Risk Factors for Depression in Aphasia: Clinical Implications

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 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). Speech pathologists agree that treatment should be relevant and useful to patients and their families; thus, clinicians might consider prevention or reduction of depression a desirable treatment outcome.

Current means by which to address post-stroke depression are limited, however, by inadequate knowledge of its cause(s) (Sharpe et al., 1994; Spencer, Tompkins, Schulz, & Rau, 1995). 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, time post-onset of stroke or severity of disability. Other researchers (e.g., Gainotti, Azzoni, & Marra, 1999) propose a psychosocial etiology and examine variables indirectly related to the brain lesion(s), for example, perceived lack of control or feelings of isolation in response to disability resulting from stroke. Still others (e.g., Paradiso & Robinson, 1998) examine variables unrelated to stroke, for example, demographic characteristics that might predispose an individual to depressive disorder. However, the cause(s) of depression in adults with aphasia, and thus the means by which it may best be addressed by speech-language pathologists, have not been determined.

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

Methods of Data Acquisition

Twenty-six adults with aphasia and 21 normal controls completed the protocol. Participants with aphasia had a history of one or more strokes; brain damage confined to the left hemisphere, as confirmed by neuroradiological data; no history of other disease that would affect communicative ability; and, a diagnosis of aphasia, as determined by the principal investigator, using an operational definition (Rosenbek, LaPointe, & Wertz, 1989). Normal controls, by self-report, had no history of brain injury or other disease that would affect communicative ability.

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

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

Biological variables (participants with aphasia): months post-stroke; language impairment (Porch Index of Communicative Ability, PICA, Porch, 1981); and, functional communication (Communication Activities in Daily Living, 2nd Edition, CADL-2, Holland, Frattali, & Fromm, 1999)

Psychosocial variables (all participants): loneliness (Revised UCLA Loneliness Scale, RULS, Russell, Peplau, & Cutrona, 1980); social support (Inventory of Socially Supportive Behaviors, ISSB, Barrera, Sandler, & Ramsey, 1981); desired control over

everyday events (Desired Control Scale, Short Form, DCS-SF, Reid & Zeigler, 1981); and, recent life events experienced (Recent Life Changes Questionnaire, RLCQ, Rahe, 1975)

To determine differences in continuous variables between groups, to examine relationships between discrete variables and severity of depression within groups, and to examine relationships between continuous variables and presence of depression within groups, independent samples *t*-tests were used. To determine differences in discrete variables between groups and to examine relationships between discrete variables and presence of depression within groups, chi-square tests were used. To examine relationships between continuous variables and severity of depression within groups, bivariate correlational analyses were performed. 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. Significantly more normal controls were female, and significantly more normal controls were employed. Table 2 shows stroke-related biological variables for participants with aphasia. Participants ranged from early to late post-onset of stroke and from moderately severe to mild communicative disability. Table 3 shows psychosocial variables for all participants. Participants with aphasia reported greater loneliness than normal controls.

Table 4 shows that participants with aphasia did not differ significantly from normal controls in presence or severity of depression. However, Tables 5 and 6 show that possible causes of depression differed between groups.

For normal controls, the demographic variable of work status and the psychosocial variable of loneliness were significant factors in severity of depressive symptomatology (Table 5). The psychosocial variable of loneliness was also a significant factor in the presence of clinically significant depression, as was the demographic variable of gender (Table 6).

For participants with aphasia, the biological variables months post-stroke and severity of language impairment and the psychosocial variable of loneliness were significant factors in severity of depressive symptomatology (Table 5). The biological variables months post-stroke and severity of language impairment and the psychosocial variable loneliness were also significant factors in the presence of clinically significant depression, as was the psychosocial variable desired control over everyday events (Table 6). No demographic variables were significantly related with depression in our sample of adults with aphasia.

