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THE EFFECT OF SLOW SPEECH ON TONGUE MOVEMENTS & ACOUSTIC VOWEL  
SPACE DISTANCE IN SPEAKERS WITH AMYOTROPHIC LATERAL SCLEROSIS

A Thesis by

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Bachelor of Science, Kansas State University, 2011

Submitted to the Department of Communication Sciences and Disorders  
and the faculty of the Graduate School of  
Wichita State University  
in partial fulfillment of  
the requirements for the degree of  
Master of Arts

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The following faculty members have examined the final copy of this thesis for form and content, and recommend that it be accepted in partial fulfillment of the requirement for the degree of Master of Arts with a major in Communication Sciences and Disorders.

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## ABSTRACT

With disease progression, the speech of talkers with Amyotrophic Lateral Sclerosis (ALS) becomes increasingly imprecise and unintelligible. Although speaking rate reduction is commonly used as a treatment approach to enhance speech intelligibility in these talkers, its effect on tongue movements and speech acoustics is not well understood. Thus, the purpose of this study was to determine how slow speech affects tongue excursions and speech acoustics in persons with ALS. Further, this study investigated how tongue excursions and speech acoustics differed between persons with ALS and healthy controls. Lastly, this study sought to determine how predictable tongue excursions are based on speech acoustics in persons with ALS.

3D electromagnetic articulography was used to capture tongue movements during speech in five talkers with ALS and five healthy controls. Tongue excursions and the vowel space distance during the production of the vowels /a/ and /i/ in the word “kite” embedded in the sentence “See a kite again” were measured during typical and slow speech.

Results showed increased tongue excursions and acoustic vowel space in response to slow speech for persons with ALS and controls; however, the effect was larger in the control group than in the ALS group. These outcomes support the current clinical assumption that slow speech increases tongue excursions and expanded vowel space in persons with speech impairments due to ALS. Although tongue excursions tended to be slightly larger in persons with ALS than controls, vowel space tended to be smaller in persons with ALS than controls. These findings challenged the current assumption that a small vowel space indicate small tongue excursion. The predictability of change in tongue excursions based on change in speech acoustics in response to slow speech was much lower in persons with ALS than controls. Thus, acoustic measures should not be used to infer the underlying speech movements in persons with ALS.

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# CHAPTER 1

## INTRODUCTION

Amyotrophic Lateral Sclerosis (ALS) is a neurodegenerative disease resulting in progressive muscular paralysis due to motor neuron cell death. “Amyotrophy” refers to the atrophy of muscle tissue (Rowland & Schneider, 2001). “Lateral sclerosis” signifies the scarring of the lateral and anterior corticobulbar and corticospinal tracts due to motor neuron cell death. These tracts are responsible for directing motor commands from the upper motor neurons (UMN) in the brain via the lower motor neurons (LMN) in the brainstem and spinal cord to the head, neck, and spinal muscles (Rowland & Schneider).

ALS is known to affect both upper and lower motor neurons. As the disease progresses, muscle functions become progressively affected and patients who are in the later stages of the disease may become completely paralyzed (Wijesekera & Leigh, 2009). In Europe and North America, the incidence of ALS is between 1-2 cases per every 100,000 people a year (Rowland & Schneider, 2001). Studies have consistently shown that the disease more often affects males than females and that the age of onset for ALS is between 55-65 years of age (Wijesekera & Leigh). Research has shown that 50% of ALS patients die within three years of onset (Rowland & Schneider), typically from respiratory failure or other pulmonary complications (Wijesekera & Leigh).

### 1.1 Classification of ALS

ALS can be classified in several ways. One classification is based on the location of the initial lesion. In the spinal form of the disease, symptoms can start both distally or proximally in the upper and lower limbs. Around two thirds of patients with ALS have the spinal form of the disease (Wijesekera & Leigh, 2009). Patients with bulbar onset experience initial muscular

weakness in the area of the lower part of the face, mouth, and throat (see Figure 1). It is, however, important to note that the disease will spread to the mouth and face muscles in many people with ALS within 1-2 years of disease onset even when their initial symptoms started in the limbs (Wijesekera & Leigh).

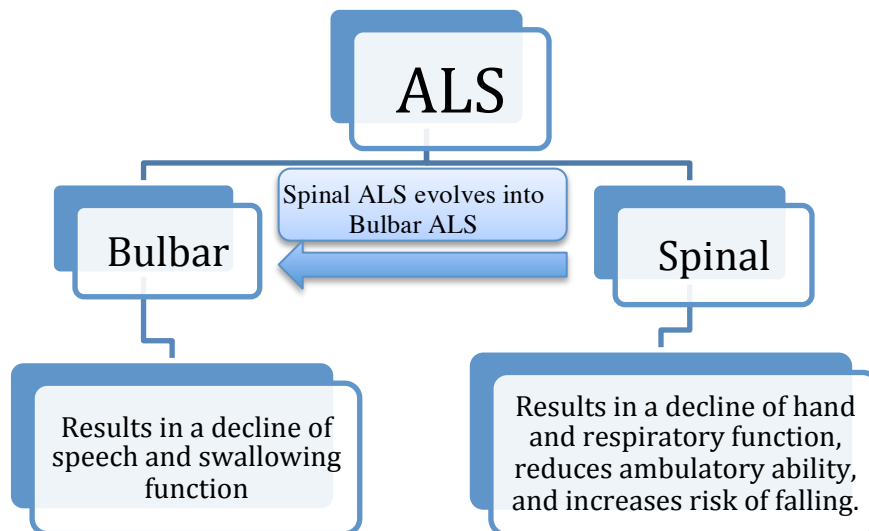


Figure 1. Classifications of ALS and the areas impaired

Another way to classify ALS is by etiology. A majority of ALS cases are sporadic with no known cause. However, approximately 5-10% of ALS is familial with a genetic component (Rowland & Schneider, 2001). Of those with a family history of ALS, only in a small percentage of this population has a gene defect been identified that can be linked to ALS (Rowland & Schneider). The remaining percentage is caused by mutations in other genes. In addition, those with familial ALS have an earlier onset of the disease and have a shorter survival rate (Wijesekera & Leigh, 2009).

## 1.2 Diagnosing ALS

It is difficult to diagnose ALS because currently no tests are available that can give a clear answer whether or not someone has ALS. Therefore, ALS is often diagnosed by excluding other diseases. Several tests are available that are typically completed during the diagnostic

procedure: neuroimaging studies, electrophysiological studies, muscle biopsy and neuropathological studies, and several laboratory tests (Wijesekera & Leigh, 2009).

Neuroimaging studies using magnetic resonance imaging (MRI) are important for excluding diseases that mimic ALS. More recently, MRIs have been shown to help identify the lesions that are associated with ALS through looking at the structure and function of the white and grey matter in the brain (Douaud, Filippini, Knight, Talbot, & Turner, 2011).

Electrophysiological studies are done to identify central or peripheral neuromuscular dysfunctions. Nerve conduction studies, such as conventional and quantitative electromyography (EMG), as well as transcranial magnetic stimulation (TMS) and central motor conduction studies are specific types of electrophysiological assessments completed to rule out other diseases and come to the diagnosis of ALS (Wijesekera & Leigh, 2009; Rowland & Schneider, 2001).

Muscle biopsy of skeletal muscle and other tissues and neuropathological studies are other diagnostic procedures that are completed. The biopsy is not a requirement for a diagnosis, but does assist in ruling out any other syndrome and provides additional information on the function of the lower motor neurons (Wijesekera & Leigh, 2009). Additional laboratory tests can also be completed to assess other areas such as cerebrospinal fluid (CSF) proteins or muscle enzymes. A definite diagnosis of ALS is rare, but is indicated when a worsening of the UMN and LMN is evident in at least three regions of the body (Wijesekera & Leigh).

ALS is easily misdiagnosed for other diseases that present themselves in a similar manner, for example cerebral lesions, primary lateral sclerosis, multifocal motor neuropathy, multiple sclerosis, Kennedy's disease, and cervical spondylotic myelopathy. Because of this easy misdiagnosis, it is important that a diagnosis is not formulated on the basis of

electromyography (EMG) alone, but also includes the patient's clinical history and thorough testing (Wijesekera & Leigh, 2009).

