The relationship of lesion location, resting-state EEG and aphasia severity in post-stroke aphasia

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Background

With up to 64% of stroke survivors meeting criteria for expressive and receptive language difficulties[1,2], impairments in speech production after stroke can leave devastating impacts on mental health, daily functioning and overall quality of life.[3]

Resting state electroencephalographic (EEG) data have the potential to provide insight into the neural mechanisms of speech in participants with post-stroke aphasia. Recent evidence demonstrates that using spectral power, participants with chronic stroke exhibit increased delta (0.5-3.5 Hz) and theta (4-7.5 Hz) and decreased alpha (8-12.5 Hz), beta (13-30 Hz), and gamma (>30 Hz) band activities during resting eyes-closed and opened conditions compared to controls.[4]

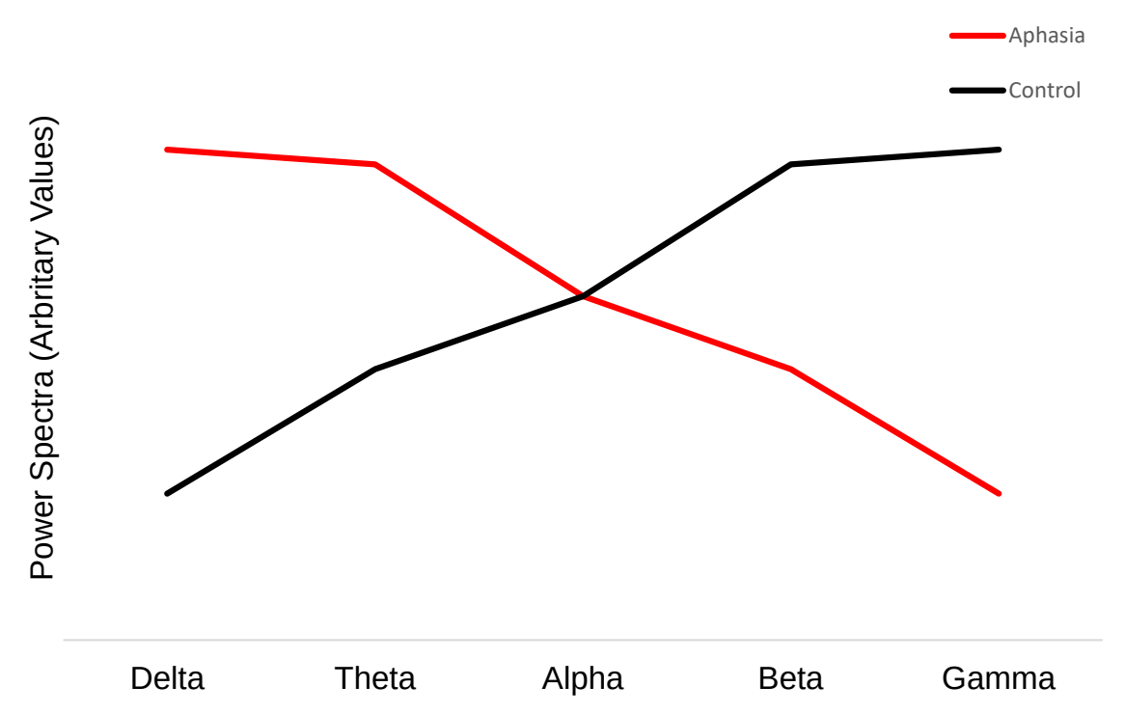

Figure 1: General trend of resting state data (both eyes closed, and eyes opened) conditions in participants with post-stroke aphasia.

However only a few studies have investigated the role of EEG data in relation to the language impairments that are present in left-hemisphere stroke survivors.[4,5,6,7] In this pilot study, we investigated the role of spectral power during the resting state (i.e., eyes-closed and eyes-opened conditions) and its relationship with lesion location and language impairment as a consequence of left-hemisphere stroke.

