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Research Article

Diurnal Cortisol Dynamics, Perceived Stress, and Language Production in Aphasia

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AQ1 **Purpose:** The current study investigated diurnal cortisol dynamics in adults with and without aphasia, along with subjective reports of stress and measures of language production. Dysregulation of cortisol, a common biomarker of stress, is associated with cognitive dysfunction in different clinical populations. However, little is known about the consequences of stress-induced cortisol disturbances for stroke survivors, including those with aphasia.

Method: Nineteen participants with aphasia and 14 age-matched neurotypical adults were tested. Saliva samples were collected from participants to assess the cortisol awakening response, a marker of the integrity of the hypothalamic-pituitary-adrenal (HPA) axis. Participants also completed 2 subjective stress questionnaires. Language was evaluated using 3 short, picture description narratives, analyzed for discourse (dys)fluency and productivity markers.

Results: In contrast to neurotypical participants, adults with aphasia did not show the predictable cortisol awakening response. Participants with aphasia also showed an unusual heightened level of cortisol upon awakening. Additionally, neurotypical participants demonstrated an association between intact language performance and the cortisol awakening response, whereas the participants with aphasia did not, although they did perceive the language tasks as stressful.

Conclusion: This study indicates that the functionality of the HPA axis, as indexed by cortisol, contributes to optimal language performance in healthy adults. The absence of an awakening response among participants with aphasia suggests that stroke leads to dysregulation of the HPA axis, although the degree to which this impairment affects language deficits in this population requires further investigation.

The notion that stress and language are linked in adults with aphasia is rooted in long-standing clinical observations (e.g., Goldstein, 1948; Sapir & Aronson, 1990), with growing empirical evidence for a connection between the two (for a review, see Laures-Gore & Buchanan, 2015). Specifically, one physiological stress system, the hypothalamic-pituitary-adrenal (HPA) axis, has been used to measure levels of stress in adults with aphasia (Laures-Gore, 2012; Laures-Gore, Heim, & Hsu, 2007; Sharp, Shaughnessy, Berk, & Daher, 2013). The stress measure used in these and many other stress studies is the end product of the HPA axis, cortisol, a glucorticoid hormone whose primary metabolic function is to provide fuel to working cells (Munck, Guyre, & Holbrook, 1984). Glucocorticoid receptors are distributed throughout the brain, including in the hippocampus, amygdala, and prefrontal cortex (Lovallo, Robinson, Glahn, & Fox, 2010; McEwen, Weiss, & Schwartz, 1968).

As part of normal HPA axis functioning, cortisol is released throughout the day following a predictable diurnal cycle (Lovallo & Buchanan, 2017). This cycle involves a relatively high level of cortisol at awakening, an increase within the first hour after awakening, followed by a decline of cortisol throughout the day. These dynamics reflect that the health of the HPA axis and measures of HPA health are correlated with psychosocial, psychiatric, and physical health outcomes (Adam & Gunnar, 2001; Adam & Kumari, 2009; Stalder et al., 2016). The diurnal cycle of cortisol reflects its metabolic function; the hormone is highest in the morning, in order to provide fuel during the transition from sleep to wake, and lowest in the evening, as humans transition back to sleep (Adam et al., 2017). The cortisol awakening response prepares us for the physical and cognitive demands of the day to come. For example, the cortisol awakening response is higher on weekdays than on weekends, suggesting that a higher cortisol awakening response plays a role in

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supporting the greater daily obligations associated with weekdays, as compared to weekends (Schlotz, Hellhammer, Schulz, & Stone, 2004). Further, the cortisol awakening response is positively associated with afternoon and evening cognitive performance in children, at home, and under lab conditions (Bäumler, Kliegel, et al., 2014). Recent research in older adults has similarly demonstrated that the cortisol awakening response is positively associated with cognitive measures, such as memory and processing speed (Ennis, Moffatt, & Herzog, 2016), as well as with indicators of physical performance, such as walking speed (Pulopulos, Puig-Perez, Hidalgo, Villada, & Salvador, 2016). Together, this work suggests that the cortisol awakening response may serve as a positive index of performance on various cognitive and behavioral tasks.

Alterations in the cortisol awakening response or an attenuated diurnal slope of cortisol have been related to presence of chronic stress (McEwen, 2012; Ockenfels et al., 1995), as well as a host of negative health outcomes in the general adult population (Adam & Kumari, 2009; Adam et al., 2017; Stalder et al., 2016). Disruption of HPA axis function has also been observed following stroke, characterized by high levels of cortisol immediately poststroke (Feibel, Hardy, Campbell, Goldstein, & Joynt, 1977; Olsson, 1990). The poststroke cortisol response has been found to predict acute stroke outcomes, including mortality (Christensen, Boysen, & Johannesen, 2004; Marklund, Peltonen, Nilsson, & Olsson, 2004).

Few studies have examined the impact of stroke on diurnal cortisol dynamics, let alone the relation with poststroke behaviors such as speech and language variables. The three studies that have investigated stroke and diurnal cortisol dynamics suggest that (a) a blunted cortisol awakening response is related to poststroke depression (Kwon, Kim, Lee, Sung, & Lee, 2015); (b) a lack of diurnal variation exists immediately poststroke with resolution of the variation 10 days poststroke (Franceschini et al., 1994); and (c) high levels of morning cortisol are observed in stroke patients immediately following stroke, but no difference in diurnal variation (Franceschini et al., 1994). This scant literature reveals gaping holes in our current knowledge about diurnal cortisol dynamics and its relation to poststroke recovery—cognitive or other.