Conclusions

In this study, adults with aphasia did not differ from normal controls in presence or severity of depression, but instead differed in its possible causes. In our normal controls, demographic variables – being male and being employed – were significant factors in depression. Because our samples differed significantly in gender and work status, further study is need to conclude a differential influence of these variables on depression in adults with aphasia. In our adults with aphasia, biological variables – longer time post-onset and greater severity of language impairment – were significant factors in depression. In both groups, the psychosocial variable frequency of social support was not significant. However, in both groups, loneliness was a significant factor in depression. And, our adults with aphasia reported greater loneliness than our normal controls. Finally, in both groups, the psychosocial variable number of recent life events

experienced was not significant. However, in our adults with aphasia, perceived lack of control over everyday events was a significant factor in the presence of clinically significant depression.

Clinical Implications

One third of adults with aphasia in our sample appear at risk for clinically significant depression. 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 aphasia is recommended. Because our data indicate that presence and severity of depression in aphasia may increase with time, early intervention seems prudent.

Validation of our results with samples large enough to permit causal modeling techniques may advocate a broader focus for aphasia therapy. Traditional therapy targets external, behavioral components of communication by improving patients' language skills. In our participants with aphasia, depression was associated with severity of language impairment. Thus, traditional therapy may also address depression. However, depression was also associated with loneliness and perceived lack of control over everyday events, which may represent an emotional response to communicative disability resulting from stroke. Thus, nontraditional therapy directed at internal, psychological constructs – perhaps at improving patients' feelings of fitting with and being a valued part of a group or environment (Hagerty, Lynch-Sauer, Patusky, Bouwsema, & Collier, 1992) or at increasing their sense of control (Renwick, Brown, & Raphael, 2000) – may also be warranted.

References

Barrera, M., Sandler, I. N., & Ramsey, T. B. (1981). Preliminary development of a scale of social support: Studies on college students. *American Journal of Community Psychology, 9,* 435-447.

Bates, B., Choi, J. Y., Duncan, P. W., Glasberg, J. J., Graham, G. D., Katz, R. C., Lamberty, K., Reker, D., & Zorowitz, R. (2005). Veterans Affairs/Department of Defense clinical practice guideline for the management of adult stroke rehabilitation care: Executive summary. *Stroke, 36,* 2049-2056.

Cushman, L. (1988). Secondary neuropsychiatric complications in stroke: Implications for acute care. *Archives of Physical Medicine and Rehabilitation, 69,* 877-879.

Gainotti, G., Azzoni, A., & Marra, C. (1999). Frequency, phenomenology and anatomical-clinical correlates of major post-stroke depression. *British Journal of Psychiatry, 175,* 163-167.

Hagerty, B. M. K., Lynch-Sauer, J., Patusky, K. L., Bouwsema, M., & Collier, P. (1992). Sense of belonging: A vital mental health concept. *Archives of Psychiatric Nursing, 6,* 172-177.

Holland, A. L., Frattali, C., & Fromm, D. (1999). *Communication activities of daily living – Second edition.* Austin: Pro-ed.

House, A., Knapp, B., Bamford, J., & Vail, A. (2001). Mortality at 12 and 24 months after stroke may be associated with depressive symptoms at 1 month. *Stroke, 32,* 696-701.

Jaracz, K., Jaracz, J., Kozubski, W., & Rybakowski, J. K. (2002). Post-stroke quality of life and depression. *Acta Neuropsychiatrica Scandinavica, 14,* 219-225.

Kneebone, I. I., & Dunmore, E. (2000). Psychological management of post-stroke depression. *British Journal of Clinical Psychology, 39,* 53-65.

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

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

Porch, B. E. (1981). *Porch index of communicative ability – 3rd edition.* Palo Alto, CA: Consulting Psychologists Press.

Rahe, R. H. (1975). Epidemiological studies of life change and illness. *International Journal of Psychiatric Medicine, 6,* 133-146.

Reid, D. W., & Zeigler, M. (1981). The Desired Control Measure and adjustment among the elderly. In H. M. Lefcourt (Ed.), *Research with the locus of control construct* (Vol. 1). New York: Academic Press, pp. 127-157.

