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Depression and anxiety 3 months post stroke: Prevalence and correlates

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Abstract

This study examined prevalence of depression and anxiety as well as the relationships of age, gender, hemisphere of lesion, functional independence, and cognitive functioning (i.e., memory, attention/impulsivity, cognitive speed) to depression and anxiety at 3 months post stroke in 73 individuals. Prevalence of moderate to severe depression and anxiety in the sample were high (22.8 and 21.1%, respectively), with co-morbidity in 12.3% of cases. In regression analysis, 74.6% of variance in depression was explained, with significant relationships between increased depression and younger age, reduced cognitive speed, poorer verbal memory, left hemisphere lesion, and increased impact of interference (Stroop ratio). Left hemisphere of lesion also contributed to prediction of anxiety, as did cognitive speed, explaining 50.7% of the variance. The findings suggest that individuals with left hemisphere lesions may be particularly at risk of developing depression and anxiety after stroke, with younger individuals also at heightened risk of depression. While age and hemisphere of lesion contributed, cognitive performance explained the greatest proportion of variance in both depression and anxiety (51.3 and 38.5%, respectively). The findings suggest that cognition and mood are linked over and above physical independence and that both should be addressed as part of the rehabilitative process.

Keywords: Stroke; Depression; Anxiety; Cognition

This study examined the prevalence and correlates of post-stroke anxiety (PSA) and depression. The paragraphs that follow review the literature regarding prevalence, and then the correlates of post-stroke depression (PSD) and PSA, in turn.

1. Prevalence

The study of mood disorders after stroke has focused largely on depression. Reported prevalence of PSD varies widely, though most studies place prevalence between 20 and 50%, and indicate that depression persists 3–6 months post stroke (Fedoroff, Starkstein, Parikh, Price, & Robinson, 1991; Hosking, Marsh, & Friedman, 2000; Lyketsos, Treisman, Lipsey, Morris, & Robinson, 1998; Parikh, Lipsey, Robinson, & Price, 1988; Schubert, Taylor, Lee, Mentari, & Tamaklo, 1992; Schwartz et al., 1993; Starkstein, Bryer, Berthier, & Cohen, 1991; Starkstein & Robinson, 1991a, 1991b). PSD has a negative impact on case fatality and rehabilitation (Whyte & Mulsant, 2002), and functional outcomes (Herrmann, Black, Lawrence, Szekely, & Szalai, 1998). In contrast, PSA has only recently begun to be investigated (Castillo,

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Schultz, & Robinson, 1995; Castillo, Starkstein, Fedoroff, & Price, 1993; Chemerinski & Robinson, 2000; Dennis, O'Rourke, Lewis, Sharpe, & Warlow, 2000; Robinson, 1997, 1998; Shimoda & Robinson, 1998) with prevalence reports ranging from 4 to 28% (Astrom, 1996; House et al., 1991). As with PSD, the course of PSA has been found to remain fairly constant up to 3 years post stroke (Astrom, 1996; Robinson, 1998). Co-morbidity of PSA and PSD is high, with as many as 85% of people with generalised anxiety having co-morbid depression during the 3 years post stroke (Castillo et al., 1993, 1995).

2. Post-stroke depression

The literature generally points to the roles of demographics and injury characteristics, as well as physical independence and cognitive functioning as predictors of both PSD and PSA.

In terms of demographic and injury characteristics, depression in acute recovery from brain injury is related to the site of injury, while delayed onset depression is linked to psychological mechanisms (Astrom, 1996). Specifically, acute PSD is related to left hemisphere lesion, but this relationship disappears at, or beyond, 3 months post injury (Astrom, Adolfsson, & Asplund, 1993; Bhogal, Teasell, Foley, & Speechley, 2004; Hosking et al., 2000; Jorge, Robinson, Starkstein, & Arndt, 1993).

While most studies have not found a correlation between age and PSD (Anderson, Vestergaard, Riis, & Lauritzen, 1994; Herrmann, 1999; Lipsey, 1983; Morris, Robinson, Andrzejewski, & Samuels, 1993; Starkstein, Robinson, Berthier, Parikh, & Price, 1988; van de Weg, Kuik, & Lankhorst, 1999; Chemerinski, Robinson, & Kosier, 2001) those that do find a relationship consistently report that increased age is related to increased likelihood of depression (Fruehwald, Loffler, Eher, Saletu, & Baumhackl, 2001; Giaquinto et al., 1999). Contrary to the general population, higher prevalence of depression among women is not found post stroke (Burvill, Johnson, Jamrozik, & Anderson, 1995), with most studies finding minimal or no effect of gender (Giaquinto et al., 1999; Hosking et al., 2000; Ramasubbu, Robinson, Flint, Kosier, & Price, 1998). However, Paradiso and Robinson (1998) report that after brain injury, depressed women have a greater frequency of left hemisphere lesions and prior psychiatric and cognitive impairments than men, while depressed men have greater impairment in activities of daily living and social functioning.

In addition to demographic and injury characteristics, PSD has been associated with significantly greater cognitive impairment than is reported for patients without depression matched for size and location of lesion (Downhill & Robinson, 1994; House & Dennis, 1990; Robinson, Bolla-Wilson, Kaplan, & Lipsey, 1986; Starkstein, Robinson, & Price, 1988). Depressions' effects on cognition are differentially linked to left hemisphere lesions (Bolla-Wilson, Robinson, Starkstein, & Boston, 1989; Morris, Robinson, & Raphael, 1990; Murata, Kimura, & Robinson, 2000). While information on specific cognitive correlates are lacking, some aspects of cognition have been implicated. Hosking et al. (2000) found a correlation between PSD and attention deficits but not memory or verbal fluency. Nys (2005) reports that patients with moderate to severe PSD performed significantly worse than those with no or mild PSD on measures of memory, visual perception and construction, and language. While it is clear that cognitive impairment and depression are linked, the direction of causal mechanisms are still being debated (Morris et al., 1990; Paradiso & Robinson, 1998).