Given that diurnal cortisol dynamics have been related to a variety of cognitive functions (Ennis et al., 2016; Evans et al., 2011), we propose in the context of the current study that cortisol may mobilize energy for language processing. This proposal is supported by evidence from a study in which cortisol reactivity was associated with greater language fluency (shorter pause times) among neurotypical adults and with greater language productivity (as measured by number of words) among people with aphasia (Buchanan, Laures-Gore, & Duff, 2014; Laures-Gore, DuBay, Duff, & Buchanan, 2010). Understanding cortisol dynamics and their relation to language could thus provide valuable insights into underlying contributors to a disrupted language system following stroke above and beyond the neurological damage observed in people with aphasia.

Characterizing the diurnal cortisol dynamics in adults with aphasia is also important for understanding how chronic stress influences responses to daily stressors, which may include language demands. In earlier work, we have hypothesized that the act of verbal communication is a chronic stressor for some individuals and an important aspect of aphasia (Cahana-Amitay, Albert, et al., 2011; Laures-Gore & Buchanan, 2015). If a link between cortisol and language output in aphasia does, in fact, exist, health care professionals may target the underlying cortisol dynamics during aphasia rehabilitation to produce better language outcomes.

Because diurnal cortisol dynamics have been shown to be affected by perceived chronic stress (Slavich & Shields, 2018), it is important to assess stroke survivors' perceived chronic stress as related to their speech and language outcomes. Adults with aphasia report higher perceived stress than those without stroke and aphasia in normal daily living (Laures-Gore, Hamilton, & Matheny, 2007). Their perceived stress is especially heightened in response to linguistic tasks (Cahana-Amitay, Oveis, et al., 2015; Laures-Gore, Heim, et al., 2007). Although a direct link between language production and perceived stress has not been reliably established, considering the subjective feeling of participants with aphasia in clinical or research settings may be important for optimizing treatment of their language deficits.

The purpose of the current study was to (a) map the diurnal cortisol dynamics of adults with poststroke aphasia by measuring their cortisol awakening response and their diurnal cortisol slope, (b) characterize the relation between cortisol dynamics and language production in aphasia, (c) examine perceived stress in adults with aphasia and their relation to language production, and (d) explore the relation between perceived stress and the diurnal cortisol dynamics of adults with aphasia. We predicted that cortisol dynamics would differ between adults with aphasia and a control group, that cortisol would be related to language production in adults with aphasia, and that adults with aphasia would self-report greater perceived stress than the control group. Furthermore, although past evidence suggests no relation between perceived stress and diurnal cortisol levels, the overall literature is scarce and based on small samples, requiring replication. Results from this study will deepen our understanding of the link between the language system and stress in adults with aphasia and may offer new directions for assessment and therapeutic approaches to language impairment in this population.

Method

Participants

After obtaining approval by the institutional review board at Georgia State University, participants were recruited via fliers through the Georgia State University Speech and Hearing Clinic and through speech-language pathology departments in hospitals or rehabilitation centers throughout the metro-Atlanta area. Nineteen adults with a history

of left hemisphere stroke and aphasia participated in the study (seven women, 12 men; M_{age} = 55.47, SD = 11.86). Mean poststroke onset was 28.9 months (SD = 26.54, range: 4–110 months). Aphasia type and severity, as determined by the Western Aphasia Battery–Revised (Kertesz, 2006; n = 18) and Boston Diagnostic Aphasia Examination (Goodglass, Kaplan, & Barresi, 2001; n = 1), revealed five types of aphasia: Broca's (n = 4), global (n = 2), anomic (n = 11), transcortical sensory (n = 1), and conduction (n = 1). Mean Western Aphasia Battery–Aphasia Quotient (WAB-AQ) was 68.8 (SD = 22.5). Eighteen participants demonstrated apraxia of speech as measured by the Apraxia Battery for Adults–Second Edition (Dabul, 2000). Eligible participants had to be between 35 and 79 years of age and at least more than 1 month poststroke onset. This time, postonset was determined to allow for medical stabilization of the participants and include participants at different stages of recovery. Participants were excluded if they (a) had multiple strokes, a bilateral stroke, history of head injury, or presence of other neurological disease; (b) reported current use of corticosteroid medication, which would alter cortisol dynamics; (c) had dysphagia; and (d) reported a history of communication disorders prior to stroke. We were able to obtain neuroimaging confirmation of left hemisphere stroke for all participants with aphasia except one; however, his physical presentation (significant right hemiplegia) indicated left hemisphere stroke. Six participants had a clinical diagnosis of depression. All participants denied use of corticosteroid medication but were on other medications including antihypertensives (n = 10), antidepressants (n = 12), anticonvulsants (n = 8), and diabetes management (n = 3). See Table 1 for the description of aphasia participants.

Control participants were recruited via fliers in the metro-Atlanta area. Fourteen adults with no self-report history of neurological disease/injury or communication disorder participated (seven women, seven men; M_{age} = 55.53 years, SD = 11.9). All participants had to be between 35 and 79 years of age, with an above-the-normal cutoff of 24 on the Mini-Mental State Examination; (Folstein, Folstein, & McHugh, 1975; range: 27–30, M = 29.5). Participants were excluded if they (a) reported current use of corticosteroid medication and (b) reported a history of communication disorders. See Table 2 for the description of control participants. The number of participants included in this study is consistent with other reports of sample sizes in the aphasia literature. Post hoc sensitivity power analysis for group differences on our primary dependent measure, cortisol awakening response, demonstrated that, with our sample size, the smallest detectable effect would be a critical t value of 2.06 (see Faul, Erdfelder, Lang, & Buchner, 2007).

