

Acoustic Intensity and Speech Breathing Kinematics in a Patient with Parkinson's Disease

by

Katherine McGovern

B.A., Communication Science, University of Pittsburgh, 2018

Submitted to the Graduate Faculty of
School of Health and Rehabilitation Sciences
of the requirements for the degree of
Master of Science

University of Pittsburgh

2020

UNIVERSITY OF PITTSBURGH
SCHOOL OF HEALTH AND REHABILITATION SCIENCES

This thesis was presented

by

Katherine McGovern

It was defended on

March 19, 2020

and approved by

James Coyle, Ph.D., CCC-SLP, BCS-S, ASHA Fellow, Director of Student Financial Aid &
Awards and Professor, Communication Science and Disorders

Bharath Chandrasekaran, Ph.D., Vice Chair for Research and Professor, Communication Science
and Disorders

Thesis Advisor: Susan Shaiman, Ph.D., CCC-SLP, Director of MA/MS SLP Program and
Associate Professor, Communication Science and Disorders

Copyright © by Katherine McGovern

2020

Acoustic Intensity and Speech Breathing Kinematics in a Patient with Parkinson's Disease

Katherine McGovern, M.S.

University of Pittsburgh, 2020

Parkinson's disease (PD) is a neurodegenerative disease which affects the basal ganglia control circuit (Duffy, 2013). The motor speech disorder most strongly associated with PD is hypokinetic dysarthria, which presents with distinctive speech characteristics including reduced loudness and the inability to adequately maintain loud speech (Darley, Aronson, & Brown 1969; Duffy 2013). This is due to the variable kinematics for speech breathing associated with PD, which may result in abnormal muscular excursions, reduced vital capacity, and irregular breathing cycles (Duffy, 2013). The impaired ventilatory control can be attributed to the rigidity of muscles of inhalation and exhalation, as well as bradykinesia and hypokinesia.

The study aimed to evaluate whether a patient with PD was able to manipulate their acoustic intensity, and if such intensity changes were accompanied by changes in speech breathing kinematics in a novel intraoperative environment.

The study's data were collected intra-operatively during surgery for deep brain stimulation and recordings from the subthalamic nucleus and cortex. The patient was instructed to modulate acoustic intensity while repeating three syllable CV triplets. Speech breathing kinematics of the rib cage were obtained using a Piezo Crystal Effort Sensor with a double buckle band throughout speech production. The speech breathing kinematics of interest were duration, displacement, and peak velocity of inhalation, peak velocity of exhalation, and duration from onset of exhalation to onset of speech, as well as a descriptive comparison between tidal breathing and speech breathing.

Spearman Rho correlations indicated that there were weak to no relationships observed between speech breathing kinematics and intensity in this specific participant. However, a medium effect size (Hedge's g) was observed between tidal and speech breathing for inhalation duration, and small to medium effect size for inhalation displacement and peak velocity.

While previous literature suggests that people with PD can manipulate intensity when cued as a result of kinematic modulations for speech breathing, the current study does not support these findings for this one patient. However, previously reported differences between tidal and speech breathing were supported. Potential explanations for the lack of intensity modulation are explored, including constraints induced by the intra-operative environment.

Table of Contents

Preface.....	x
1.0 Introduction.....	1
1.1 Healthy Adults	2
1.1.1 Healthy Young Adult Physiology.....	3
1.1.2 Healthy Older Adult Physiology	7
1.1.3 Healthy Adult Kinematics for Speech Breathing.....	10
1.1.4 Healthy Adult Manipulation of Intensity.....	14
1.2 Parkinson’s Disease	18
1.2.1 Parkinson’s Disease Pathophysiology	19
1.2.2 Parkinson’s Disease Kinematics for Speech Breathing	21
1.2.3 Parkinson’s Disease Manipulation of Intensity	24
1.3 Grant Purpose.....	27
1.4 Specific Aims of the Current Study	27
2.0 Methods.....	32
2.1 Participants	32
2.2 Speech Tasks	33
2.3 Instrumentation	35
2.4 Data Analysis	36
2.4.1 Acoustic Intensity	36
2.4.2 Speech Breathing Kinematics	37
2.5 Statistical Methods	39

3.0 Results	41
4.0 Discussion.....	48
Appendix A Agonist-Antagonist Muscles for Ventilation	57
Appendix B Funding.....	59
Bibliography	60

List of Tables

Table 1 Stimulus Condition and the Dependent Variables	42
Table 2 Correlations of Intensity and Speech Breathing Kinematics (n=115)	43
Table 3 Speech Breathing and Tidal Breathing Inhalations	45

List of Figures

Figure 1 CV Stimuli	33
Figure 2 Ventilatory Displacement (Top) and Velocity (Bottom).....	38
Figure 3 Correlation of Intensity and Expiratory Peak Velocity	44
Figure 5 Tidal vs. Speech Breathing Inhalation Duration	46
Figure 6 Tidal vs. Speech Breathing Inhalation Displacement.....	46
Figure 7 Tidal vs. Speech Breathing Inhalation Peak Velocity	47

Preface

First and foremost, I would like to thank my thesis advisor, Dr. Susan Shaiman, for her unwavering guidance and support throughout this thesis. You have taught me SO much about the research process, while also guiding and mentoring me through graduate school; I simply cannot thank you enough. Thank you to my thesis committee members, Dr. Jim Coyle and Dr. Bharath Chandrasekaran, for serving on my committee and challenging me to create the best project that I could. Also, thank you to Dr. Jay Bohland for serving as my moderator during my defense.

Thank you to those involved with the grant for letting me be a part of this project, especially Dr. Mark Richardson, Dr. Alan Bush, Dr. Anna Chrabaszcz, Dr. Julie Fiez, Dr. Lori Holt, and Tara Pirnia. A special thanks to Christina Dastolfo-Hromack and Dr. Witold Lipski for their time and effort in helping me access the data and for sharing vital information. Finally, thank you to Dr. Fernando Llanos for generating Praat scripts for data analysis.

A massive thank you to my thesis partner-in-crime, Jennifer Gates, for enduring this journey with me and for creating laughter out of stress. Last and certainly not least, I would like to thank my mama for lending an ear and a shoulder to cry on when I felt overwhelmed by the process, my partner, Nicholas, for being my #1 fan, and my gal pals for listening to me talk about this project for two years! And to my feline friend, Khloé, for sitting on my lap and purring during the countless hours of data analysis, literature review, and writing.

It takes a village and I certainly could not have done it without all of those mentioned!

1.0 Introduction

Parkinson's disease (PD) is a neurodegenerative disease which affects the basal ganglia (Duffy 2013; Huber & Darling 2011). PD is not uncommon among the elderly population, with more than a half million adults older than 45 years of age living with PD in the United States (Marras et al, 2018). One of the disease symptoms is disruption of normal speech production. It affects speech production by impacting various subsystems including articulation, ventilation, and phonation, which may develop into motor speech disorders, such as a dysarthria (Duffy, 2013; Sadagopan & Huber, 2007).

The motor speech disorder most strongly associated with PD is hypokinetic dysarthria (Darley, Aronson, Brown 1969; Duffy 2013). Hypokinetic dysarthria's characteristics include reduced loudness and the inability to adequately maintain loud speech (Darley, Aronson, Brown 1969; Duffy 2013). This is due to the variable kinematics for speech breathing associated with PD, including abnormal muscular excursions, reduced vital capacity, and irregular breathing cycles (Duffy, 2013). This impaired ventilatory control can be attributed to the rigidity of muscles of inhalation and exhalation, as well as bradykinesia and hypokinesia. The functionality of the ventilatory system is key to support variation in acoustic intensity, but the disease process of PD changes this functionality. This research will evaluate whether a participant with PD is able to manipulate their acoustic intensity, and if such intensity changes are accompanied by changes in inspiratory and expiratory kinematics. Intensity of speech will be manipulated by having the participant repeat stimuli presented auditorily at both soft and loud hearing levels. If the average vowel intensities produced by the participant are significantly different between the two stimulus conditions, then this study will explore if there are differences in the duration, amplitude and peak

velocity of the ventilatory kinematic signal. If no significant difference is found in acoustic intensity, then a relationship between average vowel intensity and ventilatory kinematics will be explored.

In order to understand this research objective, we must first begin with a discussion of how healthy normal people produce loud speech. This will be discussed through an explanation of the normal physiology for speech breathing, how healthy normal adults adjust kinematics for speech breathing, and how researchers have manipulated intensity in healthy adults. These sections will be reviewed for healthy young adults, as well as healthy old adults, due to the age-related differences. Then, we will consider the literature about people with PD. A short background of PD and physiology of PD for speech breathing will be explained. Once this is understood, kinematic changes for speech breathing in people with PD will be discussed, followed by a discussion of the manipulation of intensity for people with PD. This will lead into the research questions for this paper and the methodology for investigating these questions.

1.1 Healthy Adults

Before there is a discussion of healthy adult speech breathing physiology and kinematics, terminology that will be used throughout the paper must be defined. First, *compliance* is “the ability of a structure to undergo stretching or displacement,” (p. 566) according to Rousseau & Branski (2018). Second, the authors state that the *elasticity* of the lungs is defined as the “tendency of an elastic element to snap back to its original position when displaced” (p. 571). Thus, *compliance* is the ability to be displaced and *elasticity* is the ability to return to equilibrium after displacement. *Rigidity*, however, is the resistance to movement and often is described as muscle

stiffness (Duffy, 2013; Rousseau & Branski, 2018). Rigidity prevents muscles from being stretched or displaced and can be considered the converse of compliance. (For a further analysis of rigidity, specifically in PD, please refer to Appendix A.) Elasticity, compliance, and rigidity will be referenced throughout the paper when discussing the lungs, rib cage, associated muscles, and the Parkinson's disease process. In addition, the *lung thoracic unit (LTU)* will be mentioned throughout the paper and is defined as the lungs, rib cage, and pleural linkage. With the terms defined, a discussion of healthy adult physiology can commence.

1.1.1 Healthy Young Adult Physiology

Normal physiology of the lungs and ribcage for the purpose of respiration and ventilation has been explained by various sources. Zemlin (1998) described the normal physiology in his textbook and recent textbooks have described similar physiological processes, such as Rousseau & Branski (2018) and Hixon, Weismer, & Hoit (2020). For this explanation, information from the texts of all three sources will be used.

To have an understanding of normal physiology for speech breathing, there must be a discussion of respiration and ventilation. Respiration is the exchange of gases, oxygen and carbon dioxide, through the circulatory system for life-sustaining pH balances (Rousseau & Branski, 2018). Whereas, ventilation is the transfer of air into and out of the lungs; oxygen rich air is brought into the lungs through inhalation, while air that is primarily composed of carbon dioxide is taken out of the lungs through exhalation (Hixon et al. 2020). Simply, respiration is gas exchange and ventilation is air exchange, but both are life-sustaining mechanisms. Ventilatory inhalation and exhalation are performed in order to maintain vegetative breathing, or life-sustaining breathing. However, throughout this paper, the term *speech breathing* will be used. Speech breathing is “the

process by which driving forces are supplied to generate the sounds of speech, while simultaneously serving the functions of ventilation and gas exchange” (Hixon et al., 2020, p. 60). Therefore, speech breathing is using the anatomy and physiology that is typically used for vegetative purposes to produce speech.

Prior to an explanation of vegetative and speech breathing, the anatomy for breathing will be discussed. According to Hixon et al. (2020), the anatomy of the chest wall consists of the lungs, rib cage, and abdomen. The lungs are made up of three right lobes and two left lobes, as well as the pulmonary airways consisting of the bronchi, bronchioles, and alveoli. The rib cage is composed of twelve pairs of ribs that are connected to the sternum anteriorly through costal cartilages and posteriorly to the thoracic vertebrae. The authors state that breathing is both passive and active; passive components arise from elastic recoil forces, whereas active forces arise from the muscles of the chest wall. There are muscles that are responsible for elevating and expanding the rib cage, including pectoralis major, pectoralis minor, and the scalenes, and there are muscles that contribute to depressing the rib cage, including rectus abdominus and transverse abdominus. The muscle that separates the thoracic cavity from the abdominal cavity is the diaphragm. The lungs, rib cage, and diaphragm are connected through a pleural linkage, which is a connective membrane that enables the lungs to expand as volume of the chest wall changes. Breathing is ultimately controlled by the nervous system which initiates muscles contraction for vegetative and speech breathing.

Hixon et al. (2020) explain that ventilation is produced through muscle contraction and pressure changes. For vegetative breathing, the muscle that is contracted is the diaphragm. The diaphragm contracts, causing the shape of the diaphragm to become relatively flat compared to its resting state of being dome-shaped. This diaphragmatic contraction (as well as contraction of the

intercoastal muscles) increases the volume of the lungs through rib cage expansion and connection of the pleural linkage. As the volume of the lungs increases, the alveolar pressure, or pressure within the alveoli, decreases. Thus, the alveolar pressure is less than atmospheric pressure and air from the atmosphere rushes into the lungs. As alveolar pressure begins to equal atmospheric, this marks the end of an inhalation for vegetative breathing. Then, the diaphragm relaxes and begins the expiratory phase of breathing. Once the diaphragm relaxes, elastic recoil forces of the lungs cause the lung volume to decrease and the alveolar pressure to become higher than atmospheric pressure. Due to the high alveolar pressure, air rushes out of the lungs until the alveolar pressure equals atmospheric pressure, marking the end of an exhalation. According to Zemlin (1998), the duration of an inhalation and exhalation for ventilation is relatively equal: about 50% of a vegetative breathing cycle is inhalation, and about 50% is exhalation. During vegetative breathing, people inhale a certain volume of air during any single expiratory cycle, known as tidal volume (Zemlin, 1998). The author explains that the average tidal volume for ventilation is about 500 ml, which is the quantity of air needed for adequate gas exchange. This overall process of ventilation for vegetative breathing supports the exchange of oxygen and carbon dioxide to sustain life.