Finally, a number of studies report a relationship between PSD and physical functioning (e.g., Hosking et al., 2000; Robinson, 1983, 1985, 1987; Morris, Shields, Hopwood, & Robinson, 1994; Kotila, Numminen, Waltimo, & Kaste, 1999; Evans & Whitney, 1998). While the direction of the relationship has not been established, it is clear that early identification and treatment of PSD is beneficial to physical recovery (Robinson, 1997).

3. Post-stroke anxiety

While the literature on PSA remains in its infancy, the literature has begun to examine its relationship to similar demographic, injury, cognitive, and physical characteristics as those examined for PSD. In terms of injury characteristics, PSA correlates significantly with right hemisphere lesions, while co-morbid PSA and PSD are linked to left hemisphere lesions (Astrom, 1996). Castillo et al. (1993) found anxiety more prevalent in association with posterior right hemisphere lesions, whereas worry without anxiety disorder was associated with anterior lesions.

Those studies that have found relationships between PSA and age and gender report that women (Morrison, Johnston, & Walter, 2000; Schultz, Castillo, Kosier, & Robinson, 1997) and younger patients (<59 years) are more susceptible to PSA (Schultz et al., 1997), while others report no significant relationship (Dennis et al., 2000).

Most studies that have examined cognitive function and PSA have also assessed physical impairment. Castillo et al. (1993, 1995) report that PSA is not significantly correlated with physical functioning, cognitive functioning, or social functioning. While some authors similarly report no significant correlation (Starkstein et al., 1990), others report that anxiety is linked to greater impairment in activities of daily living both acutely and up to 3 years post stroke (Schultz et al., 1997).

As reported above, demographic and injury characteristics, physical functioning, and cognitive functioning have each been examined in their relationships to PSD and PSA. To date, few studies have examined both depression and anxiety post stroke, or their differential relationships to these factors. This study examined prevalence of PSA and PSD, and the relationships of age, gender, hemisphere of lesion, functional independence, and cognitive functioning with depression and anxiety 3 months post stroke. Given the potential negative impact of depression and anxiety, it is important for clinicians to know the likelihood of negative mood outcomes and to be able to accurately identify those clients who are most at risk of these outcomes.

4. Method

4.1. Participants

Participants were consecutive admissions to an inpatient rehabilitation unit with primary diagnosis of stroke who were 3 months post-stroke onset. Excluded were 14 individuals who were unable to be assessed; 10 of whom were aphasic, and 4 who were not fluent in English. Of the 76 remaining potential participants who consented to participate, three were lost to follow-up. The 73 participants were 40 (54.8%) males and 33 (45.2%) females with a mean age of 51.7 years (S.D. = 10.19). Formal education completed ranged from 9 to 17 years (mean = 11.97 years). Most of the participants ($n = 47$; 64.4%) were married, 21 (28.8%) were single, and 5 (6.8%) were divorced. Most participants ($n = 39$; 53.4%) self-identified as European, 10 (13.7%) as Asian, 9 (12.3%) as Maori, and 15 (20.5%) as Pacific Island peoples. Most participants ($n = 58$; 79.5%) had suffered an ischemic stroke, while 15 (20.5%) had suffered a hemorrhagic stroke. According to CT scans, 31 (42.5%) participants had left hemisphere damage and 33 (45.2%) right hemisphere damage. For the remaining nine (12.3%) participants, CT scan confirmation of stroke site was not available. Based on clinical evidence (e.g., laterality of hemiplegia), six of these nine participants had experienced a right hemisphere stroke and three a left hemisphere stroke. Further information on stroke location is provided in Table 1, which presents the frequency of various stroke locations, as noted in participant medical files. Middle cerebral artery, basal ganglia, and cerebellar infarcts were the most common descriptors of stroke location found for the sample, while a large proportion of the sample had only a vague descriptor of stroke location/type (e.g., multiple infarcts, CVA).

4.2. Measures

4.2.1. Beck Depression Inventory-II (BDI-II)

The BDI-II (Beck, Steer, & Brown, 1996) was used to measure depression. Participants responded to 21 four-choice statements, selecting the statement that most accurately described him/her over the past 2 weeks in relation to emotional, behavioural, and vegetative symptoms. Total scores range from 0 to 63; with 0–13 considered as 'minimal depression',

Table 1
Frequency and percent of various stroke location descriptors as obtained from participant medical files

Location descriptor	Frequency (%)
Middle cerebral artery	24 (32.9)
'CVA'	14 (17.8)
Basal ganglia	12 (16.4)
Cerebellar	10 (13.7)
'Multiple infarcts'	4 (5.5)
Pons or medulla	4 (5.5)
Parenchymal ICH	3 (4.1)
Anterior parietal	2 (1.4)

Note. Descriptors in quotes are verbatim descriptors, no more details information was available. ICH = intracranial hemorrhage.

14–19 as 'mild', 20–28 as 'moderate', and 29–63 as 'severe'. Test–retest reliability is 0.93, with internal consistency of 0.93 (Beck et al., 1996).

4.2.2. Beck Anxiety Inventory (BAI)

The BAI (Beck & Steer, 1993) has 21 items, each presenting a symptom of anxiety that is not shared with depression. Items are rated from 0 (not at all) to 3 (severe) for the last week. Fourteen items reflect somatic symptoms (e.g., trembling hands) and seven reflect thoughts/emotions (e.g., fear of dying). Total scores range from 0 to 63; with scores of 0–7 described as 'minimal', 8–15 as 'mild', 16–25 as 'moderate', and 26–63 as 'severe' (McDowell & Newell, 1996). Research indicates high internal consistency (0.90–0.94), reasonable test–retest reliability (0.67–0.93 after 1 week), and moderate to high convergent validity with other self-report and clinical ratings of anxiety (Beck & Steer, 1993).