Procedure

Participants were seen in the Georgia State University Aphasia and Motor Speech Disorders Lab or their home for initial language, speech, and cognitive testing, as well as discourse sampling. Data were collected by a trained examiner/research assistant. After testing and discourse sampling, participants were given instructions for the saliva sampling procedures, which took place later in their homes or workplaces. The first author demonstrated the saliva sampling technique and then gave the participants the Salivette tubes, an electronic monitoring device (MEMS, 6 TrackCap Monitor, Aardex Lt.), and a daily diary to take home. She showed them how to remove the cotton pledget from the Salivette container, how to place and remove the pledget in the mouth, and how to reinsert the pledget into the Salivette container and place the Salivette into the MEMS bottle. Participants were instructed to not open the MEMS bottle unless they were placing a sample in it to avoid unnecessary time stamps. In many instances, caregivers were also present during the sampling training. All participants were asked if they understood the instructions. Verbal and visual instructions were additionally augmented with written instructions that were taken home with the participants. The first author also reviewed the diary with the participants (and caregivers when appropriate) and explained the need to complete each diary entry at the same time that the cortisol sample was taken. Participants were also told that they could contact the first author at her lab phone number with any problems with the sampling or diary. Saliva samples were collected a mean of 4.36 days (range: 1–18) following language testing for the aphasia group and 2.66 days (range: 1–8) for the control group.

Discourse Sampling

Language was assessed using discourse samples that were elicited through picture descriptions of three sets of card sequences ("Curse of Caffeine," "Modern Day Camping," "Winds of Change"; Helm-Estabrooks & Nicholas, 2003). This discourse elicitation method neutralizes the confounding effects that emerge in conversational settings and also enables systematic coding and analysis that captures the multilevel complexity associated with discourse production in aphasia (Cahana-Amitay & Jenkins, 2018). The samples were audio-recorded during the initial testing session and later transcribed and coded by trained lab personnel for markers of discourse dysfluency and discourse productivity (Cahana-Amitay, Fitzpatrick, Volz, & Finley, 2011; Cahana-Amitay et al., 2012; 2015). Markers of dysfluency included filled pauses, false starts, perseverations, paraphasia, jargon, and long silent pauses (exceeding 3 s). Markers of productivity included averages of word counts (content and function words) and clause counts (where a clause was defined as a syntactic unit containing a predicate, describing an action, event, or state). Interrater reliability of discourse coding was determined by a reanalysis of discourse measures from 18% of the total discourse samples with a second trained research assistant. The code for each discourse measure from the reanalysis was compared to the original code assigned in the first analysis. Pearson product–moment correlations conducted between original and second measurements indicated high interrater reliability (r = .981). We expected the discourse measures to reveal an association that might otherwise be hidden in larger composite scores

Table 1. Description of aphasia participants.

Subject	Gender	Age	Ethnicity	Site of stroke	MPO	WAB-R AQ	Aphasia type
1	M	76.8	C	Left temporal lobe	16	N/A	B
2	M	49.1	C	NA	14	22.3	G
3	M	69.3	C	Left MCA	14	23.5	G
4	M	45.7	AA	Left MCA	25	28	B
5	M	51.4	C	Left MCA	21	91.6	Anom
6	M	61.7	C	Left posterior/temporal/parietal	45	87.4	Anom
7	M	70.66	C	Left MCA	44	83.2	Anom
8	F	39.1	C	Left ischemic	50	93.2	Anom
9	F	36.3	C	Left MCA	36	54.6	B
10	M	49.2	C	Left MCA	24	83.3	Anom
11	F	54.6	AA	Left temporal/frontal	110	82.2	Anom
12	F	46.9	C	Left MCA	11	71.6	Anom
13	M	36.4	A	Left MCA	10	69.9	Anom
14	M	62.9	C	Left temporal	13	68.8	TS
15	M	52.3	C	Left MCA	4	75.8	B
16	F	67.5	C	Left MCA	6	68.1	Anom
17	F	66.5	C	Left MCA	28	87.3	Anom
18	M	58.5	C	Left posterior temporal/parietal	7	73.2	C
19	F	58.8	C	Left MCA	72	76.0	Anom

Note. MPO = months postonset; WAB-R AQ = Western Aphasia Battery–Revised Aphasia Quotient; M = male; C = Caucasian; B = Broca's; G = global; MCA = left middle cerebral artery; AA = African American; Anom = anomic; F = female; A = Asian; TS = transcortical sensory; C = conduction.

typically used in standardized language tests. This expectation was based, in part, on previous work by Laures-Gore and colleagues showing no correlation between cortisol and grosser measures of language in an aphasia group, such as the Western Aphasia Battery–Revised Aphasia Quotient and Boston Naming Test (Laures-Gore, 2012; Laures-Gore, Heim, et al., 2007). It was also based on findings that discourse measures such as those used here tap subtle changes in language performance in aphasia when language tasks vary in the degree of stress they induce (Cahana-Amitay et al., 2015).

