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Association Between the Beta Band Neural Response and the Behavioral Performance in Aphasic and Neurologically Intact Individuals

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Association Between the Beta Band Neural Response and the Behavioral Performance in Aphasic and Neurologically Intact Individuals

by

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Abstract

The complex motor act of speech requires integrating linguistic and sensorimotor processes. Sensorimotor interaction mainly supports speech production in the form of state feedback control architecture. While speaking, subjects react to perturbations in the pitch of voice auditory feedback by changing their tone in the opposite direction to pitch-shift stimuli to compensate for the perceived pitch shift. Aphasia is a communication impairment affecting patients' speaking, understanding, reading, and writing. The present study aims to examine the association between brain neural activity and the ability for speech auditory feedback error correction in both post-stroke aphasia and neurologically intact individuals. There are 34 aphasic individuals (age: 61+/-11.2 years) and 46 neurologically intact participants (age: 64+/-7.9 years). Participants were asked to produce a vowel sound /a/ under altered auditory feedback (AAF) during the experiment. The vocalization signals were recorded, while EEG signals were simultaneously recorded from 64 scalp electrodes following a standard 10-10 montage. A multivariate linear regression model was fitted to examine the association between the EEG beta band power (13-25 Hz) and the vocal compensation. Results show a significant negative linear association between the two variables under the downward pitch-shift AAF condition. However, such association is not detected for the upward pitch-shift AAF condition. In addition, the aphasia group had significantly reduced power of beta band de-synchronization compared with controls. The findings of the current study reveal that diminished neural de-synchronization of the beta band activities is related to the poorer performance on speech auditory feedback error correction under the downward pitch-shift AAF condition. Such relation is not found under the upward pitch-shift AAF condition. The reason might be that the

participants failed to reach the desired low F0 trajectory under the upward pitch-shift stimulus. Furthermore, the beta band de-synchronization is diminished for the aphasia group compared to the healthy control group, suggesting that aphasic individuals have deficits in the underlying neural mechanisms for the sensorimotor system.

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Chapter 1

Introduction

Speech is a complex medium that conveys our thoughts and mediates cognition. It not only refers to speech production (i.e., overt speech) but also refers to covert self-talk (i.e., inner speech). The complex motor act of speech requires the integration of linguistic (i.e., lemma retrieval and selection, phonological code retrieval, and phonological encoding) and sensorimotor processes (e.g., articulatory control and feedback processes)(Alderson-Day and Fernyhough 2015). Previous research has found that, while speaking, subjects react to perturbations in the pitch of voice auditory feedback by changing their tone in the opposite direction to pitch-shift stimuli to compensate for the perceived pitch shift (Xu et al. 2004) (Behroozmand et al. 2012).

It has been demonstrated that persistent alterations in sensory feedback results in sensorimotor adaptation in the vocal motor systems(Nasir and Ostry 2008). According to Hickok (2012), sensorimotor integration in the domain of speech has two main ideas. First, the auditory system is critically involved in speech production. Second, the motor system is critically involved in speech perception. Sensorimotor interaction mainly supports speech production in the form of state feedback control architecture (Hickok 2012). Figure 1 Hickok et al., (2012) shows the mechanism of the sensorimotor integration.

Aphasia is a communication impairment affecting patients' speaking, understanding, reading, and writing. Aphasia is often induced by stroke damage to the left side of the brain (Black-Schaffer and Osberg 1990; Engelter et al. 2006; Flowers et al. 2016; Hilari 2011). It affects over 2 million people in the US. Speech sensorimotor

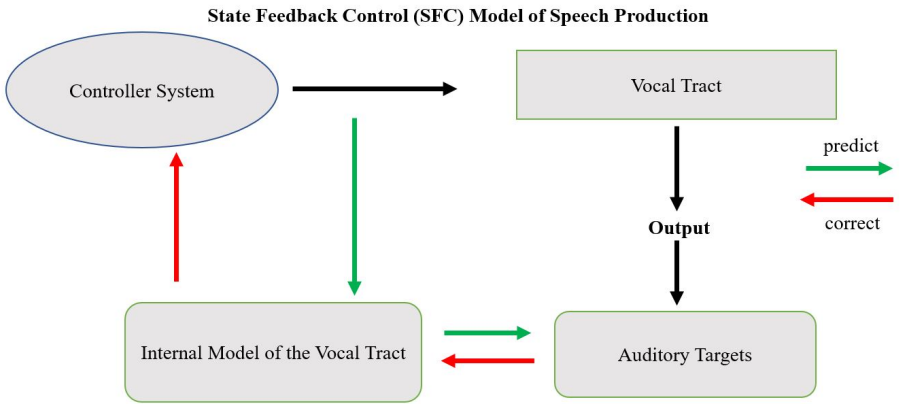

Figure 1.1: The controller generates motion commands to the vocal tract. In the meantime, it also sends the command to the internal model, which forward predicts the dynamic state of the vocal tract and the sensory results of these states. The deviation between the predicted auditory state and the actual sensory feedback generates an error signal that is used to correct and update the internal model of the vocal tract.

integration for speech error processing is impaired in stroke patients with aphasia due to damage to the left hemisphere of the brain.

Impairment of the sensorimotor network that supports auditory feedback processing in the speech process causes certain aspects of aphasia behavior disorder (Buchsbaum et al. 2011; Hickok and Poeppel 2000; Hickok et al. 2000; 2011). These aspects of aphasia behavior disorder include several subtypes of aphasia, such as conduction aphasia, anomic aphasia, and Broca's aphasia. Conduction aphasia, characterized by phonetic errors and difficulty in repetition, is the result of damage to the sensorimotor integration circuit, which leads to the limitation of speech auditory representation and the impairment of the ability to guide corresponding pronunciation representations (Buchsbaum et al. 2011). Other subtypes of aphasia, such as Broca's and anomic, also have the characteristics of errors in speech production and impairment with repetition (Fridriksson et al. 2008, 2009, 2016). Broca aphasia is associated with damage to frontal motor-related regions (Hillis 2007). Impaired motor planning and programming of motor speech are frequently observed in Broca's aphasia (Hillis

2007). Anomic aphasia is thought to be caused by a disconnection between sensory language processes and semantic knowledge of objects [17].

An EEG test is helpful for research on human sensorimotor processing as it uses the small, metal electrodes attached to people's scalp to trace electrical activation of brain areas involved in sensorimotor processing EEGs are typically non-invasive and painless yet provide fine temporal resolution. Previous studies have investigated the neural base of sensorimotor adaption that record electroencephalogram (EEG) signals during altered auditory feedback (AAF) paradigms(Behroozmand and Sangtian 2018). The authors use event-related potential (ERP) recordings to investigate the neural mechanisms of sensorimotor adaptation in response to altered auditory feedback (AAF) during vocal production and they find that adaptive vocal responses are negatively correlated with ERPs over the parietal and positively correlated with those over the fronto-central areas after vocalization onset. The electroencephalogram (EEG) contains information about the brain; hence the sub-band decomposition of EEG is used for analyzing many brain diseases. There are several methods for the EEG decomposition, such as Fourier transformation. EEG signal could be decomposed to various brain waves with different frequency bands, such as gamma band, beta band, alpha band, theta band, delta band, etc.

Transient increases and decreases in spectral power recorded in the human EEG have been traditionally termed event-related synchronization (ERS) and desynchronization (ERD), respectively (Leocani et al. 1997; Pfurtscheller and Aranibar 1977; Pfurtscheller et al. 1996). Cortical beta-band oscillations are actively involved in sensorimotor processing. Beta-band oscillations exhibit event-related desynchronization (ERD) during active states of the sensorimotor system (Vinding et al. 2019).

1.1 Thesis Goals

This thesis aims to identify the association between the magnitude of the beta band de-synchronization and the vocal compensation for healthy and aphasic individuals.

Simulation studies are conducted to assess the performance of the general linear regression and the multivariate linear regression models in detecting the true association between the beta band power and the vocal compensation. The values of sensitivity and specificity are calculated to compare the performance of the models.

Chapter 2

Experimental Design and Data

2.1 Alter Auditory Feedback (AAF)

An online altered auditory feedback (AAF) stimulus is used to simulate overt speech errors in the current study. It functions in the way that externally induces a mismatch between the predicted and overtly detected speech. One of the most common types of AAF is a pitch-shift stimulus (PSS). The PSS involves experimentally changing the subject's vocal pitch feedback to them over earphones (Larson 1998). Delivering PSS generates a mismatch (or error) between the internally predicted and actual speech feedback, which the auditory system detects (Behroozmand et al.,2009, Behroozmand et al., 2016 Greenlee et al., 2013). Figure 2.1 shows the altered auditory feedback process.

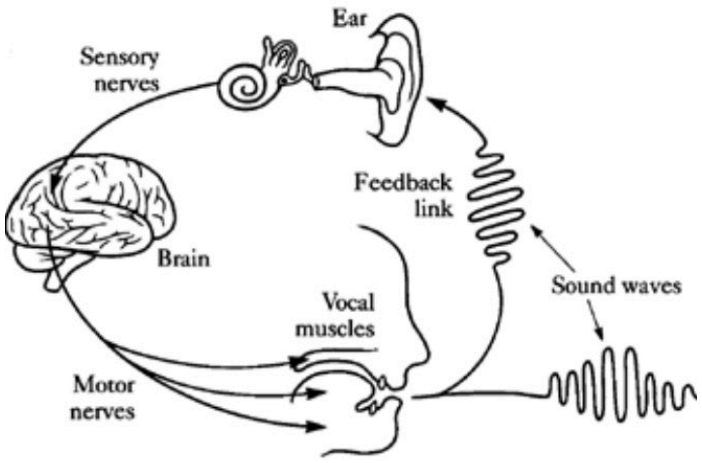

Figure 2.1: Pitch-Shift-Stimulus(PSS): 100-110 trials per pitch-shift stimulus direction

2.2 Experimental Design

The brain signals involved in speech sensorimotor processing of both aphasia and neurologically intact participants are investigated by using an altered auditory feedback (AAF) experiment. There are 34 aphasics (average age is 59) and 46 neurologically intact participants (average age is 64) participate in this study. The process of the experiment is shown in Figure 2.2.