4.2.3. Functional Index Measure (FIM; Granger, Hamilton, & Sherwin, 1986)

The FIM contains 13 motor items and 5 cognitive items, and is commonly used to assess outcomes of inpatient rehabilitation. Items cover self-care, sphincter control, mobility, locomotion, and social cognition. Though not used as a cognitive measure in this study, the five 'cognitive' items on the FIM include independence in comprehension of communication, expressive communication, social interactions, problem solving, and memory. The scale was rated by trained on-site nurses on performance rather than capacity, based on the amount of assistance needed to complete a task. Scores range from 126 to 18, with high scores indicating greater independence. The FIM is widely used post-stroke (Granger, Divan, & Fiedler, 1995; Hinkle, 2000; McDowell & Newell, 1996). Validity and reliability are equal to or better than that of other measures of physical functioning (Duncan et al., 2002; Granger, Hamilton, Linacre, Heinemann, & Wright, 1993; Hobart et al., 2001a; Kidd, Stewart, & Landry, 1995).

4.2.4. California Verbal Learning Test-II (CVLT-II; Delis, Kramer, Kaplan, & Ober, 2000)

The CVLT-II measures recall of a word list over a number of trials, and recall for an interference list; followed by immediate and delayed free and category-cued recall trials and a delayed recognition trial. Moderate to high levels of internal consistency have been reported for the CVLT-II (Delis et al., 2000). For this study, performances on immediate and delayed free recall trials were used to indicate unassisted recall memory.

4.2.5. Visual Paired Associates (VPA; Wechsler, 1987)

This test of visual learning and memory requires the examinee to learn and recall colours that are paired with six abstract line drawings over a number of trials. A delayed recall trial for the figure–colour pairs is administered after a 30 min delay (Wechsler, 1987). The reliability coefficients for learning trials range from 0.52 to 0.68 with an average of 0.58, while coefficients for delayed recall range from 0.31 to 0.69 with an average of 0.58 (Wechsler, 1987).

4.2.6. Digit and spatial spans (Wechsler, 1997)

These were included because the CVLT-II and VPA do not provide specific measures of working memory. In the digit span subtest, the subject is required to repeat an auditory string of digits of increasing length, and then do the same in reverse order from the examiner. Each correctly recalled string receives 1 point. Scores for forward trials range from 0 to 16 and backwards trials from 0 to 14. The two scores are summed to produce a digit span total score. Digit span internal consistency (0.90) and test–retest reliability (0.83) are high (Wechsler, 1997). Spatial span is analogous to digit span; however, subjects are required to tap out specific visual sequences of blocks on a 3-D board in the same or reverse order as presented by the examiner. Scores for both forward and backward spatial spans range from 0 to 16. Spatial span subtest reliability coefficients range from 0.71 to 0.85 across all age groups, and test–retest correlation coefficients are 0.71 (Wechsler, 1997). Raw scores are converted to standard scores that have a mean of 10 and standard deviation of 3.

4.2.7. Integrated Visual Auditory Continuous Performance Test (IVA-CPT; Sandford & Turner, 2000)

The IVA-CPT is a computerized assessment requiring the examinee to press a button when he/she sees or hears a '1' (target) and not to press when he/she sees or hears a '2' (foil). The task starts with a 1.5 min warm-up, followed by 32 practice items. The test has 500 trials and lasts for approximately 13 min. An equal number of auditory and visual stimuli are presented in a pseudo-random order. Prudence scores indicate errors of commission, with low prudence scores indicate carelessness or over-reactivity, and high scores indicate cautious, careful responding. As a measure of

attention vigilance scores indicate errors of omission. IVA-CPT scores are calculated as both raw scores and quotient scores that have a mean of 100 and a standard deviation of 15. The labels mildly, moderately, severely, and extremely impaired are reflected in IVA quotient scores that are less than 90, 80, 70, and 60, respectively.

4.2.8. Victoria Stroop

Scores on the Stroop test reflect the ability to suppress an automatic reading response and to shift sets to conform to changing demands while under a time constraint (Spreen & Strauss, 1989). Participants are shown three cards with dots, words, or colour names printed in red, green, blue, and yellow ink in pseudo-random order. The participant must name the colour of each dot, word, or colour name, moving from left to right, as quickly as possible. Colour names are printed in a colour that is never the same as the word (e.g., 'yellow' printed in blue ink). For each card, the time taken to say the correct colour of all items is recorded. A discrepancy score is obtained by comparing time taken to finish colour names with the baseline speed dots condition. Test–retest reliability coefficients of 0.90, 0.83, and 0.91 have been reported for the three trials.

4.3. Procedure

Ethical approval was obtained from the Auckland Ethics Committee. All new referrals over a 24-month period to an inpatient rehabilitation unit who met inclusion criteria were approached. At the time they were first approached, potential participants were 16–37 days post stroke. Variability in the timing of first contact was largely related to delays in referral to rehabilitation resulting from medical complications. All potential participants reviewed a Participant Information Sheets and Consent form, and it was made clear that consent was purely voluntary and that participation would not impact the rehabilitation services received. Consent forms highlighted the right to withdraw from the study at any time without penalty. For those who gave consent, historical information such as demographics and injury characteristics were gathered from patient files. At this time, an appointment was made to conduct the study interview at approximately 3-month post-stroke onset. Of the 76 individuals who consented to participate, only 3 were lost to follow-up at 3 months. The exact timing of the assessment ranged from 79 to 98 days post stroke (mean = 89 days). Neuropsychological and mood assessments were conducted according to standardised procedures and took approximately 120 min to complete.

All tests were scored according to standardised procedures and the data were entered into an SPSS 14.0 file for analysis. An alpha level of 0.05 was used to determine statistical significance. Preliminary to conducting regression analyses, multicollinearity of variables was addressed by combining those variables whose correlation was greater than 0.70, following the recommendation of Tabachnick and Fidell (1989).

5. Results

The results will be presented in three sections. First, descriptive data in regards to performance across the various measures is presented. This includes data relevant to the first question of interest of this study—the prevalence of PSD and PSA. This will be followed by presentation of correlations between the variables of interest, which leads to answering the main research question via regression analyses.

