

Depression in Right Hemisphere Disorder

Research Problem and Rationale

Estimates of the prevalence of post-stroke depression range from 25-79% (Kneebone & Dunmore, 2000; Thomas & Lincoln, 2006). Negative outcomes associated with post-stroke depression include increased use of health services (Cushman, 1988), longer hospitalization (Cushman, 1988), limited recovery of physical and cognitive functions (Morris, Raphael, & Robinson, 1992), decreased quality of life (Jaracz, Jaracz, Kozubski, & Rybakowski, 2002), and increased mortality post-stroke (House, Knapp, Bamford, & Vail, 2001). Growing consensus among speech pathologists that treatment should be relevant and useful to patients and their families might thus spur clinicians to consider decreased depression a desirable treatment outcome.

Current means by which to reduce or eliminate post-stroke depression are limited, however, by inadequate knowledge of its cause(s) (Thomas & Lincoln, 2006). Some researchers (e.g., Robinson, Kubos, Starr, Rao, & Price, 1984) propose a biological etiology and examine variables that are directly related to the brain lesion(s), for example, location of lesion or severity of disability (Sharpe et al., 1994; Thomas & Lincoln, 2006). Other researchers (e.g., Gainotti, Azzoni, & Marra, 1999) instead propose a psychosocial etiology, and examine variables unrelated or indirectly related to the brain lesion(s), for example, perceived lack of control or feelings of hopelessness in response to disability resulting from stroke (Sharpe et al., 1994; Thomas & Lincoln, 2006). Because adults with RHD may have limited insight into both physical and cognitive disability (Myers, 1999), psychosocial reaction to lost motor or cognitive-communicative function may be less likely than demographic or biological factors to cause depression in this population. However, the cause(s) of depression in adults with RHD, and thus the means by which it may best be addressed by speech-language pathologists, have not been determined.

In this pilot study, our aims were to determine whether depression in adults with right hemisphere disorder differs significantly from depression in normal controls, and, if so, to identify possible, treatable variables associated with increased depression in adults with RHD.

Methods of Data Acquisition

Sixteen adults with RHD and 16 normal controls completed the protocol. Participants with RHD had a history of one or more strokes; brain damage confined to the right hemisphere, as confirmed by neuroradiological data; no history of other disease that would affect communicative ability; and, a diagnosis of RHD, as determined by the principal investigator, using the presence of one or more of the following signs and/or symptoms resulting from acquired damage to the right hemisphere: attentional deficits, neglect, visuoperceptual deficits, cognitive and communicative deficits, and/or affective and emotional deficits (Myers, 1999). Normal controls had no history of brain injury or other disease that would affect communicative ability. Absence of brain damage in normal controls was based on history by self-report.

To compare presence and severity of depression between groups, all participants were administered the Self-Rating Depression Scale (Zung, 1965). To identify possible, treatable variables associated with depression within groups, the following additional data were collected:

Demographic variables (all participants): age, gender, education, marital status, and work status

Biological variables (participants with RHD): months post-stroke; physical disability (Modified Rankin Scale, MRS, van Swieten, Koudstaal, Visser, Schouten, & van Gijn, 1988); language impairment (Porch Index of Communicative Ability, PICA, Porch, 1981); functional communication (Communication Activities in Daily Living, 2nd Edition, CADL-2, Holland, Frattali, & Fromm, 1999); and neglect (Behavioural Inattention Test, BIT, Wilson, Cockburn, & Halligan, 1987)

Psychosocial variables (all participants): loneliness (UCLA Loneliness Scale, Version 2, Russell, Peplau, & Cutrona, 1980); social support (Inventory of Socially Supportive Behaviors, ISSB, Barrera, Sandler, & Ramsey, 1981); stress (Recent Life Changes Questionnaire, Rahe, 1975); and desired control over everyday events (Desired Control Scale, Short Form, Reid & Zeigler, 1981)

To determine differences in continuous variables between groups, independent samples *t*-tests were used. To determine differences in discrete variables between groups, chi-square tests were used. To examine relationships between continuous demographic, biological, and psychosocial variables and depression within groups, bivariate correlational analyses were performed. To examine relationships between discrete demographic, biological, and psychosocial variables and depression within groups, analyses of variance and *post hoc* testing were used. For this pilot study, an alpha level of .05 was used to establish statistical significance.

Results and Analysis

Table 1 shows demographic information for all participants. Groups differed only in age (on average, participants with RHD were significantly older than normal controls) and current work status (significantly more normal controls than participants with RHD were currently employed). Table 2 shows stroke-related biological variables for participants with RHD. Participants were, on average, two years post-stroke, with residual physical disability that exceeded cognitive or communicative disability. Table 3 shows psychosocial variables for all participants. There were no significant differences in loneliness, social support, recently experienced stress, or desired control over everyday events between groups.

Table 4 shows that participants with RHD scored significantly higher on the Self-Rating Depression Scale Index than normal controls and that significantly more participants with RHD than normal controls were depressed (i.e., scored at least 50 on the SDS Index). Almost two thirds of participants with RHD were depressed, and more than a third of participants with RHD had at least moderately severe depression. Instead, less than one fifth of normal controls were depressed, and all normal controls who were depressed had only minimal to mild depression.

Table 5 shows that, within participants with RHD, the psychosocial variables loneliness and social support were significantly related with depression. No demographic or biological variables were significantly related with depression in this sample of adults with RHD.

Table 6 shows that, within normal controls, the psychosocial variable loneliness was significantly related with depression. No demographic or biological variables were significantly related with depression in this sample of normal controls.