Perception of Stress

Chronic stress was assessed with the Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983) during the initial testing session. The PSS has been used in previous studies of stress and aphasia (Laures-Gore & Defife, 2013; Laures-Gore, Farina, Moore, Russell, 2016; Laures-Gore, Hamilton, et al., 2007). The administration of the PSS in the current study is similar to previous administration with participants with aphasia. The first author read the instructions and possible responses aloud while the participant followed along. Similarly, the first author read each statement in the questionnaire aloud while the participant followed along. The participant responded either verbally or pointed. The first author repeated statements and possible responses upon participant request. Daily stress was measured with a stress scale developed by the first author, adapted from a comparable scale used in previous studies (Gillespie, Laures-Gore, Moore, Farina, & Russell, 2018; Laures-Gore, Heim, et al., 2007). The current stress scale required participants to rate their stress level on a 5- rather than on a 7-point Likert scale, with *not at all* corresponding to a 1 and *very* corresponding to a 5. The scale was modified from the one described in Gillespie et al. (2018) to capture more specifically perceived stress in the time period close to the cortisol sampling, as participants were asked to rank their stress level in the last hour. The scale was included on each diary page that participants were required to complete for each saliva collection for a total of seven ratings across 1 day. As noted in Gillespie et al., the stress scale was highly correlated with the Self-Assessment Manikin Arousal (Bradley & Lang, 1994), suggesting that it measures the construct of stress.

Table 2. Description of control participants.

Subject	Gender	Age	Ethnicity	MMSE
1	M	52.3	C	34
2	F	61.7	C	34
3	F	59.2	AA	35
4	M	42.05	C	34
5	F	42.6	AA	32
6	F	42.3	C	35
7	M	57.4	C	35
8	M	69.3	C	35
9	M	42.06	AA	33
10	F	65.4	AA	35
11	F	38.6	AA	35
12	F	65.7	C	34
13	M	66.03	C	35
14	M	72.4	AA	32

Note. MMSE = Mini-Mental State Examination; M = male; C = Caucasian; F = female; AA = African American.

Cortisol Sampling

Saliva was collected via Salivette tubes (Sarstedt) at the participant's home seven times throughout 1 day.

Collection times were upon awakening, 30 and 60 min later, AQ8 and then at 1100 hr, 1500 hr, 1800 hr, and bedtime. Adherence to the sampling strategy was checked with an electronic monitoring device (MEMS 6 TrackCap Monitor, Aardex Lt.). Participants were instructed to complete their saliva sampling during a weekday and to abstain from exercise, ingestion of nicotine, alcohol, or food in the hour before each sample. At each collection point, participants completed a diary to record exercise, ingestion of nicotine, alcohol, or food and to scale their current level of stress. Saliva samples and diaries were retrieved by lab personnel the day following saliva collection. Saliva samples were stored and frozen on the Georgia State University campus at −20° until later assayed by immunoassay using a commercially prepared kit produced by Diagnostics Systems Laboratory at the Neuroscience Institute Core Lab, Georgia State University, Atlanta, Georgia. Intra-assay coefficients of variation were less than 10%.

Data Analysis

Cortisol and perceived stress were analyzed using 2 Group (Aphasia, Control) × 7 Time (Wake, Wake + 30, AQ9 Wake + 60, 1100 hr, 1500 hr, 1800 hr, Bedtime) repeated measures analyses of variance (ANOVAs). The cortisol awakening response includes the sample at awakening and 30 and 60 min after awakening. The area under the curve with respect to increase (AUCi) was calculated according to the Pruessner, Kirschbaum, Meinlschmid, and Hellhammer (2003) study to serve as a single summary statistic of the cortisol awakening response. Group differences in AUCi and chronic stress assessed via the PSS were examined using independent-samples *t* tests. Relations among cortisol, self-reported stress, and language measures were examined using Pearson *r* correlation coefficient. Due to the likely violation of the sphericity assumption in repeated-measures designs, Greenhouse–Geisser–corrected ANOVAs are reported, in order to avoid the inflated Type I error rate when the sphericity assumption is not met (Maxwell & Delaney, 1990). Effect sizes are reported using partial eta squared for ANOVAs.