5.1. Descriptive data

Means and standard deviations across measures of physical and cognitive functioning are presented in Table 2. For the purposes of this study, performances that fell more than 1 standard deviation below the normative mean were considered below average, while those that fell more than 2 standard deviations below the mean were considered impaired. The average participant experienced a relatively good physical outcome (FIM). However, verbal memory was below average (CVLT-II short and long delayed cued recall). Both digit and spatial spans performances and visual learning and memory (VPA) were average, falling within 1 S.D. below the normative mean. On the IVA-CPT, visual prudence was within normal limits, auditory prudence was mildly impaired, auditory attention was moderately impaired, and visual attention was severely impaired. Performance on the Stroop indicates impaired speed of processing (Stroop dots) and impaired speed with a minimal cognitive load (Stroop words). Performance on the Stroop interference condition (Stroop colour names) and the Stroop ratio were both below average.

Table 2
Means and standard deviations of performance on neuropsychological measures at 3 months post stroke

Measure	Mean	S.D.
FIM raw score	116.36	11.04
California Verbal Learning Test		
Trial 1	−1.51	1.93
List B	−1.48	1.16
Short free recall	−1.59	1.48
Short cued recall	−1.77	1.76
Long free recall	−1.75	1.64
Long cued recall	−1.85	1.67
Recognition hits	−1.18	1.60
Visual Paired Associated		
Learning	−0.035	0.97
Recall	−0.282	0.92
Digit span[a]	8.28	2.71
Spatial span[a]	9.40	5.23
IVA-CPT		
Visual attention quotient	61.20	38.9
Auditory attention quotient	71.20	26.3
Visual prudence quotient	93.72	29.6
Auditory prudence quotient	88.20	31.7
Victoria Stroop		
Dots	−2.27	5.17
Words	−2.59	6.21
Colours	−1.21	4.18
Dot/colour ratio	−1.03	0.76

Note. All memory scores and Stroop scores are *z* scores.
[a] Note that for digit and spatial spans, scaled scores have a mean of 10 and S.D. of 3.

Table 3 presents findings for the BDI and BAI. Average scores fell within the minimal range for depression and the mild range for anxiety; however, 22.8% of participants experienced moderate or severe depression and 21.1% experienced moderate or severe anxiety. Five participants (6.8%) had co-morbid moderate depression and anxiety; an additional four participants (5.5%) had severe depression co-morbid with severe anxiety.

Having presented data on the prevalence of depression and anxiety, it must be determined whether this prevalence is falsely elevated due to the overlap of the physical outcomes of stroke and symptoms of PSD and PSA measured in this study. In order to examine this issue, symptoms that were ranked highly (items rated 2 or 3) by more than half of the participants were examined. In regards to PSD, the most commonly reported symptoms were feeling discouraged/hopeless (73%), feelings of being punished (67%), being self-critical (63%), and feeling tired (52%). This would suggest that, with the exception of fatigue, high scores on this measure of depression were most reflective of non-physical symptoms. Similarly, for anxiety, the most commonly reported symptoms on the BAI were fearing the worst (63%), scared (60%), fear of dying (59%), and wobbliness (53%), suggesting that cases of 'anxiety' reported here were contributed to by both emotional and somatic items, though emotional items were more commonly endorsed.

Table 3
Depression and anxiety at 3 months post stroke

	Mean	S.D.	Minimal	Mild	Moderate	Severe
BDI-II	12.49	11.85	31 (54.4%)	13 (22.8%)	7 (12.3%)	6 (10.5%)
BAI	9.35	9.05	35 (61.4%)	10 (17.5%)	8 (14.1%)	4 (7.0%)

5.2. Correlations

The results of correlational analysis are presented in Table 4. Parametric (Pearson's) and non-parametric (Spearman's) correlations have been used as appropriate to the data being examined. While Table 4 presents all correlation values, due to the large number of comparisons conducted, only those where $p < 0.01$ should be considered significant. In order to reduce the number of contrasts performed, digit span and spatial span, which both fell within 1 standard deviation of the normative mean, were excluded form this analysis. Increased BDI score was related to left hemisphere lesion and to reduced physical functioning (i.e., FIM), CVLT indices of verbal memory, IVA-CPT indices of impulsivity (Prudence), cognitive speed (Stroop dots trial), and ability to suppress habitual responses (Stroop ratio). Increased BAI score was significantly related to left hemisphere lesion and to poorer performance across measures of impulse control (IVA-CPT Prudence), and cognitive speed (Stroop dots trial).

In terms of interrelationships between demographic and injury characteristics and physical and cognitive variables, increased age at time of stroke was significantly related to increased Stroop ratio at 3 months post stroke. Male gender was associated with better cognitive speed (Stroop dots). Right hemisphere lesions were related to improved CVLT verbal delay memory. In terms of interrelationships between physical and cognitive measures, a better FIM independence scores was related to better cognitive speed (Stroop dots). There were large number of significant correlations between measures of cognitive functioning; this was particularly true of CVLT short delay recall, for which better performance was associated with better performance on all other cognitive measures except Stroop ratio. The fewest significant relationships were found between VPA delayed memory and other cognitive measures.

5.3. Regression analyses

Following from correlational analyses, due to multicollinearity, IVA-CPT prudence and vigilance scores were combined by addition to produce a 'response control' score. CVLT immediate and delayed free-recall scores were also combined through conversion of both scores to z scores, followed by addition. Following from correlational analyses performance on the VPA test (i.e., visual memory), which did not relate significantly to depression or anxiety, was excluded.

Hierarchical regression analyses were used to determine if demographic (gender, age), injury (hemisphere), physical (FIM), and the remaining cognitive variables as assessed at 3 months post stroke related to either depression or anxiety at 3 months post stroke. While the term prediction is being used, as this is the convention when using regression models, it must be noted here that the cross-sectional design of this study does not allow a determination of the direction of causal influences. Aspects of cognitive functioning included in the regression were the combined IVA-CPT vigilance and prudence 'response control' score, Stroop dots trial performance as an indication of cognitive speed, the combined verbal memory performance score, and Stroop ratio. Table 5 presents the findings.