### 1.3 Symptoms of ALS

It is rare that an ALS patient presents solely upper or lower motor neuron involvement. Symptoms are often evident for both UMN and LMN decline. It is important to note that the impact of the UMN and LMN loss is not easily distinguishable, which is why thorough testing is necessary for a reliable diagnosis (Langmore & Lehman, 1994).

#### 1.3.1 Upper Motor Neuron Lesions

Loss of UMNs decreases the contraction speed of muscles and, therefore, causes slowed repetitive movements, hyperreflexia (over-responsive reflexes), and spasticity (Kent-Braun, Walker, Weiner, & Miller, 1998). Spasticity is associated with an increase in muscle tone, also called hypertonia. When spasticity is present in bulbar muscles, it is associated with bilateral UMN lesions. Spasticity in spinal muscles can be caused by unilateral UMN lesions. Often, however, bilateral worsening of the limbs innervated by the UMN is common due to the nature of the disease resulting in spasticity on both sides of the body (Wijesekera & Leigh, 2009).

#### 1.3.2 Symptoms of Lower Motor Neuron Lesions

Loss of LMNs can lead to muscle atrophy, fasciculations, hyporeflexia (decreased response time), and hypotonia (Kent-Braun, et al., 1998). Fasciculations refer to the twitching of the voluntary muscles and are associated with lower motor neuron decline. These fasciculations are not limited to one specific muscle group but evident anywhere in the body. Flaccidity is associated with a decreased muscle tone (hypotonia) and eventually, fasciculations, flaccidity, and muscle atrophy are very prominent, particularly in the tongue of people with ALS. The

primary involvement of the tongue in this disease results in an inevitable decline of the ability to speak and swallow (Wijesekera & Leigh, 2009).

### 1.3.3 Spinal ALS

Those persons affected with the spinal form of ALS present muscle weakness that starts either distally or proximally in the upper and/or lower limbs (Wijesekera & Leigh, 2009). Spinal ALS causes muscle atrophy of the hands and shoulders in the upper limbs, and muscle atrophy of the proximal thigh or distal foot muscles in the lower limbs (Wijesekera & Leigh). Fasciculations or cramps are common symptoms of spinal ALS. The weakness that does occur is asymmetrical; however, as the disease progresses other limbs continue to degenerate and become weak. Those with spinal ALS eventually go on to develop bulbar symptoms (Wijesekera & Leigh).

### 1.3.4 Bulbar ALS

Bulbar ALS causes symptoms in the face and mouth muscle groups bilaterally and as the disease progresses, speech impairments (dysarthria) and swallowing problems (dysphagia) develop (Wijesekera & Leigh, 2009). Bulbar ALS can involve LMNs (also known as bulbar palsy), UMNs (known as pseudobulbar palsy), or both (Mitchell & Borasio, 2007). Within 1-2 years, a majority of those with bulbar signs will also develop limb weakness (Wijesekera & Leigh).

Weakness and fasciculation of the tongue are common in ALS patients and research has shown consistently that between the lip, tongue, and jaw, the tongue is the most severely affected structure (Langmore & Lehman, 1994; DePaul & Brooks, 1993). The weakness of mouth and face muscles is likely due to a worsening of several motor neurons associated with the following cranial nerves: the hypoglossal, the trigeminal, and the facial (DePaul & Brooks; Lawyer & Netsky, 1953; Bonduelle, 1975). Tongue weakness is present in both persons with mostly signs

of LMN deterioration and persons with mostly UMN deterioration (DePaul & Brooks); however, those with mostly LMN involvement demonstrate significantly reduced tongue strength compared to those with mostly UMN involvement (Langmore & Lehman).

### 1.3.5 Speech Characteristics

Dysarthria is common in ALS due to declining motor control necessary to articulate speech sounds precisely. The type of dysarthria associated in those with ALS is a mixed flaccid-spastic dysarthria (Darley, Aronson, & Brown, 1969). Slower than normal speaking rate, prolonged and distorted vowels, and shortened phrases are examples of speech characteristics linked with ALS (Weismer, Jeng, Laures, Kent, & Kent, 2000). In addition, it has been shown that vowels produced by speakers with ALS have reduced formant transitions, shallower transition, and a collapsed vowel space (Weismer et al.,).

Due to a deterioration of the muscles in the face and mouth that are under the control of the individual, speaking rate and speech intelligibility decline as the disease progresses resulting eventually in an inability to communicate orally. It is known that a change in speaking rate is evident before intelligibility declines and that at first speaking rate may slow, but overall intelligibility may stay high (Ball, Beukelman, & Pattee, 2004; Nishio & Nimi, 2000; Yunusova, Green, Greenwood, Wand, Pattee & Zunman, 2012). As the disease progresses, intelligibility then starts to decrease as well as speaking rate.

## CHAPTER 2

### MOTIVATION OF RESEARCH STUDY

Although many attempts have been made to delineate the underlying factors that contribute to speech intelligibility decline in speakers with ALS, currently very little is known about the articulatory underpinnings. In the acoustic domain, reduced vowel space has been shown to account for about 45% of intelligibility loss in speakers with dysarthria (Tjaden & Wilding, 2004; Turner, Tjaden, & Weismer, 1995). Thus, expanding vowel space is thought to be crucial for therapeutic success aiming to improve or to maintain speech intelligibility in speakers with ALS (Yorkston, 1996).

A current speech treatment approach to expand vowel space in speakers with ALS is the reduction of speaking rate (Nishio & Nimi, 2000; Yorkston, 1996). As shown in Figure 2, the rationale is based upon observed behavior of healthy speakers, which showed that rate reduction elicited significantly larger articulatory excursions during vowel productions, which in turn increased the distance between vowels in F1/F2 acoustic vowel space and, therefore, positively affected speech clarity/intelligibility in healthy speakers (Gay, 1978; Lindblom, 1990; Payton, Uchanski, & Braida, 1994; Mefferd & Green, 2010). In speakers with ALS, however, speech movements have rarely been studied and an understanding about the effect of slow speech on articulation and speech acoustics is very limited. It is currently not clear how ALS affects speech movements and how slow speech changes speech movements in these affected speakers.

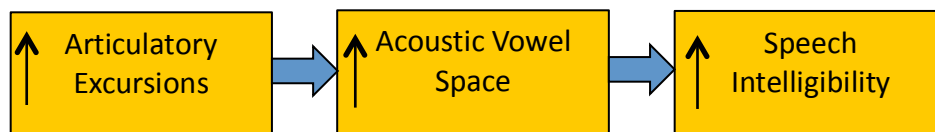


Figure 2. The effects of a slow rate of speech on articulation, acoustics, and speech intelligibility in healthy speakers.

In the past, acoustic studies have been completed in lieu of direct study on speech movements. These studies have shown that slow speech also expands vowel space in speakers with ALS; however, to a smaller extent when compared to healthy speakers (Turner, et al., 1995; Weismer et al., 2000). Currently, the underlying articulatory reasons for these differences between speakers with ALS and healthy speakers are unclear. One current assumption is that rate reductions may not elicit the same increase in tongue excursions in speakers with ALS as in healthy speakers (Weismer et al.,). To address this current gap in the literature, the purpose of this study was to:

(1) Determine how speakers with ALS and healthy controls differ in their speech movements and speech acoustics during habitual speech and to

(2) determine how slow speech affects speech movements and speech acoustics in speakers with ALS as compared to healthy speakers.

Therefore, this research study posed the following research questions:

(1) How do articulatory movements and speech acoustics differ between speakers with ALS and healthy speakers?

(2) How does speaking rate reduction affect articulatory movements and speech acoustics in speakers with ALS and healthy controls?