In contrast to vegetative breathing, speech breathing is an adaptation of the above process for communication. The movements of the breathing apparatus contribute to the control of acoustic intensity, vocal frequency, prosodic stress, and the segmentation or duration of utterances (Hixon et al. 2020). The amount of air inhaled and how quickly the air is inhaled differ based on the intended acoustic intensity, prosodic influences, linguistic goal, and goal duration of the utterance. Although the total duration of vegetative breathing is about 50% inhalation and 50% exhalation, speech breathing produces a rapid inhalation, contributing to about 10% of the total speech breathing cycle, while exhalation is a sustained and controlled duration, contributing about 90%

of the total speech breathing cycle (Hixon et al., 2020; Zemlin, 1998). The rapid inhalation provides us the necessary quantity of air in order to produce an extended exhalation since speech is produced upon exhalation. Although the exact quantity of air is dependent on the multiple factors stated above, the quantity is larger than the 500ml for vegetative breathing (Zemlin 1998). This larger quantity provides the pressure and force to generate speech through the breathing apparatus.

As stated by Hixon et al. (2020), “speech breathing is achieved through the combining of relaxation pressure and muscular pressure” (p. 60). The authors explain that *relaxation pressure* is the pressure generated from the LTU, or the natural elastic recoil pressure of the lungs and rib cage. The current relaxation pressure determines the necessary muscular forces required at any moment, in order to produce the target alveolar pressure of the utterance. The muscles and relaxation pressure work together to produce the targeted speech message. Zemlin (1998) explains this muscular act of the LTU as a “checking action.” This is the sustained contraction of the muscles of inhalation in order to counteract the relaxation pressure of the LTU. A large volume of air inhaled creates a natural elastic recoil force with which the muscles must balance out. The rate of exhalation and alveolar pressure of the utterance is controlled by the checking action, in order to produce the desired loudness, prosody, and duration. For example, loud speech is produced at a higher alveolar pressure and individuals create this high alveolar pressure by taking in a larger inhalation, yielding a larger lung volume. This larger lung volume allows individuals to take advantage of the high relaxation pressure in order to produce loud speech. The checking action enables the exhalation to be sustained over a longer period of time, or else the LTU would quickly collapse back to equilibrium. The combined efforts generate a loud and timely utterance. Thus, speech breathing uses the lung and rib cage apparatus to achieve communication, while also serving the life-sustaining function of vegetative breathing.

In normal, healthy adults, the role of the lungs, rib cage, and abdomen are essential for adequate vegetative breathing, as well as speech breathing. Vegetative breathing is performed primarily through action of the diaphragm and pressure gradient and supports life-sustaining breathing. Whereas speech breathing is dependent on the communicative purpose, muscular effort, and relaxation pressure. Muscles and pressure are the driving forces for breathing movement. The upcoming section will discuss the effects of aging on normal physiology for speech breathing.

1.1.2 Healthy Older Adult Physiology

Aging affects the body in numerous ways and the ventilatory and phonatory systems are not spared from normal aging. The description of healthy young adult ventilatory physiology and kinematics that was previously discussed remains grossly the same as we get older. However, there is evidence for a degree of age-related changes.

Janssens, Pache, & Nicod (1999) looked into physiologic changes related to speech breathing that are associated with aging. The researchers reviewed literature that considered aging across a continuum but focused on individuals older than 65 years of age when researching age-related changes. The research with which they were interested involved structural changes, pulmonary function, gas exchanges, and regulation of breathing for adults as they age. Pulmonary function tests, such as spirometry, were conducted to assess the above changes.

The authors reviewed literature that provided evidence for calcification of the rib cage, costal cartilages, and intervertebral disk spaces, which results in a reduction of chest wall compliance. The compliance of the rib cage provides information about how well it can expand so that the individual can inhale the proper amount of air. The reduction of chest wall compliance is

coupled with the lungs becoming more distensible with age (Janssens et al. 1999). In addition, the lungs begin to decrease elasticity with age, according to the literature review. Thus, with age, the rib cage becomes more resistive to movement, while the lungs become more compliant and less elastic. This rib cage and lung anatomy has major influence on the residual volume and vital capacity. The authors found that there is evidence for an increase of residual volume and vital capacity as we age due to the decreased elasticity of the lungs. Not only is the compliance and elasticity of the LTU affected, but specific parts of the lungs also change. The alveoli dilate, airspaces enlarge, surface area of the peripheral airways decrease, and there is a loss of supporting tissue within the peripheral airways with age (Janssens et al. 1999). Structural changes appear to be prominent throughout the ventilatory system as we age on a global level.

Postural changes also infringe on the LTU. There is evidence that the greater prevalence of kyphosis with age decreases the functionality of the diaphragm, resulting in decreased force generation of the diaphragm (Janssens et al. 1999). Kyphosis causes a mechanical barrier to typical diaphragmatic structure. However, there is also a decrease of overall strength of the diaphragm as we age, in addition to other muscles. Janssens et al. (1999) explain that there is a decrease in muscle strength, mass, and muscle fibers associated with aging for ventilatory and phonatory musculature.

In a related study, Hoit & Hixon (1987) confirmed similar age-related changes to the structures of the breathing apparatus across three age ranges: 25, 50, and 75 years. They assessed speech breathing through linearized magnetometry on the abdomen and rib cage, as well as acoustic signals. Data were recorded for both vegetative and speech breathing. They discussed evidence that the mechanism for age-related functional changes can be explained by structural changes. The authors indicated that this provides evidence of physiological changes including

adjustments in laryngeal valving and the LTU. The researchers explain that the main sources of age-related changes are atrophy, thinning of muscles, and ossification of the ventilatory and phonatory system. This is similar to the structural changes reported by Janssens et al. (1999).

With respect to valving changes of the larynx, the laryngeal muscles atrophy, the elastic fibers of the vocal ligaments become thin, and the laryngeal cartilages ossify (Hoit & Hixon 1987). The researchers suspect that the reduction in laryngeal airway resistance is due to the atrophy of the intrinsic laryngeal muscles, specifically. Not only do the muscles atrophy, but there is a reduction in ventilatory driving pressure within the older adult group of participants (Hoit & Hixon 1987). The reduction of driving pressure causes the phonatory system to adapt by changing laryngeal valving. The LTU also adapts in order to maintain typical function with concomitant structural changes. According to Hoit & Hixon (1987), older adults had larger lung volume and rib cage volume initiations and excursions compared to younger adults. This is likely explained by the drive of the LTU to maintain functional goals (ex. loudness manipulation) when coupled with other age-related influences.

Janssens et al. (1999) and Hoit & Hixon (1987) provide evidence for normal age-related changes of the ventilatory and phonatory system. These changes include atrophy, ossification, calcification, compliance, elasticity, and physiologic adjustments compared to younger adults. The research from Janssens et al. (1999) and Hoit & Hixon (1987) is consistent with more recent literature. Huber, Darling, Francis, & Zhang (2012), Huber & Spruill (2008), and Lalley (2013) confirmed that changes in physiology for speech breathing in older adults includes increased compliance of the lungs, decreased compliance of the rib cage, decreased elastic recoil forces, and decreased strength of the muscles of ventilation. By changing the structure and physiology of the

ventilatory and phonatory system due to aging, there also are changes in kinematics for speech breathing. A discussion of normal kinematic characteristics of speech inhalation follows.

1.1.3 Healthy Adult Kinematics for Speech Breathing

Normal kinematics for speech breathing begins with normal musculature explained above. As the muscles of inhalation and exhalation contract for speech breathing, there are reciprocal movements of the abdomen and ribcage that occur. These movements allow researchers to infer changes within the lungs, due to the connection of pleural linkage within the LTU. Researchers have studied kinematics of the chest wall in order to understand lung volume changes during different speech tasks by using tools, such as a magnetometer. The measurement of chest wall displacements gives researchers an estimation for lung volume due to the reciprocal displacement maintained by the pleural linkage. Kinematics for speech breathing will be discussed in this section through an explanation of past research on normal function. The authors used various methods to measure chest wall movement and used this information to determine lung volume changes.

As early as 1973, Hixon, Goldman, & Mead researched the kinematics of the chest wall during speech production. They looked at six healthy young adults and analyzed the kinematics of the ribcage and abdomen through magnetometers. The subjects performed non speech tasks, such as vegetative breathing, as well as speech tasks in utterance form, such as spontaneous conversation, and in CV repetitions. Subjects produced the speech tasks in normal, loud, and soft speech for each CV repetition and in upright and supine positions. The authors examined the volume displacements of the abdomen, rib cage, and lungs. They explained that healthy adults manipulate the chest wall in various ways for speech breathing. Not all of the healthy adult subjects changed the volumes of the rib cage and lungs in the same way, but the speech output was

successful and consistent among subjects. They determined that in order to produce louder speech, some participants took in a larger inhalation using the muscles of inhalation to take advantage of the elastic recoil forces during exhalation, whereas some participants expired greater amounts of air using the muscles of exhalation, exclusively or in addition to elastic recoil forces. However, a larger inhalation was generally used to produce loud speech because it allowed for the least amount of muscular energy. This allowed for greater elastic recoil forces at higher volumes to produce louder speech through an increase of pressure. So, the researchers concluded that there is a wide range of acceptable kinematics of the chest wall in healthy young adults for speech production.

Hixon et al. (1973) determined that there is more than one way to produce normal speech, but they did not consider how healthy adults determine the kinematic variation for speech output. Winkworth, Davis, Adams, & Ellis (1995) outlined how the kinematic variation can be chosen among healthy young adults. They studied the lung volumes, acoustic intensity, and linguistic elements of spontaneous speech using inductance plethysmography. The authors confirmed Hixon et al.'s (1973) findings that kinematics differ between normal speakers, but they also determined that there are individual differences within speakers. They reported physiologic and linguistic influences on kinematic variation. One of the primary reasons for normal lung volume variation were the linguistic factors of the message, suggesting an influence from neural planning on motor function for speech breathing. Consistent with the results from Hixon et al. (1973), Winkworth et al. (1995) described that acoustic intensity and lung volume are not exclusively associated. Some subjects took a larger inhalation to produce loud speech, but other subjects used different strategies to produce loud speech as outlined by Hixon et al. (1973). Winkworth et al. (1995) confirmed the previous evidence that there is normal kinematic variation that healthy young adults use to produce a larger acoustic intensity.

In addition to Hixon et al. (1973) and Winkworth et al. (1995), Huber, Chandrasekaran, & Wolstencroft (2005) looked into the different cues that increase loudness in normal subjects. The study examined the ventilatory mechanisms used to increase loudness in three loud conditions: asking subjects to reach a specific SPL value, asking subjects to speak twice as loud, and asking subjects to speak while listening to noise. The authors measured chest wall kinematics congruent with the previous research in order to estimate lung volumes. Resptrace bands were placed on the rib cage and abdomen to transduce movements and calculate estimated lung volumes. They identified that the three conditions produced three different kinematic variations of the chest wall for speech breathing, but each produced similar intensity levels. In the specific SPL condition, participants tended to use an increased lung volume to take advantage of high elastic recoil pressures to produce loud speech and when participants were to speak twice as loud as comfortable, they tended to use increased muscle tension to produce loud speech. Yet, when participants spoke in noise, they used a combination of high elastic recoil pressure and muscle tension to speak loudly. In all conditions, participants were all able to increase loudness, regardless of the kinematic variation of the LTU. Interestingly, the researchers found that the abdomen did not play a crucial role in increasing loudness, which they found to be in agreement with previous studies. This is notable because it provides evidence for the crucial role of the rib cage and lungs in producing loud speech. Overall, the results are consistent with findings from Hixon et al. (1973) and Winkworth et al. (1995) that kinematics for speech breathing may differ among and within individuals based on the goal of the utterance.

The kinematics for speech breathing of healthy young adults is similar to the kinematics for speech breathing of healthy older adults, with the exception of age-related changes. Regardless

of age, normal movement of the rib cage gives insight into lung volume. Kinematic changes due to aging influence lung volumes and impact speech breathing.

Janssens et al. (1999) and Hoit & Hixon (1987) discussed the changes in kinematics with age, in addition to changes in physiology. As previously stated, there is evidence for atrophy, ossification, increased compliance of the lungs, decreased compliance of the chest wall, decreased elastic recoil forces, and decreased strength of muscles of ventilation as we age. According to Hoit & Hixon (1987), kinematics for speech breathing were also found to differ with age through changes in lung volume excursions, rib cage initiations, number of syllables per breath group, and lung volume expended per syllable. When compared to healthy young adults, healthy older adults produced shorter utterances on average and fewer syllables per breath group, which may be attributed to the physiologic changes in the LTU, including valving differences within the breathing apparatus (Hoit & Hixon 1987). Huber et. al (2012) also supported the findings that there is an increase of effort from muscles of inhalation. The age-related changes make it difficult for older adults to inhale to larger lung volumes and utilize the muscles for checking action when speech breathing. Thus, older adults produce shorter utterances compared to their younger counterparts. In addition to fewer syllables per breath group, Janssens et al. (1999) explained that muscle performance for speech breathing and elastic recoil differences cause an increase in functional residual capacity for older adults. This means that older adults begin inhalation at higher lung volumes when speech breathing compared to young adults, although total lung volume does not differ with age. The researchers also stated that a higher functional residual volume is associated with an additional burden on the ventilatory musculature, which is already burdened with age-related changes. Therefore, aging influences kinematics for speech breathing by

increasing the functional residual capacity, variation in muscular effort of the muscles of inhalation, and the production of shorter utterances on average among older adults.