In predicting depression, in step 1, the variables age, gender, and hemisphere of lesion explained only 2.4% of the variance in depression and was not significant ($p > 0.05$). Addition of the FIM to the equation increased variance explained to only 3.0% ($p > 0.05$). With demographic, injury, physical, and cognitive variables entered into the equation, 74.6% of the variance in depression was explained, $R = 0.864$, $F(8, 62) = 14.411$, $p < 0.001$; with cognitive variables accounting for 51.3% of variance explained. Contributing significantly to prediction were age ($p < 0.001$), hemisphere of lesion ($p < 0.001$), the combined verbal memory ($p < 0.05$), Stroop dots trial performance ($p < 0.001$), and Stroop ratio score ($p < 0.001$). Beta weights (β) presented in Table 5 suggest that increased BDI scores were associated with younger age, left hemisphere lesions, poorer scores on verbal memory and Stroop dots, and a higher Stroop ratio score (indicating that performance with increased cognitive load/interference was much worse than performance without).

In predicting anxiety age, gender, and hemisphere of lesion explained 12.1% of the variance, which was not significant ($p > 0.05$). With FIM added to the equation, only 12.2% of the variance was explained ($p > 0.05$). With the addition of cognitive variables to the equation, 50.7% of the variance in anxiety was explained, $R = 0.712$, $F(8, 62) = 4.999$, $p < 0.001$; with cognitive variables explaining 38.5% of the variance. Hemisphere of lesion ($p < 0.05$) and Stroop dots trial performance ($p < 0.001$) contributed significantly to the prediction. Increased BAI scores were related to left hemisphere lesion and poorer performance on the Stroop dots trial (i.e., reduced cognitive speed).

Table 4
Correlations of variables at 3 month follow-up

	BDI	BAI	Age	Gender	Hemisphere	FIM	CVLT short free	CVLT long free	VPA immediate	VPA delayed	IVA-CPT attention	IVA-CPT prudence	Stroop dots
BDI													
BAI	0.628**												
Age	0.073	−0.010											
Gender	0.086	0.327*	−0.106										
Hemisphere	−0.401**	0.372**	0.153	0.163									
FIM	−0.218*	−0.221	−0.239*	−0.056	−0.371*								
CVLT short free	−0.400**	−0.257	0.080	−0.357*	0.189	0.301*							
CVLT long free	−0.385**	−0.322*	0.059	−0.456**	0.334**	0.249	0.357*						
VPA immediate	−0.013	0.042	−0.091	0.062	−0.186	0.126	0.521**	0.295*					
VPA delayed	−0.160	−0.041	−0.012	0.166	−0.272	0.237	0.414**	0.204	0.751**				
IVA-CPT attention	−0.295*	−0.119	0.042	−0.182	0.159	0.417*	0.622**	0.564**	0.395**	0.351**			
IVA-CPT prudence	−0.395**	−0.391**	−0.090	−0.302*	0.307*	−0.183	0.422**	0.453**	0.258	0.008	0.782**		
Stroop dots	−0.654**	−0.715**	−0.083	−0.344**	0.089	0.349**	0.497**	0.532**	0.307*	−0.098	0.461**	0.589**	
Stroop ratio[a]	0.624**	0.181	0.405**	−0.201	−0.114	−0.263*	−0.225	−0.229	−0.098	−0.169	−0.567**	−0.695**	−0.405**

[a] Stroop ratio refers to the ratio of colour name trial/dot trial.
* $p < 0.05$.
** $p < 0.01$.

Table 5
Summary of hierarchical regression predicting depression and anxiety at 3 months follow-up

	Variable	*B*	*β*	*t*
Depression				
Step 1	Gender	0.125	0.005	0.06
	Age	−0.380	−0.372	−3.89
	Hemisphere	7.38	0.318	3.24
Step 2	FIM	0.184	0.186	2.01
Step 3	Verbal memory	−1.39	−0.282	−2.15
	Response control	0.45	0.228	1.90
	Cognitive speed	−1.06	−0.491	−4.53
	Ratio	8.66	0.748	6.78
Anxiety				
Step 1	Gender	2.60	0.150	0.255
	Age	−0.076	−0.097	0.471
	Hemisphere	6.348	0.359	0.012
Step 2	FIM	0.096	0.127	0.325
Step 3	Verbal memory	−0.554	−0.148	0.423
	Response control	0.018	0.118	0.484
	Cognitive speed	−1.09	−0.667	0.000
	Ratio	0.590	0.067	0.666

6. Discussion

This study examined prevalence of depression and anxiety, and their relationships to physical independence, demographic and injury characteristics, and cognitive abilities at 3 months post stroke. Differences in reported prevalence of previous studies are likely a result of differences in measurement procedures and a function of timing of the assessment (Hosking et al., 2000). Prevalence rates for moderate to severe depression and anxiety in the present sample were 22.8 and 21.1%, respectively. If one includes those with mild depression or anxiety, these increase to 45.6 and 38.6%, respectively. The reported rate of depression is similar to that reported at 3 months post stroke by Hosking et al. (2000). The prevalence of anxiety in this sample is consistent with existing data which states that 25% of people will suffer anxiety acutely post stroke, with 20% having anxiety at 3–6 months follow-up (Robinson, 1998). Co-morbid moderate anxiety and depression was found in 6.8% of participants, while an additional 5.5% had severe depression co-morbid with severe anxiety. None of the participants had co-morbid moderate anxiety with severe depression; or moderate depression with severe anxiety. Though this finding requires further investigation, it suggests that when depression and anxiety coexist, their perceived severities are congruent. While this may be reflective of severity of specific symptoms assessed, it is alternatively possible that this can be taken as a global indication of distress. A further alternative explanation is shared method variance. In this study, patient self-report was used to assess both depression and anxiety. In this case, we would expect to find some degree of association between depression and anxiety due to the use of the same data collection method. For example, a respondent who is generally miserable might rate his or her current anxiety level as high and at the same time indicate that he/she experiences a number of depressive symptoms.