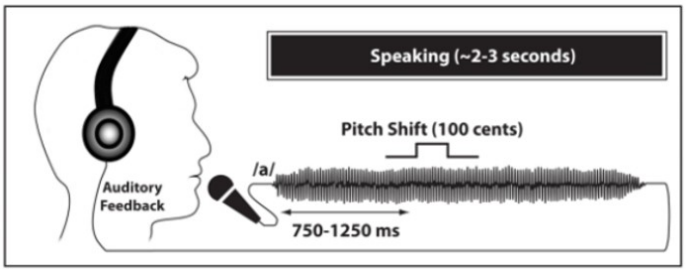

Figure 2.2: Pitch-Shift-Stimulus(PSS): 100-110 trials per pitch-shift stimulus direction. Two types of pitch-shift stimuli (PSS) are applied to elicit altered auditory feedback: upward and downward pitch shift. Following the PSS, the 34 participants are asked to produce the steady speech vowel sound /a/ at a constant pitch for 2-3 seconds after seeing a "GO" visual cue on the screen. Each participant is recorded 200-220 trials of the speech signals (and 100-110 trials per PSS direction). A 200 ms PSS with randomized $\pm$100 cents magnitude is delivered during each speech production trial to alter speech auditory feedback online. The onset time of PSS randomly falls into the time interval between 750 and 1250 ms after speech onset.

The experiment procedures, including recruitment, data acquisition, and informed consent, are approved by the University of South Carolina Institutional Review Board.

2.3 EEG Data Preprocessing

The 10-20 system is an internationally recognized method to describe and apply the location of scalp electrodes in the context of an EEG exam. Most of the newer EEG systems, such as the 64 channels system, are derived from this system. In the current study, the EEG signals are recorded from 64 sites on the subjects' scalp using the Brain Vision active electrode system (Brain Products GmbH, Germany) placed on an electrode cap (Easy-Cap GmbH, Germany) with the standard 10-20 montage.

Data are recorded using a typical average reference with a BrainVision actiCHamp EEG amplifier (Brain Products GmbH, Germany) in PyCorder software at a 1 kHz sampling rate after applying a low-pass anti-aliasing filter with a sampling frequency of 200Hz.

The layout of the EEG electrodes used in the current experiment is shown in Figure 2.3.

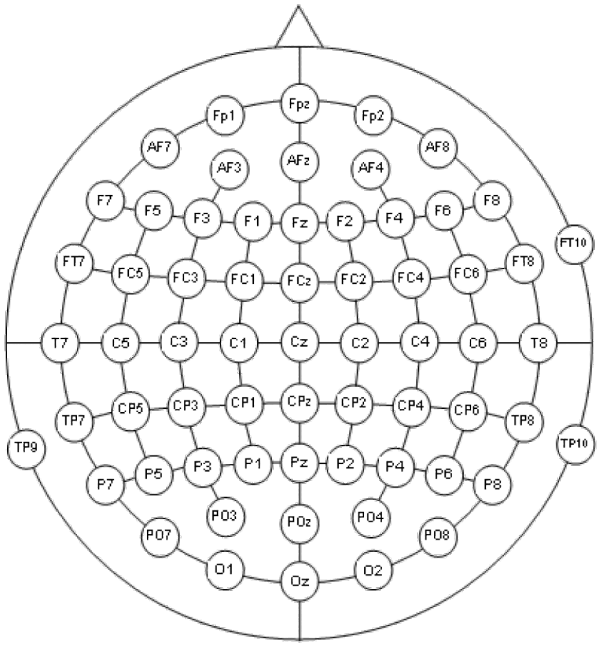

Figure 2.3: The figure shows the locations and the names of the 64 EEG sites on the subjects' scalp using the Brain Vision active electrode system.

A band-pass filter is used to extract the beta band neural activities from the EEG signal. We applied the Independent Component Analysis (ICA) to remove eye movement, blinks, muscle, and line noise artifacts. Then the EEG signals were segmented into epochs ranging from -200 ms before to 1000 ms after the onset of the pitch-shift stimulus. Individual beta-band EEG data are then subjected to baseline correction by removing the amplitude of the pre-stimulus time window from -200 to 0 ms for each channel. The choice of the baseline is based on visual inspection to

identify the pre-stimulus time window within which the induced neural activities are relatively stable. A complex Morlet wavelet transformation (1-30 Hz) is performed to extract the event-related spectral power of induced neural activities on a trial-by-trial basis for each experimental condition separately. The changes in beta band signal power in response to speech onset are calculated using the log transformation of the ratio between neural response power (PResponse) and the baseline signal power (P Baseline) according to equation 1.

$$Power[dB] = 20 * log_{10}\ (Presponse/Pbaseline). \tag{2.1}$$

2.4 Vocal Compensation Data Preprocessing

Vocal compensation can measure participants' ability to compensate for errors in the speech auditory feedback process. The pitch frequency of the produced voice signals is extracted in Praat (Boersma and Weenink, 2001) using an autocorrelation method. Then they are exported to MATLAB for further processing. The extracted pitch frequencies are segmented into epochs ranging from 100 ms before to 500 ms after the onset of pitch-shift stimuli. Individual vocal compensation data is then subjected to baseline correction by removing the amplitude of the pre-stimulus time window from -100 to 0 ms. The following formula is used to convert the pitch frequencies from Hertz to Cents scale in order to measure the vocal compensation in response to the pitch-shift stimulus:

$$Magnitude[Cents] = 1200 * (F/FBaseline). \tag{2.2}$$

We seek to determine for each AAF condition the ensemble of channels that best describe the reduction of beta-band activity during the speech production task. The goal is to aggregate channels that provide the same information about beta-band ERD and, consequently, improve the signal-to-noise ratio.

2.4.1 Upward Pitch-shift Stimulus

Figure 2.4 displays the topographical distribution maps for grand-average beta band powers within the time window for the upward pitch-shift AAF condition for aphasia and control groups, respectively. From the head maps, we observe that the average beta band powers for the control group attenuate clearly at the nine channels centered at FC2. The nine channels are Fz, F2, F4, FCz, FC2, FC4, C2, C4, and Cz. The beta band desynchronization at these nine channels reaches the peak at the time window [200,300] ms.

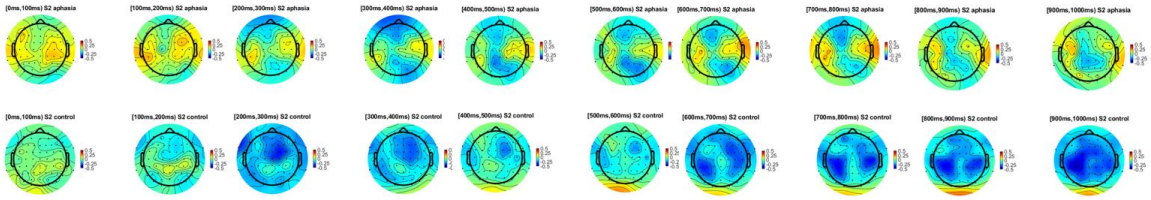

Figure 2.4: The head maps are the top view of scalp showing the topographical distribution maps of the mean beta band power for 64 EEG channels at each time window under upward pitch-shift AAF condition.

Figure 2.5 shows the average vocal compensation and the average relative beta band power. From the mean beta band power plot on the left, we observe that the mean beta-band power for the control group starts desynchronizing 100 ms before the onset of the stimulus and reaches the peak around the time 300 ms after the onset of the pitch-shift stimulus. Furthermore, this effect seems diminished for the aphasia group. From the mean vocal compensation plot on the right, it seems that the magnitude of vocal compensation starts increasing from 100 ms after the pitch-shift stimulus and reaches its peak around 300 ms after the onset of the pitch-shift stimulus. This effect is diminished for the aphasia group, as the magnitude of the compensatory vocal responses is more significant for the control than the aphasia group.

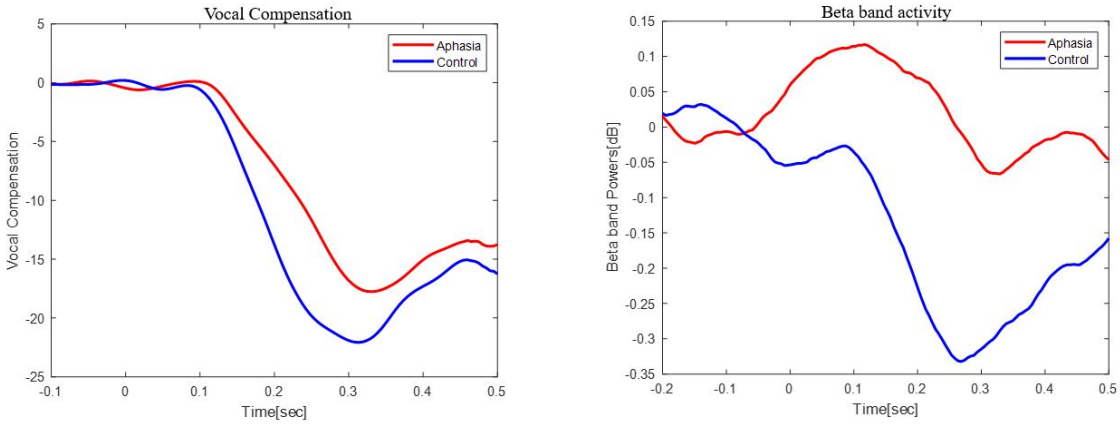

Figure 2.5: The mean relative beta band power figure (on the left) shows the time course of the mean relative beta-band power averaged over the nine channels aligned to the onset of the stimulus. The mean vocal compensation plot (on the right) shows the time course of the mean compensation value aligned to the onset of the stimulus.