The research discussed in this section explains normal variations in kinematics for speech breathing in healthy adults. Taking a larger inhalation and utilizing elastic recoil forces during exhalation may be the primary method to increase acoustic intensity, but relying more substantially on the muscles of exhalation may be chosen instead. Healthy young adults manipulate their rib cage and abdomen in different ways, which impacts lung volume and how the intended speech is produced. The kinematic manipulation of the chest wall is based on the internal goal, such as linguistic influences or the intended target, such as loud speech. Older adults have similar kinematics for speech breathing, with the exception of shorter utterances per breath group, differences in muscular effort, and an increase in functional residual capacity of the lungs. These kinematic adaptations are due to the physiologic changes seen in aging. However, these are a range of normal functions and normal variations for adults to produce their targeted speech output. Since kinematics for speech breathing are dependent on the characteristics of the intended utterance, such as loudness, a discussion of the manipulation of intensity will follow.

1.1.4 Healthy Adult Manipulation of Intensity

During this section, there will be a discussion of manipulation of intensity, as well as loudness. The distinction between intensity and loudness will be defined prior to beginning the section. According to Hixon et al. (2020), *intensity* is the magnitude of sound energy, whereas *loudness* is the subjective perception of intensity, according to Rousseau & Branski (2018). Acoustic intensity is typically measured in dB SPL, while vocal loudness is individually perceived by each listener (Rousseau & Branski, 2018; Zemlin, 1999). However, the terms *intensity* and

loudness will be used somewhat interchangeably throughout the section, based on the terms that the cited authors used in their research.

To begin, intensity can be manipulated by researchers through a variety of methods. A well-researched technique to increase acoustic intensity in any individual is the Lombard effect. The Lombard effect is conducted by playing ambient noise in a speaker's ears and having them produce speech (Lane & Tranel 1971; Winkworth & Davis 1997). When ambient noise is introduced, the speaker talks at a higher intensity (Lane & Tranel 1971; Winkworth & Davis 1997). In Winkworth & Davis' (1997) study, background noise was presented via headphones at 55 and 70 dB and participants were instructed to simultaneously read orally or give a spontaneous monologue. No instructions were given about changing acoustic intensity, yet a Lombard effect was observed. All subjects increased acoustic intensity during the noise conditions through various kinematic methods. Lung volumes were shown to be more variable and larger during noise conditions, consistent with the previous section's findings on kinematics for speech breathing. The research provided evidence that healthy adults increase acoustic intensity when presented with background noise and that they tend to increase acoustic intensity by taking in a larger inhalation during these conditions. In addition to the previous study, Lane & Tranel (1971) reviewed literature and explained that the Lombard effect occurs in healthy adults. They confirmed that people try to create a signal-to-noise ratio that is beneficial for communication, thus increasing their intensity in the presence of noise. The Lombard effect remains stable with age. Matheron, Stathopoulos, Huber, & Sussman (2017) compared laryngeal aerodynamic measures among healthy older adults and people with PD by presenting the participants with multi-talker background noise. The results focusing on people with PD will be discussed in a later section, but

the researchers confirmed that the Lombard effect is constant over time as we age by concluding that the acoustic intensity of healthy older adults increased in the presence of background noise.

In addition to speaking in noise, researchers have studied other methods to increase acoustic intensity among individuals. Huber et al. (2005), who were mentioned in the previous section, considered different cues to increase loudness among healthy adults. They explored three loud conditions: asking participants to reach a specific SPL value, asking subjects to speak twice as loud, and asking subjects to speak while listening to noise (prompting the Lombard effect). As formerly stated, the first condition resulted in increased lung volume, the second condition resulted in increased muscular effort from the muscles of exhalation, and the last used a combination of the two. The internal target of the utterance was shown to influence the kinematics for speech breathing that were used. However, all of the conditions were shown to elicit similar increases in acoustic intensity among the participants. Parallel results were seen in Huber (2007), who used similar methodology to further explore the kinematics for speech breathing when different loudness cues were presented. These findings suggest that the methods to manipulate intensity that were used (including targeting an SPL value, speaking twice as loud as comfortable, and talking in noise) are effective.

Manipulating intensity through various cues is shown to be successful in healthy adults, but Baker et al. (2001) considered how young adults and older adults compared. Four young participants and five older participants were asked to produce a series of syllables at soft, comfortable, and loud levels of loudness. The authors were curious about the effect of aging on the mechanisms for speech breathing, including ventilatory and laryngeal mechanisms. Across the three loudness conditions, healthy older participants generated SPL values that were lower in magnitude compared to their younger counterparts. However, the researchers also found that the

older adults modulated loudness in similar ways compared to healthy young adults. This provides evidence that healthy older adults are able to manipulate acoustic intensity levels depending on the goal of the utterance, but they tend to manipulate acoustic intensity at lower magnitudes overall. For example, if a young adult produces loud speech at 90 dB SPL, then an older adult may produce loud speech at 80 dB SPL. Both loudness levels are considered “loud,” but the older adults are not as loud as the young adults.

As can be seen, researchers have used a variety of techniques to manipulate acoustic intensity in healthy adults, including elicitation of the Lombard effect, targeting an SPL value, speaking twice as loud as possible, and speaking at different subjective loudness levels. All of the conditions produced successful modulations of acoustic intensity in participants. Although healthy older adults produced speech at lower intensity levels when compared to younger adults, they still successfully modulated their intensity levels. This section supports the effectiveness of acoustic intensity modulation in healthy adults when presented with various intensity manipulation conditions.

With the understanding of healthy physiology, kinematics for speech breathing, and manipulation of acoustic intensity, there now can be consideration of how disease processes influence normal function. In the following sections, there will be a discussion of the influence of Parkinson’s disease on physiology, kinematics for speech breathing, and manipulation of acoustic intensity compared to healthy adults. To begin, a brief overview of PD will be provided.

1.2 Parkinson's Disease

Parkinson's disease (PD) is a common neurodegenerative disorder that affects the elderly population. PD is a parkinsonism of unknown etiology, whereas neurodegenerative diseases that go beyond the typically signs and symptoms of idiopathic PD are referred to as Parkinson's plus disorders (Duffy 2013). The Parkinson's plus disorders are Multi System Atrophy (MSA), Progressive Supranuclear Palsy (PSP), Diffuse Lewy Body disease, and Cortico Basal Ganglionic Degeneration (CBGD) (Ramig, Meyer, Fox, Blitzer, & Tagliati, 2005). However, for this research, there is a focus on idiopathic PD specifically. PD is typically associated with a decrease in the production of dopamine, which can commonly be treated by pharmaceuticals, such as levodopa (Duffy 2013; Zigmond & Burke, 2002). When motor impairments become unmanageable by levodopa alone, people with PD may elect to receive deep brain stimulation (DBS) inserted into the subthalamic nucleus (STN) in order to improve motor function and reduce tremulousness (Project Information, n.d.). The clinical signs of PD are characterized by rigidity, bradykinesia, resting tremor, and often asymmetric onset and postural abnormalities (Duffy 2013; Ramig et al 2005; Zigmond & Burke, 2002). Not all of these features must be present for a PD diagnosis, but each provides further evidence for a certain diagnosis. In addition to the clinical signs of PD, Zigmond & Burke (2002) report that there are often concomitant cognitive and psychiatric disorders associated with PD, such as dementia. In fact, Duffy (2013) states the prevalence of dementia is about 40% and the prevalence of depression is about 40-60% in people with PD. Although these motor, cognitive, and psychiatric manifestations are key components of PD, there are also clear speech disturbances associated with PD. These speech disturbances are influenced by physiology of the disease process. Thus, the pathophysiology of PD will be further explained in the following section.

1.2.1 Parkinson's Disease Pathophysiology

As previously discussed, aging causes normal changes in phonation and ventilation. People with PD have these changes along with concomitant changes due to the disease process. While the four principal manifestations of PD are rigidity, bradykinesia, tremor, and postural instability, there will be a focus on rigidity and bradykinesia because of their influence on speech disturbances. *Rigidity* is a resistance of movement, thus creating reduced range of motion of the articulators, laryngeal musculature, and ventilatory musculature (Darling-White & Huber 2017; Duffy 2013; Zigmond & Burke, 2002). The rigidity associated with PD goes beyond the effects of calcification and ossification associated with age. In addition, kinematics for speech breathing are impacted by bradykinesia and hypokinesia (Darley, Aronson & Brown 1969; Duffy 2013). *Bradykinesia* reduces the speed of muscles and generates problems with initiation of movement (Duffy 2013). Essentially, bradykinesia is slow movement. Along with slow movement, *hypokinesia* reduces the range of motion of automatic and voluntary movements (Duffy 2013). Thus, initiation, rate, and range of motion for speech is disrupted, specifically disruption of the mechanisms used for articulation, phonation, and ventilation. There is also evidence for a decrease in muscular strength and coordination that goes beyond the natural aging process, according to Darling-White & Huber (2017). All of these factors influence speech production and eventually form into a motor speech disorder.

Darley, Aronson & Brown (1969) and Duffy (2013) explain that the hallmark motor speech disorder of PD is hypokinetic dysarthria (HKD). The authors state that HKD affects more than 90% of people with PD. HKD is distinct from other dysarthria types through association with the basal ganglia control circuit, which includes the cortex, thalamus, striatum, lentiform nucleus,

substantia nigra, and subthalamic nuclei. This dysarthria cannot be explained by rigidity or bradykinesia alone, rather there are multiple factors that produce HKD. The basal ganglia control circuit dynamically processes and integrates motor, sensory, temporal, affective, cognitive, and executive functions (Sapir, 2014). According to Duffy (2013), the basal ganglia is involved in allowing intended voluntary movements, prevents unwanted and competing movement, and balances these two movements without perseverating. The basal ganglia's role in speech is to provide voluntary motor control. Duffy (2013) explains that the voluntary motor control of speech is disrupted in HKD, such as the preparation, maintenance, and switching of motor programs. Characteristics of HKD are most apparent in prosody, articulation, and voice, but it affects all aspects of speech. According to Duffy (2013), people with PD often complain that their voice is 'quieter' or 'weak,' as well as that they 'talk too fast' and it's 'hard to get speech started.' Prominent disrupted speech characteristics include rapid and atypical speech, specifically syllable repetitions and lengthened syllables. There is also reduced range of movement (hypokinesia) of the speech mechanisms, which produces imprecise articulation and reduced loudness. Duffy (2013) continues that voice abnormalities also are present and characterized by hoarseness, breathiness, and tremulousness. Overall, the most typical characteristics of HKD in people with PD are "monopitch, monoloudness, reduced loudness, reduced stress, short phrases, variable rate, short rushes of speech, and imprecise consonants" (Duffy, 2013, p.173). These can all affect a person's comprehensibility, which can influence her/his social interactions and possibly quality of life. As the disease progresses, the signs and symptoms of HKD also might develop. In addition to speech disturbances, there are other signs of the disease including tremor in the jaw and lips, masked facies (expressionless facial expressions), and dysphagia.

Rigidity, bradykinesia, hypokinesia, and disruptions in the basal ganglia control circuit play a role in the signs and symptoms of HKD. Once again, HKD is primarily characterized by “monopitch, monoloudness, reduced loudness, reduced stress, short phrases, variable rate, short rushes of speech, and imprecise consonants” (Duffy, 2013, p. 173). The speech disturbances associated with HKD, especially the prosodic abnormalities, are closely related to changes in kinematics for speech breathing. Therefore, kinematics for speech breathing in people with PD will be discussed in the following section.

1.2.2 Parkinson’s Disease Kinematics for Speech Breathing

Kinematics for speech breathing contribute to some of the prominent features in HKD, primarily those related to prosody, loudness, and utterance length. Duffy (2013) details the ventilatory changes associated with HKD and PD. The author states that there is evidence for “reduced vital capacity, amplitude of chest wall movements, and respiratory muscle strength and endurance, as well as irregularities in breathing patterns and increased respiratory rates” (p. 175). Many of these changes can be attributed to the rigidity of the rib cage and ventilatory muscles during movement for speech breathing. Specifically related to speech, Duffy (2013) explains that there is documentation of reduced maximum vowel duration, fewer syllables per breath group, and shorter utterance lengths in people with PD that goes beyond normal aging, from sources such as Huber & Darling (2011). Therefore, utterance length is affected by the pathophysiology. However, during longer utterances, research suggests increased inhalation duration and increased breath groups. People with PD also have been noted to have difficulty coordinating breath groups with utterance length, often breathing at inappropriate times within the utterance. So, there is difficulty producing long utterances and coordinating breathing with speech. More problems with

coordination are evident in people with PD. There tends to be a delay for beginning exhalation after inhalation, as well as delayed initiation of phonation after exhalation begins, according to Duffy (2013). This may be influenced by bradykinesia and hypokinesia because there is difficulty initiating and coordinating movements for speech breathing.

