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Naming errors and dysfunctional tissue metrics predict language recovery after acute left hemisphere stroke

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ARTICLE INFO
Keywords:
Left hemisphere stroke
Language
Naming errors
Longitudinal
Recovery
Structural imaging

ABSTRACT
Language recovery following acute left hemisphere (LH) stroke is notoriously difficult to predict. Global language measures (e.g., overall aphasia severity) and gross lesion metrics (e.g., size) provide incomplete recovery predictions. In this study, we test the hypothesis that the types of naming errors patients produce, combined with dysfunctional brain tissue metrics, can provide additional insight into recovery following acute LH stroke. One hundred forty-eight individuals who were hospitalized with a new LH stroke completed clinical neuroimaging and assessments of naming and global language skills. A subset of participants again completed language testing at subacute, early (5–7 months post-stroke), and late (≥11 months post-stroke) chronic phases. At each time point, we coded naming errors into four types (semantic, phonological, mixed and unrelated) and determined error type totals and proportions. Dysfunctional tissue measures included the percentage of damage to language network regions and hypoperfusion in vascular territories. A higher proportion of semantic errors was associated with better acute naming, but higher proportions of other error types was related to poorer accuracy. Naming and global language skills significantly improved over time, but naming error profiles did not change. Fewer acute unrelated errors and less damage to left angular gyrus resulted in optimal naming and language recovery by the final testing time point, yet patients with more acute errors and damage to left middle temporal gyrus demonstrated the greatest increases in language over time. These results illustrate that naming error profiles, particularly unrelated errors, add power to predictions of language recovery after stroke.

1. Introduction
In the first days following stroke, approximately 30% of survivors experience aphasia (Flowers et al., 2016; Laska et al., 2001; Pedersen et al., 2003; Wade et al., 1986), a language disorder characterized by receptive and expressive language deficits. Deficits persist into the chronic phase of recovery (around six months post-stroke and onwards) in upwards of 30% of these individuals (Engelter et al., 2006; Flowers et al., 2016). Despite endeavors to identify factors that determine aphasia prognosis (Laska et al., 2001; Lazar et al., 2008; Lazar and Boehme, 2017; Pedersen et al., 2003; Plowman et al., 2012), the ability to predict whether a patient will recover following acute left hemisphere (LH) stroke remains elusive. Lazar et al. (2008) found that the combined variables of age, lesion size, and initial aphasia severity at the acute phase explain only a small percentage of variance in language recovery by the 90-day mark (Lazar et al., 2008). Furthermore, recovery mechanisms—specifically reorganization of functional networks (Hillis and Heidler, 2002)—continue beyond the subacute phase, and as such, identifying specific neural and behavioral factors that correspond to optimal acute to chronic longitudinal outcomes is crucial.

1.1. Anomia and naming errors as markers of severity
Anomia, or impaired word retrieval, is the most persistent deficit in long-term aphasia (Goodglass and Wingfield, 1997). While psycholinguistic models of lexical access (Dell et al., 1997; Dell & O’Seaghdha, 1992; Foygel and Dell, 2000; Indefrey and Levelt, 2004; Levelt et al., 1999; Rapp and Goldrick, 2000; Schwartz et al., 2006) differ in terms of...
information flow and levels of representation, most models propose separate semantic and phonological processing stages (cf. theories more aligned with parallel distributed processing accounts, e.g., Lambon Ralph et al., 2002; Plaut, 1995). It is believed that the types of errors patients make during naming attempts (along with patterns of performance across different tasks) reflect impairments within distinct semantic or phonological systems.

For example, semantic errors during object naming are those that are conceptually related to the target item and often manifest as category coordinate (e.g., cat for “rabbit”), category superordinate (e.g., vegetable for “asparagus”) or semantic associate (e.g., milk for “cow”) errors. In post-stroke aphasia, these errors are attributed to deficits in access or retrieval of semantic information, weak connections between semantic and lexical levels, and/or degraded lexical representations (Caramazza and Hillis, 1990; Dell et al., 1997; Foygel and Dell, 2000; Hillis and Caramazza, 1995; Jefferies et al., 2007, 2008; Jefferies and Lambon Ralph, 2006; Mirman and Britt, 2013; Schwartz et al., 2006). Phonological paraphasias most prototypically are non-word errors that either have some degree of phonemic overlap with target items (e.g., kittel for “kitten”) or are neologisms with little to no phonemic overlap with the target (e.g., flugger for “kitten”). These errors most often occur due to impaired access to phonological codes or errors in phonological assembly (Dell et al., 1997; Dell & O’Seaghdha, 1992; Schwartz et al., 2006). Mixed paraphasias (e.g., cat for “rat”) have semantic and phonemic similarity with the target item and occur due to breakdown at the semantic-word level (Dell et al., 1997; Foygel and Dell, 2006; Schwartz et al., 2006) or due to cascading activation and/or feedback connectivity between lexical and phonological layers (Rapp and Goldrick, 2000). Dell et al. (2004) proposed that omissions, or failures at attempting naming altogether, can occur due to breakdown at any point in the processing stream, yet some evidence exists for a semantic locus for such errors (Chen et al., 2019; Lambon Ralph et al., 2002).

Prior evidence indicates that certain types of object naming errors are more sensitive to anoma severity and are more likely to decrease with recovery than other error types. Specifically, low naming accuracy has been linked to a greater number of non-single word responses (omissions and circumlocutions) and words unrelated to the target item (Le Dorze and Nespoulous, 1989; Mitchum et al., 1990; Moerman et al., 1983; Schuell and Jenkins, 1961; Schwartz and Brecher, 2000). Based on the weight and decay computational model by Dell et al. (1997), Schwartz and Brecher (2000) proposed—and found evidence suggesting—that formal errors (i.e., phonologically-related real words), non-words and unrelated word errors occur most often in severe anoma but that semantic and mixed errors span the range of anoma severity. The literature (e.g., Basso et al., 1996; Mitchum et al., 1990; Schwartz and Brecher, 2000) generally suggests that severe anoma and production of predominantly semantic errors do not go hand in hand (cf. Rapp and Goldrick, 2000). As naming recovers over time, patients produce higher proportions of responses that are either semantically or phonologically related to target items (Basso et al., 1996; Crary and Kertesz, 1988; Kohn and Smith, 1994; Mitchum et al., 1990; Schwartz and Brecher, 2000), although error profile evolution varies between patients (Crary and Kertesz, 1988; Schwartz and Brecher, 2000). Naming deficit profiles identified early in recovery, such as the types of errors patients produce, also may be informative in predicting future language gains, although no study has explicitly tested this hypothesis.