Based on previous studies on typical speakers, we expected to see significant increases in articulatory excursions and vowel space distance in response to slow speech (Mefferd & Green, 2010) in our control group. In speakers with ALS, however, we could not make any predictions for articulatory measures due the lack of literature available on direct speech movement analysis. Vowel space distance was expected to be reduced in speakers with ALS relative to healthy speakers as reported in previous studies (i.e., Turner et al., 1995). Although vowel space distance

was also expected to increase in speakers with ALS, the effect of slow speech on vowel space distance was expected to be smaller than in healthy speakers.

A secondary aim of this study was to determine the strength of association between changes in articulatory movements and speech acoustics (i.e., acoustic vowel precision).

Although it has been established that there is a strong linear relationship between articulatory and acoustic change in healthy speakers, this relationship has not been tested in impaired speakers. Such information, however, is clinically highly relevant to better predict changes in the acoustic signal of impaired speakers that will positively affect speech clarity and speech intelligibility since this is important for speech treatment outcomes.

Further, acoustic data acquisition is less costly and less time-consuming than speech movement acquisition; however, it is currently unknown if acoustic measures can be used instead of kinematic measures. If so, then they could be used in lieu of speech movement (kinematic) data to document disease progression and treatment effectiveness. Thus, the third research question in this study was:

(3) How strong is the association between articulatory and acoustic changes in response to speaking rate manipulation in speakers with ALS and healthy controls?

We expected to replicate previously reported strong correlations between increases in articulatory excursions and vowel space expansion in response to slow speech in healthy speakers. In speaker with ALS, we could not make any predictions because there was not sufficient literature available addressing articulatory-to-acoustic relations in impaired speakers.

## CHAPTER 3

### METHODS

#### 3.1 Participants

This study included five speakers with ALS (Mean age = 61.8 years,  $SD = 6.41$ ), and five gender-matched healthy controls (Mean age = 59.8 years,  $SD = 7.59$ ). All participants with ALS were diagnosed by a board-certified neurologist and none of the participants reported any other neurological disease. All participants passed a hearing screening at 0.5, 1, 2, and 4 kHz at 30 dB to assure adequate hearing. Table 1 provides further demographic information and prominent speech characteristics of speakers with ALS.

TABLE 1

ARTICULATORY RATE (AR) AND SENTENCE INTELLIGIBILITY (SIT) FOR SPEAKERS WITH ALS

| ID | Sex    | AR  | SIT | Severity |
|----|--------|-----|-----|----------|
| A1 | male   | 170 | 98  | mild     |
| A2 | male   | 211 | 99  | mild     |
| A5 | male   | 127 | 95  | moderate |
| A6 | female | 116 | 96  | moderate |
| A7 | male   | 96  | 55  | severe   |

Note: AR=Articulatory Rate, SIT=Sentence Intelligibility Test

#### 3.2 Experimental Tasks

First, all participants completed the Speech Intelligibility Test (SIT) (Beukelman, Yorkston, Hakel, & Dorsey, 2007) to determine their typical articulatory rate (AR) and the speech severity of speakers with ALS. This involved reading ten sentences that were mixed in

complexity and yielded minimal predictability of word use. An untrained listener orthographically transcribed the sentences using a software program. As shown in Table 1, each participant with ALS was given a rating of mild, moderate, or severe based upon their sentence intelligibility test.

Next, all participants were asked to complete five repetitions of the sentence “See a kite again” at a typical speaking rate and again at approximately half of their typical speaking rate. The diphthong /ai/ in “kite” was analyzed in the study.

### 3.3 Experimental Setup

Articulatory movements were tracked using the 3D Electromagnetic Articulograph (AG500, Medizintechnik Carstens). The participant was seated inside a plexiglass cube, which was surrounded by six electromagnets. The electromagnets established a low electromagnetic field inside of the cube. When small sensor coils are placed in the electromagnetic field, a low-grade current is evoked and depends on the relative distance between the sensor and the six electromagnets. Voltages are amplified and submitted to an algorithm that then establishes the 3D position of each sensor in the field at a sampling rate of 200Hz with sub-milimeter accuracy (Yunusova, Green, & Mefferd, 2009).

For the purpose of this study, two sensors were placed on the midline of the tongue approximately 1.5cm (TT) and 4cm (TB) posterior to the tip of the tongue. However, only the TB sensor was analyzed to answer our research question. Two sensors were placed on the right and left lower canine teeth to track jaw movements. Those sensors were not analyzed in this study. Further, three sensors were placed on plastic goggles (head right, head left, head center), which the participant wore during the data collection. These sensors served as head reference sensors.

The acoustic data was collected with a professional grade lavalier microphone, which was positioned approximately 20 cm away from the participant's mouth. Acoustic data was synchronized with the kinematic data and recorded with a sampling rate of 16 kHz.

#### 3.4 Data Analysis

The diphthong /ai/ in “kite” was analyzed for the purpose of this study. Acoustic data was displayed in the spectrographic view in TF32 (Milenkovic, 2005). As shown in Figure 3, the diphthong /ai/ was identified using the stop plosive characteristics as landmarks. The first glottal impulse of the diphthong determined the onset; the last glottal impulse of the diphthong determined the offset of the diphthong production. The time between the onset and offset of the diphthong production was recorded as a durational measure to evaluate if speakers successfully changed their speech durations from typical to slow speech. For each participant, mean durations across all five repetitions at the typical and at the slow speaking rate were averaged and submitted for further statistical analysis.

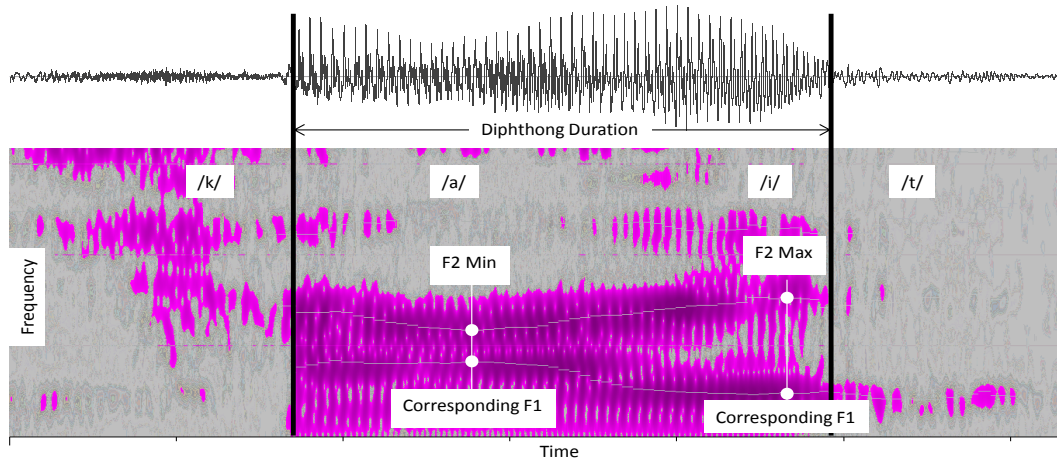


Figure 3. Measuring vowel duration and vowel space distance. The black lines indicate the onset and offset of the target /ai/ in the “kite” used to determine the duration of /ai/. The white points illustrate the landmarks for extracting F1 and F2 formant frequencies, which were used to determine the vowel space distance between /a/ and /i/.

The LPC algorithm in TF32 was used to determine the first and second formants (F1 and F2, respectively) during the diphthong /ai/ in “kite.” The LPC algorithm was used at its default setting and formant mis-trackings were corrected manually. Because F2 has been shown to be more sensitive to speaking rate changes than F1 (Turner et al., 1995) the F2 minimum and the corresponding F1 value and F2 maximum and the corresponding F1 values were selected for the vowels /a/ and /i/, respectively (Figure 3) and entered into an Excel spreadsheet.

To determine vowel space distance, the 2D Euclidean distance between /a/ and /i/ in F1/F2 vowel space was calculated based on the extracted F1 and F2 values of /a/ and /i/ (Figure 4). For each participant, the resulting values of five repetitions at a typical and slow speaking rate were averaged and submitted for further statistical analysis.