Similar to research cited by Duffy (2013), Bunton (2005) conducted research to examine patterns of lung volumes in people with PD. She compared acoustic, kinematic, and linguistic measures between people with PD and a control group. Lung volumes were estimated through measurement of kinematic movement of the abdomen and rib cage. She found that speakers with PD began speaking at lower lung volumes and that lung volume initiations were more variable compared to the control group. These findings are consistent with the findings from Huber & Darling-White (2017) who examined the changes in speech breathing and speech production in older adults with and without PD. The researchers found that people with PD had significant decreases in lung volume initiation and termination compared to older adults. Thus, lung volumes are impacted by the disease process. The findings from Bunton (2005) about linguistic influences were consistent with the information provided by Duffy (2013). Within her research, there were abnormalities when the participants began or terminated inhalations, often inhaling at inappropriate times within the utterance. The results also were consistent about people with PD producing shorter mean durations of utterances compared to healthy older adults. However, unlike Duffy (2013) who cited a reduction of overall muscular effort, Bunton (2005) noted an increase in muscular effort, specifically abdominal effort, to counteract the rigidity of the rib cage. Huber & Darling-White (2017) also found an increase in muscular effort during exhalation in order to produce speech, while Soloman & Hixon (1993) and Huber et al. (2003) also provided evidence

for an increase in abdominal effort in people with PD. These changes may be problematic for people with PD because speech becomes more effortful.

As previously stated, in HKD, it is common to hear short rushes of speech, short phrases, and inappropriate pauses. These prosodic changes noted in utterance length and loudness provide evidence for changes in the ventilatory system as mentioned above, but also the phonatory system. Abnormal airflow patterns and reduced intraoral pressure have been reported by authors, such as Ramig et al. (2005). Ramig et al. (2005) explain the impact on voice and laryngeal function in people with PD. They describe variation in airflow resistance caused by abnormal movements of the vocal folds and supralaryngeal area. Thus, disease influenced speech cannot only be attributed to changes in LTU.

Prosody, utterance length, and phonation associated with PD are abnormalities that differ from the normal aging processes. Overall, the research shows that there are abnormal muscular excursions during speech breathing, reduced vital capacity, and irregular breathing cycles in people with PD. This impaired ventilatory control can be attributed to the rigidity of muscles of inhalation and exhalation, as well as bradykinesia and hypokinesia. These changes impact prosody and are evident in acoustic intensity, as well as speech duration, which are consistent with the characteristics of HKD. Reduced acoustic intensity is affected by the reduced vital capacity, abnormal muscular excursions, and difficulty alternating breathing for speech and vegetative breathing, according to Duffy (2013). Due to the impact on vocal loudness in HKD, there will be further investigation on how to manipulate acoustic intensity in people with PD.

1.2.3 Parkinson's Disease Manipulation of Intensity

Similar to the section on “Healthy Adult Manipulation of Intensity,” the terms intensity and loudness will be used based on the specific author’s terminology. As previously stated, people with PD and HKD have markedly reduced loudness compared to healthy older adults (Duffy, 2013). According to De Keyser et al. (2016), people with PD may have difficulty naturally producing and maintaining a louder vocal quality, but often can increase their acoustic intensity when cued. People with PD tend to spontaneously speak softly but they have the ability to speak loudly when prompted. Therefore, people with PD are still able to increase acoustic intensity using a variety of techniques.

Consistent with “Healthy Adult Manipulation of Intensity,” the Lombard effect is a phenomenon that holds true for people with PD. Adams, Moon, Dykstra, Abrams, Jenkins, & Jog (2006) examined the effects of background noise on speech intensity in participants with *hypophonia*, or reduced speech intensity, due to PD. The researchers compared the speech intensity of participants with PD and age-matched controls when multi-talker background noise between 50-70 dB SPL was played. They found that participants with PD increased speech intensity in the presence of background noise. However, the speech intensity produced by people with PD when background noise was introduced was significantly lower than the control group. Thus, the Lombard effect is preserved in people with PD, but hypophonia remains consistent compared to healthy older adults. Stathopoulos, Huber, Richardson, Kamphaus, Decicco, Darling, Fulcher, & Sussman (2014) also found that people with PD increased their acoustic intensity and that the participants used various laryngeal and ventilatory techniques to increase intensity. However, Matheron et al. (2017) also considered acoustic intensity in participants with PD and healthy older

adults when presented with background noise. Contrary to the findings of Adams et al. (2006), Matheron et al. (2017) found that participants with PD and healthy older adults spoke at similar intensities when presented with background noise. Regardless, the Lombard effect is present in people with PD.

Darling & Huber (2011) not only considered background noise as a cue to increase loudness, but they also considered targeting 10 dB above comfortable loudness level and twice as loud as the comfortable loudness level in participants with PD and healthy older adults. These research methods were similar to the methods conducted by Huber et al. (2005) with healthy young adults. Darling & Huber (2011) found that participants with PD and healthy older adults increased dB SPL in all loud conditions, although participants with PD produced lower speech intensities compared to healthy older adults. They also provided evidence that both groups had the highest dB SPL in the presence of background noise and the 10 dB condition. This shows that people with PD produce similar acoustic intensity trends when cued to increase loudness compared to both healthy young and older adults; however, people with PD are impacted by the effects of hypophonia associated with the disease process.

Another notable technique for increasing acoustic intensity in people with PD is through the Lee Silverman Voice Treatment (LSVT). This is a common therapy technique for speech language pathologists working with individuals with PD. Spielman, Mahler, Halpern, Gilley, Klepitskaya, & Ramig (2011) describe LSVT as a relatively simple and intensive program that aims to retrain the sensorimotor system to generalize increased intelligibility and loudness to everyday speech. LSVT has been established as an efficacious speech treatment to increase loudness in people with PD (Ramig et al., 2004; Spielman et al., 2011). The body of evidence to support LSVT as a speech treatment is vast, but a few will be mentioned. First, Ramig et al. (2004)

reviewed the literature about LSVT outcome data in people with PD and determined that there are significant long-term improvements in speech, including increased acoustic intensity, in people with PD. Specifically, Huber, Stathopoulos, Ramig, & Lancaster (2003) examined the mechanism for speech breathing and acoustic intensity pre-LSVT and post-LSVT in participants with PD. Although the authors found variability in the kinematics for speech breathing, all of the participants increased acoustic intensity when cued using LSVT and post-LSVT. Thus, the research suggests that people with PD have beneficial speech outcomes when participating in LSVT treatment. However, Spielman et al. (2011) questioned whether DBS would have an impact on LSVT outcomes. They considered the speech in participants with PD who had DBS and participants with PD who did not have DBS before and after LSVT treatment, in order to determine if outcomes were similar regardless of DBS. The researchers found that both groups had significant increases in acoustic intensity from pre-LSVT to post-LSVT, as well as during the six month follow up. This research suggests that LSVT's efficacy is stable among diverse medical groups of people with PD.

In addition to the evidence from Adams et al. (2006) focusing on the Lombard effect, the researchers also examined whether people with PD were capable of imitating three different intensity levels (from 60-80 dB SPL) that were presented auditorily. They discovered that people with PD were able to increase speech intensity as the auditory cues became louder. However, compared to normal controls, people with PD had reliably lower speech intensities. Regardless, people with PD were able to have higher speech intensities when asked to imitate loud speech. This data is relevant because it directly relates to the purpose of the present study, which will be provided in the following sections.

1.3 Grant Purpose

As mentioned above, Duffy (2013) explains that the basal ganglia control circuit, and specifically the STN, play a crucial role in proper motor function. In PD, there are motor disturbances that develop due to the impact of the disease on the basal ganglia control circuit, including the speech disturbances discussed previously. However, there is varying evidence on the role of the STN in speech production. Spielman et al. (2011) describe that there is some positive evidence for DBS in the STN for managing PD speech symptoms, but that there is also evidence that suggests negative impact on speech disturbances. Thus, the grant purpose is to continue to research the role of the STN on speech production. The overall research is an NIH-funded study that investigates “how motor and linguistic speech information is encoded within the STN-cortical network, and to determine the relationship between neural activity within the STN-cortical network and the gain of vocal output” (Project Information, n.d.).

1.4 Specific Aims of the Current Study

The purpose of this project is to determine whether patients with PD are able to manipulate their acoustic intensity, and if such intensity changes are accompanied by changes in speech breathing kinematics. The research shows that people with PD have pathophysiology that influences their kinematics for speech breathing, often producing variable kinematics (Bunton, 2005; Huber et al., 2003; Soloman & Hixon, 1993; Stathopoulos et al., 2014.) Although kinematics are altered, people with PD have the ability to modulate their acoustic intensity when cued (Adams et al., 2006; De Keyser et al., 2016; Huber et al., 2003; Ramig et al., 2004; Spielman et al., 2011;

Stathopoulos et al., 2014). Thus, this research is contributing to the present literature by examining the kinematics of the rib cage and acoustic intensity output in people with PD who are undergoing DBS surgery for motor impairments. The study's data were collected intra-operatively and patients were instructed to modulate loudness (soft vs. loud speech) while repeating three syllable CV strings that were presented auditorily. Specifically, this research aims to answer:

1. Are the acoustic intensities of the first vowel produced by the participant significantly higher for the loud stimulus presentation condition compared to the acoustic intensities of the first vowel for the soft stimulus presentation condition?
2. Are the speech breathing kinematics produced by the participant different in the loud vs. soft stimulus presentation condition?
 - a. Is the duration of inhalation different in the loud vs. soft stimulus presentation condition?
 - b. Is the displacement of inhalation different in the loud vs. soft stimulus presentation condition?
 - c. Is the peak velocity of inhalation different in the loud vs. soft stimulus presentation condition?
 - d. Does the peak velocity of exhalation produced by the participant differ in the loud vs. soft stimulus presentation condition?
 - e. Does the time from the onset of exhalation to the onset of speech differ in the loud vs. soft stimulus presentation condition?
3. How do the inspiratory kinematics of tidal breathing compare to the inspiratory kinematics of speech breathing?

Although previous literature suggests that the acoustic intensity may be reduced compared to healthy adults, there is still expected to be acoustic intensity modulation when cued, based on the evidence in previous studies. It is hypothesized that a higher acoustic intensity will correspond with a different kinematic change when compared to a lower acoustic intensity. Thus, it is hypothesized that the duration of inhalation, magnitude of inhalation, and peak velocity of inhalation will be different for a higher acoustic intensity, as compared to a lower acoustic intensity, during utterance imitation production for participants with PD.

Peak velocity of exhalation will be considered because the literature suggests there may be input from the expiratory musculature when adjusting acoustic intensity. For healthy older adults, variation in kinematics for speech breathing is normal, such as using the muscles of inhalation to take in a larger inhalation and then taking advantage of the elastic recoil forces during speech, as well as expiring greater amounts of air by using the muscles of exhalation, exclusively or in addition to elastic recoil forces (Hixon, Goldman, & Mead, 1973; Winkworth Davis, Adams, & Ellis, 1995; Huber, Chandrasekaran, & Wolstencroft, 2005). Expiratory effort is also evident in people with PD. Huber & Darling-White (2017) found an increase in muscular effort during exhalation in order to produce speech, while Solomon & Hixon (1993), Huber et al. (2003), and Bunton (2005) also provided evidence for an increase in abdominal effort in people with PD when expiring.

Time of onset of the exhalation to the onset of speech will be considered because there is evidence that people with PD tend to delay initiation of phonation after exhalation begins, according to Duffy (2013). This is considered to be influenced by difficulty initiating and coordinating movements in PD (Duffy, 2013).

There are a few tidal breaths at the beginning of the participant's data collection that can be considered for analysis. According to Zemlin (1998), the duration of an inhalation and exhalation for tidal breathing is relatively equal: about 50% of a vegetative breathing cycle is inhalation, and about 50% is exhalation. Although the total duration of vegetative breathing is about 50% inhalation and 50% exhalation, speech breathing produces a rapid inhalation, contributing to about 10% of the total speech breathing cycle, while exhalation is a sustained and controlled duration, contributing about 90% of the total speech breathing cycle (Hixon et al., 2020; Zemlin, 1998). Thus, a comparison of tidal breathing and speech breathing will be considered for the participant because there is evidence for a duration of inhalation for speech breathing in people with PD, due to the bradykinesia and rigidity of the chest wall (Duffy, 2013).

While the research questions for this study likely will not provide novel findings for the relationship between ventilatory kinematics and acoustic intensity for people with PD, the results are anticipated to replicate previous findings in a novel environment of intra-operative data collection, which can be used for subsequent neural mapping. The overall purpose of this research is to investigate kinematics for speech breathing and acoustic intensity of people with PD prior to DBS implantation, in order to have data for later mapping of the neural signals in the subthalamic nucleus (STN). After our preliminary research on ventilatory kinematics and acoustic intensity, the data will be matched to STN activity at the corresponding time point that was attained intra-operatively (Project Information, n.d.). Not only will the findings be used to determine the relationship between neural activity within the STN-cortical network and the kinematics used for speech production in patients with PD, but the methodology of the research will also provide a protocol for future data collection of this type (Project Information, n.d.). This protocol will be accessible to future researchers who plan to map neural activity to kinematic activity. Thus, the

grand research aim is to investigate the contribution of the STN on speech production and the results of this project will be used to further achieve that research aim, while also providing protocol for future research.