Renwick, R., Brown, I., & Raphael, D. (2000). Person-centered quality of life contributions from Canada to an international understanding. In R. Schalock and K. Keith (Eds.), *Cross-cultural perspectives on quality of life* (pp. 5-21). Washington, DC: American Association on Mental Retardation.

Robinson, R. G., Kubos, K. L., Starr, L. B., Rao, K., & Price, T. R. (1984). Mood disorders in stroke patients. Importance of location of lesion. *Brain, 107,* 81-93.

Rosenbek, J. C., LaPointe, L. L., & Wertz, R. T. (1989). Aphasia: A clinical approach. Austin, TX: Pro-ed, p.53.

Russell, D., Peplau, L. A., & Cutrona, C. E. (1980). The revised UCLA loneliness scale: Concurrent and discriminant validity evidence. *Journal of Personality and Social Psychology, 39,* 472-480.

Sharpe, M., Hawton, K., Seagroatt, V., Bamford, J., House, A., Molyneux, A., Sandercock, P., & Warlow, C. (1994). Depressive disorders in long-term survivors of stroke: Associations with demographic and social factors, functional status, and brain lesion volume. *British Journal of Psychiatry, 164,* 380-386.

Spencer, K. A., Tompkins, C. A., Schulz, R., & Rau, M. T. (1995). The psychosocial outcomes of stroke: A longitudinal study of depression risk. *Clinical Aphasiology, 23,* 9-23.

Thomas, S. A., & Lincoln, N. B. (2006). Factors relating to depression after stroke. *British Journal of Clinical Psychology, 45,* 46-91.

Zung, W. W. K. (1965). A self-rating depression scale. *Archives of General Psychiatry, 12,* 63-70.

Table 1

Demographic variables: All participants

Variable				
Age (Years)	Mean	Range	*SD*	Difference
Participants with aphasia	56.31	41-77	9.69	$t(45) = 1.42$, $p = .164$
Normal controls	52.67	42-76	7.46	
Education (Years)	Mean	Range	*SD*	
Participants with aphasia	14.46	10-18	2.20	$t(45) = .58$, $p = .564$
Normal controls	14.83	12-20	2.16	
Gender*	% Female			
Participants with aphasia	8			$\chi^2 = 11.60$, $p = .001$
Normal controls	52			
Marital Status	% Married			
Participants with aphasia	54			$\chi^2 = .01$, $p = .920$
Normal controls	52			
Work Status*	% Employed			
Participants with aphasia	4			$\chi^2 = 32.33$, $p = .000$
Normal controls	86			

*Differences between groups are statistically significant.

Table 2

Biological variables: Participants with aphasia

Variable	Mean	Range	*SD*
Months Post Stroke	38.08	1-120	35.52
Language Impairment (PICA, 1-16 scale)	11.88	8.78-14.25	1.65
Functional Communication (CADL-2, 0-100 scale)	83.85	60-96	10.95

Table 3

Psychosocial variables: All participants

Variable	Mean	Range	*SD*	Difference
Loneliness* (RULS, 20-80 scale)				
Participants with aphasia	45.27	23-67	13.57	$t(45) = 2.34$, $p = .024$
Normal controls	37.24	25-57	8.88	
Social Support (ISSB, 40-200 scale)				
Participants with aphasia	88.19	41-141	24.80	$t(45) = 1.87$, $p = .069$
Normal controls	76.52	59-112	15.92	
Desired Control (DCS-SF, 16-400 scale)				
Participants with aphasia	213.69	137-269	34.08	$t(45) = 1.29$, $p = .204$
Normal controls	225.05	188-276	23.95	
Recent Life Events (RLCQ, 0-3545 scale)				
Participants with aphasia	310.15	24-721	192.80	$t(45) = 1.86$, $p = .069$
Normal controls	205.38	0-695	191.06	

* Difference between groups is statistically significant.

