion of more extensive measures to determine initial and 6mo. cognitive deficit been possible, differences may have been detected regarding illness representations. With regard to the differences between patients with 1yr. cognitive deficit and patients without cognitive deficit at 1yr., on 1yr. illness representations, there were no consistent findings between the studies. Though there were significant differences, these differences did not occur on the same measures between studies 1 and 2.

Finding 5: Patients who have cognitive deficit, due to stroke, do not have significantly different mood than patients with out cognitive deficit.

Evidence: H2.d (study 2), H2.e (study 2) H2.b (study 1), H2.f (study 2): In general, patients with cognitive deficit, at all time points, did not report having significantly different mood than patients without cognitive deficit. While there were significant differences, there were no consistent patterns in the data between the two studies to strongly support the hypotheses. While some differences, in the first study, suggested that patients with cognitive deficit had

lower mood than patients without cognitive deficit, other results suggested the opposite relationship. As these results were not replicated in the second study, the findings were inconclusive. Again, initial cognitive deficit data may have been affected by insensitivity in the measure or, the possibility that initial cognitive deficit patients were excluded due to screening. Also, differences in mood may have been detected had there been more extensive testing to determine cognitive deficit initially and, at 6mo.

Finding 6: There is a curvilinear relationship between cognitive status and mood; whereby better mood occurs when cognitive status is both low and high.

Evidence: H2.g (study 2): Using cognitive measures where data was both concurrent and predictive, there was evidence to suggest a significant curvilinear relationship between cognitive status and mood in the hypothesised direction. This finding may account for the fact that there have been results in the literature which suggest both positive and negative relationships between cognitive status and mood (Robinson et al., 1985 and Kenealy et al., 2000 respectively). This may only be true with regard to specific cognitive status as there was also a significant finding in the opposite direction on a general cognitive status measure (MMSE). The presence of curvilinear relationships between cognitive deficit and mood could decrease the strengths of linear relationships and therefore, account for the non-significant relationships found regarding the linear hypotheses. This possibility should be further examined in future studies between cognitive deficit and mood.

LIMITATIONS

- 1.) The design of study 2 (choice of measures) was limited by the practical constraints of being conducted in concurrence with the SWOT study. While conducting the second study concurrently with the SWOT study allowed data to be gathered at two previous time-points from 1 yr., there were practical considerations regarding the amount of testing that could be managed by other researchers. In future studies, patients should be administered the same measures, by the same examiner(s), at each of the chosen time-points.

- 2.) The measure of disability (Barthel Index) may not have been sensitive enough to identify the true extent of disability for the patients used in this thesis. Therefore, there may have been a greater range of recovery for the patients. While the findings in the literature regarding perceived control and attention control predicting recovery used the Barthel Index as their measure of disability, from which recovery residuals were based, had a more sensitive measure of disability in this thesis been used, e.g., SIP (Gilson et al., 1975)/FLP (Patrick & Peach, 1978), significant predictions of recovery from levels of perceived control and attention control may have occurred.

- 3.) The definition of control as a health psychology construct, and indeed 'perceived control' varies among researchers. As discussed in the second chapter, there does not seem to be a singular definition of 'perceived control'. As such, there is ambiguity between the measure of perceived control used in this thesis and the theoretical construct being measured. The measure used in this thesis to assess levels of perceived control was the RLOC. In its

development, interrater agreement for internal/external items was used in the selection process. As a measure of perceived control, the RLOC is therefore mainly defined in terms of assessing *locus of control* as defined by Rotter (1966), i.e., internal vs. external control of outcome. Future studies may find it beneficial to include questions which are specific to the other domains of perceived control as was defined by Wallston et al. (1989) in chapter 2. Other than outcome, questions may be more specific with regard to control over internal states, behaviour and environment.

4.) There is no standardised method for defining 'cognitive deficit'. As with perceived control, there is ambiguity between the measures used to assess cognitive deficit and the theoretical concept of cognitive deficit itself. While the measures chosen, and methods used, to determine cognitive deficit attempted to equate with the theoretical concept of cognitive deficit, it is possible that patients with true cognitive deficit were not identified. Future studies should place more emphasis on identifying true cognitive deficit, e.g., using patients with identified brain lesions, recruiting from neurological rehabilitation centres, etc.

5.) The initial measure of cognitive deficit may not have been sensitive enough to identify true cognitive impairment. As mentioned in the second chapter, the MSQ is considered to be a brief measure of cognitive impairment. Had another measure with greater sensitivity been used, the 'initial cognitive deficit' group may have included individuals with significantly different illness representations and mood. Future studies should include measures sensitive enough to detect true cognitive impairment at early time-points. Using the measures administered

in this thesis as an example, a full initial WAIS-R score or MMSE score would have been beneficial in determining a truer level of initial cognitive deficit.