2.0 Methods

2.1 Participants

The participants were patients with PD who had agreed to undergo STN deep-brain stimulation to address tremors that were no longer being managed with medication. All research procedures were approved by the University of Pittsburgh Institutional Review Board (IRB Protocol # PRO13110420), and all participants provided informed consent to participate in this study. Participants were off of dopaminergic medication for at least twelve hours prior to the initiation of surgery. While data were collected for sixteen participants, after beginning data analysis, it became evident that there was inadequate ventilatory data for most of the participants due to improper placement of the equipment during the DBS surgery. It was determined that there was only a single participant for whom there was a clean ventilatory signal. This participant was a 69-year-old female who was diagnosed with Parkinson's Disease nine years prior to surgery. Her UPDRS score was 45. According to Goetz et al. (2008), the UPDRS is scored from 0, indicating no disability, to 199, indicating a total disability. No official diagnoses of dyspnea, dysarthria, or voice disorders had been assigned to any participants, and all participants underwent pure-tone hearing screenings at 500Hz, 1kHz, 2kHz, and 4kHz at 25 and 40 dB HL.

2.2 Speech Tasks

The participant performed a three-syllable triplet speech repetition task during the surgical intervention. These triplets were derived from a sample of 16 unique CV syllables. The triplets were constructed based on four consonants: /g, t, s, v/ and three vowels: /i, a, u/, in order to make the three syllable triplets, such as /si tu ga/. The CV stimuli were chosen to ensure that they include various combinations of articulator features (AF) and phonetic state spaces that can be seen in Figure 1 (AF in the top half, phonetic state space in the bottom half). The triplets were created based on the features and frequency of occurrence in order to probe multiple levels of encoding within one speech production task and dataset. The syllable triplets were chosen pseudo-randomly, so that the initial phoneme of the triplets was balanced, allowing each consonant to be presented in the initial position 30 times within a session. (Note: the vowel /ε/ was not included in the final stimulus set.)

Feature(s)	vee	vah	voo	veh	jee	jah	joo	jeh	tee	tah	too	teh	see	sah	soo	seh
lips AF	1	1	1	1	0	0	1	0	0	1	0	0	0	0	1	0
tongue tip AF	0	0	0	0	1	1	1	1	1	1	1	1	1	1	1	1
larynx AF	1	1	1	1	1	1	1	1	0	0	0	0	0	0	0	0
low tongue/jaw AF	0	1	0	1	0	1	0	1	0	1	0	1	0	1	0	1
front tongue AF	1	0	0	1	1	0	0	1	1	1	1	1	1	0	0	1
dorsal tongue C	0	0	0	0	1	1	1	1	0	0	0	0	0	0	0	0
labial C	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0
coronal tongue tip C	0	0	0	0	0	0	0	0	1	1	1	1	0	0	0	0
coronal sibilant C	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1
high front V	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0
high back V	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0
low front V	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1
low back V	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0

Figure 1 CV Stimuli

In order to form the triplet stimuli, an adult male speaker produced each of the CV syllables using “normal voice.” Using Praat (PSOLA_script), the duration of each recorded CV syllable was equated to 500 ms, and the CV syllables then were combined into triplets, as described above

(Boersma & Weenink, 2020). Praat (ScaleIntensity script) was used to scale the relative intensity of the triplets in order to create a sufficient contrast between soft and loud conditions (that is, uncalibrated levels within Praat of 50 and 75 dB).

The triplet acoustic stimuli were presented to the participants, who were instructed to match their production with the perceived loudness of the auditory signal. The intensity of the acoustic signal was balanced within each three-syllable triplet. Five triplets were presented at a soft intensity and five were at a loud intensity. Similar to the order of each CV syllable within each triplet, the order of soft and loud conditions within each list was pseudo-randomized, but the order remained fixed during presentation to participants.

Participants completed a pre-surgical training session involving the syllable triplet production task. Prior to the training, participants completed the informed consent and completed pure-tone hearing screenings at 500Hz, 1kHz, 2kHz, and 4kHz at intensity levels of 25 and 40 dB HL. The presentation levels of the acoustic stimuli were adjusted preoperatively for each patient, with the soft signal being increased to a level where it was comfortably audible and perceived as soft (Cox & Gray, 2001). Then, participants were given standardized instructions to repeat the three-syllable triplets that were presented auditorily. They were told that some sounds that they heard would be soft, while others would be loud, and to use a vocal loudness that matched the acoustic stimuli. Next, participants completed practice trials with the acoustic stimuli. Feedback was given for loudness production during the practice trials, and patients were occasionally reminded during data collection to ensure target loudness was achieved. If they could not produce the target loud amplitude after feedback was given, participants were excluded from the study. In the operating room, following the initial placement of an electrode arrays within the STN and on the sensorimotor cortex, anesthesia was adjusted, and the supine participants were awoken and

instructed to repeat the three-syllable triplets at the perceived loudness level, similar to the pre-operative training. The order of the triplets remained consistent when presented to each participant. During the experimental procedure, the electrode array was moved to different recording depths within the STN and the speech task was repeated at each depth. This corresponded to 2-4 sessions of speech tasks, depending on surgical concerns and participant fatigue.

2.3 Instrumentation

Acoustic triplet stimuli were presented to the participants through an audio amplifier (PreSonus, AudioBox iTwo) via ER38-14F foam tip ear inserts (Etymotic Research, Inc.) The microphone (AT875R, Broadcast & Production Microphones) was positioned at a mouth-to-microphone distance of 15 cm, 45 degrees below the horizontal level of the left oral angle. Acoustic data were digitized at 44.1kHz with a 7.5 kHz low-pass anti-aliasing filter (H6 Handy Recorder, Zoom Inc.) Due to the restrictions of the intra-operative environment, the acoustic intensity signal obtained from the microphone was uncalibrated. However, since data were analyzed within-subject, amplitude settings and background noise should be similar within a participant. Ventilatory kinematic data were obtained using a Piezo Crystal Effort Sensor with a double buckle band. This sensor converts LTU motion to a small analog voltage that translates to a ventilation waveform (Scientific Laboratory Products, SleepSense Respiratory Effort Sensors). The sensors are designed to pull on the band attachments and convert these subsequent signals to a waveform. However, the bands were utilized correctly for only one participant. For all other participants, the sensors were taped to the skin overlying the xiphoid process of the sternum without the use of the bands. Thus, the only adequate data came from the first participant that the bands were utilized.

The data from this participant were digitized at 1 kHz with a 250 Hz low-pass antialiasing filter. Like the uncalibrated acoustic recordings, the sensors also were uncalibrated due to the intra-operative environment. This prevents the kinematic data from having exact units of measurement, so data are reported as “uncalibrated units.”

2.4 Data Analysis

2.4.1 Acoustic Intensity

Analysis of acoustic signals first was completed utilizing MATLAB 2017a (Mathworks Inc., Natick, MA, USA), R version 3.4. 4 (R Development Core Team, 2018). Acoustic signals were displayed in MatLab as spectrograms with a customized graphical user interface (GUI) to allow for detailed marking of consonants and vowels, voice and transcription of speech. Vowel onset was identified as the onset of voicing (vertical glottal pulses in the spectrogram); if the preceding consonant was voiced, vowel onset was identified as the initiation of formant structure. The above analysis was completed prior to the author’s involvement in this study; reliability data are not available at this time.

The average intensity of the first vowel of the triplet was measured for the acoustic intensity data. The first vowel was selected for analysis because this likely represents the highest intensity that the participant achieved (based on previous literature considering hypophonia and maintaining loudness). The intensity of the first consonant was not analyzed in order to eliminate differences in intensity across the produced consonants. The average intensity (in uncalibrated units from Praat) was calculated through a custom acoustic intensity script in Praat (Boersma & Weenink,

2020). The script used time points that were collected by research assistants (described above) from the onset, offset, and duration of the first vowel in each utterance. The vowel duration, from onset to offset, was used to calculate acoustic intensity using the Praat “energy” method ($10 \log_{10} \left\{ \frac{1}{(t_2 - t_1)} \int_{t_1}^{t_2} 10^{x(t)/10} dt \right\}$). The stimuli condition (loud vs. soft) was noted for each utterance intensity value for comparison.

2.4.2 Speech Breathing Kinematics

The displacement signal generated by the transducer was filtered through Matlab using a 4 Hz lowpass filter to mitigate noise. After the displacement signal was filtered for noise, the signal was resampled from 96,000 Hz to 50 Hz in order to successfully produce the first derivative. The first derivative, velocity, was obtained using the derivative function in Matlab once the displacement signal was downsampled.

Like the acoustic intensity signal, the auditory stimulus target was noted for each speech breathing kinematic value. Using Praat, the onset of inhalation for speech breathing first was visually identified as occurring shortly before the acoustic onset of the triplet production. Subsequently, the onset of inhalation was marked automatically by the time of the zero crossing on the velocity trace in the negative direction (negative direction indicating inhalation). The Praat function “move cursor to nearest zero crossing” can be seen in Figure 2 (the vertical red line identifying the zero crossing), as an example of how onset of inhalation was identified. The offset of the inhalation was marked by the zero crossing in the velocity trace in the positive direction (movement in the direction of exhalation). This method of using the zero crossing on the velocity curve also was used to mark onset and offset of exhalation. The offset of inhalation and onset of exhalation tended to be congruent.

The time of peak velocity for both inhalation and exhalation were identified manually within Praat. The cursor was manually moved in small steps along the time axis until the peak negative (inhalation) and positive (exhalation) values were identified on the y-axis. Interrater reliability for identification of the time, and thus, magnitude, of peak velocity for inhalation and exhalation was evaluated, as these measures were manually identified. Fifteen percent of the total utterances (29 utterances) were re-measured for interrater reliability. Using a Pearson R, the interrater reliability was determined to be $R=1.0$.

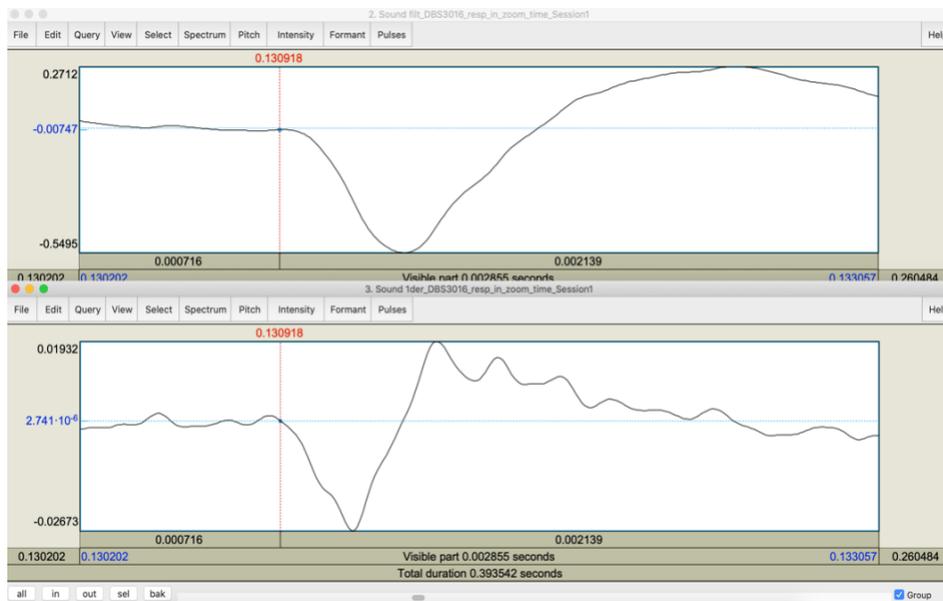


Figure 2 Ventilatory Displacement (Top) and Velocity (Bottom)

Duration of inhalation was calculated as the time (in seconds) from onset of inhalation to offset of inhalation. Magnitude of inhalation displacement (in uncalibrated units) was calculated as the difference in movement amplitudes at the onset and offset of inhalation. Finally, peak velocity of both inhalation and exhalation were measured by the largest rate of change (negative for inhalation, positive for exhalation; uncalibrated units/second) within the total duration of the ventilatory cycle.

The time of onset of exhalation and onset of speech may differ for patients with Parkinson's Disease due to difficulty initiating movements previously discussed. This was evaluated by analyzing the time difference between onset of exhalation and onset of the first vowel in the utterance.

Finally, ventilatory kinematics for tidal and speech breathing were compared. This considers the duration, magnitude, and peak velocity of inhalation described above for tidal and speech breathing. The mean and standard deviations of the inspiratory kinematics were compared using formulas in Excel.

2.5 Statistical Methods

Initially, t-tests were planned to evaluate the significance of results across multiple subjects. However, given that there was only a single participant, data were analyzed descriptively, and through correlations, and time series analyses.