2.4.2 Downward Pitch-shift Stimulus

Figure 2.6 shows the topographical distribution maps for grand-average beta band powers within the time window for the downward pitch-shift AAF condition. From the head maps, we observe that the average beta band powers for the control group attenuated clearly at the nine channels centered at CP3. The nine channels are C5, C3, C1, CP5, CP3, CP1, P5, P3, and P1.

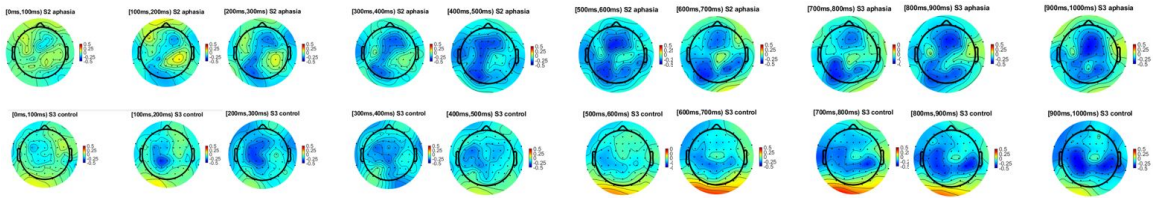

Figure 2.6: The head maps are the top view of scalp showing the topographical distribution maps of the mean beta band power for 64 EEG channels at each time window under downward pitch-shift AAF condition.

Figure 2.7 shows the average vocal compensation and the average relative beta band power. The mean relative beta band power figure (on the left) shows the time course of the mean relative beta-band power averaged over the nine channels aligned

to the onset of the stimulus. The mean vocal compensation plot (on the right) shows the time course of the mean compensation value aligned to the onset of the stimulus. From the mean beta band power plot on the left, we observe that the mean beta-band power for the control group starts desynchronizing 100 ms after the onset of the stimulus and reaches the peak around the time 300 ms after the onset of the pitch-shift stimulus. Furthermore, this effect seems diminished for the aphasia group. From the mean vocal compensation plot on the right, it seems that the magnitude of vocal compensation starts increasing from 100 ms after the pitch-shift stimulus and reaches its peak around 300 ms after the onset of the pitch-shift stimulus. This effect is diminished for the aphasia group, as the magnitude of the compensatory vocal responses is greater for the control than the aphasia group.

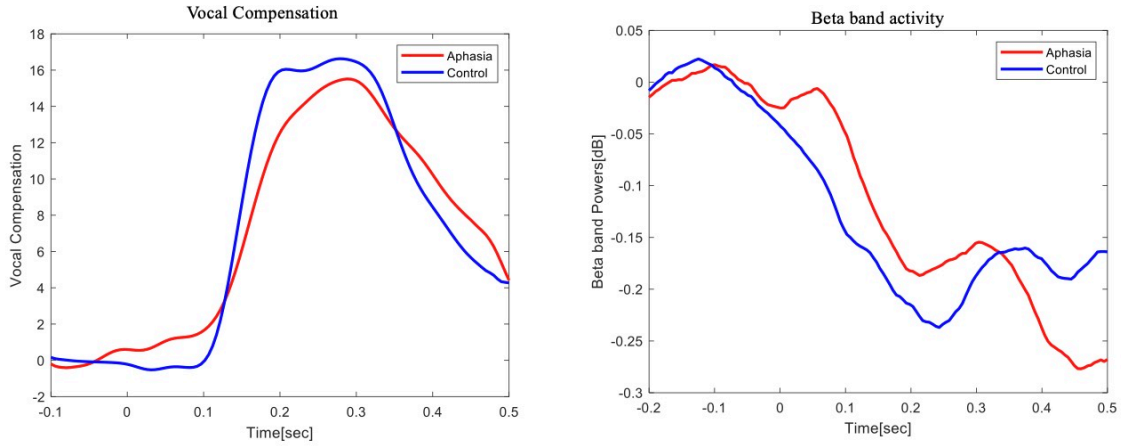

Figure 2.7: The mean relative beta band power figure (on the left) shows the time course of the mean relative beta-band power averaged over the nine channels aligned to the onset of the stimulus. The mean vocal compensation plot (on the right) shows the time course of the mean compensation value aligned to the onset of the stimulus.

Chapter 3

General Linear Model

Linear regression is a standard statistical method used to understand the relationship between a continuous dependent variable and a set of independent variables. Given a data set $\{y_i, x_{i1}, \ldots, x_{ip}\}_{i=1}^{n}$ of n statistical units, a linear regression model can be used to examine the linear relationship between the dependent variable y and the p-vector of independent variable x.

The dependent variable is a vector of observed values y_i $(i = 1, \ldots, n)$, it can be wrote as the matrix form as $\mathrm{y} = \begin{pmatrix} y_1 \\ y_2 \\ \vdots \\ y_n \end{pmatrix}$. The independent variable matrix is

$X = \begin{pmatrix} 1 & x_{11} & \cdots & x_{1p} \\ 1 & x_{21} & \cdots & x_{2p} \\ \vdots & \vdots & \ddots & \vdots \\ 1 & x_{n1} & \cdots & x_{np} \end{pmatrix}$, the vector of the $\beta = \begin{pmatrix} \beta_0 \\ \beta_1 \\ \beta_2 \\ \vdots \\ \beta_p \end{pmatrix}$,

where $\mathbf{x}_{i0} = 1$ for $i = 1, \ldots, n$. The corresponding element of β is called the intercept. The linear regression model can be expressed as $\mathbf{y} = X\boldsymbol{\beta} + \boldsymbol{\varepsilon}$. Typically, ordinary least squares (OLS) is used to estimate the regression parameters by minimizing the sum of squared errors (SSE). For a linear regression with the parameter vector $\boldsymbol{\beta}$, the fitted value is X$\boldsymbol{\beta}$, and thus the sum of squared errors is calculated by SSE($\boldsymbol{\beta}$) = (y - X$\boldsymbol{\beta}$)$^{\mathsf{T}}(y - X\boldsymbol{\beta})$. The goal is to find the vector of β that minimize the sum squared

errors. The equation could be expressed as $\hat{\boldsymbol{\beta}} = \arg\min_{\boldsymbol{\beta}} (SSE(\boldsymbol{\beta}))$. Finally, $\hat{\boldsymbol{\beta}}$ is the coefficient vector of the OLS method, calculated by $\hat{\boldsymbol{\beta}} = \left(\mathrm{X}^{\mathsf{T}}\mathrm{X}\right)^{-1}\mathrm{X}^{\mathsf{T}}\mathbf{y}$.

In order to calculate the variance of $\hat{\beta}$, we find the variance of $\left(\mathrm{X}^{\mathsf{T}}\mathrm{X}\right)^{-1}\mathrm{X}^{\mathsf{T}}\mathbf{y}$. The variance of the coefficient estimators is expressed as

$\mathrm{Var}[\,\hat{\beta} \mid X\,] = Var[\left(\mathrm{X}^{\mathsf{T}}\mathrm{X}\right)^{-1}\mathrm{X}^{\mathsf{T}}\mathbf{y}] = \left(\mathrm{X}^{\mathsf{T}}\mathrm{X}\right)^{-1}\mathrm{X}^{\mathsf{T}}Var(\mathbf{y})[\left(\mathrm{X}^{\mathsf{T}}\mathrm{X}\right)^{-1}\mathrm{X}^{\mathsf{T}}]^{\mathsf{T}}$

$= \left(\mathrm{X}^{\mathsf{T}}\mathrm{X}\right)^{-1}\mathrm{X}^{\mathsf{T}}Var(\mathbf{y})\mathrm{X}\left(\mathrm{X}^{\mathsf{T}}\mathrm{X}\right)^{-1} = \sigma^2(X^TX)^{-1}$.

The only random variable involved in the expression of $\hat{\beta}$ is Y, so the distribution of $\hat{\beta}$ is based on the distribution of Y. Therefore $\hat{\beta}$ is normally distributed. The significance of $\hat{\beta}$ could be examined by t-test using the formula $\mathrm{t} = \dfrac{\hat{\beta} - 0}{\sqrt{(Var(\hat{\beta}))}}$.

The residual vector $\hat{\epsilon}$ is $y - X\hat{\beta} = y - X(X^TX)^{-1}X^Ty$, so the residual sum of squares $\hat{\epsilon}^T\hat{\epsilon}$ (after simplification) is $SSE = y^Ty - y^TX(X^TX)^{-1}X^Ty$. Denote the sample mean of the dependent variable values in the vector y as $\bar{y}$. Then the total sum of squares is $SST = (y - \bar{y})^T(y - \bar{y}) = y^Ty - 2y^T\bar{y} + \bar{y}^T\bar{y}$. The regression sum of squares, defined as the sum of squared deviations of the predicted values from the sample mean of y_i, is $SSR = (\hat{y} - \bar{y})^T(\hat{y} - \bar{y}) = \hat{y}^T\hat{y} - 2\hat{y}^T\bar{y} + \bar{y}^T\bar{y}$.

R^2 is often interpreted as the proportion of the variation in dependent variable that is explained by the independent in the model. $R^2 = \dfrac{SSR}{SST} = 1 - \dfrac{SSE}{SST}$. The adjusted $\mathrm{R}^2(\bar{R}^2)$ is commonly used in the model selection process as it accounts for the phenomenon of the R squared automatically and spuriously increasing when extra indendent variables are added to the model.. $\bar{R}^2 = 1 - \dfrac{SSE/\mathrm{df}_{error}}{SST/\mathrm{df}_{total}}$.