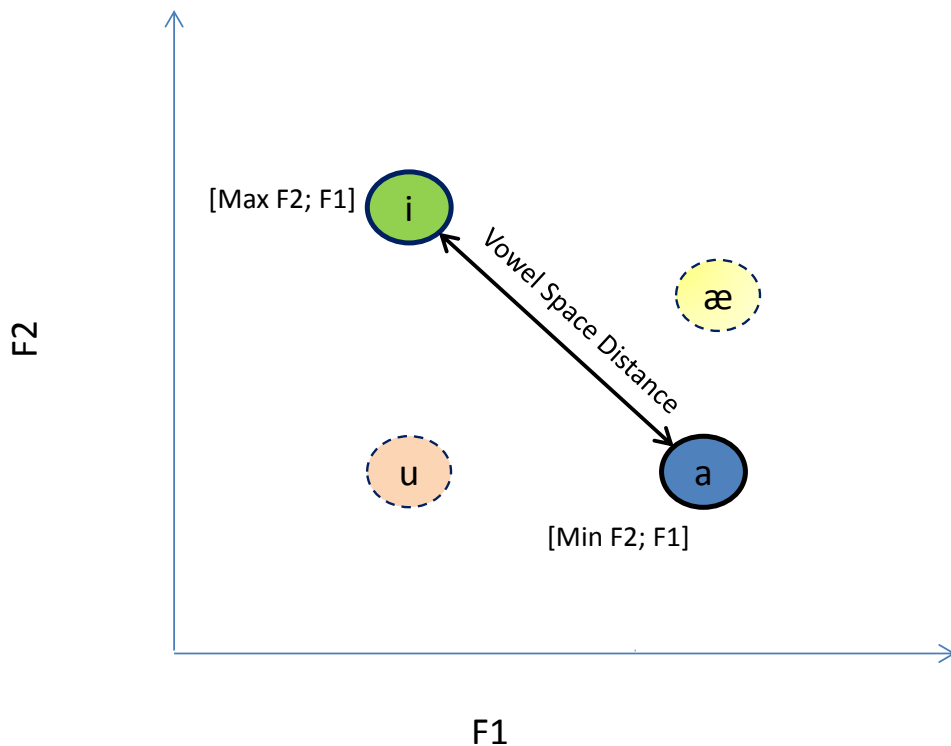


Figure 4. Corner vowels traditionally used to determine acoustic vowel space and the vowel space distance between /a/ and /i/ used in this study.

In the kinematic domain, all recorded movements were corrected for head movements using the Normpos software program provided by Medizintechnik Carstens. Further movements were filtered with a low-pass filter at 18 Hz, which is the conventional procedure for speech kinematic data. Figure 5 displays the posterior tongue movement during the production of “kite” and signals the portion of the diphthong /ai/ and the tongue excursion that was measured between /a/ to /i/.

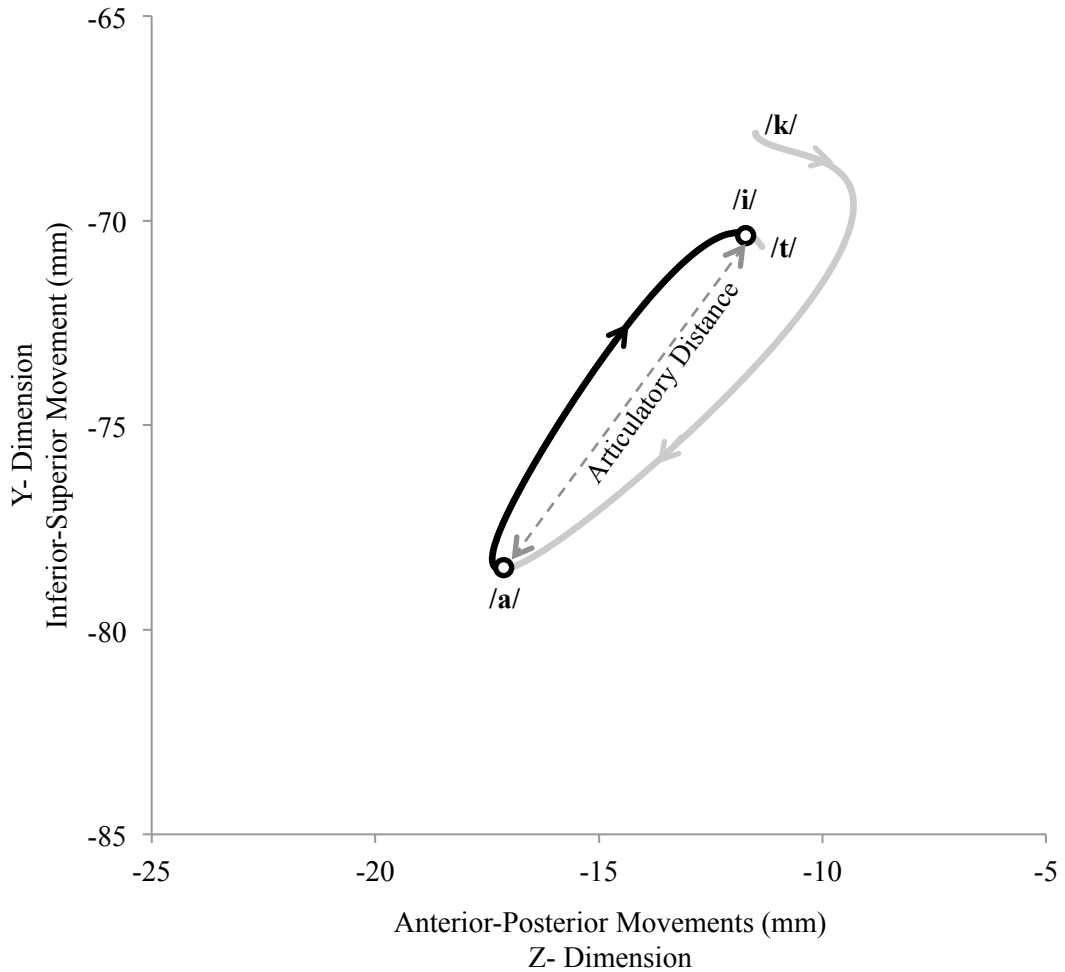


Figure 5. Posterior tongue movement during the production of “kite”. The dark line represents the tongue excursion that was measured.

In Figure 6 the kinematic data of the posterior tongue during the word “kite” is displayed for each of the three movement dimensions (X = lateral-medial; Y = superior-inferior, Z = anterior-posterior) as a function of time. The maximum and minimum tongue position in the Y-dimension and the corresponding coordinates in the X- and Z-dimensions were selected to retrieve the positional data of the tongue during the production of /a/ and /i/, respectively (Figure 5). Tongue excursions between /a/ and /i/ were then calculated based on the 3D-Euclidean distance formula. For each participant vowel space distances were averaged across the five

repetitions in typical and slow speech. Average values were submitted for further statistical analyses.