Research Aims

1. Examine differences in spectral resting state EEG (rsEEG) during eyes-closed and eyes-opened conditions between post-stroke aphasia and neurologically intact controls.
2. Investigate the association between spectral rsEEG and behavioral measures of language impairment due to left-hemisphere stroke.
3. Explore the relationship between spectral rsEEG, behavioral measures and lesion location in post-stroke aphasia.

Methods

Participants: Eight participants with chronic left-hemisphere stroke (four females, age range: 47.58-70.83 years, M: 61.16 years, SD: 6.99 years) and 4 neurologically intact control participants (3 females, age range: 43.75-68, M: 56.96, SD: 10.01) were recruited from the Aphasia Lab and C-STAR at the University of South Carolina. One aphasia participant's EEG data was considered noisy during data preprocessing and rejected from further analysis. The distribution of aphasia sub-types, based on WAB-R scores, were as follows: Anomic = 1; Broca's = 4; Conduction = 1; and 2 above the cut-of.

Data Acquisition: Participants were seated in a sound-attenuated booth where EEG signals were recorded during rest (see Figure 2) for approximately 10 minutes. EEG signals were recorded using BrainVision actiCHamp amplifier (Brain Products Gmbh, Germany) and EEG caps with 64 electrodes using standard 10-10 montage.

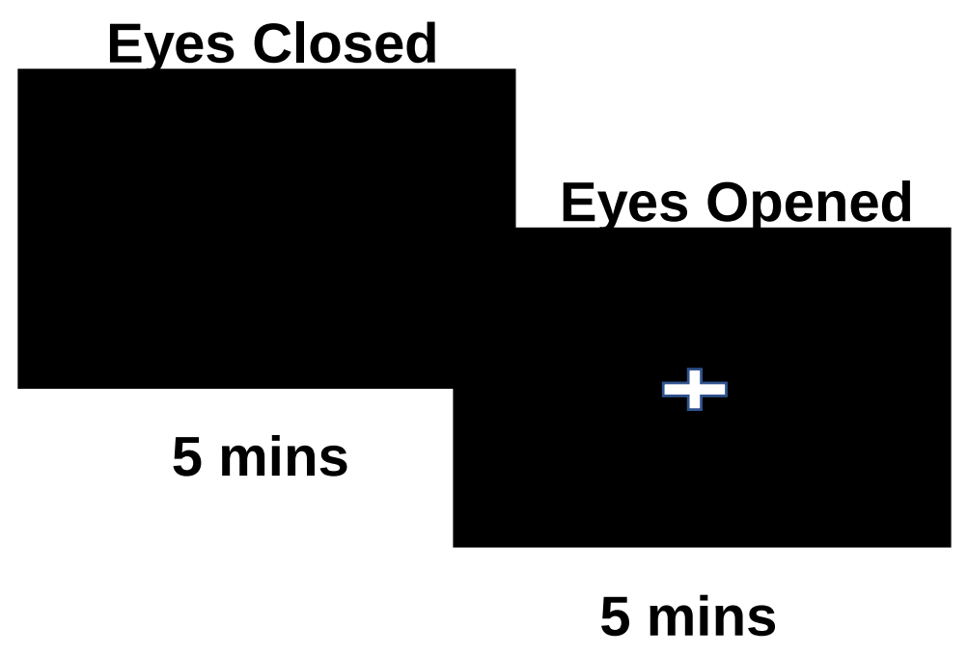

Figure 2: Experimental set-up. Participants were asked to sit still with their eyes closed for five minutes and eyes opened and fixated on the white cross in the middle of a black computer screen.

Structural MRI data were acquired using a 3T Siemens Trio scanner fitted with a 12-channel head-coil and used for demarcating left-hemisphere lesions in stroke participants.