6.) The 6mo. measure of cognitive deficit may not have been sensitive enough to detect true cognitive deficit. Again, practical constraints of conducting the research determined the number of 6mo. cognitive impairment measures administered. As discussed in the second chapter, the MMSE, while being fairly quick to administer, measures a limited amount of cognitive abilities. It is possible that cognitive deficits at this time-point were not identified simply because they were not measured. Had a more extensive measure been used, significant differences may have been identified regarding illness representations and mood. Future studies should incorporate a more sophisticated measure of cognitive deficit or, incorporate more measures to determine true cognitive deficit at 6 months. Again, using this thesis as an example, the accuracy of 6mo. cognitive deficit would have been improved had there been full WAIS-R data available.

7.) Methods for defining 1yr. cognitive deficit were based on estimations of premorbid IQ, VIQ and PIQ (estimation based on an estimation). While this is a valid method of determining actual cognitive deficit, certain limitations are inherent. The methods for measuring cognitive deficit at 1yr. were compromised by the practical considerations of conducting research (achieving an adequate sample size). The preferred methods of either selecting patients for whom actual pre and post-morbid data existed or, administering more post-morbid tests in order to improve the accuracy of proration was not feasible. The limitation of

this, however, is that the 1yr. data for general and specific cognitive deficit was based on an estimation of pre-morbid ability which, in itself has the limitation of not being a true measure of pre and post-morbid differences. This was particularly important with regard to general cognitive deficit as determined by the WAIS-R score. As the patients' WAIS-R scores were estimated by proration of two subtests and that estimation was compared with a further estimation of premorbid ability, the chance of incorrectly identifying general cognitive deficit was increased. Regarding specific cognitive deficits, there were particular limitations with comparing these to estimations of premorbid verbal and performance IQ scores (themselves being obvious approximations to FSIQ). Future studies may improve upon the method used in this thesis by two obvious solutions. Firstly, the sample being examined may be restricted in that they have actual premorbid data, e.g., earlier testing may exist for patients who were neuropsychologically assessed for a TIA before an actual full-blown stroke. Secondly, and more practically, the use of demographic variables to improve the accuracy of premorbid estimations may be used in the calculations.

8.) Results based on the IPQ are limited in what can be surmised about specific illness representations as defined by the Self-Regulation model. The IPQ assumes that it tests the Leventhal model and, that the items are of equal numeric distances apart. Results in this thesis regarding illness representations (other than perceived control) are limited in so far as they reflect representations which are purported to equate with Leventhal's model, but such a relationship (between measure items and the model) has not been empirically shown in the literature to exist. It is therefore, possible that cognitive deficit patients have significantly

different representations of illness identity, cause, timeline, consequences and, control/cure. While a strength of the IPQ is that its development was based on a valid theoretical model, future studies should incorporate further measures of illness representation to increase the validity of measuring the five constructs within Leventhal's model. The incorporation of such measures as the IMIQ (Turk, Rudy & Salovey, 1986) along with the IPQ would increase the validity of the results regarding illness representations as defined by the Self-Regulation model.

9.) Data was gathered by different examiners initially and at 6 months. While this made concurrent data collection possible, interviewer biases may have been introduced. The limitation specific to the second study is that the significance of such variance was not tested. Furthermore, these biases were not constant as they may have come from different researchers at different times. Such biases would increase the error variance of the results. Results must be considered in light of how such biases may have affected them. Some styles of administration may not have been in the standardised fashion as intended by the protocol of each measure. In addition, intra-interviewer administration styles may not have been constant. The attempt to minimise this was made by the author by giving standardised instructions for the administration of the measures to other researchers who collected initial and 6mo. data for study 2.

10.) There may have been a sampling bias regarding the patients who were recruited. The patients who were recruited to the second study had to have been initially tested (participants in the SWOT study). Therefore, these were the

patients who had passed the initial cognitive screening. It is possible that the patients in the second study were not representative of the normal stroke population, i.e., less disabled. This possibility concerns results where recovery was predicted. It is possible that these patients had less room for recovery. Though recovery was regressed on individuals' measures of initial and final Barthel Index, a greater range of recovery might have been more sensitive to the predictors of perceived control and attention control.

11.) A portion of the patients in this study were recipients of the SWOT intervention. As this intervention has been shown to significantly increase recovery, there may have been a bias with the amount of recovery calculated in this study. It is possible that the recovery residual may have been different for patients who had received the intervention and therefore, results in this study regarding recovery may have been biased by the concurrent study.

12.) An important limitation regarding the statistical analyses of research question 2, in both studies, was the use of multiple t-tests without correcting for the increase of type I errors. One possibility would to have been to adjust the level of significance through Bonfferoni corrections. The use of multiple t-test analyses increased the probability of significant findings occurring by chance. Significance levels should have been adjusted to counter this increase in probability.