While the rates of PSD and PSA reported are consistent with the literature, the issue of timing of measurement must be acknowledged. A time frame of 3 months was chosen for this study, as this is the time when the majority of stroke survivors will have been or are nearing discharge into the community. Ideally, measurement would have also occurred within 1 month post stroke. Unfortunately, it was not feasible to assess the majority of potential participants acutely due to the need to prioritise medical care. Multiple assessment beginning 1 month post stroke, using tests where alternative forms are available, would have allowed for a better picture of the natural history of mood, and physical and cognitive changes post stroke. By tracking mood and physical symptoms over time, it would be possible to differentiate affective and somatic underpinnings for endorsement of some BDI and BAI items; for example, by tracking if endorsement of items such as ‘wobbliness’ declines as physical versus emotional recovery occurs.

Having noted these issues, the above data do indicate a greater likelihood of experiencing depression or anxiety after stroke than in the general population, with one in five individuals experiencing either moderate or severe depression and/or anxiety. The potential impact of this on the ability to participate fully in rehabilitation is considerable. For example, a depressed individual may lack the motivation to push him/herself during physical therapy sessions, while an anxious individual may be too fearful of falling to attempt to walk without assistance despite assurances that he/she is ready to do so. Because depression and anxiety occur more frequently in the early stages post stroke, and because this is the same period in which rehabilitative resources are offered with the most intensity, the presence of emotional difficulties will have an even greater impact on recovery. As depression and anxiety are treatable, it is important that efforts at early identification and support be provided in order to assist this large proportion of clients in optimising rehabilitation.

As noted in the literature (e.g., Hochstenbach, Mulder, van Linden, Donders, & Schoonderwaldt, 1998), few studies provide any detail as to the types and extent of cognitive difficulties encountered across cognitive domains post stroke, nor has the course of cognitive recovery been made clear. Barker-Collo and Feigin (2006) state that no accurate data exists on frequency, relationship, and predictive ability of various long-term neuropsychological outcomes of stroke in general. In this study, difficulties of varying severity were encountered across cognitive measures, with only working memory, visual memory, and impulse control falling within 1 standard deviation of the normative mean. The verbal memory, attention, speed, and executive function scores were below average despite an average FIM rating, indicating that at 3-month post-stroke participants were viewed as relatively independent of functional tasks despite experiencing cognitive difficulties. Only cognitive speed, as measured by Stroop dots, was significantly related to FIM score; which is unsurprising given the FIM's weighting towards physical independence, and the more direct impact one would expect speed to have on physical independence.

That left hemisphere lesion was related to increased likelihood of depression and anxiety is consistent with the literature if one considers 3 months to be within the acute phase of recovery (Astrom, 1996; Astrom et al., 1993; Bhogal et al., 2004). Depression and anxiety were both related to reduced cognitive speed (Stroop dots) and high scores on the Stroop ratio (also indicative of reduced cognitive speed). While the methodology used does not allow for causal inferences and, as such, depressed mood may be either a result of or a contributor to cognitive decline in these areas, it is not surprising that an individual with significant slowing of cognitive abilities might experience subsequent difficulties in adjustment. Such slowing may, for example, lead to difficulties in tracking conversations and reduction in the ability to respond quickly when needed. It is alternatively possible that the reduced speed identified here is a function of depression itself; or that both are reflections of the physiological effects of stroke.

Given that left hemisphere lesions are also associated with cognitive deficits such as impaired verbal memory, it is then not surprising to find that deficits in these areas were also linked to increased likelihood of depression. It is, however, important to note that these cognitive factors independently contributed to mood functioning. This suggests that while hemisphere of lesion may be an important factor, it may be the cognitive deficits that result during the acute stages of left hemisphere damage that lead an individual to experience depression or anxiety. For example, it is possible that in the acute stages, experiencing severe problems with verbal retrieval could lead an individual to feel depressed due to perceived memory deficits. If this was indeed the case, then this calls into question conclusions that acute PSD is endogenous rather than reactive. To test this hypothesis, it would first be necessary to assess not only verbal memory but also participants' perceptions of the severity of their own deficits. Furthermore, it is important to note that different brain areas within the hemisphere may be differentially linked to depression and anxiety. Future studies with larger samples and access to CT scan results in a greater proportion of participants being advised to go beyond the simplistic left/right split used here and examine relationships of cognition to mood for groupings based on anterior/posterior and cortical/subcortical aspects of injury as well. By obtaining a larger sample, it may also be possible to separately examination each predictor variable of interest. Unfortunately, this was not possible in this study due to both the high correlations between the variables and the small sample size which limits the number of variables that can be examined.

7. Conclusion

The results of this study suggest that depression and anxiety are commonly experienced 3 months post stroke and that left hemisphere lesions may be linked to increased depression and anxiety scores. Cognitive factors such as speed of

processing and verbal memory were more related to mood disturbance than was level of physical independence. While further longitudinal study is required to determine the direction of causal relationships between mood and cognitive functioning, the findings thus far suggest that cognition and mood post stroke are linked and both should be addressed as part of the rehabilitative process.

References

Anderson, G., Vestergaard, K., Riis, J., & Lauritzen, L. (1994). Incidence of post-stroke depression during the first year in a large unselected stroke population determined using a valid standardized rating scale. *Acta Psychiatrica Scandinavica*, *90*, 190–195.

Astrom, M. (1996). Generalised anxiety disorder in stroke patients: A 3 year longitudinal study. *Stroke*, *27*, 270–275.

Astrom, M., Adolfsson, R., & Asplund, K. (1993). Major depression in stroke patients: A 3-year longitudinal study. *Stroke*, *24*, 976–982.

Barker-Collo, S., & Feigin, V. (2006). The impact of neuropsychological deficits on functional stroke outcomes. *Neuropsychology Review*, *16*(2), 53–64.

Beck, A. T., & Steer, R. A. (1993). *Manual for the Beck Anxiety Inventory*. San Antonio, TX: The Psychological Corporation.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck Depression Inventory* (2nd ed.). San Antonio, TX: The Psychological Corporation.

Bhogal, S. K., Teasell, R., Foley, N., & Speechley, M. (2004). Lesion location and poststroke depression: Systematic review of the methodological limitations in the literature. *Stroke*, *35*, 794–802.