3.1 Data Description

There are 34 aphasic and 46 neurologically intact subjects in the data. Each subject has the EEG signals for two AAF conditions (upward and downward pitch-shift stimuli). We use the data from 0 to 0.5 second after speech onset. For each AAF condition, there are EEG signals for the 9 channels (we extracted from the previous head maps). Each EEG signal has 100 data points (The sampling frequency is 200Hz). To make the calculation easier, we calculated the average beta band powers over the

nine channels, and then average within the 0.1 second time window. The average value of the beta band power is the dependent variable in our research. Each value of averaged beta band power (b-bar) is treated as the beta band value of one observation.

Each subject has the vocal compensation signals for two AAF conditions (upward or downward pitch-shift stimulus). Each vocal compensation signal has 100 data points (The sampling frequency is 200Hz). To make the calculation easier, we calculated the average value within the 0.1 second time window. The average value of the vocal compensation is the independent variable of interest in our research. Each value of averaged vocal compensation is treated as the vocal compensation value of one observation.

The main interest of this study is to estimate the relationship between the average beta band power and the average vocal compensation. Based off the literature review, several variables were identified as important to include in the analysis. These covariates are condition (upward or downward pitch-shift stimulus) and group (aphasia or control).

In our current models, we treat the observations as independent observations. However, the observations are actually not independent. The non-independent observation issue could be addressed by applying a mixed-effect model. And we will apply it in the future study.

3.2 Application

Before we fit the model, we make a histogram to check the distribution of the residual. It turns out that the distribution of the beta band power is left skewed (the histogram plot can be found in the appendix on my personal website). Therefore, we transformed the beta band data. The transformation of the beta band powers is

$$B_i = exp(\bar{b}_i/3) \tag{3.1}$$

$$Beta_i = B_i - mean(B_i) \tag{3.2}$$

where B_i is the original beta. $Beta_i$ is the transformed data. The transformed beta band power has a mean of 0 and standard deviation of 0.165. We excluded 6 outliers that has the beta band power greater than 0.495 or less than -0.495 (beyond three standard deviation from the mean). Below is the histogram of the transformed average beta band powers. The distribution appears to be normal distribution after the transformation (the histogram plot could be found in appendix).

We then generate a scatter plot to visualize the beta band power against the vocal compensation. It seems that there is a slightly negative association between the beta band power and the vocal compensation (the figure can be found in appendix on my personal website). In order to test the linear association between the two variables, we run a simple linear regression model. The dependent variable is the beta band power. The independent variable of interest is the vocal compensation. The simple linear regression model is

$$Beta_i = \beta_0 + \beta_1 Compensation_i + \epsilon_i \tag{3.3}$$

where i=1,...,781.

The hypothesis in this case is

$$H_0 : \beta_1 = 0 \tag{3.4}$$

$$H_1 : \beta_1 \neq 0 \tag{3.5}$$

We check the equal variance and the normality of the residuals assumptions for the linear regression model. No significant violation is found. Details can be found in Appendix on my personal website.

The estimated predictive equation is

$$\hat{Beta} = \beta_0 - 0.088 Compensation \tag{3.6}$$

We use the t-test to examine the significance of linear association between the two variables. The t-value is -2.299. With and the p-value of 0.0217, which is less than 0.05, we have sufficient evidence to reject the null hypothesis and conclude that there is significant linear association between the beta band power and the vocal compensation. As the vocal compensation increases by 1 unit, we expect that the mean of beta band power to decrease by 0.088 unit. The adjusted R-squared value is 0.0055, which indicates that the simple linear model can explain 0.55 percent of the variation in the beta band power.

Previous study has found that a downward stimulus in the 250 ms timing condition would elicit a smaller response than an upward stimulus because the latter would be perceived as a failure to reach the desired low F0 trajectory (Xu 2005). In this model, we add the condition variable to test if the beta band neural responses are different for the two AAF conditions.

We make a boxplot to compare the beta band powers for the two AAF condition. It could be seen that the beta band power for the upward pitch-shift AAF condition is slightly greater than that for the downward pitch-shift AAF condition (the figure can be found in appendix on my personal website).

We add the condition variable to our analysis. The model is:

$$Beta_i = \beta_0 + \beta_1 Compensation_i + \beta_2 Condition_i + \epsilon_i \tag{3.7}$$

where i=1,...,781

The equal variance and the normality of the residuals assumptions are checked for the linear regression model. No significant violation is detected. Details can be found in Appendix on my personal website.

We use the t-test to examine the significance of the predictors. The t-value of the beta band power is -2.36. With the p-value of 0.0185, we have sufficient evidence to reject the null hypothesis and conclude that there is a significant linear association

between the beta band power and the vocal compensation. As the vocal compensation increases by 1 unit, we expect that the mean beta band power to decrease by 0.091. The t-value of condition is 1.541. With the p-value of 0.1236, which is greater than 0.05. We do not have enough evidence to reject the null hypothesis. We conclude that the mean beta band powers are not significantly different for the two AAF conditions. The adjusted R-squared value is 0.0072, which indicates that the simple linear model can explain 0.72 percent of the variation in the average vocal compensation.

Then we are interested in examining if the linear associations between the beta band power and the vocal compensation are different for the two AAF conditions. We hypothesis that there is a significant linear association between the two variables under downward pitch-shift. However, the linear association between the two variables is weaker under upward pitch-shift AAF condition than that for the downward pitch-shift condition, because downward stimulus in the 250 ms timing condition would elicit a smaller response than an upward stimulus because the latter would be perceived as a failure to reach the desired low F0 trajectory (Xu 2005).

We made a scatter plot t0 visualize the association between the beta band power and vocal compensation for the two AAF conditions. And we observe that the slopes for the two AAF condition are slightly different (figure could be found in appendix on my personal website). For the downward pitch-shift, there is a slight negative linear association between the vocal compensation and the beta band powers. For the upward pitch-shift, we do not detect a clear linear association between the two variables. Therefore, we add an interaction term between the beta band powers and the condition. The model is

$$Beta_i = \beta_0 + \beta_1 Compensation_i + \beta_2 Condition_i + \beta_3 Compensation_i * Condition_i + \epsilon_i \tag{3.8}$$

where i=1,...,781

The equal variance and the normality of the residuals assumptions for the linear regression model are checked. No significant violation is detected. Details can be found in Appendix on my personal website.

We use the t-test to examine the significance of the predictors. The t-test of the interaction term is 1.264. With the p-value of 0.2068, we do not have enough evidence to reject the null hypothesis. We cannot conclude that the linear associations between the beta band power and vocal compensation are different for the two AAF conditions. The t-value of the vocal compensation is -2.33. With the p-value of 0.0099, we have sufficient evidence to reject the null hypothesis and conclude that there is a significant linear association between the beta band power and the vocal compensation. As the vocal compensation increases by 1, we expect that the mean of average beta band power to decrease by 0.136. The t-value of condition is 1.386. With the p-value of 0.1662, which is greater than 0.05. We do not have enough evidence to reject the null hypothesis. We conclude that the mean beta band powers are not significantly different for the two AAF conditions when beta is 0. The adjusted R-squared value is 0.0080, which indicates that the simple linear model can explain 0.8 percent of the variation in the beta band power.

The linear associations between the beta band power and the vocal compensation are not different for the two AAF conditions. Adding the interaction term between compensation and condition does not significantly increase the adjusted R-squared value. Therefore, we do not include the interaction term in the model. And the condition variable is not significant in the model, we do not keep the condition variable in the model.

People with aphasia might have impairment in detecting the speech error or change their pitch of voice after detecting the error. Therefore, the group variable should be accounted in the model. We make a boxplot to visualize the beta band powers for the two groups (figure could be found in appendix).

We include the group variable in the model, The model is:

$$Beta_i = \beta_0 + \beta_1 Compensation_i + \beta_2 Group_i + \epsilon_i \tag{3.9}$$

where i=1,...,781

We check the equal variance and the normality of the residuals assumptions for the linear regression model. No significant violation is detected. Details can be find in Appendix.

The estimated predictive equation is

$$\hat{Beta} = -0.01 - 0.08 Compensation + 0.03 Group(aphasia) \tag{3.10}$$

We use the t-test to examine the significance of the predictors. The t-value of the beta band power is -2.086. With the p-value of 0.0373, we have sufficient evidence to reject the null hypothesis and conclude that there is a significant linear association between the beta band power and the vocal compensation. As the vocal compensation increases by 1, we expect that the mean of the beta band power to decrease by 0.08. The t-value of group is 2.67. With the p-value of 0.0077, which is less than 0.05. We have enough evidence to reject the null hypothesis. We conclude that the mean beta band powers for the aphasia group is 0.03 greater than that for the control group. The adjusted R-squared value is 0.0132, which indicates that this linear model can explain 1.3 percent of the variation in the beta band power. Adding the group variable increases the adjusted R-squared value from 0.055 to 0.0132. Therefore, we include the group variable in the model.

We are interested in testing if the linear associations between the two variables are different for aphasia and control group.