IMPLICATIONS

- 1.) It may be beneficial to measure stroke patients' levels of attention control at one year to determine how much recovery over one year has occurred. Levels of attention control at time-points before recovery (6mo.) do not appear from the findings of study 2 to be predictive of recovery at one year. However, given the limitations of study 2, it may be beneficial in predicting recovery from stroke to measure patients' levels of attention control before recovery as suggested by Robertson, Ridgeway et al. (1997).
- 2.) The relationship between perceived control and attention control warrants further investigation in a longitudinal study. The clinical implications of these two constructs may be different at different time-points. In reference to the finding of 1yr. attention control significantly predicting recovery, it may be that at later time-points during a patient's recovery, attention control is a more important indicator.
- 3.) Results from this thesis tend to support past findings in the literature regarding perceived control and attention control with regard to recovery from stroke.
- 4.) Future studies examining the relationship between cognitive deficit and illness representations need to have improved methods of identifying cognitive deficit; particularly when making premorbid estimations.
- 5.) The possibility of curvilinear relationships between cognitive deficit and mood should be of concern to future studies

CONCLUSION

This thesis sought to examine the relationship between perceived control and attention control in stroke patients. It has given support to previous findings, suggesting that attention control significantly predicts recovery from stroke. This thesis has also been innovative in exploring the relationship between cognitive deficit (in both a general and specific depth) and illness representations. It has suggested that illness representations are not influenced by the patient's cognitive status. Finally, this thesis could give no support to previous findings in the literature regarding the negative linear relationship between cognitive impairment and mood; suggesting for the first time that this relationship may be curvilinear. Given the summation of the findings, there still exists a need for research to explain the incongruent relationship between impairment and disability discussed in the introduction chapter.

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Appendix A. (Recruitment Material)

Our Ref. 59/98

Tayside Committee on Medical Research Ethics

Dr R S MacWalter
 Consultant Physician
 Wards 3/4
 Ninewells Hospital
 Dundee

East Day Home
 King's Cross Hospital
 Clepington Road
 Dundee
 DD3 8EA
 Tel: 01382 660111
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1 May 1998

Enquiries to: Mr M Finlayson
 Extension No: 36926

Dear Dr MacWalter

An investigation of stroke disability as influenced by illness representations and cognitive and mood status

I refer to the above and wish to thank you and Mr Morthland for attending the meeting held on 24 April 1998.

The outstanding points having been addressed, I am pleased to advise of formal approval to proceed.

This approval is granted for a three year period on the understanding that the Committee be advised if, for any reason, the study does not proceed.

Yours sincerely



Malcolm J B Finlayson
 Secretary

Members: Dr P M Windsor (Chairman); Mr P K Brown; Dr W F M Dorward; Professor G Fenton; Dr B Green; Dr A B Lawson; Miss E S Macallan; Mr A MacConnachie; Dr M Roworth; Professor I D Willock; Mrs S Wilson. Medical Adviser: Dr D Walsh; Scientific Adviser: Dr T Smith.
 Secretary: Mr M J B Finlayson.



University of St. Andrews
School of Psychology

Date

Name

Address

Re: Stroke Study Recruitment

Dear...

I am a Ph.D. student from the University of St. Andrews, School of Psychology, working with Professor Marie Johnston and Dr. Ron MacWalter in Ninewells Hospital. I am conducting a study into recovery from stroke. I am looking at how the stroke may have interfered with everyday activities, memory, concentration and mood. I am also interested in your thoughts about the stroke and factors that may speed up recovery.

I am writing to invite you to participate in this *one time* study which, would last approximately one and half hours long. This research requires participants such as yourself who have had a stroke approximately one year ago. If you do agree, a date and time would be scheduled to meet you at your home. During the visit, questions would be asked about your stroke and questionnaires would be given that have to do with everyday activities, memory, concentration and mood. All information in this study is completely confidential and the study itself has been approved by the Tayside Medical Ethics Committee.

By participating, you are helping to further stroke research that in the future may be beneficial to others such as yourself. Participation is entirely voluntary and will not affect your medical care in any way should you not wish to participate. Enclosed is an information sheet that gives more detail into the study. I would be grateful if you would return your answer in the enclosed stamped and addressed envelope, or call: _____ where you may leave a message.

Yours sincerely,

Martin Morthland, M.A.

Information Sheet:

You are being asked to participate in a study entitled: An Investigation of Stroke Disability and Illness Representation Using Perceived Control, Attention Control and Measures of Cognitive and Mood Status. You have been chosen for this study because, you have had a stroke which enables you to reliably and verbally provide information pertinent to this study.

The aim of this study is twofold. Firstly, we want to see if we can better predict the amount of disability caused by having a stroke. Secondly, we want to look for a pattern of how, after having a stroke, people see themselves affected. These aims will be met through the administration of psychological tests.

You will be scheduled one testing date that will encompass approximately an hour and a half of testing in total. For your convenience, the testing session will be held in your home. The only foreseeable discomfort in the study is the possibility of simple fatigue due to the length of testing. The benefit, however, of being a subject for this study is that any useful and appropriate data will be given, upon your consent, to your consulting doctor. Being a subject for this study also benefits future stroke patients in that stroke disability may be better understood.