Bolla-Wilson, K., Robinson, R. G., Starkstein, S. E., & Boston, J. (1989). Lateralization of dementia of depression in stroke patients. *American Journal of Psychiatry*, *146*(5), 627–634.

Burvill, P. W., Johnson, G. A., Jamrozik, K. D., & Anderson, C. S. (1995). Prevalence of depression after stroke: The Perth Community Stroke Study. *British Journal of Psychiatry*, *166*(3), 320–327.

Castillo, C. S., Schultz, S. K., & Robinson, R. (1995). Clinical correlates of early-onset and late-onset poststroke generalised anxiety. *American Journal of Psychiatry*, *152*(8), 1174–1181.

Castillo, C. S., Starkstein, S. E., Fedoroff, J. P., & Price, T. R. (1993). Generalized anxiety disorder after stroke. *Journal of Nervous & Mental Disease*, *181*(2), 100–106.

Chemerinski, E., & Robinson, R. G. (2000). The neuropsychiatry of stroke. *Psychosomatics*, *41*(1), 5–14.

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

Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (2000). *CVLT-II. California Verbal Learning Test. Second edition manual*. USA: The Psychological Corporation.

Dennis, M., O'Rourke, S., Lewis, S., Sharpe, M., & Warlow, C. (2000). Emotional outcomes after stroke: Factors associated with poor outcome. *Journal of Neurology, Neurosurgery, & Psychiatry*, *68*, 47–52.

Downhill, J. E., & Robinson, R. G. (1994). Longitudinal assessment of depression and cognitive impairment following stroke. *Journal of Nervous & Mental Disease*, *182*(8), 425–431.

Duncan, P. W., Reker, D. M., Horner, R. D., Samsa, G. P., Hoenig, H., & LaClair, B. J. (2002). Performance of a mail-administered version of a stroke-specific outcome measure, the Stroke Impact Scale. *Clinical Rehabilitation*, *16*(5), 493–505.

Evans, G. L., & Whitney, F. W. (1998). The role of medical conditions and depression in the functional outcome of stroke survivors: A review and pilot study. *Topics in Stroke Rehabilitation*, *5*(*3*), 30–50.

Fedoroff, J., Starkstein, S., Parikh, R., Price, T., & Robinson, R. (1991). Are depressive symptoms nonspecific in patients with acute stroke? *American Journal of Psychiatry*, *148*(9), 1172–1176.

Fruehwald, S., Loffler, H., Eher, R., Saletu, B., & Baumhackl, U. (2001). Relationship between depression, anxiety and quality of life: A study of stroke patients compared to chronic low back pain and myocardial ischemia patients. *Psychopathology*, *34*(1), 50–56.

Giaquinto, S., Buzzelli, S., Francesco, L. D., Lottarini, A., Montenero, P., & Tonin, P. (1999). On the prognosis of outcome after stroke. *Acta Neurologica Scandinavica*, *100*(3), 202–208.

Granger, C., Divan, N., & Fiedler, R. (1995). Functional Assessment Scales. A study of persons with traumatic brain injury. *American Journal of Physical Medicine and Rehabilitation*, *72*, 107–113.

Granger, C., Hamilton, B., Linacre, J., Heinemann, A., & Wright, B. (1993). Performance profiles of the functional independence measure. *American Journal of Physical Medicine and Rehabilitation*, *72*, 84–89.

Granger, C., Hamilton, B., & Sherwin, F. (1986). *Guide for use of the uniform data set for medical rehabilitation*. Buffalo: Buffalo General Hospital.

Herrmann, N. (1999). Post-stroke depression. *Primary Care Psychiatry*, *5*(3), 81–87.

Herrmann, N., Black, S., Lawrence, J., Szekely, C., & Szalai, J. (1998). The Sunnybrook Stroke Study: A prospective study of depressive symptoms and functional outcome. *Stroke*, *29*(3), 618–624.

Hinkle, J. (2000). The use of the FIM instrument in the poststroke population. *Topics in Stroke Rehabilitation*, *7*(3), 47–66.

Hobart, J., Lamping, D., Freeman, J., Langdon, D., McLellan, D., & Greenwood, R. (2001). Evidence-based measurement. Which disability scale for neurologic rehabilitation. *Neurology*, *57*, 639–644.

Hochstenbach, J., Mulder, T., van Linden, J., Donders, R., & Schoonderwaldt, H. (1998). Cognitive decline following stroke: A comprehensive study of cognitive decline following stroke. *Journal of Clinical & Experimental Neuropsychology*, *20*(4), 503–517.

Hosking, S., Marsh, N., & Friedman, P. (2000). Depression at 3-months poststroke in the elderly: Predictors and indicators of prevalence. *Aging Neuropsychology & Cognition*, *7*(4), 205–216.

House, A., & Dennis, M. (1990). The relationship between intellectual impairment and mood disorder in the first year after stroke. *Psychological Medicine*, *20*, 805–814.

House, A., Dennis, M., Mogridge, L., Warlow, C., Hawton, K., & Jones, L. (1991). Mood disorders in the year after first stroke. *British Journal of Psychiatry*, *158*, 83–92.

Jorge, R., Robinson, R., Starkstein, S., & Arndt, S. (1993). Depression and anxiety following traumatic brain injury. *Journal of Neuropsychiatry & Clinical Neurosciences*, *5*(4), 369–374.

Kidd, D., Stewart, G., & Landry, A. (1995). The functional independence measure: A comparative validity and reliability study. *Disability & Rehabilitation*, *17*, 10–14.

Kotila, M., Numminen, H., Waltimo, O., & Kaste, M. (1999). Post-stroke depression and functional recovery in a population-based stroke register: The Finnstroke Study. *European Journal of Neurology*, *6*(3), 309–312.

Lipsey, J. R. (1983). Mood change following bilateral hemisphere brain injury. *British Journal of Psychiatry*, *143*, 266–273.

Lyketsos, C., Treisman, G., Lipsey, J., Morris, P., & Robinson, R. (1998). Does stroke cause depression? *Journal of Neuropsychiatry & Clinical Neurosciences*, *10*(1), 103–107.