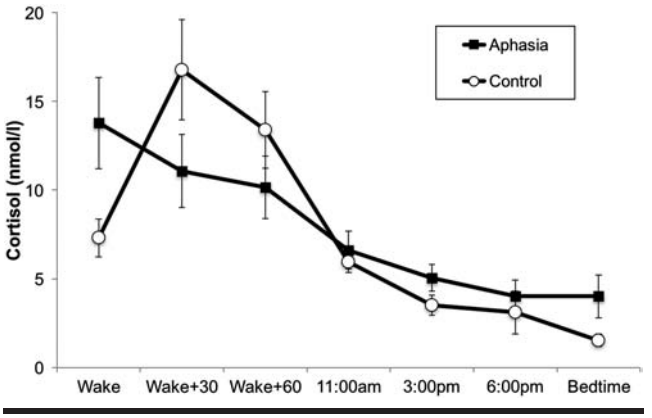
Results

Diurnal Cortisol Dynamics

One participant with aphasia was excluded from the analysis due to the MEMS cap not recording the time, and five aphasia participants were excluded due to a lab error resulting in insufficient saliva volume for analysis. The groups showed different patterns of cortisol release throughout the day as shown by a significant Group × Time interaction (see Figure 1 and Table 3), characterized by the aphasia group showing a higher cortisol level at awakening, followed by a lack of a cortisol awakening response. There was also a significant main effect of Time (indexing the typical diurnal changes in cortisol throughout the day) but no main effect of Group (see Figure 1 and Table 3). The aphasia group showed a significantly reduced cortisol awakening response, as compared to the control participants: $M_{aphasia} = -136.03$, $SD = 237.4$; $M_{control} = 337.25$, $SD = 400.56$; $t(25) = 3.7$, $p = .001$ (see Figure 2). Analysis of individual data revealed that 11 out of 14 of the control participants showed a positive cortisol awakening response, whereas only three out of 13 of the aphasia group showed a positive cortisol awakening response, a significant group difference ($\chi^2 = 8.315$, $p < .004$).

F1 F2 T3

Figure 1. Mean and standard error of the mean cortisol levels between groups across time.



Because differences in wake time and noncompliance with sampling can negatively affect the reliable measurement of the cortisol awakening response (Stalder et al., 2016), we collected indices of wake time and sampling time. The groups did not differ significantly on wake time: $M_{aphasia}$ = 7:32 a.m., SD = 1 hr 45 min; $M_{control}$ = 6:40 a.m., SD = 1 hr 29 min; $t(25) = 1.4$, $p > .18$. Despite this nonsignificant difference, wake timing may have affected the group differences in cortisol patterns (see Stalder et al., 2016); therefore, wake time was included as a covariate in follow-up analyses on all cortisol levels, as well as the cortisol awakening response. Results of these analyses showed the same pattern as those without wake time as a covariate: Group × Time interaction, $F(6, 144) = 3.3$, $p = .024$, and Group difference in cortisol awakening response, $F(1, 26) = 12.3$, $p = .002$.

Perceived Daily and Chronic Stress

The groups did not differ in self-reported stress across the day, as shown by a nonsignificant interaction from a

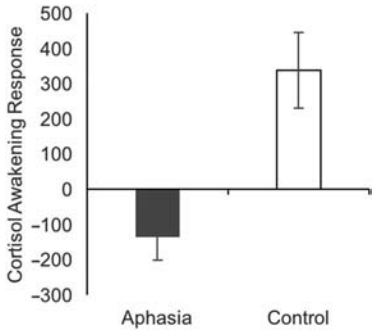
Table 3. Analysis of variance table of cortisol and perceived stress between groups across time.

	Group		Time		Group × Time	
Variable	*F*	η_p^2	*F*	η_p^2	*F*	η_p^2
Cortisol	0.25	.01	21.3**	.46	3.7*	.13
Perceived stress	1.5	.05	1.3	.05	0.86	.03

Note. Cortisol *df* = (1, 25), Minute and Condition × Minute *df* = (6, 150); Perceived Stress *df* = (1, 27), Minute and Condition × Minute *df* = (6, 162).

*$p < .05$. **$p < .01$.

Figure 2. Mean and standard error of the mean cortisol awakening response (in arbitrary units) between groups.



2 Group (Aphasia, Control) × 7 Time (Wake, Wake + 30, Wake + 60, 1100 hr, 1500 hr, 1800 hr, Bedtime) repeated-measures ANOVA (Group × Time interaction; see Table 3). Further, there were no main effects of Time, nor were the groups different in perceived stress (no main effect of Group; see Table 3). The aphasia group did not report different chronic stress levels on the PSS, as compared to controls: $M_{aphasia}$ = 23.3, SD = 8.6; $M_{control}$ = 19.6, SD = 7.2 2, $t(31) = 1.3$, $p = .196$.

Correlations Between Cortisol and Self-Reports of Stress

There were no significant correlations between the cortisol awakening response and the average score of self-reported stress throughout the day ($r = .13$, $p > .5$) or the level of chronic stress reported on the PSS ($r = -.15$, $p > .4$). This pattern was consistent across the whole sample, as well as when the analyses were run separately for each group.

Discourse Variables

One aphasia participant was excluded from the discourse analysis as there was no audio recording due to technical error. The aphasia group produced more paraphasias, jargon, filled pauses, false starts, and perseverations than the control group (see Table 4 for descriptive and inferential statistics). We next examined the relationship between diurnal cortisol and discourse variables separately for the control and aphasia groups. Several language measures were significantly correlated with the cortisol awakening response in the control group (average clauses, average number of words, average content words, average function words, and number of long pauses, rs = .798–.812; see Table 5). No discourse measures were reliably correlated with the cortisol awakening response in the aphasia group, however (rs < .35). The correlations between the cortisol awakening response and language variables in the aphasia versus the control group were compared directly using Fisher's z transformation. Table 5 shows that several of these relationships were significantly greater for the control group than for the aphasia group.

No significant correlation was revealed between PSS and discourse measures in the control group; however, the aphasia group showed significant correlations between the PSS and averages of the following variables: clauses ($r = .552$, $p = .021$), content words ($r = .498$, $p = .009$), false starts ($r = .611$, $p = .009$), and perseverations ($r = .516$, $p = .034$). To examine the potential association between depression diagnosis, WAB-AQ scores, age, or months postonset, and cortisol dynamics, correlational analysis was performed. AUCi did not correlate with any of these variables for either group. We found no significant group difference in the prevalence of depression diagnosis, $M_{aphasia}$ = 0.8, SD = 1.36; N = 20; $M_{control}$, = 0.21, SD = 0.8; N = 14, $t(32) = 1.57$, $p = .126$. Further, depression was not related to cortisol awakening response in either the whole group ($r = -.18$, $p > .37$) or in the individual groups (aphasia group: $r = -.12$, $p > .6$; controls: $r = -.21$, $p > .4$).