As a subject for this study, confidentiality is a prime importance to the examiner. All of your test information will be stored in a lock facility. Your information will be referred to by a "subject number", not by your name. To assure confidentiality and that the study is being conducted ethically, Tayside Medical Research Ethics Committee may monitor the study's progress.

If any time you wish more information on this study, contact: Martin Morthland, M.A., University of St.Andrews, School of Psychology, Fife,KY16 9JU, (tel. number). Participation in this study is entirely voluntary and you are free to refuse to take part or to withdraw from the study at any time without having to give a reason and without this affecting your future medical care.

Please place a tick-mark next to your answer.

Yes, I wish to participate in the study.

Contact me by telephone on this number: _____

and/or,

Contact me by post at this address:

No, I do not wish to participate.

Please print your name here: _____

Date

Dr. _____

Re: Patient Recruitment

Dear Dr. _____,

I am writing you in regard to your patient _____. Mr./Mrs. _____ has returned for their one-year follow-up appointment to the Stroke Centre in Ninewells Hospital (Dr. R.S. MacWalter). At this time Mr./Mrs. _____ has been invited to participate in a study involving stroke patients: An Investigation of Stroke Disability as Influenced by Illness Representations and Cognitive and Mood Status. The duration of the study lasts approximately one and a half hours and occurs one time per subject. This study has been reviewed and approved by the Tayside Medical Research Ethics Committee. Attached is the patient information sheet which gives more detail into the study. If you see any reason why your patient may be inappropriate, please contact one of the primary investigators: Martin Morthland, M.A. and Professor Marie Johnston; University of St. Andrews, School of Psychology, Fife, KY16 9JU, 01334-462072, 01334-462060; and Dr. R.S. MacWalter, Consultant Physician, Ninewells Hospital, Wards 3&4, Dundee, DD1 9SY, 01382-660111, 01382-632317.

Respectfully,

Martin Morthland, M.A.

Appendix B
(Measures)



BARTHEL INDEX

Name:

Date: Record Number:

Activity	Scoring
1. BOWEL control	10 = continent 5 = occasional accident 0 = incontinent
2. BLADDER control	10 = continent 5 = occasional accident 0 = incontinent/catheterized and unable to manage
3. PERSONAL toilet (wash face, comb hair, shave, clean teeth)	5 = independent 0 = needs help
4. FEEDING	10 = independent 5 = needs some help (cutting up food, spreading butter etc.) 0 = dependent
5. TOILET (getting on/off, handling clothes, wipe, flush)	10 = independent 5 = needs some help 0 = dependent
6. WALKING on level surface	15 = independent (may use aid) 10 = walks with help of person (verbal/physical) 5 = independent (in wheelchair) 0 = unable
7. TRANSFER (chair to bed and vice versa)	15 = independent 10 = minimal help (verbal or physical) 5 = can sit, major help 0 = unable
8. DRESSING (all fasteners, etc.)	10 = independent (including zips, buttons etc.) 5 = needs help but does at least half 0 = dependent
9. STAIRS	10 = independent 5 = needs help (verbal/physical) 0 = unable
10. BATHING	5 = independent 0 = dependent

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RECOVERY LOCUS OF CONTROL SCALE

Name:

Date: Record Number:

These are statements other people have made about their recovery. Please will you indicate the extent to which you agree or disagree with them in the right-hand columns.

	Strongly agree	Agree	Uncertain	Disagree	Strongly disagree
1. How I manage in the future depends on me, not on what other people can do for me.					
2. It's often best just to wait and see what happens.					
3. It's what I do to help myself that's really going to make all the difference.					
4. My own efforts are not very important, my recovery really depends on others.					
5. It's up to me to make sure that I make the best recovery possible under the circumstances.					
6. My own contribution to my recovery doesn't amount to much.					
7. Getting better now is a matter of my own determination rather than anything else.					
8. I have little or no control over my progress from now on.					
9. It doesn't matter how much help you get, in the end it's your own efforts that count.					

© Partridge and Johnston, 1989. From 'Perceived control of recovery from physical disability: measurement and prediction', *British Journal of Clinical Psychology*, 28, 53-9. Adaption of Table 1. Adapted with the kind permission of the author and publishers, The British Psychological Society, London.

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Code 4920 10 4



We are interested in your own personal views of how you now see your stroke. Please indicate how much you agree or disagree with the following statements about your stroke.