1.2. Brain structure integrity and post-stroke language recovery

Although gross lesion metrics (e.g., size, stroke laterality) have been implicated in post-stroke recovery in general, the integrity of specific brain structures is critical for longitudinal recovery of specific language abilities—like object naming—in post-stroke aphasia. A key mechanism of recovery in acute stroke is reperfusion of hypoperfused tissue (Hillis and Heidler, 2002). With regards to language, Hills and coworkers (2001b) found that reperfusion of tissue within Brodmann Area (BA) 22 (Wernicke’s area, including most of the left superior temporal gyrus (LSTG)) caused improvement in lexical-semantics for production and comprehension tasks in hyperacute stroke. In a subsequent study, Hills et al. (2006) reported that early reperfusion of BA 22, BA 37 (i.e., left fusiform (LfFUG), posterior middle (LpMTG) and inferior temporal (LpITG) gyri and BA 44/45 (i.e., inferior frontal gyrus, pars opercularis (LIFGop) and pars triangularis (LIFGtri)) resulted in improved confrontation naming specifically. At a further level of granularity, DeLeon and coworkers (2007) reported that impaired naming due to deficits in amodal semantic processing correlated with dysfunctional tissue (damage and/or hypoperfusion) in BA 22, whereas impaired naming due to lexical access deficits was related to damage/hypoperfusion in BA 37; overall impaired oral naming corresponded to dysfunctional tissue in BA 22, 37 and 39 (i.e., angular gyrus (AG)).

Reorganization of systems-level brain structure-function relationships and the forging of new pathways and compensatory mechanisms mediate continued recovery in the weeks into years following stroke (Hillis and Heidler, 2002). With regards to long-term recovery, Hills et al. (2013) reported that improvement in overall naming from acute to chronic post-stroke stages was linked to less damage in posterior LSTG (LpSTG) and to the left arcuate fasciculus (LAF). The integrity of LAF also has been implicated in acute to chronic longitudinal recovery from aphasia in general (Hosomi et al., 2009; Jang and Lee, 2014; Kim and Jang, 2013) and in response to a variety of therapies in individuals with chronic aphasia (Breier et al., 2011; Holland et al., 2016; Schlaug et al., 2009; van Hees et al., 2014). Other studies in chronic aphasia have highlighted the importance of ventral white matter tracts, such as the left inferior longitudinal fasciculus (LLF) (Bonilha et al., 2016; McKinnon et al., 2017; Meier et al., 2019) in response to naming therapy. Cortical necrosis also impacts naming therapy outcomes: Fridriksson (2010) found that the strongest predictor of poor response to naming treatment was damage at the junction of BA 37, 39 and 19 in LpMTG.

These collective studies indicate that the integrity of specific brain structures, particularly the LAF and many temporal lobe areas, is crucial for longitudinal naming recovery. Naming errors provide a different—but also valuable—method to index anoma severity and recovery. Yet, the relative utility of brain structure and naming error metrics in capturing acute to chronic language recovery following LH stroke is unknown.

1.3. The current study

In this study, we propose that the types of object naming errors patients produce, combined with dysfunctional brain tissue metrics, can provide insight into recovery after a new LH stroke. Our primary goals pertain to stroke recovery, but we also investigated relationships between various language and demographic variables at the acute post-stroke stage to situate the longitudinal results. We addressed the following questions: 1) What are the relationships between naming error profiles, demographic variables (sex, age, and education), and naming accuracy in acute stroke? 2) Do global language abilities, overall naming accuracy, and naming error profiles change from early (acute/subacute) to later post-stroke recovery stages? 3) To what extent do error totals, demographic variables, and dysfunctional tissue metrics from an earlier stroke recovery time point predict future language skills and longitudinal change?

We hypothesized that a high proportion of errors that are related to the target (particularly in semantics) would be associated with better acute naming and predict future recovery and language gains, whereas a high proportion of unrelated errors would be associated with poorer acute and later-stage language recovery and less improvement. We also hypothesized that naming accuracy and global language skills would improve and that the proportion of unrelated errors would decrease from early to later post-stroke stages. Finally, we predicted that when considered together, acute naming errors and dysfunctional tissue
metrics (i.e., percentage of damage to regions of interest, hyperperfusion in vascular territories, and total lesion volume) would independently predict later-stage language skills and change in language abilities over time.

2. Materials and methods

2.1. Participants

As part of an ongoing longitudinal stroke recovery project, we studied patients who presented at Johns Hopkins Hospital or Johns Hopkins Bayview Medical Center within 48 h of onset of a new LH stroke. For the current study, exclusionary criteria were lack of premorbid proficiency in English, uncorrected vision and/or hearing, factors preventing participation in testing procedures (e.g., reduced level of consciousness, intubation), and a history of dementia or other neurological disease affecting the brain (excluding stroke). Our final sample included 148 individuals (69 women; mean age: 60.74 ± 13.23 years), seen between September 2012 and June 2020. Of note, 51 participants had a history of prior stroke per MRI report; these participants were included to achieve a sample representative of the larger stroke population, as more than 20% of Americans who experience a stroke each year have a history of prior infarct (Benjamin et al., 2019). In subsequent sections, we describe the steps we took to account for multiple strokes in our analyses. Demographic information, including age, handedness, and years of education, is summarized in Table 1.

Study protocols adhere to the Declaration of Helsinki and were approved by the Institutional Review Board of Johns Hopkins University. Participants or their healthcare proxies (for patients with impaired language comprehension) provided written informed consent to all study procedures.

2.2. Language assessment

Participants completed a language battery at acute (≤11 days, n = 140), subacute (2 weeks-5 months, n = 40), early chronic (5-7 months, n = 41) and/or late chronic (≥11 months, n = 33) post-stroke stages. At each study time point, we administered either the Western Aphasia Battery-Revised (WAB-R; Kertesz, 2007) or Boston Diagnostic Aphasia Examination-3 (BDAE-3; Goodglass et al., 2001) to capture patients’ global language deficits. We used scores from similar subtests of verbal expression and auditory comprehension from each measure to generate within-measure language z-scores at each time point. We calculated z-scores according to the standard formula (x - μ)/σ, where x was the patient’s subtest score at a given time point and μ and σ were the sample mean and standard deviation, respectively, for a given subtest across time points. We averaged subtest z-scores to generate a single global language z-score for each participant at each time point. If a participant had multiple timepoints (e.g., acute and subacute or both chronic time points), we incorporated the earliest (time point 1) and latest (time point 2) data points into the calculations.