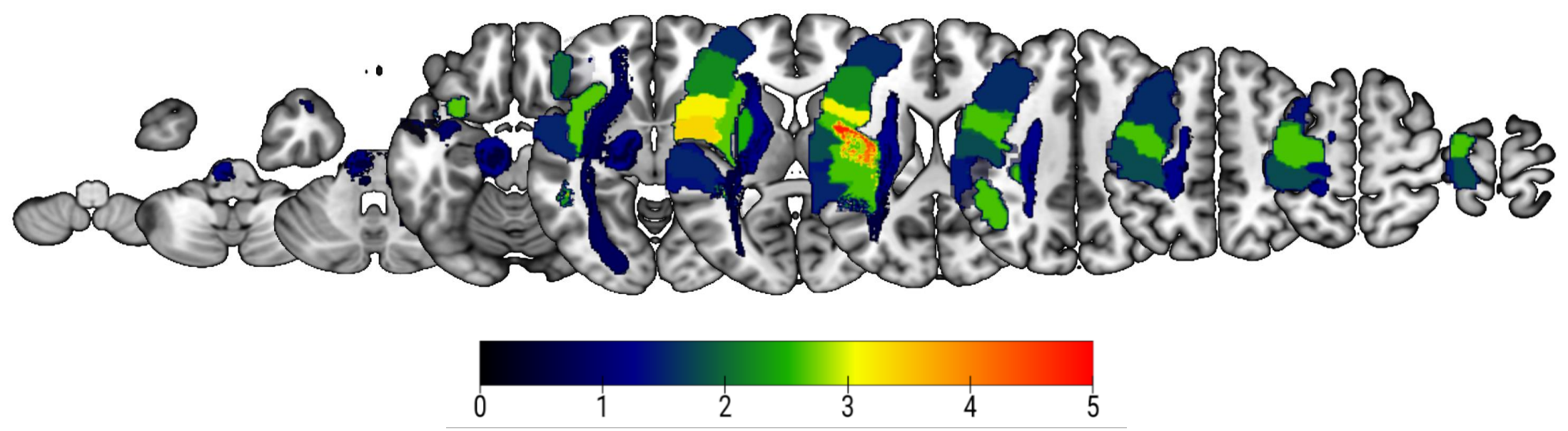

Figure 2: Lesion overlap maps in individuals with post-stroke aphasia (n = 6).

Methods (continued)

Data Analysis: EEG data were pre-processed using EEGLAB toolbox.[8] Using FieldTrip[9], power spectral density (PSD) was then calculated from pre-processed and epoched (2-s) EEG data for delta (0.5-3.5 Hz), theta (4-7.5 Hz), alpha (8-12.5 Hz), low beta (13-20.5 Hz), high beta (21-30 Hz), and low gamma (30-50 Hz) frequency bands. Regions of interest (ROIs) for lesion mapping analysis and electrodes of interest were chosen based on previous literature regarding implicated networks in speech and language and post-stroke aphasia.[4,7,10,11]

Statistical Analysis: Statistical analyses were performed using IBM SPSS (version 28.0). NiiStat (https://www.nitrc.org/projects/niistat) was used for lesion mapping analysis. The significance threshold was set at $p < .05$. Bonferroni corrections were applied where appropriate to control for errors due to multiple comparisons. As non-normality was observed (via Kolmogorov-Smirnov), Mann-Whitney *U* test and Spearman Rank Order Correlation were utilized as non-parametric statistics. ROIs chosen for analysis were pre-central gyrus (PreCG.L), inferior frontal gyrus (IFG), opercular part (IFGop.L), IFG, triangular part (IFGtriang.L), IFG, orbital part (IFGorb.L), Rolandic operculum (ROL.L), left insula (Ins.L) inferior parietal (IPL.L), supramarginal gyrus (SMG.L), angular gyrus (ANG.L). ROI means were calculated between lesion locations in at least 6 out of 7 participants and power spectra during eyes-closed and eyes-opened conditions. Based on the 10-10 International EEG system, we focused on power spectra related to the frontal, fronto-temporal, fronto-central, central, centro-parietal and parietal networks.

Results

Figure 3 shows the averaged topographical distribution maps from delta to low-gamma frequency bands between participants with aphasia (PWAs) and controls during eyes-closed and eyes-opened conditions. Following Mann-Whitney U tests for both conditions, there were no significant differences between the PWAs and control participants in any frequency bands and conditions.