The model is

$$\hat{Beta_i} = \beta_0 + \beta_1 Compensation_i + \beta_2 Group_i + \beta_3 Compensation_i * Group_i + \epsilon_i \tag{3.11}$$

We use the t-test to examine the significance of the predictors. The t-test of the interaction term is 0.664. With the p-value of 0.5071, we do not have enough evidence to reject the null hypothesis. We conclude that the linear associations between the beta band power and the vocal compensation are not different for the two groups. The t-value of the vocal compensation is -2.036. With the p-value less than 0.0421, we have sufficient evidence to reject the null hypothesis and conclude that there is a significant linear association between the beta band power and the vocal compensation. As the vocal compensation increases by 1, we expect that the mean of the beta band power to decrease by 0.101. The t-value of group is 2.603. With the p-value of 0.0094, which is less than 0.05, we have enough evidence to reject the null hypothesis and conclude that the mean beta band powers are significantly different for the two groups when beta is 0. The adjusted R-squared value is 0.0125, which indicates that the simple linear model can explain 1.25 percent of the variation in the average vocal compensation. Adding the interaction term between compensation and group does not significantly increase the adjusted R-squared value. Therefore, we do not include the interaction term in the model.

Therefore, our final model for the linear regression is

$$\hat{Beta} = -0.01 - 0.08Compensation + 0.03Group(aphasia) \tag{3.12}$$

3.3 Conclusion

Using the general linear regression model, we detect a significant negative linear association between the beta band power and the vocal compensation. We conclude that as the vocal compensation increases by 1, we expect that the mean of the beta band power to decrease by 0.08. We also have sufficient evidence to conclude that the mean beta band power for the aphasia group is greater than that for the control group. In other words, the magnitude of the beta band de-synchronization is greater for the control group than the aphasia group.

Chapter 4

Multivariate Linear Model

For the general linear model, the dependent variable is the average beta band powers over the 9 different channels. One limitation of the general linear model is that the mean beta band power is sensitive to the extreme values, which may induce inaccurate results. Therefore we treat the beta band powers at the 9 different channels as separate dependent variables. A multivariate linear model is needed to account for the multivariate patterns.

The multivariate linear regression model could be expressed as

$$Y_{n,k} = \begin{pmatrix} Y_{1,1} & Y_{1,2} & \cdots & Y_{1,k} \\ Y_{2,1} & Y_{2,2} & \cdots & Y_{2,k} \\ \vdots & \vdots & \ddots & \vdots \\ Y_{n,1} & Y_{n,2} & \cdots & Y_{n,k} \end{pmatrix} \tag{4.1}$$

$$= \begin{pmatrix} 1 & X_{11} & X_{12} & \ddots & X_{1p} \\ 1 & X_{21} & X_{22} & \ddots & X_{2p} \\ \vdots & \vdots & \ddots & \vdots & \\ 1 & X_{n1} & X_{n2} & \ddots & X_{np} \end{pmatrix} \begin{pmatrix} \beta_{01} & \beta_{02} & \cdots & \beta_{0k} \\ \beta_{11} & \beta_{12} & \cdots & \beta_{1k} \\ \beta_{21} & \beta_{22} & \cdots & \beta_{2k} \\ \vdots & \vdots & \ddots & \vdots \\ \beta_{p1} & \beta_{p2} & \cdots & \beta_{pk} \end{pmatrix} + \begin{pmatrix} \epsilon_{1,1} & \epsilon_{1,2} & \cdots & \epsilon_{1,k} \\ \epsilon_{2,1} & \epsilon_{2,2} & \cdots & \epsilon_{2,k} \\ \vdots & \vdots & \ddots & \vdots \\ \epsilon_{n,1} & \epsilon_{n,2} & \cdots & \epsilon_{n,k} \end{pmatrix}$$

For any choice of parameters $\boldsymbol{\beta} = \boldsymbol{b}$, where $\boldsymbol{b}$ is a (p+1) by k matrix, the matrix of errors is $(y - Xb)$. The error sum of squares is

(y - X$\boldsymbol{b}$)$^{\mathsf{T}}(y - X\boldsymbol{b})$ =

$$\begin{pmatrix} (Y_{[,1]} - Xb_{[,1]})^{\mathsf{T}}(Y_{[,1]} - Xb_{[,1]}) & \cdots & (Y_{[,1]} - Xb_{[,1]})^{\mathsf{T}}(Y_{[,k]} - Xb_{[,k]}) \\ (Y_{[,2]} - Xb_{[,2]})^{\mathsf{T}}(Y_{[,1]} - Xb_{[,1]}) & \cdots & (Y_{[,2]} - Xb_{[,2]})^{\mathsf{T}}(Y_{[,k]} - Xb_{[,k]}) \\ \vdots & \vdots & \ddots & \vdots \\ (Y_{[,k]} - Xb_{[,k]})^{\mathsf{T}}(Y_{[,1]} - Xb_{[,1]}) & \cdots & (Y_{[,k]} - Xb_{[,k]})^{\mathsf{T}}(Y_{[,k]} - Xb_{[,k]}) \end{pmatrix}$$

The selection of $\boldsymbol{b}_{[,i]}$ minimizes the ith diagnal sum of squares $(\mathrm{Y}_{[,i]} - Xb_{[,i]})^{\mathsf{T}}(Y_{[,i]} - Xb_{[,i]})$. Consequently, tr[(y - X$\boldsymbol{b}$)$^{\mathsf{T}}(y - X\boldsymbol{b})$] is minimized by the choice of $\boldsymbol{b}$. The least squares estimates $\boldsymbol{b} = \left(\mathrm{X}^{\mathsf{T}}\mathrm{X}\right)^{-1}\mathrm{X}^{\mathsf{T}}\mathbf{Y}$. The variance-covariance matrix of $\boldsymbol{b}$ is $\hat{\Sigma} = \frac{1}{n}(y - X\boldsymbol{b})^{\mathsf{T}}(y - X\boldsymbol{b})$. $n\hat{\Sigma}$ is distributed as $\mathrm{W}_{p,n-k-1}(\hat{\Sigma})$. The maximized likelihood $\mathrm{L}(\hat{\mu}, \hat{\Sigma}) = (2\pi)^{-kn/2}|\hat{\Sigma}|^{n/2}e^{-kn/2}$

The likelihood ratio test could be used to test if the independent variable is significant in the multivariate linear regression model. The hypothesis is

$\mathrm{H}_0 : \beta_{[j,]} = 0$

$H_1 : \beta_{[j,]} \neq 0$

where $\beta = \begin{pmatrix} \beta_{[j,]} \\ \beta_{[r,]} \end{pmatrix}$, $\beta_{[r,]}$ is the reduced matrix (p by k), which is identical to the matrix $\boldsymbol{\beta}$ without the jth row. Set $\mathrm{X} = \left(X_{[,j]}, X_{[,r]}\right)$, where $\mathrm{X}_{[,j]}$ is the jth row of the matrix X, $\mathrm{X}_{[,r]}$ is the X matrix except for the jth column. $\mathrm{E(Y)} = \mathrm{X}\boldsymbol{\beta} = X_{[,j]}\beta_{[j,]} + X_{[,j]}\beta_{[r,]}$. Under the null hypothesis, where $\beta_{[j,]} = 0, Y = X\boldsymbol{\beta} = X_{[,r]}\beta_{[r,]}$

The estimated coefficient of the variable j is $\hat{\beta_{[j,]}} = (\mathrm{X_{[,j]}}^{\mathsf{T}}\mathrm{X_{[,j]}})^{-1}\mathrm{X_{[,j]}}^{\mathsf{T}}\mathbf{Y}$. The covariance matrix of the reduced model is $\hat{\boldsymbol{\Sigma}}_{\boldsymbol{r}} = (y - X_{[,r]}\hat{\beta_{[r,]}})^{\mathsf{T}}(y - X_{[,r]}\hat{\beta_{[r,]}})/n$ The maximized likelihood $\mathrm{L}(\hat{\mu_r}, \hat{\Sigma_r}) = (2\pi)^{-kn/2}|\hat{\Sigma_r}|^{n/2}e^{-kn/2}$ The likelihood ratio is $\Lambda = \frac{\underset{\beta_{[r,]},\Sigma_r}{\arg\max}\, \widehat{L}(\beta_{[r,]}, \Sigma)}{\underset{\beta,\Sigma}{\arg\max}\, \widehat{L}(\beta, \Sigma)} = \frac{\widehat{L}((\hat{\boldsymbol{\beta}_{[\boldsymbol{r},]}}, \hat{\boldsymbol{\Sigma}}_{\boldsymbol{r}}))}{\widehat{L}((\hat{\boldsymbol{\beta}}, \hat{\boldsymbol{\Sigma}}))} = (\frac{|\hat{\boldsymbol{\Sigma}}|}{|\hat{\boldsymbol{\Sigma}}_{\boldsymbol{r}}|})^{(\frac{n}{2})}$ The likelihood ratio test statistics is

$$-2\boldsymbol{ln}\boldsymbol{\Lambda} = -nln(\frac{|\hat{\boldsymbol{\Sigma}}|}{|\hat{\boldsymbol{\Sigma}}_{\boldsymbol{r}}|}) = -nln(\frac{|\boldsymbol{n}\hat{\boldsymbol{\Sigma}}|}{|\boldsymbol{n}\hat{\boldsymbol{\Sigma}} + n(\hat{\boldsymbol{\Sigma}}_{\boldsymbol{r}} - \hat{\boldsymbol{\Sigma}})|})$$

The residual sum of squares $\boldsymbol{E} = n\hat{\boldsymbol{\Sigma}}$. The extra sum of squares is the difference of residual sum of squares between the full model and the reduced model, which can

be expressed as $\boldsymbol{H} = \boldsymbol{n}(\hat{\boldsymbol{\Sigma}}_{\boldsymbol{r}} - \hat{\boldsymbol{\Sigma}})$. The statistics can be defined in terms of $\boldsymbol{E}$ and $\boldsymbol{H}$ directly, Wilks' lambda is a test statistic used in multivariate analysis. Wilk's lambda tests how well each level of independent variable contributes to the model. The scale ranges from 0 to 1, where 0 means total discrimination, and 1 means no discrimination. Each independent variable is tested by putting it into the model and then taking it out - generating a Λ statistic. The significance of the change in Λ is measured with an F-test; if the F-value is greater than the critical value, the variable is kept in the model. $\Lambda = |\dfrac{\boldsymbol{E}}{\boldsymbol{E}+\boldsymbol{H}}|$.