Table 4. Means and standard deviations for language variables between groups.

Variable	Control group	Aphasia group	*t*
Clauses	10.2 (6.2)	9.9 (5.8)	–0.17
Words	86.0 (46.0)	79.5 (51.9)	–0.37
Words per clause	9.1 (2.0)	8.0 (2.4)	–1.4
Content words	46.2 (24.9)	37.5 (24.7)	–0.99
Function words	38.2 (21.1)	31.1 (21.7)	–0.93
Paraphasias	0.0 (0.0)	1.7 (2.6)	**2.4***
Jargon	0.0 (0.0)	0.61 (0.9)	**2.3***
Filled pauses	1.2 (1.5)	7.5 (5.3)	**4.3****
False starts	0.7 (0.7)	5.5 (7.3)	**2.5***
Perseverations	0.2 (0.3)	2.0 (2.4)	**2.8***
Long pauses	0.6 (0.6)	1.8 (2.3)	1.9

Note. *t*-Statistic report results of independent-samples *t* tests for between-groups comparison.

*$p < .05$. **$p < .01$.

Discussion

The current study compared diurnal cortisol dynamics in poststroke adults with aphasia to neurotypical age-matched

Table 5. Correlations between cortisol awakening response and language variables between groups.

Variable	Control group	Aphasia group	*z*
Clauses	.79	.15	**2.1***
Words	.75	.14	**1.8***
Words per clause	–.39	.001	–0.89
Content words	.72	.12	**1.7***
Function words	.71	.10	**1.7***
Filled pauses	–.03	.12	–0.33
False starts	.05	.32	–0.83
Perseverations	–.24	.22	–1.0
Long pauses	.81	.10	**2.2***

Note. Entries show Pearson *r* coefficients and the *z* score differences between groups' correlations using Fisher's *z* transformation. Note that control participants did not produce enough paraphasias or jargon to be included in these analyses.

*$p < .05$.

adults and examined these dynamics in relation to subjective reports of stress and measures of discourse production. The diurnal cortisol profiles that emerged suggest that, whereas healthy controls presented a typical cortisol awakening response, adults with aphasia showed no cortisol awakening response. These participants presented with a high cortisol level at awakening and then showed a decline in cortisol at 30 and 60 min postawakening. This pattern contrasts with the typical increase in cortisol levels during the hour after awakening observed in the control group. The remainder of the diurnal slope appeared similar in both groups and follows a predictable pattern of decline throughout the day.

We are confident that the cortisol awakening responses discussed below are reliable indicators of cortisol levels among all study participants, as we examined the potential effects of the timing of saliva collection on the accuracy of the cortisol awakening responses reported here (for a discussion of sampling error issues, see Stalder et al., 2016, for a review). Using objective timing indicators (MEMS 6 TrackCap Monitor) that are designed to track timing of saliva collection, we determined that there was no difference in sample timing between the two groups. The participants in the aphasia group did wake up later than the control group, but statistically controlling for this difference in wake time did not affect the pattern of results.

The heightened level of cortisol upon awakening and the absence of the awakening response that we found among the participants with aphasia is unusual and suggests dysregulation of the HPA axis similar to that reported for other clinical populations, such as people with posttraumatic stress disorder, hippocampal amnesia, and Type II diabetes (Bruehl, Wolf, & Convit, 2009; Buchanan, Kern, Allen, Tranel, & Kirschbaum, 2004). These neurological patient groups also show a disruption of the cortisol awakening response (Boggero, Hostinar, Haak, Murphy, & Segerstrom, 2017). Among people with aphasia, it is possible that the disruption of cortisol awakening response is related to the neurological damage associated with their stroke. Because the majority of the participants with aphasia in the current study were well into the chronic stage of the disease, we propose that the effects associated with dysregulation of the cortisol awakening response may continue for a protracted period in this population. This claim should be confirmed in future studies, as the immediate and long-term impact of stroke on the HPA axis remains an unexplored research area (Franceschini et al., 1994).

We do not believe that the absence of the cortisol awakening response among the participants with aphasia is attributable to the medications that they were taking during the study, especially because none were corticosteroid medications. Several medications are known to have an effect on the HPA axis (see Granger, Hibel, Fortunato, & Kapelewski, 2009, for a review). However, cortisol levels and ranges reported here were within normal limits for all participants and did not differ by group. Further, there is no indication from the literature that the medications are directly linked to a dysregulation specifically of the cortisol awakening response in the current sample; however, this variable should be studied further in future investigations.

Beyond the neurological damage associated with the stroke, other factors potentially contributing to the lack of a cortisol awakening response in the participants with aphasia could include poor sleep. Disrupted sleep patterns have been noted following stroke (for a review, see Wallace, Ramos, & Rundek, 2012); however, the specific relation between the cortisol awakening response and sleep disturbances remains unclear (Elder, Wetherell, Barclay, & Ellis, 2014; Hansen et al., 2012). Given the heightened cortisol level immediately upon awakening among the participants with aphasia in our sample, it is possible that a disrupted sleep pattern interferes with the timing of their cortisol release. This is an area demanding further examination in the aphasia population.