We used the 30-item version of the Boston Naming Test (BNT) (Fisher et al., 1999) to measure confrontation naming abilities. Each item was scored as either correct (1) or incorrect (0). For incorrect items, a trained speech-language pathologist (CRH) coded errors using the 10 classifications from Goodglass et al. (2001) and conferred with other study authors (ELM and SMS) regarding challenging cases. In the event that a patient provided multiple responses, the final, most complete response was coded. We then condensed errors into four main error types. Semantic errors included verbal paraphasias semantically-related to the target such as category coordinates (e.g., “hammer” for saw, “squirrel” for beaver) and semantic associates (e.g., “explosion” for volcano, “pyramid” for sphinx) as well as circumscriptions that described a semantic feature of the target (e.g., “used in math” for protractor). Phonological errors included nonword and real-word phonemically-based paraphasias with greater than 50% preservation of target phonology (including phonemic transpositions and phonemes within one position of its target position) and neologisms with less than 50% overlap with the phonology of the target word. Mixed errors included single-word responses that were semantically related to the target and retained approximately 50% of the target word phonology (e.g., “elevator” for escalator) and multi-word responses that included a semantic and phonological error, as described previously. Unrelated errors included verbal (real word) paraphasias unrelated to the target, multi-word paraphasic errors, off-topic utterances including omissions (e.g., “I don’t know”), perseverations, and perceptual errors (e.g., “ghost” for octopus).

Another trained speech-language pathologist (EBG) who was blinded to the original error codes also completed error-coding using the four-code system. Inter-rater reliability according to Cohen’s weighted kappa was excellent, ranging from 0.911 to 0.979 (p < 0.0001) across time points. For each participant, we calculated error totals as well as the proportion of each error type (sum of each error type divided by the total number of errors) at every time point. In certain analyses, we used error type proportions (rather than error totals) in order to mitigate the confound of anomia severity on the results. Table 1 provides a summary of language performance at each time point.

2.3. Neuroimaging data

During their acute hospitalization, participants underwent a clinical imaging protocol that included diffusion-weighted imaging (DWI), T1-weighted, and fluid attenuated inversion recovery (FLAIR) sequences.3 Trained technicians manually delineated acute lesions slice-by-slice on DWIs for patients in MRIcron (https://www.nitrc.org/projects/mricron). We traced prior strokes on FLAIR scans for participants with a history of previous infarct per the clinical MRI report.

Images were normalized to MNI space using SPM12 (Statistical Parameter Mapping; https://www.fil.ion.ucl.ac.uk/spm/software/spm12/). For acute lesions, the DWI b0 image was warped to a template from older healthy adults (Rorden et al., 2012), and normalization parameters were applied to the corresponding acute lesion maps. For prior strokes, we used the MR segment-normalize routine in Clinical Toolbox (Rorden et al., 2012). Within this pipeline, FLAIR images and the corresponding lesion maps were coregistered to T1-weighted images; the lesion was masked out via enantiomorphic warping; and segmentation and bias correction routines from SPM12 were applied to the anatomical image. The corrected T1-weighted image and lesion map were warped to the same template used in normalization of the DWIs.

For patients with prior stroke, we combined the normalized acute and chronic lesion maps into one total lesion map in MarsBaR (Brett et al., 2002). We used NiStat (https://www.nitrc.org/projects/nistat/) to determine acute and total stroke volumes. Most prior strokes were small (mean size: 1.944 ± 2.766 cc) in the participants who completed follow-up testing and were included in brain-behavior prediction

3 We remotely administered follow-up assessments to one participant because we were unable to complete in-person testing during the COVID-19 pandemic.
2 WAB-R subtests included Information Content; Fluency, Grammatical Competence, and Paraphasias; Yes/No Questions; Auditory Word Recognition; Sequential Commands; Repetition; Object Naming; Word Fluency; Sentence Completion; and Responsive Speech. BDAE-3 subtests included Simple Social Responses; Word Comprehension; Complex Ideational Material; Automatized Sequences; Repetition of Single Words; Repetition of Sentences; and Responsive Naming.
3 Four participants had contraindications for MRI and instead received a CT scan. Due to differences between CT and MR-DWI in detecting acute stroke, these patients were excluded from imaging analyses.
analyses. Acute and total lesion volumes did not significantly differ in the subgroup of participants with multiple strokes who completed follow-up testing ($t = -0.168, p = 0.868$). Nonetheless, to ensure that prior stroke did not unduly influence the results, we extracted the percentage of damaged tissue in regions of interest (ROIs) from niiStat based on the intersection of combined (acute plus prior stroke) lesion maps with template regions for patients with prior stroke. We also controlled for total lesion volume in all lesion analyses. ROIs included 21 cortical regions implicated in aphasia recovery (Fridriksson et al., 2018), including left middle frontal gyrus (LMFG), LIFGtri, LIFGop, LIFG pars orbitalis (LIFGorb), anterior insula (Alinsula), posterior insula (PInsla), portions of the basal ganglia (putamen, globus pallidus), precentral gyrus (PrCG), postcentral gyrus (PoCG), LSTG, LMTG, LITG, the superior and middle temporal poles (LSTpole, LMTpole, respectively), LpSTG, LpMTG, LpITG, supramarginal gyrus (LSMG), LAG, and middle occipital gyrus (LMOG) extracted from the Johns Hopkins University (JHU) atlas (Faria et al., 2012; Mori et al., 2008) as well as four white matter tracts, i.e., LAF, LIIIF, inferior fronto-occipital (LIFO), and uncinate (LUIF) fasciculi, extracted from the atlas by Catani and Thiebaut de Schotten (2008).

In addition to infarct visible on DWI, hypoperfusion surrounding the infarct (the ischemic penumbra) contributes to deficits in patients with acute stroke (Beaulieu et al., 1999; Hillis et al., 2006; Hillis et al., 2001a, b; Sorensen et al., 1996). Although perfusion-weighted imaging (PWI) is often included in clinical scanning protocols in suspected infarcts, it is not always performed due to time constraints and/or contraindication for intravenous contrast. Due to lack of flow void, vessels that supply hypoperfused regions appear hyperintense on T2-weighted sequences such as FLAIR. As such, as an alternative to PWI, we estimated hypoperfusion in six vascular territories within the left hemisphere (anterior cerebral artery, posterior cerebral artery, middle cerebral artery (MCA)-frontal, MCA-temporal, MCA-insular, MCA-parietal) using the NIH-FLAIR Hyperintense Vessel (FHV) scoring system (Reyes et al., 2017). Within this system, raters provide a score for each vascular territory that reflects the number of FHVs, where $0 = 0$ FHVs, $1 = 1–2$ FHVs and $2 = 3$ or more FHVs.