(1) (2) (3) (4) (5)
strongly disagree disagree neither agree nor disagree agree strongly agree

- _____ **A germ or virus caused my stroke.**
- _____ **Diet played a major role in causing my stroke.**
- _____ **Pollution of the environment caused my stroke.**
- _____ **My stroke is hereditary - it runs in my family.**
- _____ **It was just chance that I had my stroke.**
- _____ **Stress was a major factor in causing my stroke.**
- _____ **My stroke is largely due to my own behaviour.**
- _____ **Other people played a large role in causing my stroke.**
- _____ **My stroke was caused by poor medical care in the past.**
- _____ **My state of mind played a major part in causing my stroke.**
-
- _____ **My stroke will last a short time.***
- _____ **My stroke is likely to be permanent rather than temporary.**
- _____ **My stroke will last for a long time.**

(1) (2) (3) (4) (5)
strongly disagree disagree neither agree nor disagree agree strongly agree

_____ **My stroke is a serious condition.**

_____ **My stroke has had a major consequence in my life.**

_____ **My stroke has become easier to live with.***

_____ **My stroke has not had much effect on my life.***

_____ **My stroke has strongly affected the way others see me.**

_____ **My stroke has serious economic and financial consequences.**

_____ **My stroke has strongly affected the way I see myself as a person.**

_____ **My stroke will improve in time.**

_____ **There is a lot I can do to control my symptoms.**

_____ **There is very little that can be done to improve my stroke.***

_____ **My treatment will be effective in curing my stroke.**

_____ **Recovery from stroke is largely dependent on chance or fate.***

_____ **What I do can determine whether my stroke gets better or worse.**

Elevator Counting Protocol

Ex1.)

Ex.2)

1. 3 tones

2. 5

3. 6

4. 8

5. 11

6. 9

7. 14

Elevator Counting With Distraction

Ex 1.)

Ex 2.)

1. 2 tones

2. 4

3. 6

4. 8

5. 7

6. 10

7. 9

8. 12

9. 11

10. 14

Patient _____

Examiner _____

Date _____

MINI MENTAL STATE

Score Orientation

- () What is the (year) (season) (month) (date) (day)? (5 points)
- () Where are we? (state) (county) (town) (hospital) (floor)
(5 points)

Registration

- () Name 3 objects: 1 second to say each. Then ask the patient to repeat all three after you have said them. 1 point for each correct. Then repeat them until he learns them. Count trials and record _____ (3 points)

Attention and Calculation

- () Serial 7's. 1 point for each correct. Stop at 5 answers.
Or spell "world" backwards. (Number correct equals letters before first mistake - i.e., d l o r w = 2 correct).
(5 points)

Recall

- () Ask for the objects above. 1 point for each correct. (3 points)

Language Tests

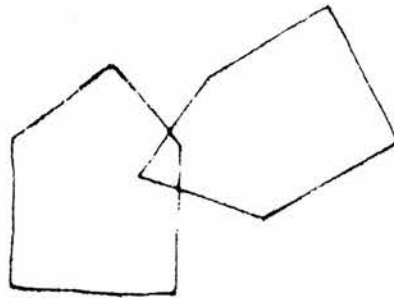
- () name - pencil, watch (2 points)
- () repeat - no ifs, ands or buts (1 point)
- () follow a 3 stage command: "Take the paper in your right hand, fold it in half, and put it on the floor." (3 points)

Mini Mental State
Page 2

Score

Read and obey the following:

- () CLOSE YOUR EYES. (1 point)
- () Write a sentence spontaneously below. (1 point)
- () Copy design below. (1 point)



() TOTAL 30 POINTS

CHORD	körd	SUPERFLUOUS	sōo-pûr'flōō-əs, sū-pûr'flōō-əs
ACHE	āk	SIMILE	sim'i-li
DEPOT	dep'ō	BANAL	bən-al'
AISLE	īl	QUADRUPED	kwod'rōō-ped
BOUQUET	bōōk'a, bōōkā', bōkā'	CELLIST	chel'ist
PSALM	sām	FACADE	fa-sād'
CAPON	kā'pn	ZEALOT	zel'ət
DENY	di-nī	DRACHM	dram
NAUSEA	nō'si-ə,nō'zhə	AEON	ē'on
DEBT	det	PLACEBO	plə-sē'bo
COURTEOUS	kûrt'yəs	ABSTEMIOUS	ab-stē'mi'əs
RAREFY	rār'-i-fi	DETENTE	dā-tāt (Fr.)
EQUIVOCAL	i-kwiv'ə-kl	IDYLL	id'il, id'əl
NAIVE	nā-ēv	PUERPERAL	pū-ûr'pər-əl
CATACOMB	kat'ə-kōōm	AVER	ə-vûr'
GAOLED	jāld	GAUCHE	gō sh
THYME	tīm	TOPIARY	tō'pi-ə-ri
HEIR	ār	LEVIATHAN	le-vī'ə-then
RADIX	rā'diks	BEATIFY	bi-at'i-fi
ASSIGNATE	as'-ig-nāt	PRELATE	prel'it
HIATUS	hī-ā'təs	SIDEREAL	sī-dē'ri-əl
SUBTLE	sut'l	DEMESNE	di-mān', di-mēn'
PROCREATE	prō'kri-āt	SYNCOPE	sing'kə-pē
GIST	jist	LABILE	lā'bīl
GOUGE	gowj	CAMPANILE	kam-pan-ē'lā, kam-pan-ē'lē

vocabulary

1. bed _____
2. ship _____
3. penny _____
4. **winter** _____
5. breakfast _____
6. repair _____
7. fabric _____
8. assemble _____
9. enormous _____
10. conceal _____
11. sentence _____
12. consume _____
13. regulate _____
14. terminate _____
15. commence _____
16. domestic _____
17. tranquil _____
18. ponder _____
19. designate _____
20. reluctant _____
21. obstruct _____
22. sanctuary _____
23. compassion _____
24. evasive _____

vocabulary cont.

