While much research effort has focused on the organic link between neurologic lesion and depressive symptoms (Spalletta et al., 2006), there is also an acknowledgement of the importance of psychosocial factors in depression etiology. This "secondary" post-stroke depression (Code & Herrmann, 2003; Herrmann & Wallesch, 1993) evolves as patients enter the chronic phase and reflects their reaction to the impairments associated with their stroke. Degree of functional disability and presence of motor, cognitive, or communication impairments (including aphasia) have been identified as contributors to post-stroke depression (Hilari et al., 2010; Kauhanen et al., 2000).

In the general adult population, it is well accepted that depression and stress are closely related (e.g., Bao, Meynen, & Swaab, 2008). Indeed, depression can be considered a response to stress. Although stressful life events in and of themselves have been identified as precursors to depression (Brown & Harris, 1978; Kendler et al., 1999), an individual's *perception* of stress appears to be an important variable affecting the development of depression. Hewitt, Flett, and Mosher (1992) demonstrated that perceived stress is predictive of depression; in their clinical sample of depressed patients, greater perceived stress was associated with more severe depression.

Laures-Gore, Hamilton, & Matheny (2007) found that left hemisphere post-stroke patients living with aphasia demonstrate greater perceived stress than adults without stroke. However, it is not known whether heightened perceptions of stress are related to depression in the post-stroke population. While perceived stress and depression in stroke patients have previously been explored independently, little empirical evidence has explicitly investigated their relation in this population. Differences in perceptions of stress among stroke patients with and without aphasia may contribute to the high degree of individual variation observed in stress responses (Laures-Gore, 2012) that can result in post-stroke depression. A greater understanding of perceived stress and its link to post-stroke depression may aid in earlier diagnosis of depression and more focused behavioral treatments for depression in this population.

Therefore, the purpose of the current study is to determine whether perceived stress, neurological functioning, and depressive symptoms are associated in a sample of left hemisphere stroke patients with aphasia (LH) and right hemisphere stroke patients without aphasia (RH). We also explore change over time in these measures.

Methods

Nineteen LH and 12 RH participants were included in the current study. At the initiation of the study, all LH patients had aphasia and all RH patients had mild-moderate cognitive-communication disorders at the initiation of the study. Presence of aphasia was determined by the *Western Aphasia Battery-Aphasia Quotient* (*WAB-AQ*; Kertesz, 1982). Participants in the LH group demonstrated a Mean *WAB-AQ* of 57.8 at initiation of study. Presence of cognitive-communication disorder was determined by the *Mini Inventory of Right Brain Injury-2* (*MIRBI-2*; Pimental & Knight, 2000). Mean *MIRBI* score was 5.7 at initiation of study.

All participants were given the *Perceived Stress Scale* (PSS; S. Cohen et al., 1983), *Community Stroke Aphasic Depression Questionnaire-10* (*SADQ-10*; Sutcliffe & Lincoln, 1998), and the *Scandinavian Stroke Scale* (*SSS*; Scandinavian Stroke Study Group, 1985) four times over the course of three months. Questionnaires were completed either at the participant's home or on the campus of Georgia State University.

Results

There was a strong positive correlation between *PSS* and *SADQ* scores ($r(30) = .62$, $p < .001$), suggesting that across participants, higher perceived stress is associated with more severe

depressive symptoms. However, no associations between neurological impairment (*SSS*) and either *PSS* ($r(31) = -.02$, $p > .20$) or *SADQ* ($r(30) = -.09$, $p > .20$) were observed. An analysis of between-group differences in degree of neurological impairment, perceived stress, and depression for all time points was conducted. Independent samples t-tests were performed separately on the overall means of the three corresponding measures (*SSS*, *PSS*, and *SADQ*). Participants in the LH and RH groups did not differ significantly on the measure of stroke severity (*SSS*; $t(29) = -1.11$, $p > .20$), nor on perceived stress (*PSS*; $t(29) = .04$, $p > .20$) or depression (*SADQ*; $t(28) = -.73$, $p > .20$). These data do not indicate that LH stroke patients with aphasia have higher overall levels of perceived stress or depressive symptoms than those with right hemisphere stroke and no aphasia.

To examine group differences in changes over time, separate mixed-design analyses of variance (ANOVAs) were performed for each of the three measures, with two time points (first, last) as the within-subjects factor and group (LH, RH) as the between-subjects factor. In the analysis conducted for *SSS*, there was a large main effect of time (Wilks $\lambda = .65$, $F(1, 29) = 15.64$, $MSE = 229.97$, $p < 001$, partial $\eta^2 = .35$) but no significant effect of group ($F(1, 29) = .81$, $MSE = 92.74$, $p > .20$, partial $\eta^2 = .027$) and no interaction (Wilks $\lambda = .99$, $F(1, 29) = .04$, $MSE = 0.61$, $p > .20$, partial $\eta^2 = .001$). In the analysis for *PSS*, there was no significant main effect of time (Wilks $\lambda = .99$, $F(1, 29) = .27$, $MSE = 9.22$, $p > .20$, partial $\eta^2 = .009$) or group ($F(1, 29) = .09$, $MSE = 21.32$, $p > .20$, partial $\eta^2 = .003$). However, there was a marginally significant interaction of time and group (Wilks $\lambda = .89$, $F(1, 29) = 3.37$, $MSE = 114.64$, $p = .077$, partial $\eta^2 = .104$). This moderate-to-large effect size suggests that, despite the small sample size, there is a trend toward the LH group reporting *less* stress from the first to the last administrations of the PSS, while the RH group tended to report *more* stress. In similar analyses on the *SADQ*, the effect of time was a trend that did not approach statistical significance (Wilks $\lambda = .92$, $F(1, 29) = 2.34$, $MSE = 24.04$, $p = .14$); however, the effect size was nonetheless moderate (partial $\eta^2 = .077$). There was no main effect of group ($F(1, 28) = .30$, $MSE = 13.21$, $p > .20$, partial $\eta^2 = .01$) or interaction of time and group (Wilks $\lambda = 1.00$, $F(1, 28) = .003$, $MSE = 0.04$, $p > .20$, partial $\eta^2 = .000$).

Discussion

Overall, results indicate that perceived stress is a predictor of depression in post-stroke patients. Furthermore, despite improvement in neurological functioning over time, both LH and RH stroke patients report similar overall levels of depressive symptoms with a trend toward more depressive symptoms over time. Correspondingly, while the RH group reported an increase in perceived stress, the LH group reported a decrease in perceived stress over the three months of the study.

These findings have important clinical implications for determining who may be of greatest risk for developing PSD. Our findings suggest that the perception of stress may be a more critical variable in developing depression than neurological functioning is in stroke patients. Although the lack of relation between neurological functioning and depressive symptoms or perceived stress in this sample of stroke patients was not expected, it does align nicely with the notion that the number of stressors is less important than the perception of stress in developing physical symptoms and reporting distress (Bolger & Schilling, 2006). Routine screening of perception of stress may need to occur in post-stroke patients with and without aphasia to avoid development of depression.

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