### 2.4. Statistical analyses

All analyses were conducted in R (R Core Team, 2020). To address aim #1, we determined relationships between acute language variables (i.e., error type proportions and total correct on the BNT) and demographic variables using non-parametric statistics, either Wilcoxon rank sum tests (for sex) or Spearman correlations (for age and years of education). To further explore acute naming error profiles, we used Spearman correlations to determine the relationships between the different types of acute error proportions as well as associations between error type proportions and naming accuracy.

To address aim #2, we determined change in naming over time using a binomial linear mixed effects model predicting item accuracy (coded as 1s and 0s) from fixed effects of time point (acute, subacute, early chronic, and late chronic), lexical frequency of items (from Brookshire and Nicholas, 1995), age, and education with random intercepts for participants and items. We determined change in global skills by conducting a Welch’s two-sided t-test. We used analysis of covariance (ANCOVA) to determine if error type proportions—reflecting different naming error profiles—changed from early (acute/subacute) to later time points, controlling for the percentage of coded errors, days between testing time points, age, and education.

To address aim #3, we first conducted four linear regression analyses to determine if the total number of each error type produced at an early recovery stage (acute/subacute) predicted either: 1) later-stage naming accuracy (i.e., total correct items on the BNT at the latest testing time point), 2) global language skills (i.e., z-scores at the latest testing time point), 3) change in naming accuracy (i.e., the difference in BNT total correct at the latest versus the earliest time points), or 4) change in global language skills (i.e., the difference in z-scores at latest versus the earliest testing time points). Within each of these models, we controlled for days between testing time points, age, years of education, and the percentage of coded errors.

To gain insight into neurobehavioral profiles of recovery, we conducted four Least Absolute Shrinkage and Selection Operator (LASSO) regression analyses, predicting either later-stage language skills (naming accuracy or global language skills) or change in language (naming or global language skills) from first to later time points. Predictors included error totals and demographic variables significant in prior analyses, dysfunctional tissue metrics (i.e., percentage of damaged tissue in ROIs, FHV ratings and total lesion volume) and nuisance variables (days between testing time points, percentage of coded errors). LASSO regression is suitable for models with predictors that exhibit high levels of multicollinearity, which was likely in the present study due to the fact that adjacent ROIs often are damaged in conjunction. LASSO regression adds a penalty that shrinks coefficients towards zero, resulting in a simple model with maximal prediction capacity. We ran 5000 permutations and a cross-fold validation equal to $n$ per LASSO model. We excluded brain metrics (percent damage to ROIs, FHV ratings) for which $<10\%$ of the sample had values indicating damage or dysfunction.

For results significant at an uncorrected threshold, we performed multiple comparison correction using the false discovery rate ($q < 0.05$) at the model level across correlation, ANCOVA and linear regression analyses.

### 3. Results

#### 3.1. Relationships between error type proportions, demographic variables and naming in acute stroke

At the acute post-stroke time point, naming errors were recorded for

Table 1
Demographic and language information.

|                | Time 1 |          | Time 2 |          |
|----------------|--------|----------|--------|----------|
|                | $n$    | Range    | Mean   | SD       |
| Age            | 148    | 28.05–91.43 | 60.74 (13.23) |  |
| Years of education | 145    | 5–21     | 13.89 (2.88) |  |
| Handedness (R:L) | 136:12 |          |        |          |
| BNT (total correct) | 148    | 0–30     | 19.32 (8.99) | 63 |
| Language z-scores | 130    | –3.27–0.63 | –0.100 (0.958) | 61 |

Notes: $n$ reflects the number of participants for whom data were available at each time point. Time 1 corresponds to the earliest data point, equivalent to the acute time point for 140 patients and the subacute time point for 8 patients. Time 2 corresponds to the latest data point in participants with follow-up testing, either the subacute (n = 11), early chronic (n = 19) or late chronic (n = 33) time point. R = right, L = left.
115 participants. On average, participants produced a greater proportion of semantic (62.15 ± 36.02%) and unrelated (25.25 ± 31.28%) errors compared to phonological (10.97 ± 18.04%) and mixed (1.57 ± 7.09%) errors. Error type proportions did not differ between men and women (q ≥ 0.572) and were not related to either age (q ≥ 0.120) or years of education (q ≥ 0.359) (see Supplemental Table 1 for complete results). Acute BNT accuracy did not vary by sex (W = 1538.5, p = 0.534) or age (r = 0.009, p = 0.926), but there was a trending, weak association between total correctly-named items and years of education (r = 0.167, p = 0.078).

A higher proportion of semantic errors was strongly associated with lower proportions of phonological (r = −0.572, q < 0.001) and unrelated (r = −0.824, q < 0.001) errors and weakly associated with a lower proportion of mixed errors (r = −0.261, q < 0.009). A greater proportion of phonological errors weakly correlated with a greater proportion of mixed errors (r = 0.241, q = 0.014). We found an insignificant relationship between unrelated and mixed error proportions (r = 0.037, q = 0.698) but a trending association between unrelated and phonological error rates (r = 0.175, q = 0.073). A higher proportion of semantic errors was associated with better naming accuracy (r = 0.550, q < 0.001), whereas higher proportions of phonological (r = −0.367, q < 0.001), mixed (r = −0.219, q = 0.019), and unrelated (r = −0.554, q < 0.001) errors were associated with poorer naming.

3.2. Change in language over time

The linear mixed effects model revealed overall naming accuracy significantly improved from early to later recovery stages (β = 0.597, SE = 0.046, t = 13.003, p < 0.001), controlling for lexical frequency of target items (β = 0.007, SE = 0.003, t = 2.607, p = 0.009), age (β = 0.013, SE = 0.022, t = 0.577, p = 0.564), and education (β = 0.334, SE = 0.104, t = 3.222, p = 0.001). Global language skills (according to summary z-scores) also significantly improved from early to later recovery stages (β = −4.859, q < 0.001). In contrast, controlling for the percentage of coded errors per time point, days post-stroke onset, age and years of education, the proportion of semantic (F1,92 = 1.176, p = 0.281), phonological (F1,92 = 0.012, p = 0.914), mixed (F1,92 = 1.604, p = 0.209), and unrelated (F1,92 = 0.782, p = 0.379) errors that patients produced did not change significantly over time (see Supplemental Table 2 for full ANCOVA models). The null findings may have been caused by patients with mild anoma with higher proportions of semantic and lower proportions of other error types, leading to little room for group-level change. As such, we re-ran the analysis only with patients whose early-stage naming accuracy fell within the lowest quartile, i.e., fewer than 15 items correct (n = 18). However, there was still no significant effect of time for any error type (p ≥ 0.086) in the subsample. Supplemental Table 3 presents summary data for overall language skills, naming, and error types for each discrete time point (i.e., acute, subacute, 6-months, and 12-months post-stroke) for the longitudinal errors sample.