The first aim, observing vowel intensity production as a function of stimulus condition, was evaluated through means and standard deviations. These were calculated through functions in Excel. Next, the speech breathing kinematic variables were evaluated with correlational analyses. The Spearman Rho coefficient was used because the data were for a single subject and were nonparametric. An online statistical calculator (N. Vasavada, 2016), using R code, was used to calculate Spearman Rho and the related p-values. Lastly, tidal breathing and speech breathing were compared descriptively using mean and standard deviation of inspiratory kinematics. Once again, these were calculated with functions in Excel and compared for appreciable differences. To

calculate effect sizes between tidal breathing and speech breathing, there was consideration to use Glass' delta, which uses the standard deviation as the control group (i.e. tidal breathing). However, a Hedge's g was determined to be a more appropriate measure because it calculates effects size based on the relative size of each sample. In this study, the sample sizes were not equivalent, but rather vastly different ($n=115$ for speech and $n=8$ for tidal); thus, Hedge's g provided an evaluation of effect size for the two samples through an online calculator.

3.0 Results

This research aimed to evaluate whether a patient with PD was able to manipulate their acoustic intensity, and if such intensity changes were accompanied by changes in speech breathing kinematics in a novel intraoperative environment. Intensity changes were expected to reflect stimulus conditions presented. For example, a loud stimulus condition was expected to result in the production of a greater acoustic intensity produced. Therefore, stimulus condition and intensity, as well as stimulus condition and speech breathing kinematics, were described descriptively through central tendency analyses. Means and standard deviations for each of the dependent variables, by stimulus presentation condition and across stimulus conditions, are presented in Table 1, below.

Table 1 Stimulus Condition and the Dependent Variables

		Loud	Soft	Across Conditions
Intensity (uncalibrated dB)	Mean	68.921	69.046	68.985
	SD	10.896	8.142	9.540
Inhalation Duration (seconds)	Mean	1.218	1.112	1.164
	SD	0.490	0.356	0.428
Inhalation Displacement (uncalibrated units)	Mean	54.416	52.514	53.440
	SD	15.408	10.253	12.998
Inhalation Peak Velocity (uncalibrated units/second)	Mean	-0.021	-0.025	-0.023
	SD	0.006	0.025	0.018
Exhalation Peak Velocity (uncalibrated units/second)	Mean	0.017	0.017	0.017
	SD	0.004	0.004	0.004
Duration from onset of exhalation to onset of speech (seconds)	Mean	0.440	0.436	0.438
	SD	0.283	0.316	0.299

Descriptively, intensity did not substantively differ across the two loudness stimulus conditions. Similarly, mean speech breathing kinematic variables for loud and soft stimulus presentation conditions did not differ by an appreciable value. Rather, the means tended to be nearly equivalent for each stimulus condition. While the experimental manipulation of loudness condition did not result in the anticipated intensity difference, it was deemed likely that the participant was producing some degree of variable vocal intensities across the two data collection sessions. Thus, data were collapsed across the stimulus conditions and the relationships between intensity and the speech breathing kinematic variables were analyzed, irrespective of stimulus

condition. Table 2, below, presents the Spearman Rho correlation coefficients and related p-values between intensity and inhalation and exhalation kinematics.

Table 2 Correlations of Intensity and Speech Breathing Kinematics (n=115)

	Intensity - Spearman Rho	p-value
Inhalation Duration	0.004	0.965
Inhalation Displacement	0.090	0.342
Inhalation Peak Velocity Magnitude	0.116	0.221
Exhalation Peak Velocity Magnitude	0.160	0.089
Duration from onset of exhalation to onset of speech	-0.128	0.175

The results above indicate that there were very weak to no relationships between intensity and inhalation duration and inhalation displacement. There were very weak positive (although nonsignificant) correlations between intensity and the peak velocities of both inhalation and exhalation, as well as a very weak negative correlation between intensity and the duration from onset of exhalation to onset of speech. This shows that as peak velocities of both inhalation and exhalation increased, the acoustic intensities increased, but only slightly. Similarly, as the duration from onset of exhalation to onset of speech decreased, the intensity increased slightly. However, none of the correlations were significant. In Figure 3 below, the strongest of the 5 correlations, between intensity and exhalation of peak velocity, can be observed.

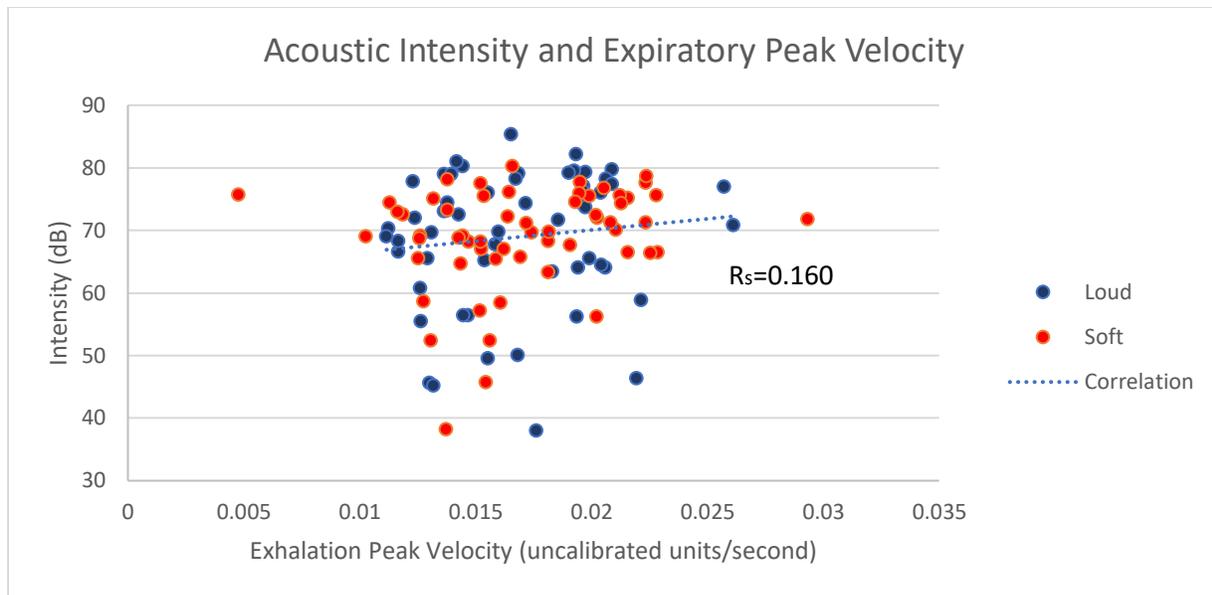


Figure 3 Correlation of Intensity and Expiratory Peak Velocity

During the two sessions of data collection, multiple cycles of the patient’s tidal breathing were collected both before and after the speech task. Therefore, this study also aimed to compare the inspiratory kinematics of tidal and speech breathing. As seen in Table 3 below, on average, the duration of tidal breathing was longer than speech breathing. The average duration of inhalation of tidal breathing was determined to be 1.424 seconds. Thus, the average cycle duration is approximated to be about 2.8 seconds (based on the assumption that tidal breathing is expected to be approximately 50% inhalation and 50% exhalation (Hixon et al., 2020; Zemlin, 1998)). With a 2.8 second cycle, this participant’s respiratory rate is estimated to be 21.4 breaths per minute, which is within the normal range for respiratory rate (Zemlin, 1998).

The average inhalation displacement and peak velocity of speech breathing were greater than the average inhalation displacement and peak velocity of tidal breathing. These results were consistent with the hypotheses that inhalation duration of tidal breathing would be longer than inhalation duration of speech breathing, and that displacement and peak velocity of inhalation for speech breathing would be greater than inhalation for tidal breathing. Hedge’s *g* provided a

measure of effect size between speech and tidal breathing. There was a medium effect size for duration of inhalation (0.619), a small effect size for displacement of inhalation (0.101), and a small to medium effect size for peak velocity of inhalation (0.400).

Table 3 Speech Breathing and Tidal Breathing Inhalations

		Duration (seconds)	Displacement (uncalibrated units)	Peak Velocity (uncalibrated units/second)
Speech (n=115)	Mean	1.164	53.440	-0.023
	SD	0.428	12.998	0.018
Tidal (n=8)	Mean	1.424	52.156	-0.016
	SD	0.250	5.353	0.002
Effect size	Hedge's g	0.619	0.101	0.400

A time series analysis of the patient's tidal and speech breathing was then conducted. Figure 4 below suggests that inhalation duration was somewhat longer and less variable during tidal breathing compared to speech breathing. Interestingly, and maybe not surprisingly, the inhalation duration for tidal breathing decreased across the duration of data collection during the surgical procedure. Figure 5 shows that inhalation displacement decreased during both of the speech production tasks, while the inhalation displacement remained relatively stable for tidal breathing. Figure 6 shows that peak velocity of inhalation for tidal breathing was slower (negative values closer to 0 indicate decreased velocity), less variable and highly stable across the duration of data collection than was speech.

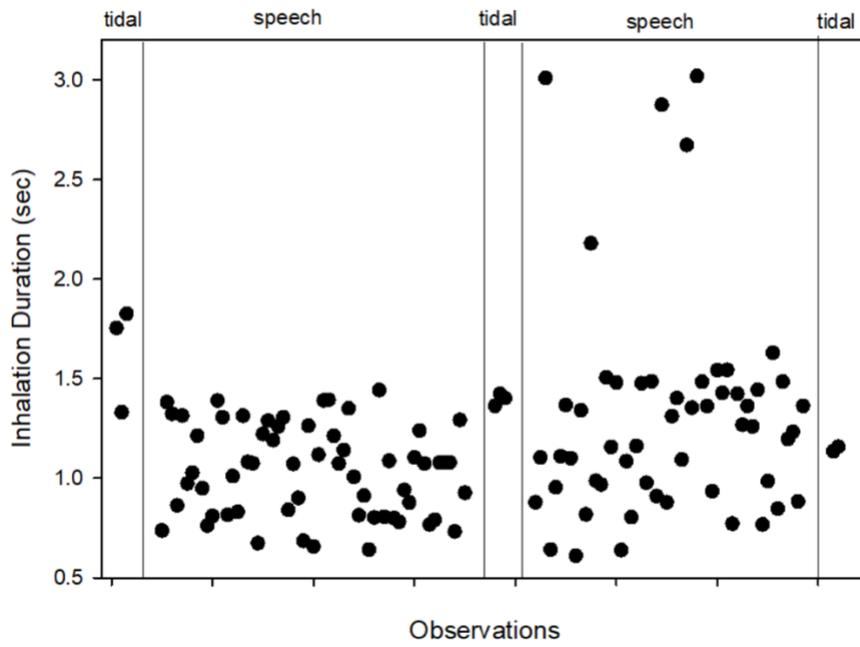


Figure 4 Tidal vs. Speech Breathing Inhalation Duration

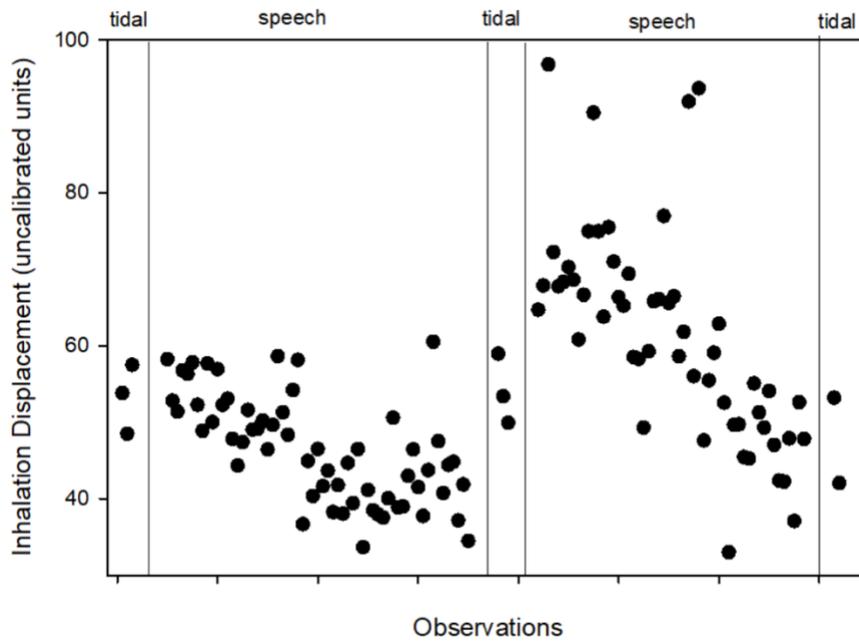


Figure 5 Tidal vs. Speech Breathing Inhalation Displacement

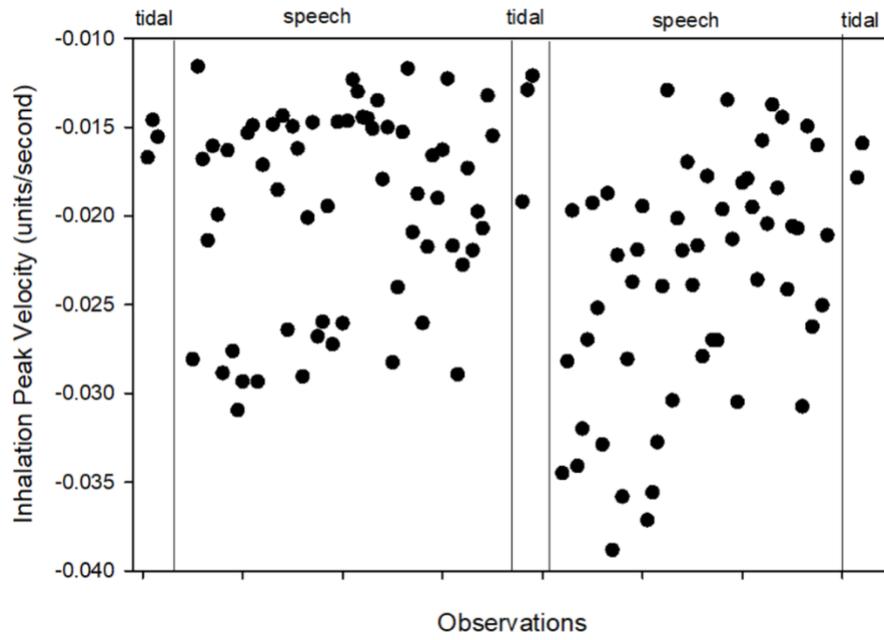


Figure 6 Tidal vs. Speech Breathing Inhalation Peak Velocity

4.0 Discussion

The experimental data collection for this research project was focused on neural recording in the STN and sensorimotor cortex, as well as the acoustic speech signal. Therefore, limited attention was paid to the signal from the ventilator bands. As a result of improper connection of the ventilator band subsequent to the first patient, the current study is limited to the results of one participant. This prevents the ability to draw general conclusions from the data analyses. The subsequent participants demonstrated intensity changes for the loudness conditions that align with previous research. Unfortunately, their kinematic data were inadequate for analysis. Nonetheless, the discussion that follows describes the results for the single participant.