Table 4

Depression: All participants

Variable				
Severity of Depressive Symptomatology (SDS Index, 25-100 scale)	Mean	Range	*SD*	Difference
Participants with aphasia	44.08	28-69	12.89	$t(45) = 1.40$, $p = .169$
Normal controls	39.43	25-55	9.02	
Presence of Clinically Significant Depression				$\chi^2 = 1.76$, $p = .185$
No Depression (SDS Index below 50)	%			
Participants with aphasia	69			
Normal controls	86			
Presence of Depression (SDS Index above 50)	%			
Participants with aphasia	31			
Normal controls	14			

Table 5

Risk factors in severity of depressive symptomatology

Variable	Relationship with SDS Index
Demographic	
Age	
Participants with aphasia	$r = .19$, $p = .344$
Normal controls	$r = -.17$, $p = .453$
Education	
Participants with aphasia	$r = .26$, $p = .202$
Normal controls	$r = -.18$, $p = .427$
Gender	
Participants with aphasia	$t(24) = .35$, $p = .733$
Normal controls	$t(19) = .41$, $p = .684$
Marital Status	
Participants with aphasia	$t(24) = .48$, $p = .637$
Normal controls	$t(19) = 1.21$, $p = .241$
Work Status	
Participants with aphasia	$t(24) = .71$, $p = .484$
Normal controls*	$t(19) = 2.73$, $p = .013$
Biological (participants with aphasia)	
Months Post Stroke*	$r = .41$, $p = .040$
Language Impairment*	$r = .44$, $p = .024$
Functional Communication	$r = .27$, $p = .186$
Psychosocial	
Loneliness	
Participants with aphasia*	$r = .73$, $p = .000$
Normal controls*	$r = .67$, $p = .001$
Social Support	
Participants with aphasia	$r = .02$, $p = .932$
Normal controls	$r = -.42$, $p = .061$
Desired Control	
Participants with aphasia	$r = -.32$, $p = .113$
Normal controls	$r = -.01$, $p = .993$
Recent Life Events	
Participants with aphasia	$r = .12$, $p = .556$
Normal controls	$r = .31$, $p = .168$

*Factor is statistically significant.

Table 6

Risk factors in presence of clinically significant depression

Variable	Presence or Absence of Depression
Demographic	
Age	
Participants with aphasia	$t(24) = 1.86$, $p = .075$
Normal controls	$t(19) = .74$, $p = .466$
Education	
Participants with aphasia	$t(24) = 1.45$, $p = .161$
Normal controls	$t(19) = .86$, $p = .401$
Gender	
Participants with aphasia	$\chi^2 = .96$, $p = .326$
Normal controls*	$\chi^2 = 3.85$, $p = .050$
Marital Status	
Participants with aphasia	$\chi^2 = .35$, $p = .555$
Normal controls	$\chi^2 = .51$, $p = .476$
Work Status	
Participants with aphasia	$\chi^2 = .46$, $p = .497$
Normal controls	$\chi^2 = .58$, $p = .445$
Biological (participants with aphasia)	
Months Post Stroke*	$t(23) = 2.69$, $p = .013$
Language Impairment*	$t(24) = 2.42$, $p = .024$
Functional Communication	$t(24) = 1.10$, $p = .282$
Psychosocial	
Loneliness	
Participants with aphasia*	$t(24) = 4.41$, $p = .000$
Normal controls*	$t(19) = 3.56$, $p = .002$
Social Support	
Participants with aphasia	$t(24) = .08$, $p = .941$
Normal controls	$t(19) = 1.39$, $p = .182$
Desired Control	
Participants with aphasia*	$t(24) = 2.27$, $p = .033$
Normal controls	$t(19) = .07$, $p = .943$
Recent Life Changes	
Participants with aphasia	$t(24) = .50$, $p = .623$
Normal controls	$t(19) = .79$, $p = .442$

*Differences in depressed and non-depressed subgroups are statistically significant.