3.3. Predictions of language recovery and gains by error types, demographic variables and dysfunctional tissue measures

In the remainder of analyses, we used the total number of each error type—rather than error type proportions—in order to capture the severity of underlying impairments. Mixed errors were excluded from the analyses as no patients produced more than one mixed error. Therefore, predictors within the multivariable regression analyses included the total number of semantic, phonological, and unrelated errors, in addition to demographic variables previously-cited as critical for aphasia recovery (age and education), days between testing time points, and percentage of coded errors.

The regression model predicting later-stage naming accuracy (i.e., total correct on the BNT at the latest time point for each patient) was significant (F7,49 = 9.608, q < 0.001, adjusted R2 = 0.518). Within this model, controlling for other factors, fewer total unrelated errors produced early in recovery (β = −0.762, SE = 0.124, t = −6.155, p < 0.001) and more years of education (β = 0.831, SE = 0.293, t = 2.834, p = 0.007) predicted better future naming. The multivariable model predicting recovery of global language skills (i.e., z-scores at the latest testing time point) also was significant (F7,46 = 5.642, q < 0.001, adjusted R2 = 0.380), and within this model, fewer early-stage unrelated errors (β = −0.955, SE = 0.011, t = −4.865, p < 0.001) predicted better later-stage overall language abilities. See Table 2 for the full results of regression models predicting recovery of naming and global language skills.

The multivariable model predicting change in naming over time (i.e., total correct on the BNT at the latest minus the earliest time points) was significant (F7,49 = 7.694, q < 0.001, adjusted R2 = 0.456). Controlling for both other model factors, more early-stage semantic (β = −0.737, SE = 0.248, t = 2.976, p = 0.005), phonological (β = 1.265, SE = 0.361, t = 3.503, p = 0.001) and unrelated (β = 0.423, SE = 0.103, t = 4.106, p < 0.001) errors—as well as more years of education (β = 0.858, SE = 0.244, t = 3.520, p < 0.001)—predicted greater naming improvement. The model predicting change in global language abilities (i.e., language z-scores from the latest minus the earliest time points) was significant (F7,42 = 4.808, q < 0.001, adjusted R2 = 0.352) as well; controlling for other factors, the only significant independent predictor was unrelated errors, such that more unrelated errors at an early stage predicted greater gains in overall language skills (β = 0.588, SE = 0.011, t = 5.225, p < 0.001). Complete results for the longitudinal change prediction models are reported in Table 3.

Fig. 1 shows the lesion overlay for the patients included in the follow-up analyses including dysfunctional tissue measures. Of note, 12 participants had some degree of damage in the right hemisphere (in addition to left hemisphere damage), mostly within deep subcortical structures. Nine participants had infarct in either the cerebellum or brainstem in addition to cerebral involvement. To inform the LASSO regressions, we used ANOVA to determine if language scores varied by broad lesion classifications (i.e., LH-only, bilateral, or other [cerebellum or brainstem with cerebral involvement]). We found no significant relationships between lesion group and BNT total correct at the latest time point (W = 221.5, p = 0.680), language z-scores at the latest time point (W = 223, p = 0.588), change in BNT (W = 259, p = 0.672) or change in z-scores (W = 135, p = 0.338). As such, we did not include lesion classification in the LASSO regressions.

Table 4 includes the complete results from the LASSO models. Controlling for other model variables, we found that fewer acute unrelated errors, more years of education, and less damage to LAG significantly predicted better recovery of naming by the final testing time point. Less damage to LAG was the sole significant predictor of recovery of global language abilities, controlling for other factors. The model predicting change in naming abilities over time included many variables; controlling for these factors, more acute unrelated errors and less damage to LAG predicted greater naming gains. On the other hand, patients who produced a greater number of acute unrelated errors and had damage in LpMTG demonstrated the greatest improvement in global language skills from early to later stroke recovery stages.

4. Discussion

We investigated the relationship between types of naming errors, dysfunctional brain tissue, and acute to chronic post-stroke recovery of naming and language abilities. In acute stroke, a greater proportion of semantic errors was associated with higher naming accuracy, but the
opposite pattern (i.e., higher proportions, lower accuracy) was true of other error types. Naming and global language abilities improved from early to later post-stroke recovery stages, but naming error profiles (i.e., error type proportions) did not evolve over time. The strongest and most consistent predictors of language recovery and longitudinal change were the number of unrelated errors produced early in recovery, years of education, and the integrity of tissue within temporoparietal cortex.

4.1. Naming error profiles

In order to better understand the role of naming errors in recovery, we first investigated relationships between error type proportions and language and demographic variables in acute stroke. We found that a higher proportion of semantic errors produced at the acute time point was significantly associated with lower proportions of other error types and with better naming accuracy. On the other hand, a higher proportion of phonological errors coincided with higher proportions of mixed and unrelated errors, and poorer acute naming abilities correlated with higher proportions of these three error types. In other words, when patients made errors in the acute stage, they were better off if they produced more semantic errors relative to other error types.

These results partially align with prior work and also provide novel insights into the nature of semantic errors in acute stroke. In this study, semantic errors included verbal paraphasias related to target items (often a semantic feature of the target). These errors indicate some degree of successful semantic access, which may reflect a stronger lexical-semantic system and a higher likelihood of successful lexical access in general. The strong positive relationship between semantic error proportions and naming accuracy supports this interpretation and coheres with data suggesting individuals with mild anomia (often a semantic feature of the target) and with better naming accuracy. On the other hand, a higher proportion of phonological errors coincided with higher proportions of mixed and unrelated errors, and poorer acute naming abilities correlated with higher proportions of these three error types. In other words, when patients made errors in the acute stage, they were better off if they produced more semantic errors relative to other error types.