The previous literature suggests that people with PD can manipulate intensity when cued as a result of kinematic modulations in speech breathing kinematics (Adams et al., 2006; Bunton, 2005; Huber et al., 2003; Ramig et al., 2004; Soloman & Hixon, 1993; Stathopoulos et al., 2014.) The main aim of this study was to determine whether a participant with PD can manipulate acoustic intensity using variable speech breathing kinematics in a novel intraoperative environment.

First, the participant was cued to produce loud or soft speech with the expectation that a loud cue would result in a higher acoustic intensity. However, the participant produced similar acoustic intensities for both cues. This conclusion is not consistent with the previous literature that a person with PD can manipulate intensity when cued. One reason for this outcome could be that the participant may have difficulty perceiving loudness variations. Several studies have suggested that people with PD have abnormal perception of vocal loudness. Clark et al. (2014) found that people with PD had a speech loudness perception deficit compared to controls, which was

evidenced by a different pattern and range of perceptual loudness, imitation of acoustic intensity, and self-generated estimates of loudness. Furthermore, Ho et al. (2000) stated that people with PD perceive their speech to be louder as compared to self-perception of loudness for controls. Thus, there is evidence that people with PD not only have difficulty generating loud speech, but they also perceive their speech to be louder than the true intensity. The participant in this study may have had difficulty perceiving her loudness output, especially in a noisy intraoperative environment.

Another reason for the unexpected intensity modulations could be the disease progression of PD for this particular participant. Compared to the other participants for which speech kinematics were planned to be assessed, this single participant had a relatively higher UPDRS score. She also had a longer length of diagnosis compared to the other participants (nine years vs. 5.5-year average). The longer duration from diagnosis and greater perception of disability associated with PD may have impacted performance.

Next, speech breathing kinematics and intensity were assessed for potential relationships in the second research question. As discussed in the beginning sections of this document, healthy normal adults perform ventilation for life-sustaining purposes primarily through kinematics of the diaphragm and driving forces of changing pressure gradients (Hixon et al., 2020; Zemlin, 1998). Vegetative breathing physiology is generally maintained with age, but aging effects can be observed with decreased nerve conduction velocity, reduced alveolar surface area, and diminished oxygen extraction (Levitzky, 2018). This affects proper ventilation and chemical processes to sustain life.

In addition to vegetative breathing, breathing for speech production was discussed for healthy normal adults based on literature from Hixon et. al (2020) and Zemlin (1998). For speech

breathing, there is recruitment of the muscles of inhalation, elastic recoil forces, and relaxation pressure to produce the communicative goal. This is maintained as we age, with some altered physiology including atrophy, ossification, and decreased compliance, elasticity, and strength of muscles. Although speech breathing kinematics are similar, there is evidence of shorter utterances per breath group, differences in muscular excursions and effort, as well as increase in functional residual capacity as we age. For people with PD, the physiology and kinematics of breathing for older adults is changed further (Duffy, 2013). Compliance of the chest wall decreases more than typical aging due to rigidity of the rib cage. There is an even more significant reduction in vital capacity and more significant abnormality of muscular excursions that goes beyond typical aging. One main change in muscular kinematics for breathing is reliance on the abdominal musculature (Bunton, 2005; Huber et al., 2003; Soloman & Hixon, 1993). This results in irregular breathing cycles and impaired ventilatory control due to bradykinesia. Furthermore, there is difficulty alternating tidal breathing and speech breathing, and inappropriate initiation of inhalation during speech. Speech is affected by these neurologic and physiologic changes. HKD is characterized by speech disturbances such as reduced loudness and short utterances.

Although reduced loudness is a hallmark characteristic of HKD and 90% of people with PD are diagnosed with HKD, acoustic intensity was expected to modulate when cued (Duffy, 2013). People with PD had variable kinematic changes when producing larger acoustic intensities, including altering inhalation kinematics, exhalation kinematics, or both (Bunton, 2005; Hixon, Goldman, & Mead, 1973; Winkworth Davis, Adams, & Ellis, 1995; Huber, Chandrasekaran, & Wolstencroft, 2005; Huber & Darling-White, 2017). In this research, it was expected that larger speech breathing kinematic changes, inhalation and/or exhalation, would correlate with larger intensities. The results from this study suggest that, while there were very weak relationships

between acoustic intensity and some measures of speech breathing kinematics, none reached a level of significance. Two potential reasons for this result are the position of the participant and the placement of the sensors.

The supine position is a limitation to functional speech breathing kinematics and the acoustic intensities. As stated above, ventilation and speech breathing are impacted by the disease processes of PD, but the supine position amplifies this impact. Hixon et al. (2020) state that gravity facilitates expiration for both the rib cage and abdominal wall, which causes the relaxation pressure to be greater in the supine position. The authors also explain that gravity impacts the resting state of the LTU and results in a reduced vital capacity in the supine position by about half. This becomes a daunting challenge for a person with PD who already has reduced vital capacity and abnormal muscular excursions during speech breathing in the upright position due to muscle rigidity. For a healthy adult to achieve the target acoustic intensity in the supine position, there must be greater inspiratory effort of the muscles so that the individual can generate an adequate alveolar pressure (Hixon et al., 2020). Rigidity, bradykinesia, and hypokinesia would make these greater muscular efforts even more difficult for this study's participant with PD. (Refer to Appendix A Agonist-Antagonist Muscles for Ventilation for a more detailed explanation of the neurology associated with rigidity).

Then, in order to sustain the targeted alveolar pressure in the supine position, the authors state that breathing action is accomplished almost entirely by the diaphragm. This leads to the next limitation of sensor placement. The sensors were placed on the ribcage near the xiphoid process, so kinematic motion of the abdomen and diaphragm were not recorded. Although the supine position may have provided mechanical advantages from the force of gravity on the chest wall, the greater muscular effort from the diaphragm for inhalation (potentially resulting in greater

displacement of the abdominal cavity) and greater muscular effort of checking action for production of speech were not considered. The supine position may increase muscular effort by the diaphragm, but people with PD also rely more on muscular effort from the abdomen when producing speech (Bunton, 2005; Huber et al., 2003; Soloman & Hixon, 1993). Thus, the speech breathing kinematics reported may not be the true, holistic kinematic values used for speech breathing by the participant. To mitigate this limitation, a sensor on the abdomen, as well as the rib cage, could be used.

When comparing speech breathing kinematics and intensity, the correlational relationships were found to be nonsignificant. Nonetheless, the correlation between intensity and expiratory peak velocity was the strongest of the correlations reported at $R_s=0.160$. The supine position may have been a limitation of the study, but it also could have aided in producing the relationship between expiratory peak velocity and intensity. In order to produce a greater intensity, there must be adequate subglottal pressure created from the muscular effort of the rib cage and abdomen during inhalation and exhalation (Hixon et al., 2020). The results suggest that the expiratory peak velocity is correlated with creating a greater subglottal pressure, in order to produce a greater acoustic intensity. In fact, the supine position may benefit the patient's exhalation for speech breathing due to the force of gravity, in addition to the abdominal effort to "push out" air while speaking that is common in people with PD (Duffy, 2013; Hixon et al., 2020). Given this evidence for greater muscular effort during exhalation, a transducer placed on the abdomen may have provided a stronger correlation between expiratory peak velocity and acoustic intensity.

The final aim of the present study was to compare tidal breathing and speech breathing inhalations. It was expected that duration of inhalation would be greatest during tidal breathing, and that displacement and peak velocity of inhalation would be greatest during speech breathing.

These results were expected because tidal breathing is found to be relatively 50% inhalation and 50% exhalation, while speech breathing is about 10% inhalation and 90% exhalation (Hixon et al., 2020; Zemlin, 1998). The authors also explain that speech breathing produces a rapid inhalation with a greater displacement compared to vegetative breathing, in order to produce a controlled subglottal pressure during exhalation. The results of the current study were consistent with this evidence. Duration of inhalation during tidal breathing was greater than speech breathing (effect size of 0.619), while displacement and peak velocity of inhalation of speech breathing was greater than tidal breathing (effect size of 0.101 and 0.400, respectively).

Inhalation duration of tidal breathing was found to be relatively longer, and less variable compared to speech breathing, yet the inhalation duration for tidal breathing also was found to decrease across time. Likewise, inhalation displacement decreased during speech breathing over time. These were interesting findings to observe, yet not necessarily surprising because the findings could be explained by patient fatigue during the research tasks and within the intraoperative environment, as well as the effects of the supine position stated above. Furthermore, inhalation displacement of tidal breathing remained stable throughout the sessions, while duration decreased over time. This would suggest that the breathing rate for tidal breathing would increase over time. However, peak velocity of inhalation for tidal breathing was relatively stable across time, as seen in the time series analysis. This could be explained by a greater expiratory velocity, although these kinematic values were not considered. A greater expiratory velocity for tidal breathing would be expected given the evidence of increased relaxation pressure and gravitational effects on the LTU in the supine position (Hixon et al., 2020).

The participant's average inhalation duration for tidal breathing was 1.424 seconds, estimating a respiratory rate of 21.4 breaths per minute. The average respiratory rate for healthy

adults is between 12-24 breaths per minute, so the participant's respiratory rate falls within the normal range (Zemlin, 1998). However, Solomon & Hixon (1993) found that the respiratory rate and minute volume for tidal breathing was greater for people with PD as compared to controls. However, the results of this study are not consistent with Solomon & Hixon's findings. Once again, the relatively normal minute volume of tidal breathing could be explained by the supine position and the positive effects of gravity on the LTU mentioned previously.

In addition to the primary limitation of a single subject analysis and the various limitations stated above, there are several other limitations that exist as confounds for this research. First, the task instructions and speech kinematic protocol may have influenced performance. This was the first participant for whom speech breathing kinematics were considered, so the protocol was novel. There may have been inadequate consideration of the participant's understanding of the directions and motivation to perform the speech tasks sufficiently. The protocol may have benefitted from specific regard of the participant's perception of her loudness production and understanding of the speech task. Plus, participants were awoken during electrode placement and asked to perform speech tasks like they did in the training prior to surgery. However, the environment was not consistent and might have led to different behaviors or performance for each environment.

Although the environment within the operation room was novel, this also is a clear limitation to data collection. The primary goal was a successful STN implantation surgery, so the research was not the prime objective. The active operating room created a noisy environment from machinery and medical professionals, rather than a controlled, quiet environment. This background noise may have interfered with the data analysis of the speech signal recordings. The environment also may have caused the participant to become anxious or fatigued more easily.

Within the task protocol, participants were asked to express fatigue when necessary, which often led to early cessation of the research tasks.

In addition to the task protocol and the environment of the operation room, the anesthesia and lack of PD medication could have influenced participant performance. The impact of pharmaceuticals may have altered typical speech breathing performance. Particularly, the anesthesia used for the operation have may sedated the participant and altered typical functioning. In addition, participants were required to be off of their medication for a minimum of twelve hours prior to the surgery. The lack of PD medication, such as Levodopa, may increase the rigidity of the lung thoracic unit. This rigidity would influence speech breathing and laryngeal kinematics needed for phonation. (See Appendix A Agonist-Antagonist Muscles for Ventilation). Regardless, both pharmaceutical modifications could impact typical speech breathing kinematics and acoustic intensity for the particular participant.

Once the aforementioned limitations are mitigated, there is room for additional improvement in the research design. Although a single participant was analyzed in this study, sixteen participants were planned to be analyzed. These sixteen participants may have provided more significant results, so future research may discover more significant results if this protocol can be repeated with more participants. Plus, since DBS is performed on people with movement disorders, the participants are solely people with PD, so a control group could provide grounds for experimental conclusions to be drawn. In addition to a greater sample size, the intensity and ventilatory kinematic signals were unable to be calibrated due to the time, technology, and space constraints of the intraoperative environment. With instrumentation and signal calibration, more valid data could be collected.