25. remorse _____

26. perimeter _____

27. generate _____

28. matchless _____

29. fortitude _____

30. tangible _____

31. plagerize _____

32. ominous _____

33. encumber _____

34. audacious _____

35. tirade _____

total (max = 70) =

Comprehension (stop after 4 failures)

score(2,1,0)

1.) Clothes

2.) Envelope

*3.) Foods

*4.) Child labor

5.) Deaf

6.) Borrow

7.) Movies

8.) License

9.) Taxes

10.) Forest

11.) Prescription

12.) Iron

13.) Land

14.) Brooks

15.) Swallow

16.) Press

*If only one idea, say "Tell me another reason why..."

total (max=32) _____

Block Design

design	time	pass-fail	score(circle the appropriate score)				
1.) 60"	1						2
	2		0	1			
2.) 60"	1						2
	2		0	1			
3.) 60"			0	4(16-60)	5(11-15)	6(1-10)	
4.) 60"			0	4(16-60)	5(11-15)	6(1-10)	
5.) 60"			0	4(21-60)	5(16-20)	6(11-15)	7(1-10)
6.) 120"			0	4(36-120)	5(26-35)	6(21-25)	7(1-20)
7.) 120"			0	4(61-120)	5(46-60)	6(31-45)	7(1-30)
8.) 120"			0	4(76-120)	5(56-75)	6(41-55)	7(1-40)
9.) 120"			0	4(76-120)	5(56-75)	6(41-55)	7(1-40)

total(max=51)_____

Digit Span

Forward

	<u>pass-fail</u>	<u>score (2,1,0)</u>
1.) 5-8-2 6-9-4	____ ____	_____
2.) 6-4-3-9 7-2-8-6	____ ____	_____
3.) 4-2-7-3-1 7-5-8-3-6	____ ____	_____
4.) 6-1-9-4-7-3 3-9-2-4-8-7	____ ____	_____
5.) 5-9-1-7-4-2-8 4-1-7-9-3-8-6	____ ____	_____
6.) 5-8-1-9-2-6-4-7 3-8-2-9-5-1-7-4	____ ____	_____
7.) 2-7-5-8-6-2-5-8-4 7-1-3-9-4-2-5-6-8	____ ____	_____
		_____ max=14

Backward

1.) 2-4 5-8	____ ____	_____
2.) 6-9-2 4-1-5	____ ____	_____
3.) 3-2-7-9 4-9-6-8	____ ____	_____
4.) 1-5-2-8-6 6-1-8-4-3	____ ____	_____
5.) 5-3-9-4-1-8 7-2-4-8-5-6	____ ____	_____