Table 2

| Dependent variable | Multivariable model | Univariate predictor | β     | SE    | t-stat | p-value |
|--------------------|---------------------|----------------------|-------|-------|--------|---------|
| BNT total at T2    | (7,49)              | Semantic             | -0.465| 0.298 | -1.561 | 0.125   |
|                    | 9.608               | Phonological         | 0.151 | 0.434 | 0.347  | 0.730   |
|                    |                     | Unrelated            | -0.762| 0.124 | -6.155 | <0.001  |
|                    |                     | T2-T1days            | 0.001 | 0.004 | 0.392  | 0.697   |
|                    |                     | Age                  | 0.041 | 0.063 | 0.647  | 0.521   |
|                    |                     | Education            | 0.831 | 0.293 | 2.834  | 0.007   |
|                    |                     | %coded               | 0.127 | 0.033 | 3.879  | <0.001  |
| Language z-score at T2 | (7,46) | Semantic             | 0.016 | 0.027 | 0.592  | 0.557   |
|                    | 5.642               | Phonological         | 0.045 | 0.040 | 1.112  | 0.272   |
|                    |                     | Unrelated            | -0.055| 0.011 | -4.865 | <0.001  |
|                    |                     | T2-T1days            | -0.0002| 0.0003| -0.475 | 0.637   |
|                    |                     | Age                  | 0.005 | 0.006 | 0.883  | 0.382   |
|                    |                     | Education            | 0.047 | 0.028 | 1.683  | 0.099   |
|                    |                     | %coded               | 0.007 | 0.003 | 2.397  | 0.021   |

Notes: BNT = Boston Naming Testing, T2 = second testing time point, T1 = first testing time point, T2-T1days = number of days between testing time points, %coded = percentage of errors coded at time point 1. Semantic/Phonological/Unrelated reflect error totals of each type at T1.

Table 3

| Dependent variable | Multivariable model | Univariate predictor | β     | SE    | t-stat | p-value |
|--------------------|---------------------|----------------------|-------|-------|--------|---------|
| Δ in BNT total (T2-T1) | (7,49) | Semantic             | 0.737 | 0.248 | 2.976  | 0.005   |
|                     | 7.694               | Phonological         | 1.265 | 0.361 | 3.503  | <0.001  |
|                     |                     | Unrelated            | 0.423 | 0.103 | 4.106  | <0.001  |
|                     |                     | T2-T1days            | 0.005 | 0.003 | 1.639  | 0.108   |
|                     |                     | Age                  | 0.0007| 0.052 | 0.014  | 0.989   |
|                     |                     | Education            | 0.858 | 0.244 | 3.520  | <0.001  |
|                     |                     | %coded               | -0.072| 0.027 | -2.638 | 0.011   |
| Δ in language z-score (T2-T1) | (7,42) | Semantic             | 0.031 | 0.027 | 1.168  | 0.249   |
|                     | 4.808               | Phonological         | 0.023 | 0.039 | 0.596  | 0.554   |
|                     |                     | Unrelated            | 0.058 | 0.011 | 5.225  | <0.001  |
|                     |                     | T2-T1days            | 0.0001| 0.0003| 0.276  | 0.784   |
|                     |                     | Age                  | 0.004 | 0.006 | 0.723  | 0.474   |
|                     |                     | Education            | 0.007 | 0.027 | 0.269  | 0.790   |
|                     |                     | %coded               | -0.004| 0.003 | -1.217 | 0.220   |

Notes: Δ = change, BNT = Boston Naming Testing, T2 = second testing time point, T1 = first testing time point, T2-T1days = number of days between testing time points, %coded = percentage of errors coded at time point 1. Semantic/Phonological/Unrelated reflect error totals of each type at T1.
errors (Grima and Franklin, 2017; Nicholas et al., 1989; Tallberg, 2005). However, this result does not reflect that semantic errors (or mixed errors) are severity-insensitive like some researchers (e.g., Dell et al., 1997; Schwartz and Brecher, 2000) previously proposed; given the relationship we found, it is unlikely that we would find similar semantic error totals among patients with mild, moderate and severe anomia. Furthermore, we found a much higher proportion of semantic errors compared to other error types, unlike Basso et al. (1996), who reported only 3.6% of errors as being semantic in patients within the first four months of recovery. Similarly, only two (of 28) patients made predominantly semantic errors within one month of stroke in Mitchum et al. (1990). Reasons for this discrepancy could be differences between studies in stroke duration (i.e., on the order of days in our study versus months in the others), semantic error definitions (e.g., inclusion of circumlocutions in our study but not in others, e.g., Schwartz and Brecher, 2000) and anomia severity. The latter point is especially likely since we recruited patients who experienced a new left hemisphere stroke but did not necessitate that they present with persistent anomia and aphasia. A central conclusion that can be drawn from our acute findings is that the production of semantic errors alone does not necessarily reflect a severe semantic deficit. Instead, it is more likely that low performance on neuropsychological assessments of semantics, combined with a high total number of semantic errors, truly indicate an impaired semantic system.

Our results linking low naming correctness with high phonological and unrelated error proportions are more consistent with prior work (Le Dorze and Nespoulous, 1989; Mitchum et al., 1990; Moerman et al., 1983; Schuell and Jenkins, 1961; Schwartz and Brecher, 2000), with some important caveats. For example, Schwartz and Brecher (2000) provided more nuanced distinctions between types of phonological errors (e.g., close versus remote neologisms, formal (real-word) errors versus nonwords), whereas we grouped these error types together. Although outside the primary scope of this paper, future work delving into differences between phonological error types in terms of phonemic overlap and lexicality and their relationship with naming correctness could complement prior findings. Similarly, we classified unrelated errors as responses with essentially no semantic or phonological overlap with targets, including many different types of errors: off-target utterances (e.g., “couple of flowers” for volcano), omissions (e.g., “I don’t know”), perseverations and perceptual errors. Although this error type somewhat serves as a catch-all category, this classification method makes conceptual sense, given that patients who produce any of these errors likely have a weaker lexical system and/or demonstrate other deficits that interfere with lexical retrieval. Chen et al. (2019) proposed different probable causes for omission errors, including core deficits within the semantic system (either deficient semantic representations or disconnect between semantic representations and lexical items), impairments in correct selection of competing lexical representations, deficits in the speech motor system and deficits in other perceptual (e.g., visual) or cognitive systems. Based on our limited language battery, we cannot specify the locus of impairment for unrelated errors (or the subtypes therein), but doing so represents an important future direction of this work.