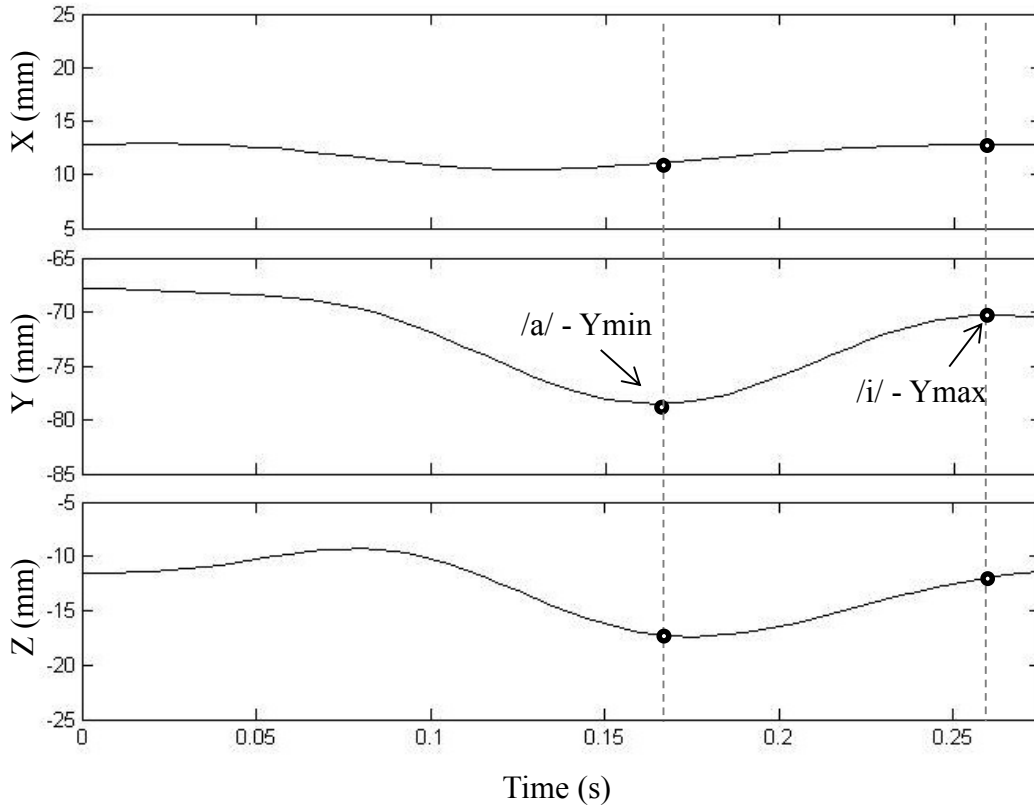


Figure 6. Measuring and calculating the tongue excursions during the production of /a/ and /i/ in “kite”.

### 3.5 Statistical Analysis

In this study, only data of the four speakers with mild-moderate ALS and controls were compared using inferential statistics. The speaker with severe ALS was analyzed in a descriptive fashion due to the severity of speaking rate and articulatory rate. Non-parametric tests were chosen in this study due to the small sample size and violation of assumptions required for parametric statistics. A disadvantage of the non-parametric tests, however, is that they are rather conservative. Based on the mild-moderate speech severity of our four speakers with ALS, we

expected small to moderate effect sizes. Our a priori power analysis using a Cohen's  $d$  value of 0.4 and a desired statistical power of 80% returns a sample size of 30 speakers in each experimental group. Therefore, clearly, this study serves as a pilot study and is very low in statistical power.

Speaking rate changes from typical to slow speech were evaluated by comparing durations of /ai/ between typical and slow speech within each group using the Wilcoxon signed rank test, which parallels the parametric paired  $t$ -test, and between groups (ALS vs. Controls) using the Mann-Whitney  $U$  test, which parallels the parametric independent  $t$ -test. In a similar fashion, the speaking rate effects on kinematic and acoustic measures were determined within each group by submitting tongue excursions and vowel space distances separately to Wilcoxon signed rank tests. Between-group differences for typical and slow speech were statistically analyzed using the Mann-Whitney  $U$  test. Lastly, the raw data (the measured value of each repetition during typical and slow speech) of articulatory excursions and vowel space distances were submitted to a bivariate correlational analysis (Pearson's correlation) for each participant in this study to determine the strength of association between articulatory and vowel acoustic change.

### 3.6 Inter-rater Reliability

Whereas the kinematic measurements were based on customized algorithms and do not yield the potential of a rater bias, an inter-rater reliability analysis was completed for the acoustic measures. For this purpose, the data of two healthy speakers and two speakers with ALS were randomly selected and re-analyzed by a second experimenter. Inter-rater reliability was significant for diphthong durations [ $r(48) = .96, p < .001$ ] and vowel space [ $r(48) = .96, p < .001$ ]. The average measurement error for diphthong duration was 0.021 seconds and the average

error vowel space was 43 Hertz. These reliability measures were similar to those of previous acoustic studies (e.g., Weismer et al., 2000).

## CHAPTER 4

### RESULTS

#### 4.1. Task Performance - Diphthong Durations

Figure 7 shows group means of diphthong durations for typical and slow speech as well as the individual means across five repetitions for the speakers with severe ALS. The Wilcoxon signed rank test showed a significant speaking rate effect on diphthong duration in the control group ( $p < .025$ ) and ALS group ( $p < .046$ ). In both groups, slow speech was associated with significantly longer diphthong durations than typical speech. The speaker with severe ALS showed a minimal increase in diphthong duration from typical speech ( $M = 0.370$ ) to slow speech ( $M = 0.400$ ).

Between-group comparisons returned no statistical significant results. However, it is noteworthy that during typical speech diphthong durations of speakers with ALS ( $M = .243$ ,  $SE = 0.6$ ) tended to be longer than those of healthy controls ( $M = 0.150$ ,  $SE = 1.3$ );  $U = 3.00$ ,  $p = .085$ . For slow speech, diphthong durations tended to be similar between groups (ALS  $M = .323$ ,  $SE = 1.9$ ; Controls  $M = .300$ ,  $SE = 0.8$ );  $U = 9.00$ ,  $p = .806$ . The speaker with severe ALS had longer diphthong durations than all other speakers with mild ALS and the control group during typical speech and slow speech (Figure 7).

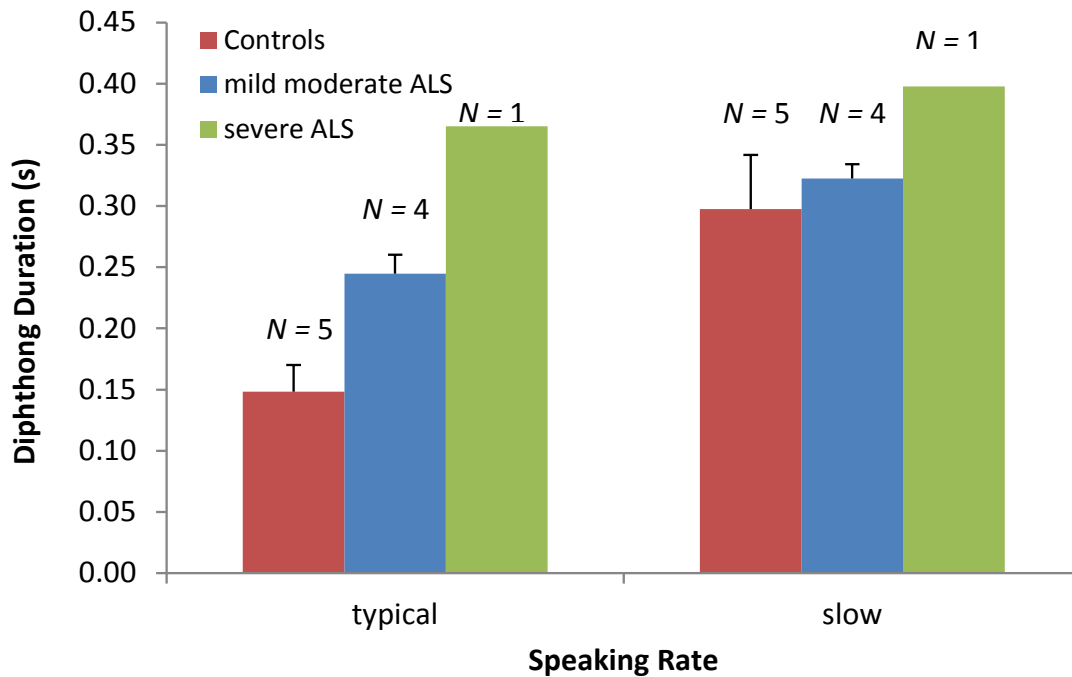


Figure 7. Mean diphthong durations of control group and speakers with ALS as a function of speaking rate.