6.) 8-1-2-9-3-6-5
4-7-3-9-1-2-8

—
—

—

7.) 9-4-3-7-6-2-5-8
7-2-8-1-9-6-5-3

—
—

—

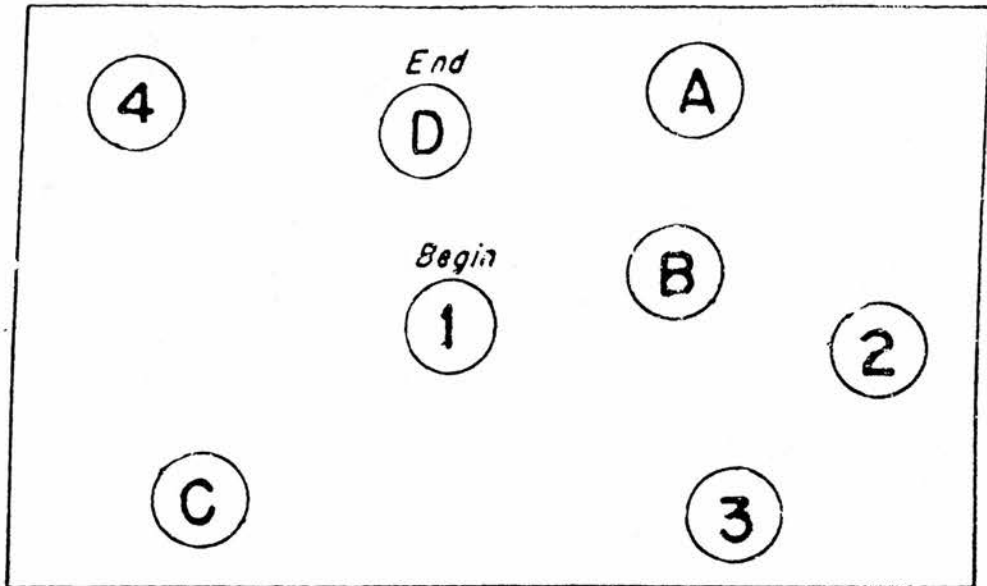
— **max=14**

— + — = — **max=28**

TRAIL MAKING

Part B

SAMPLE



End

13

8

9

I

D

B

4

3

Begin

7

1

5

H

C

12

G

A

J

2

6

L

E

F

K

11

G
 E
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 U
 DAY
 HER
 READ
 TEN
 GET
 N
 E
 O
 M
 K
 MAN
 N
 STAR
 LEG
 LEG
 ARE





Hospital Anxiety and Depression Scale

Name Date

Clinicians are aware that emotions play an important part in most illnesses. If your clinician knows about these feelings she or he will be able to help you more.

This questionnaire is designed to help your clinician to know how you feel. Ignore the numbers printed on the left of the questionnaire. Read each item and **underline** the reply which comes closest to how you have been feeling in the past week.

Don't take too long over your replies; your immediate reaction to each item will probably be more accurate than a long thought-out response.

fold along dashed line

A

3

2

1

0

D

0

1

2

3

A

3

2

1

0

I feel tense or 'wound up':

- Most of the time
- A lot of the time
- From time to time, occasionally
- Not at all

I still enjoy the things I used to enjoy:

- Definitely as much
- Not quite so much
- Only a little
- Hardly at all

I get a sort of frightened feeling as if something awful is about to happen:

- Very definitely and quite badly
- Yes, but not too badly
- A little, but it doesn't worry me
- Not at all

(continued overleaf)



HOSPITAL ANXIETY AND DEPRESSION SCALE

D	
0	
1	
2	
3	
fold along dashed line	
A	
3	
2	
1	
0	
D	
3	
2	
1	
0	
A	
0	
1	
2	
3	
D	
3	
2	
1	
0	
A	
0	
1	
2	
3	

I can laugh and see the funny side of things:

- As much as I always could
- Not quite so much now
- Definitely not so much now
- Not at all

Worrying thoughts go through my mind:

- A great deal of the time
- A lot of the time
- From time to time but not too often
- Only occasionally

I feel cheerful:

- Not at all
- Not often
- Sometimes
- Most of the time

I can sit at ease and feel relaxed:

- Definitely
- Usually
- Not often
- Not at all

I feel as if I am slowed down:

- Nearly all the time
- Very often
- Sometimes
- Not at all

I get a sort of frightened feeling like 'butterflies' in the stomach:

- Not at all
- Occasionally
- Quite often
- Very often

(continued overleaf)



HOSPITAL ANXIETY AND DEPRESSION SCALE

D	
3	
2	
1	
0	
A	
3	
2	
1	
0	
D	
0	
1	
2	
3	
A	
3	
2	
1	
0	
D	
0	
1	
2	
3	
D	A

fold along dashed line

I have lost interest in my appearance:

- Definitely
- I don't take as much care as I should
- I may not take quite as much care
- I take just as much care as ever

I feel restless as if I have to be on the move:

- Very much indeed
- Quite a lot
- Not very much
- Not at all

I look forward with enjoyment to things:

- As much as ever I did
- Rather less than I used to
- Definitely less than I used to
- Hardly at all

I get sudden feelings of panic:

- Very often indeed
- Quite often
- Not very often
- Not at all

I can enjoy a good book or radio or TV programme:

- Often
- Sometimes
- Not often
- Very seldom

Now check that you have answered all the questions

For office use only:

D : Borderline 8-10A : Borderline 8-10

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Appendix C
(Descriptive Data For Study 1)

Appendix C1: Demographic Data For Patients in Study 1

	N	Mean Age	Std. Deviation
Females	20	61.21	9.27
Males	36	60.9	9.2

Appendix C2: Descriptives of Measures Used in Study 1

	N	Minimum	Maximum	Mean	Std. Deviation	Score Range
Barthel Index	56	45	100	93.3929	10.4493	0 to 100
RLOC	56	26	45	35.2857	5.1229	0 to 45
IPQ identity	56	0	11	4.6786	3.0814	0 to 12
IPQ timeline	56	1	5	2.8869	0.8511	1 to 5
IPQ consequences	56	1.86	4.71	3.0816	0.6998	1 to 5
IPQ control/cure	56	2.83	5	3.5238	0.4339	1 to 5
elevator counting	56	0	7	0.6964	1.3338	0 to 7
elevator counting with distraction	56	0	10	3.8571	3.3217	0 to 10
Mini Mental State Exam	56	18	30	26.5	3.2079	0 to 30
NART	56	3	44	18.5893	10.2386	0 to 50
vocabulary scaled score	56	2	18	9.5536	2.7298	1 to 19
comprehension scaled score	56	3	19	9.6786	3.5732	1 to 19
block design scaled score	56	1	19	9.7321	3.4245	1 to 19
digit span scaled score	56	3	15	8.8214	3.0398	1 to 19
Trail Making	56	50	280	150.9643	62.3652	na
Mental State Questionnaire	41	0	10	9.0488	1.8702	0 to 10
HADS anxiety	56	0	21	8.8214	5.514	0 to 21
HADS depression	56	0	17	6.4464	4.6823	0 to 21