The assumptions of the multivariate linear regression are similar with that for the general linear regression. The assumptions are: 1. The observations are independent from each other. 2. Dependent variables are multivariate normally distributed. 3. The variance-covariance matrices are equal for all the fitted values. 4. There are linear associations between the dependent variables.

The multivariate normally distribution could be tested using the Shapiro-Wilk multivariate normality test in R. There is no well known existed package that could be used to test the equality of variance-covariance matrix assumption for the multivariate linear regression. We developed a function based on the score test of nonconstant variance (Fox 2009) and the Box M test (Box 1949).

For the GLM, define the OLS residuals of the linear regression as $\hat{\epsilon}$ and the residual variance $\hat{\sigma}^2 = \dfrac{\sum_{i=1}^{n} \epsilon_i^2}{n}$. In the score test of nonconstant variance (ncvTest) by John Fox, the score statistic for testing the homoscedastic is $\mathrm{U}_i = \frac{\hat{\epsilon_i}^2}{\hat{\sigma}^2}$, the derivation of the test statistics could be found at the articles(Breusch and Pagan 1979) (Cook and Weisberg 1983).

For the multivariate linear regression, where the residuals are n by p matrix, the variance of the $\mathrm{j}^{th}(j = 1, ..., k)$ column is $\hat{\sigma_j^2} = \dfrac{\sum_{i=1}^{n} \epsilon_{ij}^2}{n}$. The score statistic for testing the homoscedastic is $\mathrm{U}_{ij} = \frac{\hat{\epsilon_{ij}}^2}{\hat{\sigma_j}^2}$. For m = 1,...k, j = 1,...,k, and m$\neq j$, the covariance

of the residuals between column m and j is $\hat{\sigma_{mj}} = \frac{\sum_{i=1}^{n} \epsilon_{im}\epsilon_{ij}}{n}$. The score statistic for testing the homoscedastic is $\mathrm{U}_{imj} = \frac{\hat{\epsilon_{ij}}\hat{\epsilon_{im}}}{\hat{\sigma_{mj}}}$

Therefore, for each observation i, the score statistics of the variance-covariance matrix of the errors U_i could be calculated with themselves.

$$\mathrm{U}_i = \begin{pmatrix} U_{i,11} & U_{i,12} & \cdots & U_{i,1p} \\ U_{i,21} & U_{i,22} & \cdots & U_{i,2p} \\ \vdots & \vdots & \ddots & \vdots \\ U_{i,p1} & U_{i,p2} & \cdots & U_{i,pp} \end{pmatrix}$$

The score statistics of the ith observation's variances are displayed on the diagonal. The score statistics of the ith observation's covariances are in the lower-left portion and the upper-right portion.

In the Box's M test (Box 1949), which could be used to test the homogeneity of variances-covariances for the multivariate model when the independent variable is categorical, the logarithm of the determinant of each covariance matrices ($\log|\mathrm{S}_i|$) for each group is calculated in order to compare the covariance matrices for different groups.

In the multivariate linear model with continuous independent variable, instead of the variance-covariance matrix for each group, we have the score statistics matrix U_i for each observation. We calculate the logarithm of the determinant of each score statistics matrix, define as S_i. Then we examine if there is significant association between S_i and the fitted values by fitting the linear regression $\hat{y}_i = \alpha_0 + \alpha_1 S_i + e_i$. If there is significant linear association between the fitted values and S_i, we reject the null hypothesis and conclude that the variance-covariances are not constant for different fitted values.

4.1 Application

Before we fit the model, we make a histogram to check the distribution of the residual. It turns out that the distribution of the beta band power is left skewed (the histogram

plot could be found in appendix). Therefore, we transformed the beta band data. The transformation of the beta band powers is

$$Beta = exp(\bar{b}_i/3) - mean(exp(\bar{b}_i/3)) \tag{4.2}$$

The transformed beta band power has a mean of 0. The standard deviation of the beta band powers at different channels are not the same. We exclude 27 outliers that have a beta band power of more than three standard deviations from the mean. The distribution appears to be normal distribution after the transformation (the histogram plot could be found in appendix).

Since the channels that we are interested in are different for the two AAF conditions, we run separate models for the two AAF conditions. Below are the correlation matrices that show the correlation between the beta band powers and the vocal compensation for the two conditions.

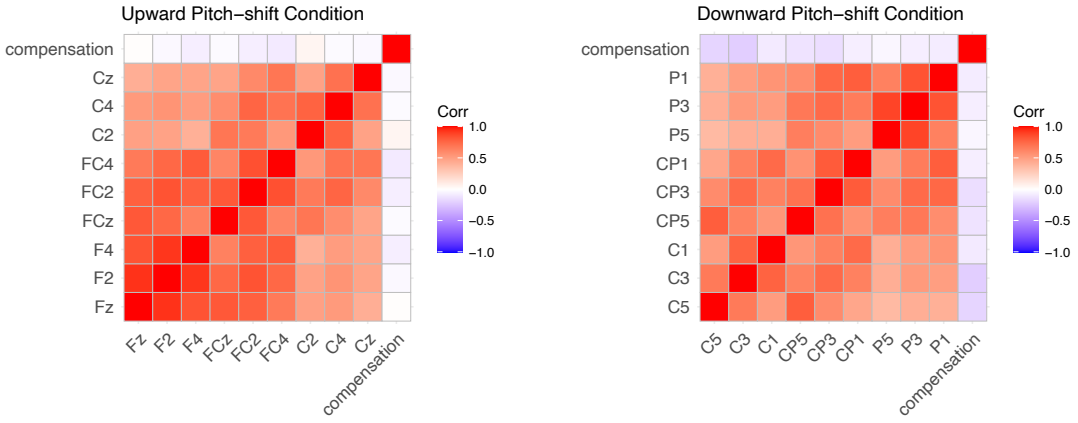

Figure 4.1: Correlation matrices

From Figure 4.1, we can observe that there are linear associations between beta band powers and the vocal compensation. However, we do not observe linear association between the beta band power and the vocal compensation for the upward pitch-shift condition. Therefore, we only run the multivariate linear regression for the downward AAF condition.

The initial model is

$$Beta_{ik} = \beta_{0k} + \beta_1 Compensation_{ik} + \epsilon_{ik} \tag{4.3}$$

where k = 1,...,9, i=1,...,375

We check the assumptions for the model. The equal variance-covariance matrix assumption and the multivariate normal distribution assumptions are not violated. Details can be found at Figure B.1 in Appendix.

The Wilks' lambda test statistics of the MANCOVA test is 0.952 for the vocal compensation. The according F-value is 2.26 (p = 0.0142). We have enough evidence to reject the null hypothesis that there is no linear association between the set of beta band powers and the vocal compensation. We conclude that there is significant linear association between the set of beta band powers and vocal compensation. With the p-value of 0.001, 0.0001, 0.021, and 0.009, we have enough evidence to conclude that there are significant negative linear association between the vocal compensation and the beta band power at the channels 1, 2,4,5. We expect the mean beta band power at these channels to decrease by 0.200, 0.257, 0.141, and 0.169, respectively, as one unit increase in vocal compensation (the coefficient table can be found in appendix).

Then we add group into the model.

The model equation is

$$Beta_{ik} = \beta_{0k} + \beta_1 Compensation_{ik} + \beta_2 group_{ik} + \epsilon_{ik} \tag{4.4}$$

where k = 1,...,9, i=1,...,375

We check the assumptions for the model. The equal variance-covariance matrix assumption and the multivariate normal distribution assumptions are not violated. Details can be found at Figure B.2 in Appendix.

The Wilks' lambda test statistics of the MANCOVA test is 0.937 for the vocal compensation. The according F-value is 2.74 (p = 0.0042). We have enough evidence

to reject the null hypothesis that there is no linear association between the set of beta band powers and the vocal compensation. We conclude that there is significant linear association between the set of beta band powers and vocal compensation. With the p-value of 0.001, 0.0001, 0.021, and 0.009, we have enough evidence to conclude that there are significant negative linear association between the vocal compensation and the beta band power at the channels 1, 2,4,5. We expect the mean beta band power at these channels to decrease by 0.2, 0.257, 0.141, and 0.168, respectively, as one unit increase in vocal compensation. The Wilks' lambda test statistics of the MANCOVA test is 0.916 for the group variable. The according F-value is 3.71 ($p = 0.0002$). We have enough evidence to reject the null hypothesis that the set of beta band powers are different for the two groups. With the p-value of 0.004, we conclude that the mean beta band power for the aphasia group is 0.056 greater than that for the control group at channel 2 (the coefficient table can be found in appendix).

Then we examine if the linear associations between the beta band power and vocal compensation are different for the two groups. The plot below shows the correlation matrix for the two groups.