#### 4.2. Speaking Rate Effect – Articulatory Excursions

Figure 8 shows the mean articulatory tongue excursions of speakers with mild-moderate ALS, severe ALS, and healthy controls during typical and slow speech. To determine if tongue excursions significantly changed between the two speech tasks, mean tongue excursions were compared between typical and slow speech. In the control group, slow speech elicited significantly larger tongue excursions ( $M = 14.36$ ,  $SE = 1.51$ ) than during typical speech ( $M = 10.66$ ,  $SE = 0.8$ );  $Z = 2.023$   $p = .043$ . In the ALS group, however, no significant difference was found between the slow ( $M = 13.8$ ,  $SE = 1.9$ ) and typical speech conditions ( $M = 11.6$ ,  $SE = 1.3$ ). For the speaker with severe ALS, there was little change in the diphthong duration from typical ( $M = 9.86$ ) to slow ( $M = 8.57$ ).

To determine if speakers with ALS had significantly different tongue excursions from healthy controls, tongue excursions were compared during typical and slow speech between the

speakers with ALS and healthy controls. No significant results were found; however, healthy speakers tended to have smaller articulatory excursions compared to speakers with mild-moderate ALS during typical speech. During slow speech, healthy speakers tended to have larger tongue excursions than speakers with mild ALS. The speaker with severe ALS had smaller tongue excursions than healthy speakers and the mild ALS during both, typical and slow speech.

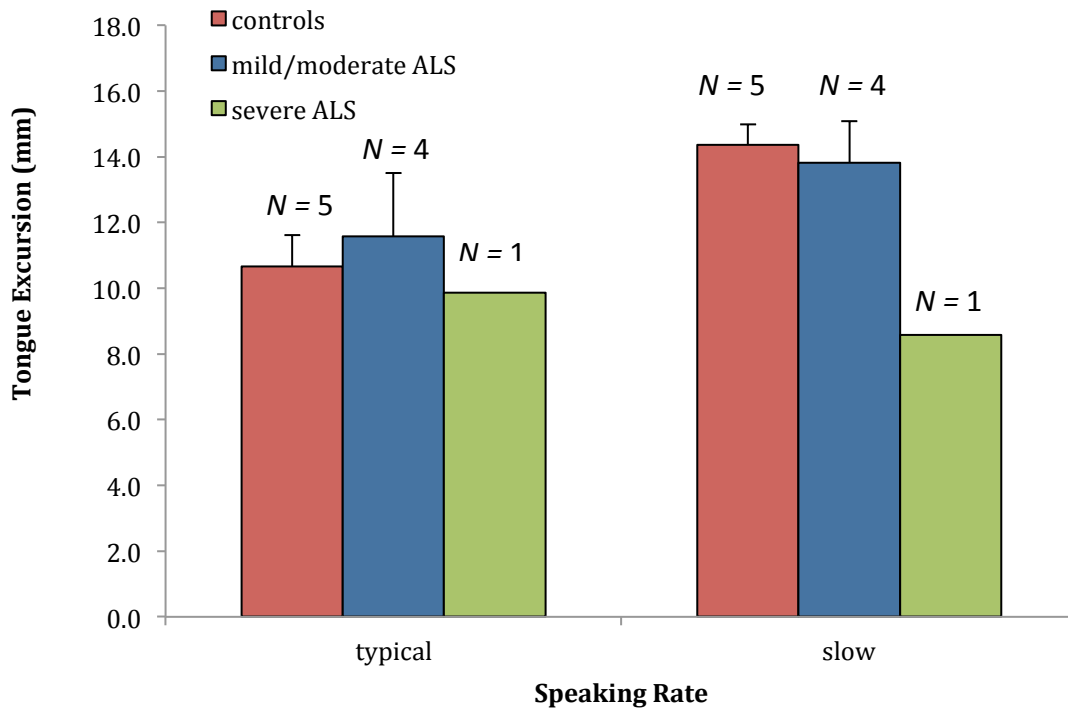


Figure 8. Mean tongue excursions of control group and speakers with ALS as a function of speaking rate.

#### 4.3. Speaking Rate Effect –Vowel Space Distance

Figure 9 shows the mean vowel space distances during typical and slow speech for speakers with mild-moderate ALS, severe ALS, and healthy controls. To determine if vowel space distances significantly differed between typical and slow speech, vowel space distances were compared between typical and slow speech in each group. In the control group, significant increases in vowel space distance were found from typical speech ( $M = 810.9, SE = 51.0$ ) to slow

speech ( $M = 1051.4$ ,  $SE = 101.0$ );  $Z = 2.023$ ,  $p = .043$ . In the mild-moderate ALS group, increases in vowel space distance were not significant; however, vowel space also tended to increase from typical speech ( $M = 615.2$ ,  $SE = 65.1$ ) to slow speech ( $M = 808.8$ ,  $SE = 116.4$ );  $Z = 1.826$ ,  $p = .068$ . For the speaker with severe ALS, there was a minimal increase in vowel space distance from typical speech ( $M = 518.78$ ) to slow speech ( $M = 541.45$ ).

To determine if vowel space distance differed between speakers with mild-moderate ALS and controls, vowel space speech distances were compared during typical and slow speech between the two groups. Although no significant differences were found, healthy controls tended to have a larger vowel space distances than speakers with mild-moderate ALS during typical and slow speech;  $Z = 1.915$ ,  $p = .086$  and  $Z = 1.015$ ,  $p = .086$ , respectively). For the speaker with severe ALS, vowel space distance increased slightly in response to a slow rate of speech.

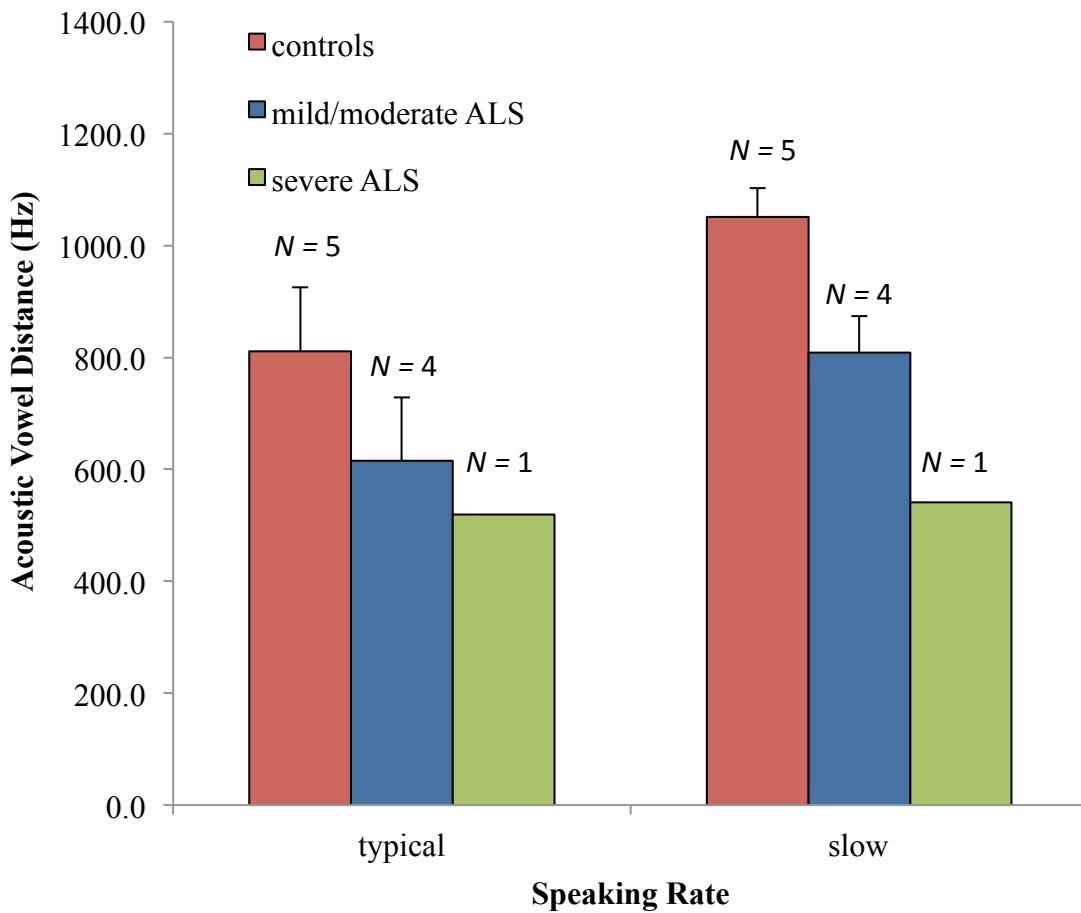


Figure 9. Mean acoustic vowel space of control group and speakers with ALS as a function of speaking rate.