Although chronic stress influences the cortisol awakening response (Boggero et al., 2017; Labonté, Azoulay, Yerko, Turecki, & Brunet, 2014), the current findings do not support such a relationship. Among people with aphasia, common stressors such as unemployment, changes in family roles, and social networks are combined with the daily challenges of communication using an impaired language system. However, in this study, chronic stress, as measured by the PSS (Cohen et al., 1983), along with daily reports of stress that were collected simultaneously with the cortisol sampling, did not differ between people with aphasia and their healthy counterparts. These findings leave open the possibility that either chronic stress is not contributing to the lack of the cortisol awakening response or that self-reports of stress are poorly associated with physiological indices of chronic stress. Indeed, researchers have pointed out the difficulty of reliably linking the biological and psychological components of stress (Hellhammer & Schubert, 2012; Mauss, Wilhelm, & Gross, 2004).

Another possibility is that reduced perceived stress among the adults with aphasia in the current study is due to a decrease in their coping resources that negatively impacts their ability to accurately monitor and report stress and tension. This idea is based on an earlier study exploring the types of coping resources available to adults with aphasia (DuBay, Laures-Gore, Matheny, and Romski, 2011), which found that adults with aphasia had fewer resources for monitoring stress and tension control than those of another stroke group without aphasia and a healthy control group. It is also consistent with the broader claim that coping resources are related to awareness of personal stress levels and the ability to reduce tension buildup (Curlette, Aycock, Matheny, Pugh, & Taylor, 1992).

The seemingly comparable levels of perceived stress in the aphasia and neurotypical groups reported here are inconsistent with an earlier study conducted by the first author and colleagues (Laures-Gore, Hamilton, et al., 2007), which showed greater levels of perceived stress among people with aphasia compared to those without the disorder. We do not believe this disparity is linked to demographic factors, as we compared the patient groups in the two

studies and found that they were comparable in terms of WAB-AQ scores, age, time poststroke onset, and type of aphasia. Instead, we speculate that this disparity may be linked to differences in coping mechanisms. In the 2007 study, participants completed a coping resources inventory not offered in the current study. We propose that, if coping resources were assessed in the current sample and if differences in coping resources were found between the two studies, they could have potentially accounted for the disparity in perceived levels of stress reported for the two patient groups.

In the current study, subjective reports of perceived stress as measured by the PSS among people with aphasia were, however, positively associated with certain language measures. This finding is consistent with previous studies, in which subjective reports of stress among people with aphasia were related to expressive language skills, more so than to receptive ones (Thomas & Lincoln, 2008). It is also in line with other studies indicating that, among adults with aphasia, language tasks can elicit greater perception of stress than nonlinguistic tasks (Cahana-Amitay et al., 2015; Laures-Gore, Heim, et al., 2007).

The specific discourse components associated with perception of stress in adults with aphasia involved both discourse *density*—clause and word counts—and *(dys)fluency*—false starts and perseverations. Production of these discourse components in aphasia is influenced by impairments in working memory (e.g., Cahana-Amitay & Jenkins, 2018) and executive functions (Cahana-Amitay & Albert, 2015, Chapter 4). We propose that the combined cognitive–linguistic load underpinning the production of these discourse components in aphasia could lead to a heightened awareness among participants with aphasia of the increased burden with which they are tasked (i.e., these tasks are perceived as difficult). This awareness might be the driver underlying their perceived stress.

Interestingly, measures of discourse density and fluency were related to the cortisol awakening response in the control group, but not in the aphasia group. This result suggests that diurnal dynamics may play an important role in supporting the intact language system. Although cortisol–language causality cannot be determined from the current design, we propose that these results, along with findings from previous studies, suggest that the cortisol awakening response may supply energy to normal language functioning, much like it provides energy to other cognitive processes (Bäumler, Kliegel, et al., 2014; Bäumler, Voigt, et al., 2014; Butler, Klaus, Edwards, & Pennington, 2017; Hodyl et al., 2016; Law, Evans, Thorn, Hucklebridge, & Clow, 2015). The idea is that the cortisol awakening response serves as an index of the cognitive and motivational resources necessary for the intact language system to achieve maximal/optimal performance. In the face of brain damage, such cognitive and motivational resources may be lacking, compromising language behaviors. To strengthen this argument, it is important that future studies conduct multiple days of salivary cortisol collection with synchronous language sampling to assess the reliability of the current study's cortisol results and to examine the impact of diurnal cortisol on contemporaneous language production.

The notion that energy underpins language performance is not new to the aphasiology literature. Specifically, McNeil and colleagues have argued that energy reflects the availability of attentional resources for task performance (McNeil, Odell, & Tseng, 1991; Tseng, McNeil, & Milenkovic, 1993). The current account extends this psychological explanation to a physiological system, whereby cortisol, as a glucocorticoid, serves as a biomarker of energy that could affect the language system. This idea is plausible, given the location of glucocorticoid receptors throughout the hippocampus and prefrontal cortex (Lovallo et al., 2010; McEwen et al., 1968) and reports that both these structures contribute to the language system (Bourguignon, 2014; Covington & Duff, 2016; Duff & Brown-Schmidt, 2012).