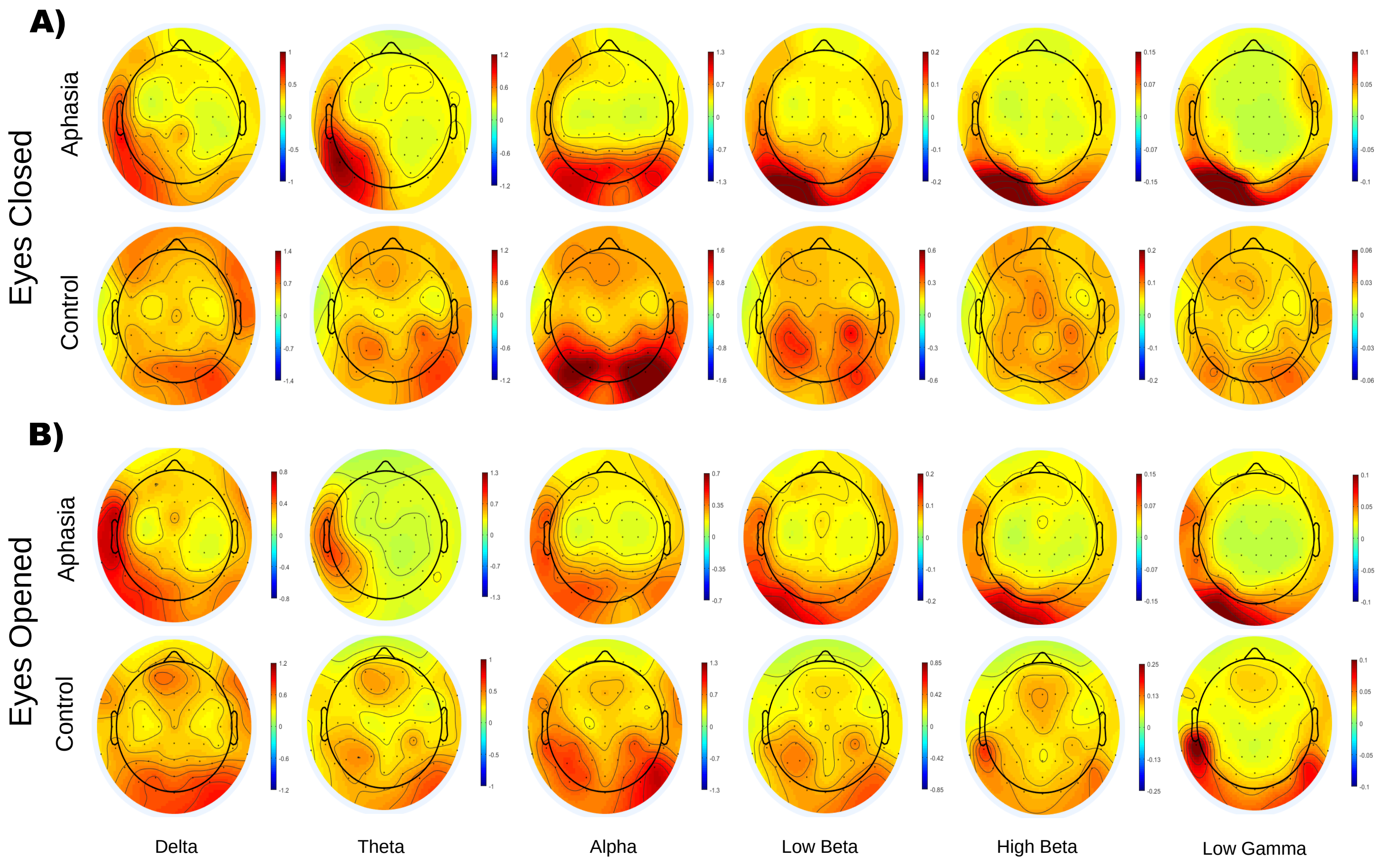

Figure 3: Topographical distributions for **A)** eyes-closed and **B)** eyes-opened conditions in speech-language network representative electrodes averaged across left-hemisphere stroke (n=7), and control (n = 4) participants in delta (0.5-3.5 Hz), theta (4-7.5 Hz), alpha (8-12.5 Hz), low beta (13-20.5 Hz), high beta (21-30 Hz) and low gamma (30-50 Hz).