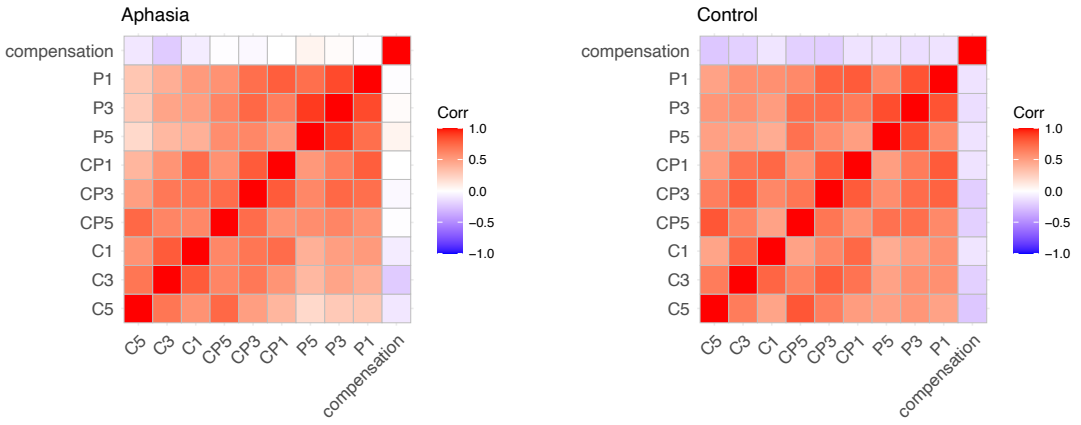

Figure 4.2: Correlation matrices

From Figure 4.2, we could observe that the associations between compensation and beta band power are different for the two groups. For the aphasia group, we only observe linear association between them at channel 2. However, for the control group, we could observe linear association at channel 1, 2, 4, and 5. Therefore, we add an interaction term to our model.

$$Beta_{ik} = \beta_{0k} + \beta_1 Compensation_{ik} + \beta_2 group_{ik} + \beta_3 Compensation_{ik} * group_{ik} + \epsilon_{ik} \tag{4.5}$$

where k = 1,...,9, i=1,...,375

We check the assumptions for the model. The equal variance-covariance matrix assumption and the multivariate normal distribution assumptions are not violated. Details can be found at Figure B.5 in Appendix.

The Wilks' lambda test statistics of the MANCOVA test is 0.917 for the interaction term. The according F-value is 1.78 (p = 0.0701). We do not have enough evidence to reject the null hypothesis. We conclude that the linear association between the set of beta band powers and the vocal compensation are not different for the two groups under downward pitch-shift AAF condition (the coefficient table can be found in appendix). Therefore, the final model for the downward pitch-shift AAF condition

is

$$Beta_{ik} = \beta_{0k} + \beta_1 Compensation_{ik} + \beta_2 group_{ik} + \epsilon_{ik} \tag{4.6}$$

Where the coefficients at different channels are listed in table 4.4.

For the upward pitch-shift AAF condition, we first test the association between the beta band power and the vocal compensation.

4.2 Conclusion

Using the multivariate linear regression model, we find that there are some strong negative linear associations between the beta band power and the vocal compensation under downward pitch-shift AAF condition. Such association is not found in upward pitch-shift AAF condition. Moreover, under the downward pitch-shift AAF condition, we do not detect a significant difference in the coefficients of vocal compensation between the two groups. Under the upward pitch-shift AAF condition, we detect a stronger negative linear association between the two variables for the control group than the aphasia group.

Chapter 5

Simulation Studies

We conduct simulation studies to identify the ability of the two models to examine the true association between the beta band powers and the vocal compensation. We have two simulation studies. In study one, there is an underlying association between the beta band powers and the vocal compensation. In study two, there is no underlying association between the beta band neural response and the vocal compensation.

5.1 Study One

For the first simulation study, we generate two groups EEG beta band powers for both the aphasia and control group as the ground truth. Base on the characteristic of our data, we make (1) the latency of the generated signals from 0 to 0.5 seconds. (2)The cut-off frequency of each signal is 200 Hz (a 0.5 second signal has 100 time points). (3)The data within the first 0.1 second is flat at the value 0 (pattern observed from the EEG beta band signal in our data). (4)The beta band powers start decreasing after 0.1 second. (5)The signal reach its peak at 0.3 second, with the value of -0.22 (mean beta band power at the peak in our data). (6)The beta band powers start increasing after 0.3 second. The plots of the generated beta band power are listed below.

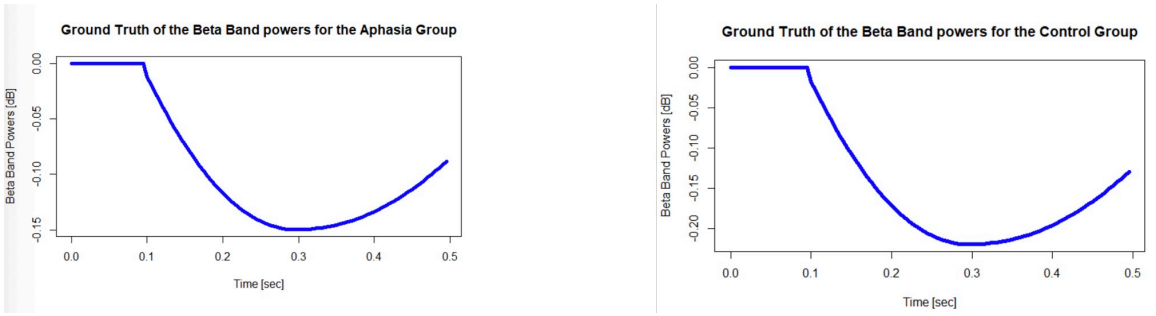

Figure 5.1: Ground truth of the beta band powers

Then we generate the ground truth of the vocal compensation signal from the EEG beta band signal for the two groups using the equation Vocal Compensation = Beta Band * (-100). The reason of using the value -100 is that the ratio between the vocal compensation and the beta band powers is approximately -100. The plots of the generated vocal compensation are listed below.

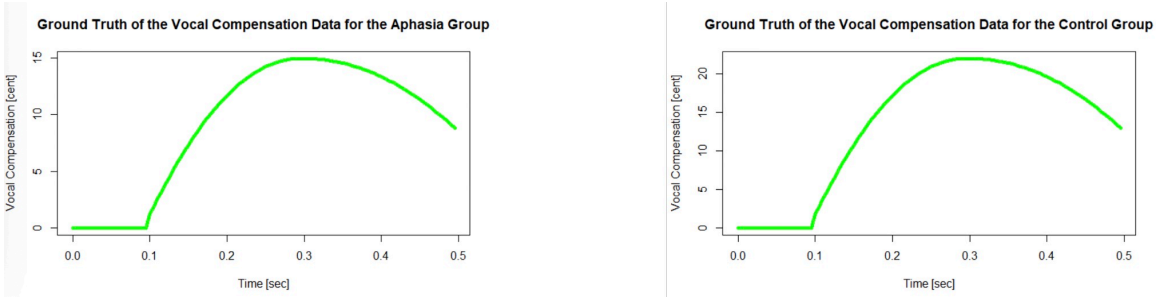

Figure 5.2: Ground truth of the vocal compensation

Then we simulate the vocal compensation and beta band power data for the 40 subjects in the aphasia group and 40 subjects in the control group at 9 different channels, respectively, by adding random errors. For the vocal compensation, the error terms added to the control group has the mean of 0 and a standard deviation of 14.8. The standard deviation of errors for the aphasia group is 12.8. The values been selected are based on the descriptive statistics of our data. For the beta band power, the error terms added to the control group at channel 1 has a mean of 0 and a standard deviation of 0.1. The error terms for the aphasia group at channel 1 has

a mean of 0 and a standard deviation of 0.1/1.47, where 1.47 is the ratio between the beta band powers for the two groups. Since the standard deviation is relatively small, we expect that there would be a strong linear association between the beta band power at channel 1 and the vocal compensation. The error terms added to the control group at channel 2 has a mean of 0 and a standard deviation of 0.5. The error terms for the aphasia group at channel 2 has a mean of 0 and a standard deviation of 0.5/1.47. We expect that the linear association between the vocal compensation and the beta band power at channel 2 is moderate. For channels 3 and 4, the means of the error terms for are both 0, and the standard deviations are 5 and 6, respectively. We expect that the linear associations between the vocal compensation and beta band power at the two channels are very weak.

Then we divide the EEG beta band power and the vocal compensation to five time-windows evenly and calculate the mean value within each window for each subject. The associations between the vocal compensation and the beta band power, and the associations between the beta band power at different channels are shown below.

From Figure 5.3, we observe a strong linear association between the vocal compensation and beta band power at channel 1, a moderate linear association between the two variables at channel 2, a weak linear association at channel 3. There is no linear association between the two variables at channel 4. We also observe that the beta band power at channels 1 and 2 has moderate linear association. The beta band power at channel 1 and 3 has a weak linear association.

Next, we construct hypothesis tests for the association between the beta band powers and the vocal compensation using the general linear model and the multivariate linear model, respectively. The equation of the model is $\text{beta}_{ik} = \beta_{0k} + \beta_{1k}compensation + \beta_{2k}group + \epsilon_{ik}$. Then we perform 700 simulations and calculate the rate of detecting the true associations between the vocal compensation and the beta band power, respectively.