Conclusions

Adults with RHD were significantly more depressed than normal controls. In both groups, depression was significantly related with the psychosocial variable loneliness. In adults with RHD, depression was also significantly related with the psychosocial variable social support. No demographic or biological variables were associated with increased depression in either group.

Clinical Implications

Adults with RHD appear at risk for depression, regardless of severity of cognitive or communicative disability. Treatment of depression with psychotherapy and/or pharmacotherapy can greatly improve rehabilitation outcomes (Bates et al., 2005). Thus, routine screening for depression in adults with RHD is recommended.

Traditional treatment of RHD consists of task- or process-oriented therapy that aims to reduce the severity of deficits that are directly related to right-hemisphere brain injury (e.g., attention, neglect, and prosody) (Myers, 1999). However, in our sample of adults with RHD, depression was not associated with biological variables that are directly related to brain lesion(s), but was associated with psychosocial variables that may represent a response to physical or cognitive disability resulting from stroke. Thus, further study, designed to determine whether treatment that aims instead to reduce loneliness and increase support also reduces depression in adults with RHD, is warranted.

References

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Table 1

Demographic variables: All participants

Variable				
Age (Years)*	Mean	Range	<i>SD</i>	Difference
Participants with RHD	60.94	53-71	5.67	$t(30) = 2.78, p = .009$
Normal Controls	54.38	46-76	7.54	
Education (Years)	Mean	Range	<i>SD</i>	
Participants with RHD	13.81	10-18	2.59	$t(30) = 1.41, p = .168$
Normal Controls	15.03	12-20	2.28	
Gender	% Female			
Participants with RHD	38			$\chi^2 = 2.00, p = .157$
Normal Controls	38			
Marital Status	% Married			
Participants with RHD	44			$\chi^2 = .13, p = .724$
Normal Controls	50			
Work Status*	% Employed			
Participants with RHD	13			$\chi^2 = 18.94, p = .001$
Normal Controls	81			

*Differences in age and work status between groups are statistically significant.

Table 2

Biological variables: Participants with RHD

Variable	Mean	Range	<i>SD</i>
Months Post Stroke	23.67	2-60	23.64
Physical Disability (MRS, 0-5 scale)	2.63	0-4	1.15
Language Impairment (PICA, 1-16 scale)	14.03	13.02-14.78	.55
Functional Communication (CADL-2, 0-100 scale)	93.50	75-100	6.34
Neglect (BIT, 0-1 scale)	.91	.59-1.00	.10

Table 3

Psychosocial variables: All participants

Variable	Mean	Range	SD	Difference
Loneliness (UCLA Loneliness Scale, 20-80 scale)				
Participants with RHD	38.31	20-63	11.97	$t(30) = .26, p = .799$
Normal Controls	37.31	25-57	10.02	
Social Support (ISSB, 40-200 scale)				
Participants with RHD	83.94	47-138	23.47	$t(30) = 1.07, p = .292$
Normal Controls	76.50	60-105	14.81	
Stress (RLCQ, 0-3545 scale)				
Participants with RHD	372.56	24-893	287.59	$t(30) = 1.67, p = .106$
Normal Controls	222.94	0-695	215.19	
Desired Control (DCS, 16-400 scale)				
Participants with RHD	218.19	93-248	36.35	$t(30) = 1.28, p = .211$
Normal Controls	231.88	199-276	22.72	

Table 4

Depression: All participants

Variable	Mean	Range	SD	Difference
SDS Index (25-100 scale)*				
Participants with RHD	51.94	31-79	14.18	$t(30) = 2.84, p = .008$
Normal Controls	39.56	25-55	10.09	
SDS Index by Category*				$\chi^2 = 22.50, p = .000$
No Depression (Below 50)	%			
Participants with RHD	37.50			
Normal Controls	81.30			
Minimal to Mild Depression (50-59)	%			
Participants with RHD	25.00			
Normal Controls	18.80			
Moderate to Marked Depression (60-69)	%			
Participants with RHD	31.30			
Normal Controls	0			
Severe to Extreme Depression (70 and over)	%			
Participants with RHD	6.30			
Normal Controls	0			

*Difference in depression between groups is statistically significant.

Table 5

Relationships among participant variables and depression: Participants with RHD

Variable	Relationship with SDS Index
Demographic	
Age	$r = -.19, p = .476$
Education	$r = -.20, p = .470$
Gender	$F(1,15) = .38, p = .546$
Marital Status	$F(4,15) = .06, p = .993$
Work Status	$F(3,15) = 1.29, p = .324$
Biological	
Months Post Stroke	$r = .07, p = .799$
Physical Disability	$r = .20, p = .450$
Language Impairment	$r = .14, p = .617$
Functional Communication	$r = .13, p = .627$
Neglect	$r = .34, p = .198$
Psychosocial	
Loneliness*	$r = .56, p = .023$
Social Support*	$r = -.56, p = .024$
Stress	$r = -.09, p = .745$
Desired Control	$r = -.45, p = .081$

*Correlations between loneliness and depression and between social support and depression within participants with RHD are statistically significant.

Table 6

Relationships among participant variables and depression: Normal controls

Variable	Relationship with SDS Index
Demographic	
Age	$r = -.26, p = .333$
Education	$r = -.28, p = .291$
Gender	$F(1,15) = .13, p = .720$
Marital Status	$F(3,15) = .53, p = .671$
Work Status	$F(3,15) = 2.52, p = .107$
Psychosocial	
Loneliness*	$r = .70, p = .003$
Social Support	$r = -.40, p = .130$
Stress	$r = .33, p = .210$
Desired Control	$r = -.01, p = .997$

*Correlation between loneliness and depression within normal controls is statistically significant.