Figure 4 displays correlation matrices for medium- (*rho* = .30 to .49) and large- (*rho* ≥ .5) sized Spearman correlation coefficients (Cohen, 1992) with $p < .05$ between WAB-R aphasia quotient and sub-item scores and rs-EEG using PSD in delta, alpha, low beta, high beta and gamma bands for PWAs. Following Bonferroni corrections, significant ($p < .001$) correlations were found between WAB-R object naming and repetition sub-items and right-hemispheric centro-parietal, parietal and temporal regions in the alpha frequency band during the eyes-closed condition. Similarly, significant ($p < .001$) correlations were found between WAB-R object naming sub-item scores and right-hemispheric centro-parietal and parietal regions in the alpha frequency band.

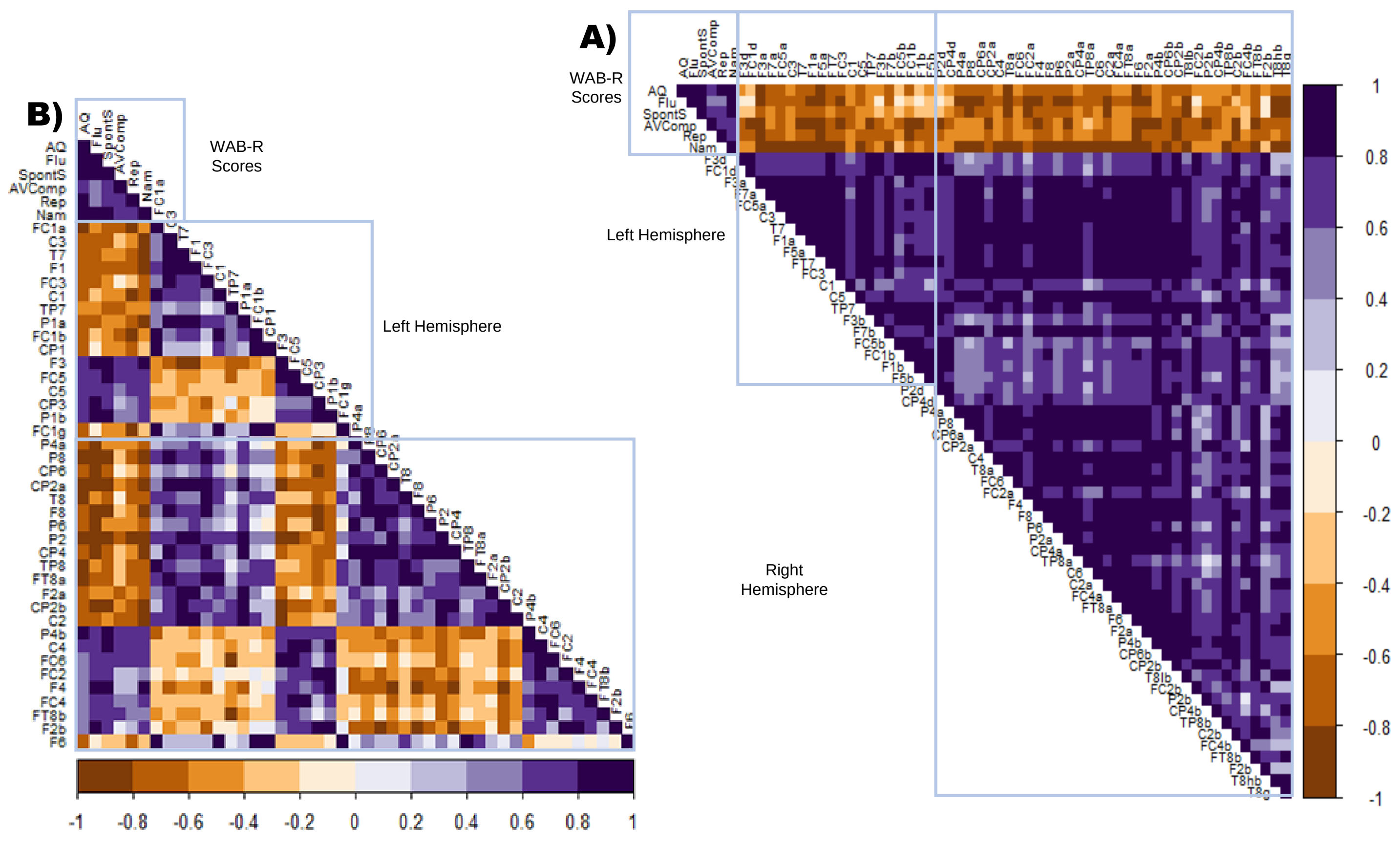

Figure 4: Correlation matrices for targeted correlations in the **A)** eyes-closed and **B)** eyes-opened conditions. Color bars indicate the strength of the correlation coefficient.

Results

ROI-based lesion mapping analysis revealed that 6 out of 7 aphasia participants had overlapping lesions in the left precentral gyrus (PreCG.L), left inferior frontal gyrus, pars opercularis (IFGob.L), Rolandic operculum (ROL.L) and left insula (Ins.L). There were significant ($p < .05$) positive correlations between lesions in the IFGob.L and ROL.L and left frontal and fronto-central alpha activity, right parietal alpha, low-beta, and low-gamma bands, and centro-parietal delta, alpha, and low-beta bands during the eyes-closed condition. Significant ($p < .05$) correlations were also observed between the Ins.L and right parietal, tempero-parietal, centro-parietal regions in both delta and alpha bands during both eyes-closed and opened. There were no significant Bonferroni-corrected correlations ($p \leq .001$).

Figure 5 shows correlations between WAB-R AQ and sub-item scores and ROI-based lesion locations. Similar to the rs-EEG data, there were no significant ($p \leq .005$) correlations between WAB-R scores and lesion locations after Bonferroni corrections.