#### 4.4. Articulatory-to-Acoustic Relations

The correlations between changes in tongue excursion and changes in vowel space distance in response to slow speech were plotted individually for each participant in Figures 10 (all controls) and 11 (all speakers with ALS). Table 2 and Table 3 display Pearson correlation  $r$ -values,  $p$ -values, and the number of data points included in the correlational analysis. Please note that due to sensor tracking errors less than ten data points were available.



TABLE 2.

CORRELATIONS OF ARTICULATORY AND ACOUSTIC CHANGES IN CONTROL GROUP

| <b>ALS</b> | <b>Severity</b> | <b>N</b> | <b>p-value</b> | <b>Pearson's r</b> |
|------------|-----------------|----------|----------------|--------------------|
| <b>A1</b>  | <b>mild</b>     | 6        | .05            | -.80               |
| <b>A2</b>  | <b>mild</b>     | 6        | .01            | .96                |
| <b>A5</b>  | <b>moderate</b> | 9        | .01            | .79                |
| <b>A6</b>  | <b>moderate</b> | 10       | .27            | .39                |
| <b>A7</b>  | <b>severe</b>   | 8        | .40            | .36                |

TABLE 3.

CORRELATIONS OF ARTICULATORY AND ACOUSTIC CHANGES IN SPEAKERS WITH ALS

| <b>Cont.</b> | <b>N</b> | <b>p-value</b> | <b>Pearson's r</b> |
|--------------|----------|----------------|--------------------|
| <b>C2</b>    | 10       | .05            | .64                |
| <b>C4</b>    | 10       | .00            | .94                |
| <b>C5</b>    | 10       | .00            | .90                |
| <b>C7</b>    | 10       | .06            | .60                |
| <b>C8</b>    | 10       | .00            | .86                |

## CHAPTER 5

### DISCUSSION

This study sought to determine how speakers with ALS and healthy controls differ in their speech tongue excursions and vowel acoustic distance during habitual speech and how a slow rate of speech affects tongue excursions and vowel space distances in speakers with ALS as compared to healthy speakers. Based on previous studies, it was hypothesized that healthy speakers would demonstrate a significant increase in tongue excursions and vowel space distances in response to a slow rate of speech. In speakers with ALS, however, we could not make predictions on the effect of slow speech on tongue excursions. Based on previous acoustic studies, we expected to find smaller effects of a slow rate of speech on vowel space distance (Turner et al., 1995; Weismer et al, 2000).

Further, this study aimed to determine the strength of association between changes in tongue excursion and vowel space distance. Based on previous studies we expected a strong correlation between changes in tongue excursions and vowel space distance in healthy speakers. Again, in speakers with ALS no predictions could be made.

#### 5.1. Speaking Rate Effects on Diphthong Durations

Our findings for diphthong durations showed that slow speech elicited durational changes of the target diphthong /ai/ in both groups; however, the effect was smaller for speakers with ALS than healthy controls. We observed that the relative change in diphthong durations from typical to slow differed between the two groups. For example, to achieve slow speech, speakers with mild to moderate ALS exhibited proportionally smaller increase in diphthong duration (a 32% increase) than did healthy controls (a 100% increase). Further, the speaker with severe

ALS, having already extremely slowed typical speech, exhibited an 8% decrease in diphthong duration. However, absolute diphthong durations during slow speech differed only slightly between the speakers with mild-moderate ALS and healthy controls. Even the durations of the speaker with severe ALS were in the range of healthy speakers. Previous studies have also reported similar segmental durations and utterance length durations during slow speech despite observable differences in durations during typical speech (Turner & Weismer, 1993; Turner et al., 1995; Weismer et al., 2000) and may suggest that speaking rate can only be slowed to a certain point before the cohesiveness of speech movements would be disrupted and speech intelligibility would be negatively affected (Weismer et al.). Thus, it appears that the slower the typical speaking rate of a speaker, the smaller the proportional decrease to achieve slow speech. This observation is of clinical importance because it poses the question if the effect of slow speech on speech movements and speech acoustics diminishes as the proportional durational change decreases. We will address this question below when we discuss our findings of the articulatory and acoustic measures.

## 5.2 Speaking Rate Effects on Tongue Excursions

The observed rate effects indicate that slow speech results in improved articulation and speech acoustics in both groups of speakers. Although slow speech elicited larger articulatory excursions and expanded vowel space in both groups of speakers, the increase tended to be slightly greater in healthy controls than in speakers with ALS. Previous data has also shown that articulatory excursions became larger in response to slow speech in healthy speakers (Gay, 1978; Lindblom, 1990; Mefferd & Green, 2010). Therefore, we were able to replicate previous findings in our study when evaluating the performance of healthy controls. For speakers with ALS, however, tongue movements in response to slow speech have not been evaluated previously. It

was often speculated that slow speech may not elicit the same increase in tongue excursions as in healthy speakers because acoustic studies on the effect of slow speech on vowel space changes could only find diminished effects for speakers with ALS when comparing results to healthy controls (Turner et al., 1995; Weismer et al., 2000). The observable trend of a relatively smaller increase in tongue excursions in response to slow speech in speakers with ALS supports this assumption.

Speech severity may impact the effect of speaking rate reduction on tongue excursion. Overall, a slight reduction in tongue excursions was noted in response to the slow rate in the speakers with severe ALS. Although the observation is only based on one speaker, it suggests that as speech may become more impaired, slow speech may not be beneficial as it was during the earlier stages of the disease or in healthy speakers. Of course, this is a preliminary observation and should only be interpreted with great caution. More data is necessary to better understand the impact of speech severity on tongue excursion changes in response to slow speech.

### 5.3. Speaking Rate Effect on Vowel Space Distance

Findings in the acoustic domain paralleled findings in articulation. Although slow speech yielded an increase in vowel space for both groups, vowel space expansions tended to be slightly greater for healthy controls than speakers with ALS. These acoustic findings are consistent with other reports on healthy speakers and speakers with ALS. For example, Lindblom (1990) as well as Mefferd and Green (2010) reported increases in vowel space in response to slow speech in healthy speakers. For speakers with ALS, Turner and colleagues (1995) also reported that speakers with ALS had relatively smaller changes in vowel space in response to speaking rate changes as compared to healthy speakers.

Speech severity may impact the effect of slow speech on vowel space distance. Overall, in the speaker with severe ALS, little change in response to rate changes were noted. Although the observation is only based on one speaker, it suggests that only little improvements are possible when the speech is already severely impaired due to disease progression. It is noteworthy that the speakers with severe ALS also achieved the smallest durational changes from typical to slow speech. It is possible that a limited ability to lengthen speech sound durations (perhaps due to already extremely long durations during typical speech) underlies the diminished changes in acoustic vowel space.

#### 5.4. Between Group Findings for Tongue Excursions

To date, it has been speculated that tongue excursions may be smaller in speakers with ALS because their vowel space sizes were relatively small when compared to healthy speakers (Turner et al., 1995; Weismer et al., 2000). However, direct studies on tongue excursions in speakers with ALS were rare. One of the few studies available on tongue excursions in speakers with ALS provided partial support for the speculations of relatively small excursions. This study reported similar or smaller articulatory excursions of the tongue for speakers with ALS and healthy controls depending on the target utterance (Yunusova et al., 2012). We observed a trend of relatively larger tongue excursions during typical speech, which is somewhat discrepant to these previous reports on tongue excursion in persons with ALS reported by Yunusova and colleagues. However, the discrepancies may be due to different methodologies in studying speech movements. For example, we analyzed the movement of the sensor attached to the posterior part of the tongue while the speaker could freely move the jaw. In the study by Yunusova and colleagues, the analyzed sensor was much closer to the tip of the tongue during vowel productions while the jaw was fixed with a 5mm bite block and could not be moved

during speech. It is possible that the relatively large movement excursions in our study are due to the influence of the jaw. Indeed, it has been previously suggested that the jaw may compensate in speakers with ALS for a progressively weakened tongue and, therefore, moves more during speech in speakers with ALS (DePaul & Brooks, 1993).