The current study has several limitations that should be considered when interpreting the findings. One limitation concerns sample size. Although small sample sizes are not uncommon in the aphasia literature, they do create the possibility of Type I errors. Indeed, the current study had a relatively small sample size, and some of the data points were, in fact, lost due to technical error or lack of adequate saliva sample volume.

Another limitation concerns our study design, specifically the use of a single-day cortisol sampling design, which could limit insights into the larger picture of day-to-day changes in diurnal cortisol dynamics (see Law, Hucklebridge, Thorn, Evans, & Clow, 2013). However, this design can still prove useful, as previous work has demonstrated substantial heritability of the cortisol awakening response (Kupper et al., 2005; Wüst, Federenko, Hellhammer, & Kirschbaum, 2000), as well as a consistent lack of response lasting several days in other neurological patient groups such as people with hippocampal amnesia (Buchanan et al., 2004).

In addition, the timing of cortisol and discourse sampling were not synchronous, potentially masking correlations between cortisol dynamics and discourse in the aphasia group that might be evident if taken simultaneously. However, the consistent correlations we found between comparison participant's cortisol awakening response and discourse measures suggest that the lack of relation between these variables in our aphasia group is specifically related to the aphasia pathology and not an idiosyncrasy of our study protocol.

Also, our assessment of self-perceived stress was based on a self-report Likert scale that has not been psychometrically tested, making interpretation of the rankings problematic. Thus, our results should be interpreted with caution. We believe they are still informative, as, to date, there exist no alternative questionnaires that offer insight into the issues at hand. Current work is being conducted to test the validity and reliability of this scale, to allow for future examination of such questions.

Finally, the current study did not collect data regarding lesion volume size, which may influence HPA axis functioning and could be an important variable to consider

in future studies. Importantly, though, the extent of the effect of this variable on HPA axis is unknown at this time. The picture emerging from this study has also been clouded by other variables such as sex and age of the participants and time postonset of stroke, which were not considered statistically in the current study due to the small sample size. These variables should be explored for their impact on the relation between cortisol dynamics and language in subsequent studies.

The direct clinical implications of the current study are not immediately obvious, as this line of inquiry is still at its inception. However, one could imagine several ways in which the long-term clinical impact of understanding the interplay between the stress and language systems can influence assessments and interventions. First, understanding the diurnal variation of stress hormones could shed light on the within-day variability in language performance in adults with aphasia and, perhaps, explain why certain patients do not respond to treatment.

Second, understanding stress patterns in aphasia may help inform individually focused pharmacological and behavioral interventions that target stress physiology. If stress is deemed to enhance language functioning in some adults with aphasia, then interventions may exploit stress as an energizing system. For those who have a negative influence of stress on their language system, pharmacological and behavioral interventions may target stress reduction in these individuals. Although the current study uses a group design, future work should focus more on individual traits that could influence stress and its management in therapies targeting improved language performance and recovery, as noted in Laures-Gore and Buchanan (2015).

Third, addressing physiological and self-perceived stress in aphasia will allow clinicians to tackle the physical and mental health variables that can become a barrier to successful recovery from aphasia. By understanding aphasia and its relation to stress, programs tailored to lessening the mental, physical, and behavioral aspects of aphasia that are detrimental to regaining poststroke neural health, including physical, mental, cognitive, and linguistic gains.

In summary, the current study is the first to map the diurnal cortisol dynamics of adults with poststroke aphasia, making novel methodological and theoretical contributions to the understanding of stress and the relation between stress and language performance in aphasia. From a methodological standpoint, we observed that the diurnal slope of the aphasia group follows the same pattern found in a neurotypical population. Along with the results of Laures-Gore (2012), which indicated no difference in afternoon cortisol levels between an aphasia group and another stroke group, it appears that sampling cortisol in the afternoon should be a reliable time for salivary cortisol collection. A reliable sampling time for the poststroke aphasia population is critical in designing future studies measuring the HPA axis response to different types of stressors.

From a theoretical perspective, the current findings revealed an association between intact language performance and the cortisol awakening response in neurotypical adults. We interpreted this pattern as evidence for the role of the HPA axis, indexed by cortisol, in providing the energy supply necessary for optimal language performance in healthy adults. The participants with aphasia in this study clearly perceived some stress, but the degree to which this stress was related to the dysfunctionality of their HPA axis requires further investigation. The vulnerability of the language system to stress remains an important topic to explore in people with aphasia, as current assessments and treatments of the disorder may be underestimating the extent to which this population experiences language as a stressor in daily and clinical settings, impeding their language recovery processes.

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AQ4: Please check if all tables if were captured properly.

AQ5: Please provide the definitions of "NA" and "N/A" as part of the Table 1 footnote.

AQ6: The acronym for "anomic," which was "A," was modified to "Anom" to distinguish it from the acronym of "Asian," which is also "A." Please check if appropriate.

AQ7: Gillespie, Laures-Gore, Moore, & Russell, 2018, was changed to Gillespie, Laures-Gore, Moore, Farina, & Russell, 2018. Please check.

AQ8: Please check if the changes made retained the intended meaning of the sentence.

AQ9: Please check if the changes made retained the intended meaning of the sentence.

AQ10: Please check if the changes made retained the intended meaning of the sentence.

AQ11: Please check if Fisher's *z* transformation was properly captured.

AQ12: Please provide significance of bold figures or remove emphases.

AQ13: Please provide significance of bold figures or remove emphases.

AQ14: Please cite this reference in the text or confirm if it should be deleted.

END OF ALL QUERIES