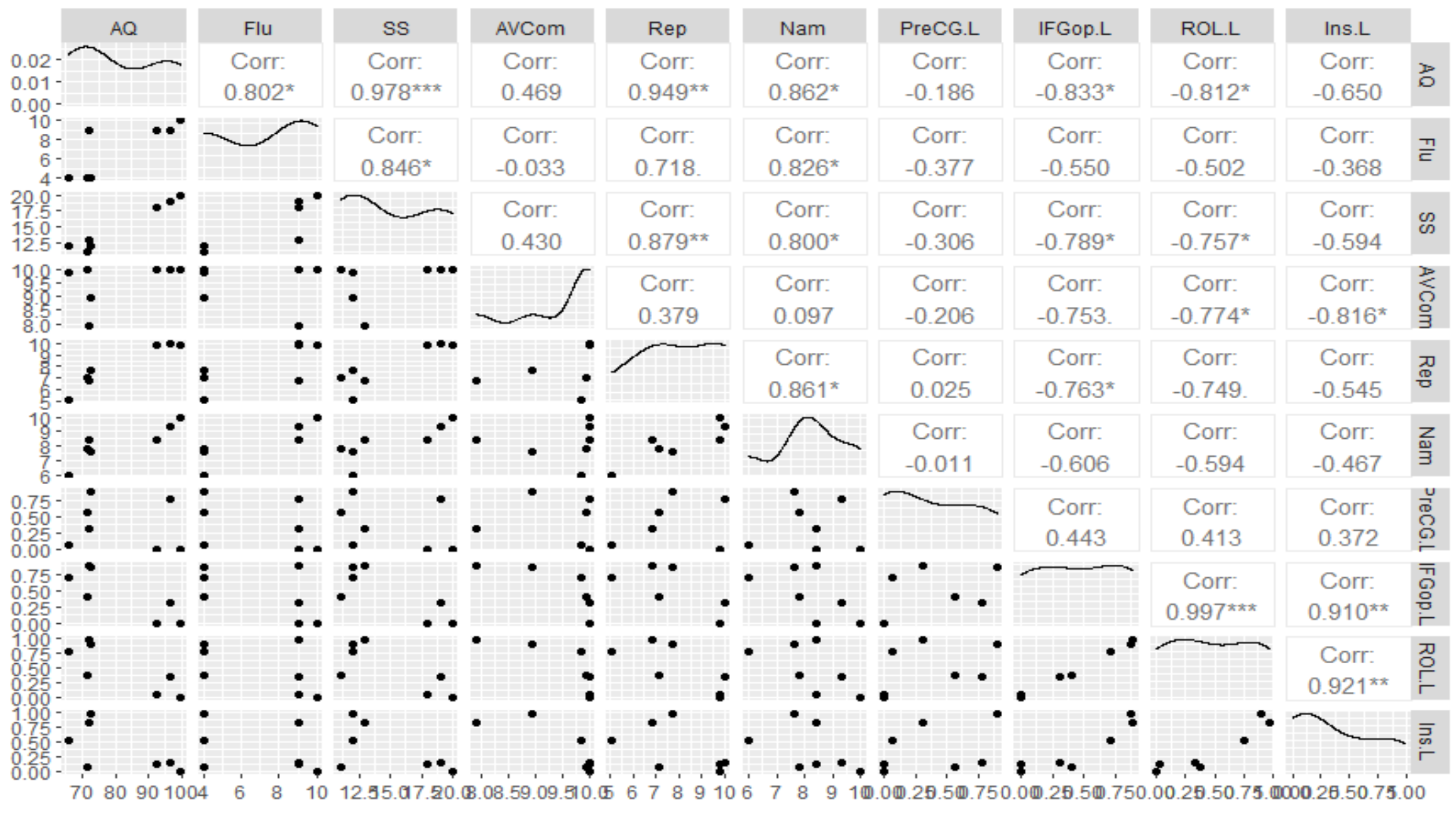

Figure 5: Correlation plots for WAB-R scores and ROI means for lesion locations in at least 6 out of 7 participants with left-hemisphere stroke. AQ= WAB-R aphasia quotient, Flu = Fluency, SS = Spontaneous speech, AVCom = Auditory verbal comprehension, Rep = Repetition, Nam = Naming, PreCG.L = Left pre-central gyrus , IFGop.L = Left inferior frontal gyrus, opercular part, ROL.L = Rolandic operculum, Ins.L = Left Insula. *$p<.05$, **$p<.01$, ***$p<.001$.

Conclusions

These preliminary results suggest increased theta power in the left temporal region, and decreased alpha, low- and high-beta, and low-gamma power in the language network as compared to neurologically intact controls. These findings are in line with previous results and demonstrate decreased alpha, low- and high-beta, and low-gamma power in resting state conditions.

Interestingly, an inverse relationship between behavioral measures of language impairment and rs-EEG power spectra was found, particularly in the eyes-closed condition, in higher frequency bands and in right centro-parietal, parietal and temporal regions. These results suggest compensatory neural responses in contra-lateral regions, reflecting functional re-organization of the language network.[12,13,14]

Using ROI-based lesion mapping analysis, there was a general positive relationship observed primarily between lesions in the left-hemispheric IFGob.L, ROL.L, Ins.L and right-hemispheric parietal, tempero-parietal and centro parietal regions in delta, alpha, low-beta and gamma bands. Following behavioral measures, there was an inverse relationship between WAB-R scores (primarily aphasia quotient, spontaneous speech, auditory comprehension) and IFGob.L and ROL.L.

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