In terms of longitudinal change, we found that overall naming ability and global language skills significantly improved from early (acute/subacute) to later post-stroke stages. However, error type proportions did not change over time, even for patients with the most severe naming impairments. It may be that the total number of each error type changes over time (reflecting overall accuracy) but that the types of errors most individual patients produce does not (Capitani and Laiacoana, 2004). That being said, prior studies (Basso et al., 1996; Crary and Kertesz, 1988; Kohn and Smith, 1994; Mitchum et al., 1990; Schwartz and Brecher, 2000) have reported evolution in error profiles. Using a different approach than ours, Crary and Kertesz (1988) reported that change in error types over the first year post-stroke mirrors the transition from one aphasia syndrome to another. Schwartz and Brecher (2000) found phonologically-related errors declined from the first to later testing points in patients with chronic aphasia and noted a general shift away from phonological and towards semantic error types. In contrast, Basso et al. (1996) found that the total numbers of no responses and neologisms patients produced during oral naming tasks decreased over time. One possible reason our results conflict with these latter two studies is that we investigated changes in error proportions, rather than error totals, to comment on error profiles without the added confound of anomia severity. Anecdotally, 17 patients did exhibit an increase of at least 20% in semantic error proportions (and a corresponding decrease

### Table 4

| Metric | T2 BNT total | T2 Language z-scores | Δ in BNT T2-T1 | Δ in z-scores T2-T1 |
|--------|--------------|----------------------|---------------|---------------------|
|        | Coefficient  | p-value              | Coefficient   | p-value             |
| T2-T1 days | –             | –                    | –             | –                   |
| Phonological errors | –0.180     | 0.017               | –             | –                   |
| Unrelated errors | 0.911     | 0.003               | –             | –                   |
| % coded errors | –             | –                    | –0.364       | 0.051               |
| Years of education | 0.321     | < 0.001             | 0.257         | 0.093               |
| % damage LIFGop | –             | –                    | –1.117       | 0.024               |
| % damage LIFGorb | –             | –                    | –0.225       | 0.240               |
| % damage LpCG | –             | –                    | –0.219       | 0.362               |
| % damage LrCG | –             | –                    | –0.091       | 0.619               |
| % damage LSMG | –             | –                    | 0.572        | 0.183               |
| % damage LAG | –0.395       | < 0.001             | –0.806       | < 0.001             |
| % damage LMOG | –             | –                    | 0.214        | 0.242               |
| % damage LpMTG | –             | –                    | –0.423       | 0.092               |
| % damage LUF | –             | –                    | 0.109        | 0.519               |
| FHV L MCA-frontal | –             | –                    | 0.155        | 0.466               |
| FHV L MCA-insula | –             | –                    | 0.189        | 0.448               |
| FHV L MCA-parietal | –             | –                    | 0.019        | 0.526               |
| Total lesion volume | –             | –                    | –            | –                   |

Notes: BNT = Boston Naming Test, T2-T1 days = number of days between first and final testing time points, L = left, p = posterior, IFGop = inferior frontal gyrus, pars opercularis, IFGorb = IFG, pars orbitalis, PoCG = postcentral gyrus, PrCG = precentral gyrus, SMG = supramarginal gyrus, AG = angular gyrus, STG = superior temporal gyrus, MTG = middle temporal gyrus, MOG = middle occipital gyrus, UF = uncinate fasciculus, FHV = FLAIR hyperintense vessel score, MCA = middle cerebral artery territory. Δ denotes change from time point 1 (T1) to time point 2 (T2). Bold font indicates significant results at p < 0.05.

The table shows the error type and dysfunctional tissue predictors of language recovery.
in other error types, especially unrelated errors) from early to later time points, but 8 patients exhibited the inverse, a shift from semantic to unrelated errors. The greater implication of our findings is that error profiles might appear resistant to change at a group level, especially in a sample of patients with varying levels of aphasia and anomaly severity, like in the current study. On an individual level, however, the types of errors patients produce are amenable to change and are likely related to global language recovery to some extent. In future studies, taking a case series approach and including in-depth neuropsychological assessment can aid in determining locus of breakdown and potential underlying causes of different error patterns in individual patients.

4.2. Predictions of longitudinal naming and global language recovery and change

Regardless of the lack of longitudinal change, error types produced at an earlier time point did predict future language recovery by the final testing time point and change in language over time. Specifically, the number of early unrelated errors was a significant predictor within all linear regression models, and semantic and phonological error totals also predicted BNT change. When error totals were considered in conjunction with demographic variables and dysfunctional tissue metrics, the number of acute unrelated errors remained a significant predictor in all models excluding the global language recovery analysis. Of note, the direction of the effects varied between recovery (i.e., performance at the latest time point) and change (i.e., the difference between scores at the latest versus earliest time points) models. The naming and global language recovery models indicated that fewer unrelated errors produced at an earlier time point predicted better language skills at a later time point. In acute stroke, low naming accuracy was significantly associated with a high proportion of unrelated errors, and therefore, it stands to reason that patients with very impaired acute naming skills may still have exhibited the most severe impairments at later recovery stages. On the other hand, patients who produced more errors at an earlier time point exhibited the greatest change in language over time. This finding likely reflects that patients who produce more early errors have the greatest room for growth. Most importantly, these results highlight the importance of further investigation into the origins of unrelated errors, as identifying their underlying cause can help tailor naming treatment protocols for patients who produce many such errors.

Within the combined error type and dysfunctional tissue models, the integrity of temporoparietal cortex was the most consistent significant neural predictor of recovery. Specifically, less damage to LAG at the acute stage predicted higher naming accuracy and global language scores at a future recovery stage as well as greater naming gains. Many structural and functional connections converge in the left temporoparietal cortex, and this area is considered a critical cross-modal hub for many processes, semantics in particular (Binder et al., 2009; Binder and Desai, 2011; Davey et al., 2015; Humphreys and Lambon Ralph, 2015; Lambon Ralph et al., 2016; Noonan et al., 2013; Seghier, 2012; Whitney et al., 2012; Xu et al., 2016, 2017). As such, it stands to reason that the preservation of tissue within LAG is crucial for receptive and expressive language skills. However, somewhat counterintuitively, patients who had highly-damaged nearby LpMTG demonstrated the greatest improvement in global language skills over time. In a recent lesion symptom mapping study of a largely-overlapping acute cohort, we found that greater damage to LpMTG and higher FHV parietal ratings were associated with acute naming deficits. In this cohort, as expected, more severe acute anoma was significantly associated with more severe global language impairments ($r = 0.750$, $p < 0.001$). Consequently, the association between LpMTG damage and greater language gains could merely reflect a pattern of LpMTG damage and very low acute language performance in certain patients, and that these individuals had more room for improvement than patients with higher acute scores. Alternatively, given LpMTG’s role as a network hub and the importance of temporoparietal cortex in normal lexical-semantic processing (Noonan et al., 2013; Xu et al., 2016, 2017)—and in people with chronic aphasia (Fridriksson, 2011; Griffis et al., 2017a, 2017b)—it may be that the brain prioritizes functional reorganization of temporoparietal-mediated processes in the event of acute dysfunction (whether restricted diffusion or hypoperfusion), ultimately leading to better long-term outcomes.