McDowell, I., & Newell, C. (1996). *Measuring Health. A guide to rating scales and questionnaires* (2nd ed.). New York: Oxford University Press.

Morris, P., Robinson, R., Andrzejewski, P., & Samuels, J. (1993). Association of depression with 10-year poststroke mortality. *American Journal of Psychiatry*, *150*(1), 124–129.

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

Morris, P., Shields, R., Hopwood, M., & Robinson, R. (1994). Are there two depressive syndromes after stroke? *Journal of Nervous & Mental Disease*, *182*(4), 230–234.

Morrison, V., Johnston, M., & Walter, R. M. (2000). Predictors of distress following an acute stroke: Disability, control cognitions, and satisfaction with care. *Psychology and Health*, *15*, 395–407.

Murata, Y., Kimura, M., & Robinson, R. G. (2000). Does cognitive impairment cause poststroke depression? *American Journal of Geriatric Psychiatry*, *8*(4), 310–317.

Nys, G. M. S. (2005). *The neuropsychology of acute stroke: Characterisation and prognostic implications*. Utrecht, Holland: Utrecht University.

Paradiso, S., & Robinson, R. G. (1998). Gender differences in poststroke depression. *Journal of Neuropsychiatry & Clinical Neurosciences*, *10*(1), 41–47.

Parikh, R., Lipsey, J., Robinson, R., & Price, T. (1988). A two year longitudinal study of post stroke mood disorders: Prognostic factors related to one and two year outcome. *International Journal of Psychiatry in Medicine*, *18*(1), 45–56.

Ramasubbu, R., Robinson, R., Flint, A., Kosier, T., & Price, T. (1998). Functional impairment associated with acute poststroke depression: The Stroke Data Bank study. *Journal of Neuropsychiatry & Clinical Neurosciences*, *10*(1), 26–33.

Robinson, R. (1983). Mood changes in stroke patients: Relationship to lesion location. *Comprehensive Psychiatry*, *24*(6), 555–566.

Robinson, R. (1985). A two-year longitudinal study of poststroke mood disorders: In-hospital prognostic factors associated with six-month outcome. *Journal of Nervous & Mental Disease*, *173*(4), 221–226.

Robinson, R. (1987). Depression and stroke. *Psychiatric Annals*, *17*(11), 731–740.

Robinson, R. (1997). Neuropsychiatric consequences of stroke. *Annual Review of Medicine*, *48*, 217–229.

Robinson, R. (1998). *The clinical neuropsychiatry of stroke*. Cambridge: Cambridge University Press.

Robinson, R., Bolla-Wilson, K., Kaplan, E., & Lipsey, J. (1986). Depression influences intellectual impairment in stroke patients. *British Journal of Psychiatry*, *148*, 541–547.

Sandford, J., & Turner, A. (2000). *Manual for the Integrated Visual and Auditory Continuous Performance Test*. Braintrain: Richmond, VA.

Schubert, D., Taylor, C., Lee, S., Mentari, A., & Tamaklo, W. (1992). Physical consequences of depression in the stroke patient. *General Hospital Psychiatry*, *14*(1), 69–76.

Schwartz, J. A., Speed, N. M., Brunberg, J. A., Brewer, T. L., Brown, M., & Greden, J. F. (1993). Depression in stroke rehabilitation. *Biological Psychiatry*, *33*(10), 694–699.

Schultz, S. K., Castillo, C. S., Kosier, J. T., & Robinson, R. G. (1997). Generalized anxiety and depression: Assessment over 2 years after stroke. *American Journal of Geriatric Psychiatry*, *5*(3), 229–237.

Shimoda, K., & Robinson, R. G. (1998). The relationship between social impairment and recovery from stroke. *Psychiatry*, *61*, 101–110.

Spreen, O., & Strauss, E. (1989). *A compendium of neuropsychological tests* (2nd ed.). New York: Oxford University Press.

Starkstein, S., Bryer, J., Berthier, M., & Cohen, B. (1991). Depression after stroke: The importance of cerebral hemisphere asymmetries. *Journal of Neuropsychiatry & Clinical Neurosciences*, *3*(3), 276–285.

Starkstein, S., Cohen, B., Fedoroff, J., Parikh, R., Price, T., & Robinson, R. (1990). Relationship between anxiety and depressive disorders in patients with cerebrovascular injury. *Archives of General Psychiatry*, *47*, 246–251.

Starkstein, S., & Robinson, R. (1991a). Dementia of depression in Parkinson's disease and stroke. *Journal of Nervous & Mental Disease*, *179*(10), 593–601.

Starkstein, S., & Robinson, R. (1991b). The role of the frontal lobes in affective disorder following stroke. In H. S. Levin & H. M. Eisenberg (Eds.), *Frontal lobe function and dysfunction* (pp. 288–303). New York, NY, USA: Oxford University Press.

Starkstein, S., Robinson, R., Berthier, M., Parikh, R., & Price, T. (1988). Differential mood changes following basal ganglia versus thalamic lesions. *Archives of Neurology*, *45*, 725–730.

Starkstein, S., Robinson, R., & Price, T. (1988). Comparison of patients with and without poststroke major depression matched for size and location of lesion. *Archives of General Psychiatry*, *45*(3), 247–252.

Tabachnick, B. G., & Fidell, L. S. (1989). *Using multivariate statistics* (2nd ed.). New York: HarperCollins.
van de Weg, F. B., Kuik, D. J., & Lankhorst, G. J. (1999). Post-stroke depression and functional outcome: A cohort study investigating the influence of depression on functional recovery from stroke. *Clinical Rehabilitation*, *13*(3), 268–272.
Wechsler, D. (1987). *Wechsler Memory Scale–Revised manual*. San Antonio, TX: The Psychological Corporation.
Wechsler, D. (1997). *Wechsler Memory Scale–Third edition manual*. San Antonio, TX: The Psychological Corporation.
Whyte, E. M., & Mulsant, B. H. (2002). Post stroke depression: Epidemiology, pathophysiology, and biological treatment. *Biological Psychiatry*, *52*, 253–264.