#### 5.5. Between-Group Findings for Vowel Space Distance

In contrast to slightly larger articulatory excursions in speakers with ALS than healthy controls during typical speech, vowel space distances were notably smaller in speakers with ALS than in healthy controls. These acoustic findings are consistent with findings from Turner, Tjaden, and Weismer (1995) and Weismer et al. (2000), who also found relatively smaller vowel space areas for speakers with ALS than controls. In addition, these acoustic findings are consistent with findings from Yunusova and colleagues (2012) who found that ALS speakers demonstrated a smaller F2 range as compared to healthy speakers. Specifically, in their study a slower rate of speech did produce a larger F2 range in speakers with ALS as compared to normal speech, but the F2 range was smaller than the control group. The healthy speakers increased their F2 range when speaking slowly. However, the speakers with ALS showed a reduced F2 range, most likely due to the progression of the disease (Yunusova et al. 2012).

#### 5.6. Articulatory and Acoustic Correlations

The second aim of the research was to determine the articulatory-to-acoustic relations in response to a slow rate of speech in speakers with ALS. As predicted our findings showed strong associations between articulatory and acoustic changes in response to slow speech in healthy speakers. As the articulatory excursions increased, the acoustic vowel space also increased. This strong linear articulatory-to-acoustic relationship in healthy controls is congruent with previous findings (Mefferd & Green, 2010). In speakers with ALS, no predictions could be

made because there were no previous studies available. Articulatory-to-acoustic relations varied greatly and included one negative correlation, two positive strong correlations, as well as a couple of non-significant weak correlations. Thus, it can be said that for some speakers with ALS, increasing the tongue excursions will also increase the vowel space distance for these speakers. However, in other speakers, no predictions can be made about how changing tongue excursions may affect the vowel space distance. One explanation for these weak correlations between articulatory and acoustic changes may be that although changes in tongue excursions were made by the impaired speaker, the appropriate tongue shape and constrictions towards the hard palate were not achieved. Support for this hypothesis is provided by Kuruvilla, Green, Hogan, Yunusova, and Ayaz (2012), who have shown previously that it becomes progressively more difficult for speakers with ALS to independently move tongue segments (i.e., tongue tip vs. tongue root).

Interestingly, speech severity did not appear to have an impact on the strength of the articulatory-to-acoustic relations. A strong correlation was observed for a speaker with moderate speech severity and a lower correlation value was observed for a speaker with very mild speech severity. However, the data of the speaker with the most severe speech impairment showed a weak correlation between articulatory and acoustic changes in response to slow speech. Thus, it is currently not clear which factors determine the strength of the association between articulation and acoustics.

A bit more puzzling is the negative correlation of one speaker with ALS. As observed in previous experiments, small changes in articulatory movements may not elicit any change in the acoustic signal due to non-linearities or quantal effects (Stevens, 1989; Perkell & Cohen, 1989). That is, the acoustic signal is thought to be insensitive to such small changes in articulation. On

the other hand, it has also been reported that very subtle change in articulatory movements can elicit a large change in the speech acoustic domain (Stevens, 1989). Yet, a negative effect has not been reported before. It is possible that such a negative relationship is a characteristic of a speech impairment and the speaker's possible attempts to compensate for it in various ways (i.e., changes in larynx height, which we did not study).

One of the most surprising findings of this study was the discrepancy between the slightly larger articulatory excursions in speakers with ALS and the relatively small vowel space distance in speakers with ALS. So far, it has always been assumed that a small vowel space area is indicative of relatively small tongue excursions (Lindblom, 1990). This assumption, however, was based on observations of healthy speakers. It appears not to hold true for speakers with speech impairments. Several explanations may be plausible. Firstly, this observation may be contributed to non-linearities between the vocal tract configurations and their acoustic consequences (Stevens, 1989). Although the movements of speakers with ALS are larger than those produced by healthy speakers, speakers with ALS may not be able to achieve the critical vowel-specific constrictions towards the hard palate. Further, as mentioned above, it is also possible that despite large articulatory excursions the overall tongue shape to produce specific sounds is not sufficiently distinguished in speakers with ALS, which would explain the poor acoustic outcome despite the relatively large tongue excursions during typical and slow speech.

### 5.7. Clinical Implications

Vowel space expansion has been shown to enhance speech intelligibility of speakers with dysarthria (Tjaden & Wilding, 2004; Weismer, et al., 2000). Thus, because overall vowel space distances increased for speakers with ALS, the findings in this study support the use of speaking rate reduction to improve intelligibility in speakers with ALS. Further, the variable correlations

between change in articulation and speech acoustics for speakers with ALS suggest that only in some cases acoustic measures can be used in lieu of direct speech movement analysis to document underlying speech processes. Along those lines, the results of our study showed that vowel space measures are not well suited to estimate between-group differences in speech movement excursions.

## 5.8 Limitations

One limitation of this study was the small sample size. A larger sample size would allow the drawing of stronger conclusions. Secondly, a wider range of speech severity in a larger sample would allow an investigation on the impact speech severity has on tongue excursions and the effects of slow speech. Further, this study investigated how acoustic measures change in response to slow speech; however, to gain more comprehensive insights, more research is needed to determine the listener's perspective about the impact of the acoustic change in a speech perceptual context. Ideally, listeners would rate the recorded speech samples at a typical and slow speech and determine under which condition speech is easier to understand. In other words, our study could only provide insights in how a slow rate of speech affects the tongue excursions and the speech acoustics, but the perceptual implications were not studied.

## CHAPTER 6

### CONCLUSION

In conclusion, this research shows that a slow rate of speech elicits improvement in tongue excursion and acoustic vowel space in both typical speakers and those with ALS. The findings of healthy speakers are harmonious with other findings (Mefferd & Green, 2010). In speakers with mild and moderate ALS, changes in tongue excursion also elicited predictable changes in acoustic vowel space in some speakers. Interestingly, relative to controls, greater tongue excursions in speakers with ALS elicited smaller acoustic vowel space. This observation may be explained by non-linearities between vocal tract configurations and their acoustic consequences (Stevens, 1989). That is, although speakers with ALS may produce larger movements than controls, they may not achieve critical vowel-specific constrictions in the vocal tract. Alternatively, difficulty to achieve distinct tongue shapes and vocal tract constrictions along the hard palate may negatively affect the acoustic signal despite relatively large overall tongue excursions.

## CHAPTER 7

### FUTURE RESEARCH

Future research should focus on the identification of factors that predict how strong the strength of articulatory to acoustic relations are in impaired speakers with ALS. This way, clinicians can use acoustic data to infer the articulatory performance in speakers with ALS and document disease progression or treatment effectiveness.

Further, future research should also investigate the specific location of constriction in the vocal tract during specific vowel sounds rather than merely measuring the relative change in distance between two speech sounds based on one single flesh point on the tongue. Such a refined approach would provide an improved understanding about weak correlations that were observed between articulatory and acoustic changes in response to slow speech in some speakers with ALS.

More research is also needed in the area of perceptual changes that occur in response to a slower rate of speech. Little is known about the relationship between articulatory and acoustic change and their effect on speech intelligibility. In other words, how much change in the articulatory movements is required to elicit change that is sufficient enough to change speech intelligibility in impaired speakers.

The current research included one speaker with ALS that is severe based on the results of the speech intelligibility test. While the data for the speaker with severe ALS are tracked separately in the graphs, it would be important to conduct larger studies with more speakers with severe speech impairments to compare the results of a group of speakers with mild-moderate speech severity due to ALS to a group of speakers with severe speech impairments. This would

provide more insight into how disease progression affects the effects of slow speech and the articulatory-to-acoustic relations.

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