The regression models also yielded some interesting findings regarding the predictive power of other variables. We found no associations between education and global language skills, which aligns with the literature suggesting that education level is not a robust predictor of overall aphasia recovery (Lazar et al., 2008; Plowman et al., 2012; Watila and Balarabe, 2015). Although we found only a trend between years of education and acute naming performance, education level consistently predicted future naming skills. Higher levels of education have been implicated in preservation of lexical-semantics for picture naming in healthy older adults (Connor et al., 2004; Le Dorze and Durocher, 1992; Neils et al., 1995; Paoli et al., 2018) and in cerebral reserve in the face of brain pathology (Staff et al., 2004). Consistent with the literature (see the review by Watila and Balarabe, 2015), smaller total lesion volume strongly correlated with better acute ($r = −0.721$, $p < 0.001$) and later-stage ($r = −0.530$, $p < 0.001$) global language skills, yet lesion volume was retained only in the BNT change LASSO regression model as an insignificant predictor. The LASSO procedure heightens predictive power by creating a sparse model containing the most optimal variables; therefore, it can be inferred that the other variable combinations—without total lesion volume—provided the best predictions of language recovery and change over time. Lesion size has long been considered a key determinant of longitudinal stroke outcomes. Yet the retention of error variables over lesion volume in the LASSO models indicates that the types of errors patients produced early in recovery—particularly unrelated errors—are even better predictors of post-stroke language recovery outcomes. These findings further emphasize the importance of early evaluation of naming and characterization of naming errors in patients following left hemisphere stroke.

4.3. Study limitations

The current study has several limitations. At each time point, certain participants had incomplete language datasets. For some participants, naming errors were not recorded for the entire BNT or were not recorded at all. To compensate for this issue, we controlled for the percentage of cued errors and excluded patients without any recorded errors in analyses. We also were unable to compute language summary z-scores for participants who completed only a handful of subtests from the WAB-R or the BDAE-3, and such participants were excluded from global language recovery analyses.

A related issue is that we were unable to collect data for all participants for all time points. Attrition at the follow-up time points was high (only 57 of 148 patients had longitudinal error data), mostly due to unsuccessful repeated contact attempts or participant transportation difficulties. Additional reasons for attrition after the acute post-stroke stage included subsequent stroke, heart attack, relocation to an assisted living facility and subsequent loss of contact, move to a location out of state to live with family, and conflicts due to rehabilitation schedules. We acknowledge that the high rate of attrition from the acute to subsequent time points is a major limitation of the current study; as such, future work is necessary to validate the current findings.

We were also unable to collect acute behavioral data for eight participants (due to medical issues, early discharge, or other time constraints), and as such, the earliest time point for these patients was the subacute phase. Given that language recovery is most rapid in the acute to subacute post-stroke stages (Hillis and Heidler, 2002), including participants without acute data may have influenced the results. Therefore, we replicated all analyses from aims #2 and #3 that used early stage data (i.e., combined acute and subacute data) and excluded participants without acute behavioral data. The main findings remained unchanged (see Supplementary Material, Tables 4–7).
One key difference between our study and prior studies is that we included participants with a history of prior stroke and participants with brain damage to regions outside of the left cerebral hemisphere. We included these participants because our primary goal was to further understanding of post-stroke language recovery (rather than informing the neurobiology of language or psycholinguistic models, per se) in a sample more representative of the stroke population than those of prior studies. Recent work by our group indicates that history of prior stroke on its own is not a significant determinant of language outcomes but that accounting for total brain damage and lesion location—as we have done in the present study—is crucial.

5. Conclusions

In this study, we found that compared to other error types, a high proportion of semantic errors produced at the acute post-stroke stage was associated with more intact naming abilities. While lexical retrieval and global language skills (specifically auditory comprehension and verbal expression) improved from early to later post-stroke stages, the proportions of error types did not change over time. Errors classified as unrelated, including omissions, off-topic utterances, perseverations, and perceptual errors, had the best predictive utility of any error type. Indeed, the most predictive factors of longitudinal improvement in naming abilities and global language skills were fewer unrelated errors produced early in recovery, in addition to the integrity of LAG. On the other hand, patients with more preserved LAG who produced more unrelated errors improved most in naming over time. Patients who demonstrated the greatest gains in global language skills from early to later post-stroke stages also produced a higher number of unrelated errors but had damage to the posterior middle temporal cortex, perhaps because they had lower baseline scores and greatest room for improvement. Considering the detrimental impact of unrelated errors on language, future studies aimed at identifying the underlying cause of unrelated errors in individual patients and determining treatment protocols to shift persistent naming errors away from unrelated and towards the semantic type are warranted.

Author contributions

Erin L. Meier, Conceptualization, Methodology, Validation, Formal analysis, Investigation, Data curation, Writing - original draft. Shannon M. Sheppard, Conceptualization, Methodology, Validation, Writing - review & editing. Emily B. Goldberg, Validation, Formal analysis, Investigation, Data curation, Writing - review & editing. Catherine R. Head, Methodology, Validation, Data curation, Writing - review & editing. Delaney M. Ubellacker, Investigation, Data curation, Writing - review & editing. Alexandra Walker, Investigation, Data curation, Writing - review & editing. Argye E. Hills, Conceptualization, Resources, Writing - review & editing, Supervision, Funding acquisition

Declaration of competing interest

None.

Acknowledgments

This work was supported by the National Institutes of Health, National Institute on Deafness and Other Communication Disorders through grant R01DC005375.

We extend our thanks to members of the Stroke Cognitive Outcomes and REcovery (S.C.O.R.E.) Lab who assisted with participant recruitment and data collection. We also gratefully acknowledge our participants who made this work possible.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.neuropsychologia.2020.107651.

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