Appendix C3: Internal Reliability of Measures Used in Study 1

Measures	N	α
Initial Barthel	53	.907
1 yr. Barthel	56	.804
RLOC	56	.790
IPQ identity	56	.883
IPQ timeline	56	.358•
IPQ consequences	56	.602
IPQ control/cure	56	.290•
HADS	56	.919

Appendix D
(Descriptive Data For Study 2)

Appendix D1: Demographic Data For Patients in Study 2

	N	Mean Age	Std. Deviation
Females	32	70.09375	9.559185331
Males	33	66.36363636	11.89227213

Appendix D2: Descriptives of Measures Used in Study 2

	N	Minimum	Maximum	Mean	Std. Deviation	Score Range
Initial Barthel	61	65	100	95.2459	7.3839	0 to 100
6mo. Barthel	64	45	100	95.7031	9.548	0 to 100
1yr. Barthel	65	75	100	98.6154	3.9039	0 to 100
Initial RLOC	64	23	45	36.4687	4.4578	0 to 45
1yr. RLOC	65	26	45	39.5538	3.7585	0 to 45
6mo. IPQ identity	64	0	8	2.75	2.4169	0 to 12
1yr. IPQ identity	65	0	8	2.1077	1.9455	0 to 12
6mo. IPQ timeline	64	1	4.33	2.8177	0.952	1 to 5
1yr. IPQ timeline	65	1	4.33	2.8462	0.8479	1 to 5
6mo. IPQ consequences	64	1.86	4.57	3.0491	0.5478	1 to 5
1yr. IPQ consequences	65	1.57	3.71	2.7451	0.5431	1 to 5
6mo. IPQ control/cure	64	2.33	4.33	3.3724	0.4014	1 to 5
1yr. IPQ control/cure	65	2.67	4.33	3.441	0.3925	1 to 5
6mo. elevator counting	55	0	7	3.4727	4.3027	0 to 7
1yr. elevator counting	65	0	7	0.6923	1.5705	0 to 7
1yr. elevator counting with distraction	65	0	8	2.9692	2.2774	0 to 10
6mo. Mini Mental State Exam	63	20	30	26.7937	2.695	0 to 30
1yr. Mini Mental State Exam	65	23	30	27.9692	1.8789	0 to 30
NART	65	3	42	17.5692	9.3491	0 to 50
vocabulary scaled score	65	2	18	9.3077	2.7382	1 to 19
comprehension scaled score	65	3	19	9.2154	3.6379	1 to 19
block design scaled score	65	3	19	9.7231	3.13	1 to 19
digit span scaled score	65	3	15	9.2769	2.8859	1 to 19
Trail Making	65	43	280	144.3692	60.9925	na
Mental State Questionnaire	61	0	10	8.3279	3.3253	0 to 10
Initial HADS anxiety	65	0	18	5.5385	4.5828	0 to 21
6mo. HADS anxiety	65	0	19	5.0154	4.8525	0 to 21
1yr. HADS anxiety	65	0	10	1.6615	2.0712	0 to 21
Initial HADS depression	65	0	19	5.8308	4.0295	0 to 21
6mo. HADS depression	65	0	13	4.9538	3.7099	0 to 21
1yr. HADS depression	65	0	9	2.9692	2.3583	0 to 21

Appendix D3: Internal Reliability of Measures Used in Study 2

Measure	N	α
initial Barthel Index	61	.60
6mo. Barthel Index	64	.78
1 yr. Barthel Index	65	.52
initial RLOC	65	.70
1yr. RLOC	65	.78
6mo. IPQ identity	64	.73
1yr. IPQ identity	65	.64
6mo. IPQ timeline	64	.70
1yr. IPQ timeline	65	.62
6mo. IPQ consequences	64	.50
1yr. IPQ consequences	65	.55
6mo. IPQ control/cure	64	.63
1yr. IPQ control/cure	65	.56
initial HADS anxiety	65	.80
6mo. HADS anxiety	65	.83
1yr. HADS anxiety	65	.78
initial HADS depression	65	.77
6mo. HADS depression	64	.74
1yr. HADS depression	65	.71

Appendix D4

One-Sample t-Test Between Study Database MMSE Scores & Double-Entry Database MMSE Scores.

MMSE								
	N	mean	sd	sem	t	df	2-tailed sig.	mean difference
Study 2 Data	21	28.23	1.58	.344	81.99	20	.00	28.23
Double-Entry	21	28.28	1.59	.346	81.74	20	.00	28.28