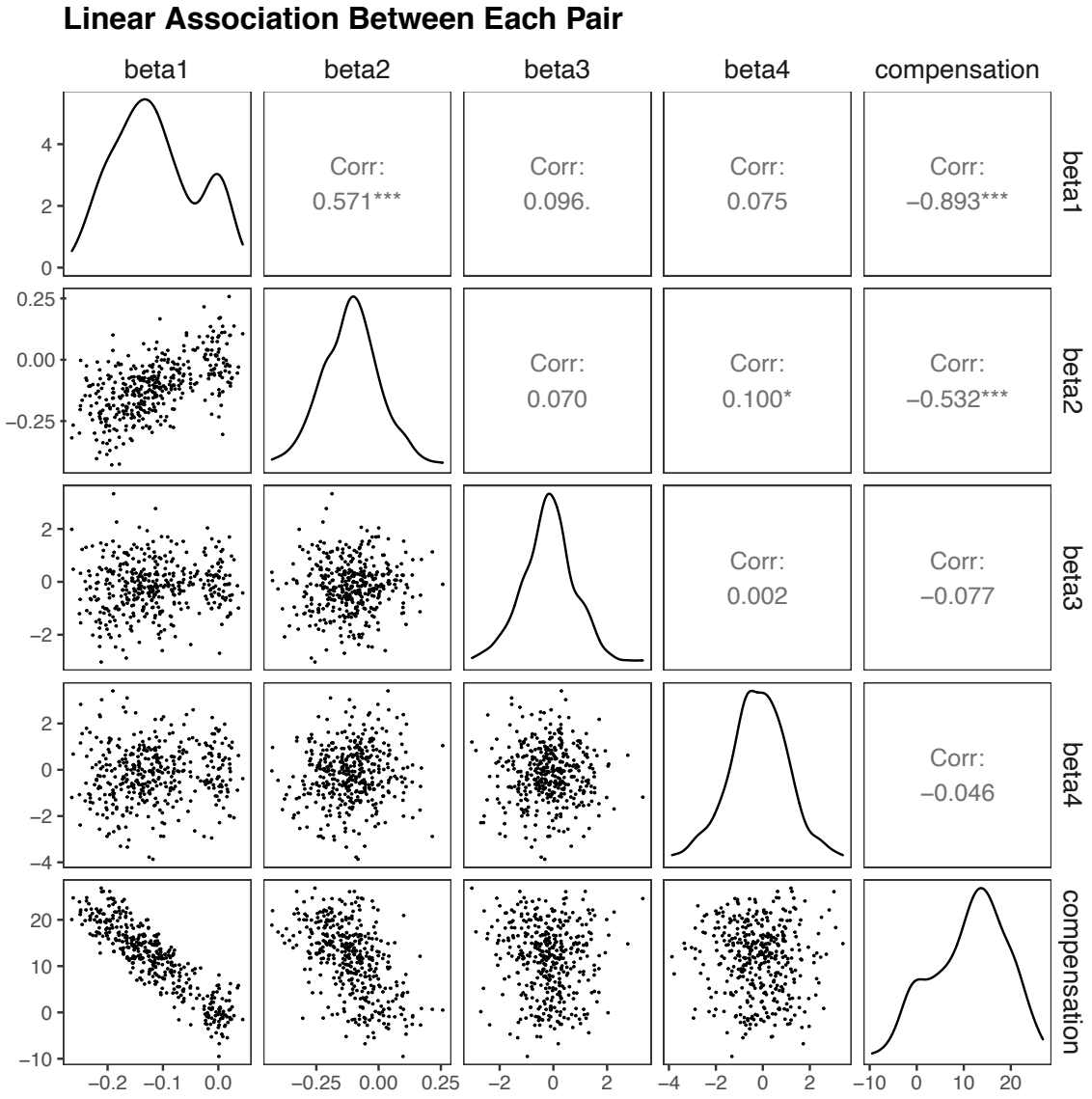

Figure 5.3: Scatterplot matrix of the associations between each of the two variables

5.2 Study Two

In simulation two, there is no linear association between the beta band power and the vocal compensation. We generate the same beta band power data as in simulation one. For the vocal compensation data, instead of using the equation Vocal Compensation = Beta Band * (-100), we generate 100 random values for each participant. For the aphasia group, the vocal compensation has a mean of 12 and a standard deviation of

8. For the control group, the vocal compensation has a mean of 14.8 and a standard deviation of 9. The values that we used are generated from the descriptive statistics of our data. Then we divide the beta band power and the vocal compensation to five time-windows evenly and calculate the mean value within each window for each subject. The associations between the vocal compensation and the beta band power, and the associations between the beta band power at different channels are shown below.

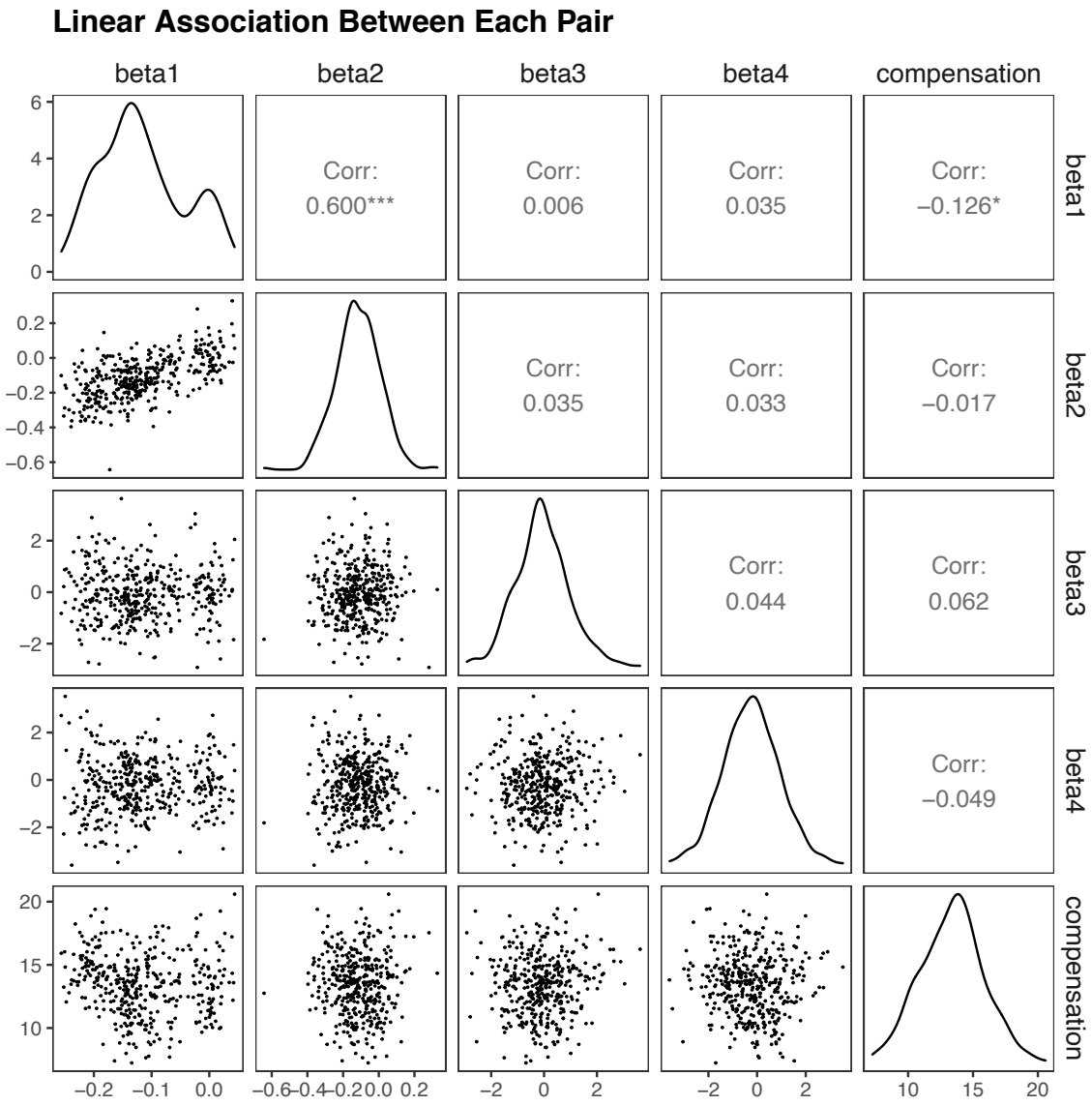

Figure 5.4: Scatterplot matrix of the associations between each of the two variables

From Figure 5.4, we observe that the beta band power at channels 1 and 2 has moderate linear association. The beta band power at channel 1 and 3 has a weak linear association. There is no linear association between the vocal compensation and the beta band power at any channel.

Next, we construct hypothesis tests for the association between the beta band powers and the vocal compensation using the general linear model and the multivariate linear model, respectively. The equation of the model is $\text{beta}_{ik} = \beta_{0k} + \beta_{1k}compensation + \beta_{2k}group + \epsilon_{ik}$. Then we perform 700 simulations and calculate the rate of detecting the true associations for the two models, respectively.

5.3 Result and Discussion

The result of the simulation studies shows that the sensitivity of the general linear regression model is 91.4%, the sensitivity of the multivariate linear regression model is 100%. The specificity of the general linear regression model is 94.4%. The specificity of the multivariate linear regression model is 93.1%. Under the situation that the independent variable has a linear association with some of the dependent variables but not all the dependent variables, the multivariate linear regression model has greater power to detect the linear association than the general linear regression model. The reason is that the multivariate linear regression model accounts for the difference in coefficients for beta band powers at different channels. However, the general linear model only predicts the mean beta band power over the nine channels, in which the set of dependent variables is affected by the beta band power at the channels that do not have a linear association with the vocal compensation.

Chapter 6

Conclusion

Using the general linear regression model, we detect a significant negative linear association between the beta band power and the vocal compensation. We conclude that as the vocal compensation increases by 1, we expect that the mean of the beta band power to decrease by 0.08. We also have sufficient evidence to conclude that the mean beta band powers for the aphasia group is greater than that for the control group. In other words, the magnitude of the beta band de-synchronization is greater for the control group than the aphasia group.

Using the multivariate linear regression model, we find that the linear associations between the set of beta band power and vocal compensation are different for the two AAF conditions. We detect a stronger negative linear association between the two variables for the downward pitch-shift AAF condition than the upward pitch-shift condition. Moreover, under the downward pitch-shift AAF condition, we do not detect a significant difference in the coefficients of vocal compensation between the two groups. Under the upward pitch-shift AAF condition, we detect a stronger negative linear association between the two variables for the control group than the aphasia group.

From the simulation studies, we conclude that, under the situation that the independent variable has a linear association with some of the dependent variables but not all the dependent variables, the multivariate linear regression model has greater power to detect the linear association than the general linear regression model.

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