Nonetheless, this research provides the opportunity for neural mapping of the STN and cortex that can be used for pilot data. The data for this project were collected as a part of the larger research study, for which the purpose of the grand study was to examine how speech information is encoded in the STN-cortical network and how that relates to speech output gain (Project Information, n.d.). The speech breathing kinematic time points can be matched to neural activity and provide further insight into planning of motor movements for speech. Not only can these findings be used to determine a relationship between neural activity within the STN-cortical network and speech breathing kinematics in the participant with PD, but the methodology provides a framework for future researchers who are interested in neural mapping of kinematic activity.

Appendix A Agonist-Antagonist Muscles for Ventilation

The disease progression of PD causes changes in neuromotor function (Duffy, 2013). One change in motor function is evident through the performance of the agonist-antagonist muscles used for speech breathing. *Agonist* muscles are defined as muscles that cause movement to occur through contraction, while *antagonist* muscles are defined as muscles that opposing motion through relaxation (Meunier et al., 2000; Gorkovenko et al., 2012). Together, these muscles work together to achieve a target function through co-activation of contraction and relaxation.

According to Duffy (2013), the basal ganglia control circuit is crucial in selecting and refining intended movements, while inhibiting competing motor patterns. The author explains that abnormalities in the basal ganglia control circuit play a role in altering agonist-antagonist muscle relationships which attribute to rigidity in PD. The speech breathing abnormalities associated with PD mentioned prior, such as reduced vital capacity and reduced chest wall movements, can be attributed to reduced agonist contraction and increased antagonist contraction. These abnormal agonist-antagonist muscle movements may be the origin for the rigidity of the rib cage, as well as the bradykinesia and hypokinesia of the intercostal muscles. Simultaneous contraction of the internal and external intercostals would result in dysfunctional initiation and discoordination of inhalation and exhalation for speech breathing. In fact, people with PD may be prescribed antiparkinsonian medications that target agonist-antagonist muscles in order to improve speech breathing kinematics and overall motor movement.

This feeling of stiffness and slowness of movement associated with rigidity is apparent through the full range of motion (Duffy, 2013). However, abnormalities in the agonist-antagonist

muscle relationship can be connected with spasticity, as well. Spasticity, often caused by upper motor neuron impairments, is velocity dependent and has the greatest impact on speech breathing at the beginning of muscle contraction. Compared to spasticity, rigidity results in resistance to movement, slowness of movement, and difficulty initiation movement in all directions due to the abnormal agonist-antagonist relationships.

Appendix B Funding

Funding was provided by NINDS U01NS098969 (PI: Richardson), the Hamot Health Foundation (PI: Richardson) and a University of Pittsburgh Brain Institute NeuroDiscovery Pilot Research Award (PI: Richardson).

Bibliography

- Adams, S., Moon, B.-H., Dykstra, A., Abrams, K., Jenkins, M., & Jog, M. (2006). Effects of multitalker noise on conversational speech intensity in Parkinson's disease. *Journal of Medical Speech Language Pathology*, 14(4), 221–228.
- Baker, K.K., Ramig, L.O., Sapis, S., Luschei, E.S., Smith, M.E. (2001). Control of vocal loudness in young and old adults. *Journal of Speech Language and Hearing Research*, 44(4), 297-305.
- Boersma, Paul & Weenink, David (2020). Praat: doing phonetics by computer [Computer program]. Version 6.1.09, retrieved 26 January 2020 from <http://www.praat.org/>
- Bunton, K. (2005). Patterns of lung volume use during an extemporaneous speech task in persons with Parkinson's disease. *Journal of Communication Disorders*, 38(5), 331–348. doi: 10.1016/j.jcomdis.2005.01.001
- Clark, J. P., Adams, S. G., Dykstra, A. D., Moodie, S., & Jog, M. (2014). Loudness perception and speech intensity control in Parkinson's disease. *Journal of Communication Disorders*, 51, 1-12.
- Cox, R. M., & Gray, G. A. (2001). Verifying Loudness Perception After Hearing Aid Fitting. *American Journal of Audiology*, 10(2), 91–98. doi: 10.1044/1059-0889(2001/009)
- Darley, F. L., Aronson, A. E., & Brown, J. R. (1969). *Motor speech disorders*. Philadelphia: W.B. Saunders.
- Darling, M., & Huber, J. E. (2011). Changes to Articulatory Kinematics in Response to Loudness Cues in Individuals With Parkinson's Disease. *Journal of Speech, Language, and Hearing Research*, 54(5), 1247–1259. doi: 10.1044/1092-4388(2011/10-0024)
- De Keyser, K., Santens, P., Bockstael, A., Botteldooren, D., Talsma, D., De Vos, S., De Letter, M. (2016). The Relationship Between Speech Production and Speech Perception Deficits in Parkinson's Disease. *Journal of Speech, Language, and Hearing Research*, 59(5), 915–931. doi: 10.1044/2016_jslhr-s-15-0197
- Duffy, J. R. (2013). *Motor speech disorders* (3rd ed.). St. Louis, MO: Elsevier Mosby.
- Goetz, C. G., Tilley, B. C., Shaftman, S. R., Stebbins, G. T., Fahn, S., Martinez-Martin, P., ... & Dubois, B. (2008). Movement Disorder Society-sponsored revision of the Unified Parkinson's Disease Rating Scale (MDS-UPDRS): scale presentation and clinimetric testing results. *Movement disorders: official journal of the Movement Disorder Society*, 23(15), 2129-2170.

- Gorkovenko, A. V., Sawczyn, S., Bulgakova, N. V., Jaszur-Nowicki, J., Mishchenko, V. S., & Kostyukov, A. I. (2012). Muscle agonist–antagonist interactions in an experimental joint model. *Experimental brain research*, 222(4), 399-414.
- Hixon, T. J., Goldman, M. D., & Mead, J. (1973). Kinematics of the Chest Wall during Speech Production: Volume Displacements of the Rib Cage, Abdomen, and Lung. *Journal of Speech and Hearing Research*, 16(1), 78–115. doi: 10.1044/jshr.1601.78
- Hixon, T. J., Weismer, G., & Hoit, J. D. (2020). *Preclinical speech science: anatomy, physiology, acoustics, and perception*. San Diego, CA: Plural Publishing Inc.
- Ho, A. K., Bradshaw, J. L., & Ianssek, R. (2000). Volume perception in parkinsonian speech. *Movement disorders: official journal of the Movement Disorder Society*, 15(6), 1125-1131.
- Hoit, J. D., & Hixon, T. J. (1987). Age and Speech Breathing. *Journal of Speech, Language, and Hearing Research*, 30(3), 351–366. doi: 10.1044/jshr.3003.351
- Huber, J.E., Stathopoulos, E.T., Ramig, L.O., & Lancaster, S.L. (2003). Respiratory Function and Variability in Individuals with Parkinson Disease: Pre- and Post-Lee Silverman Voice Treatment. *Journal of Medical Speech-Language Pathology*, 11 (4), 185-201.
- Huber, J. E., Chandrasekaran, B., & Wolstencroft, J. J. (2005). Changes to respiratory mechanisms during speech as a result of different cues to increase loudness. *Journal of Applied Physiology*, 98(6), 2177–2184. doi: 10.1152/jappphysiol.01239.2004
- Huber, J. E. (2007). Effect of Cues to Increase Sound Pressure Level on Respiratory Kinematic Patterns During Connected Speech. *Journal of Speech, Language, and Hearing Research*, 50(3), 621–634. doi: 10.1044/1092-4388(2007/044)
- Huber, J. E., & Spruill, J. (2008). Age-Related Changes to Speech Breathing with Increased Vocal Loudness. *Journal of Speech, Language, and Hearing Research*, 51(3), 651–668. doi: 10.1044/1092-4388(2008/047)
- Huber, J. E., & Darling, M. (2011). Effect of Parkinson’s Disease on the Production of Structured and Unstructured Speaking Tasks: Respiratory Physiologic and Linguistic Considerations. *Journal of Speech, Language, and Hearing Research*, 54(1), 33–46. doi: 10.1044/1092-4388(2010/09-0184)
- Huber, J. E., Darling, M., Francis, E. J., & Zhang, D. (2012). Impact of Typical Aging and Parkinson’s Disease on the Relationship Among Breath Pausing, Syntax, and Punctuation. *American Journal of Speech-Language Pathology*, 21(4), 368–379. doi: 10.1044/1058-0360(2012/11-0059)
- Huber, J.E., & Darling-White, M. (2017). Longitudinal Changes in Speech Breathing in Older Adults with and without Parkinson’s Disease. *Seminars in Speech and Language*, 38(03), 200–209. doi: 10.1055/s-0037-1602839

- Janssens, J. P., Pache, J. C., & Nicod, L. P. (1999). Physiological changes in respiratory function associated with ageing. *European Respiratory Journal*, 13(1), 197. doi: 10.1034/j.1399-3003.1999.13a36.x
- Lalley, P. M. (2013). The aging respiratory system—Pulmonary structure, function and neural control. *Respiratory Physiology & Neurobiology*, 187(3), 199–210. doi: 10.1016/j.resp.2013.03.012
- Lane, H., & Tranel, B. (1971). The Lombard Sign and the Role of Hearing in Speech. *Journal of Speech and Hearing Research*, 14(4), 677–709. doi: 10.1044/jshr.1404.677
- Levitzky, M. G. (2018). *Pulmonary physiology* (No. 1, pp. 1-viii). McGraw-Hill Education.
- Marras, C., Beck, J. C., Bower, J. H., Roberts, E., Ritz, B., ... Tanner, C. (2018). Prevalence of Parkinson's disease across North America. *Npj Parkinsons Disease*, 4(1). doi: 10.1038/s41531-018-0058-0
- Matheron, D., Stathopoulos, E. T., Huber, J. E., & Sussman, J. E. (2017). Laryngeal Aerodynamics in Healthy Older Adults and Adults With Parkinsons Disease. *Journal of Speech, Language, and Hearing Research*, 60(3), 507–524. doi: 10.1044/2016_jslhr-s-14-0314
- Meunier, S., Pol, S., Houeto, J. L., & Vidailhet, M. (2000). Abnormal reciprocal inhibition between antagonist muscles in Parkinson's disease. *Brain*, 123(5), 1017-1026.
- Project Information. (n.d.). Nih Research Portfolio Online Reporting Rools (RePORT). Retrieved From https://projectreporter.nih.gov/project_info_description.cfm?aid=9205890&icde=4498207&ddparam=&ddvalue=&ddsub=&cr=1&csb=default&cs=ASC&pball=
- Ramig, L. O., Fox, C., & Sapir, S. (2004). Parkinsons Disease: Speech and Voice Disorders and Their Treatment with the Lee Silverman Voice Treatment. *Seminars in Speech and Language*, 25(02), 169–180. doi: 10.1055/s-2004-825653
- Ramig, L., Meyer, T., Fox, C., Blitzer, A., & Tagliati, M. (2005). Laryngeal Function in Individuals with Parkinson's Disease. *Perspectives on Voice and Voice Disorders*, 15(1), 9. doi: 10.1044/vvd15.1.9
- Rousseau, B., & Branski, R. C. (2018). *Anatomy and physiology of speech and hearing*. New York, NY: Thieme.
- Sadagopan & Huber. (2007). Effects of Loudness Cues on Respiration in Individuals with Parkinson's disease. *Movement Disorders*, 22(5), 651-659. Doi: 10.1002/mds.21375
- Sapir, S. (2014). Multiple Factors Are Involved in the Dysarthria Associated With Parkinson's Disease: A Review With Implications for Clinical Practice and Research. *Journal of Speech, Language, and Hearing Research*, 57(4), 1330–1343. doi: 10.1044/2014_jslhr-s-13-0039

- Solomon, N. P., & Hixon, T. J. (1993). Speech Breathing in Parkinson's Disease. *Journal of Speech, Language, and Hearing Research*, 36(2), 294–310. doi: 10.1044/jshr.3602.294
- Spielman, J., Mahler, L., Halpern, A., Gilley, P., Klepitskaya, O., & Ramig, L. (2011). Intensive voice treatment (LSVT®LOUD) for Parkinsons disease following deep brain stimulation of the subthalamic nucleus. *Journal of Communication Disorders*, 44(6), 688–700. doi: 10.1016/j.jcomdis.2011.05.003
- Stathopoulos, E. T., Huber, J. E., Richardson, K., Kamphaus, J., Decicco, D., Darling, M., Fulcher, K., & Sussman, J. E. (2014). Increased vocal intensity due to the Lombard effect in speakers with Parkinsons disease: Simultaneous laryngeal and respiratory strategies. *Journal of Communication Disorders*, 48, 1–17. doi: 10.1016/j.jcomdis.2013.12.001
- Winkworth, A. L., Davis, P. J., Adams, R. D., & Ellis, E. (1995). Breathing Patterns During Spontaneous Speech. *Journal of Speech, Language, and Hearing Research*, 38(1), 124–144. doi: 10.1044/jshr.3801.124
- Winkworth, A. L., & Davis, P. J. (1997). Speech Breathing and the Lombard Effect. *Journal of Speech, Language, and Hearing Research*, 40(1), 159–169. doi: 10.1044/jslhr.4001.159
- Zemlin, W. R. (1998). *Speech and hearing science: anatomy and physiology*. Boston, MA: Allyn and Bacon.
- Zigmond, M. J., & Burke, R. E. (2002). 123 Pathophysiology of Parkinson's Disease. In K. L. Davis, D. Charney, J. T. Coyle, & C. Nemeroff (Eds.), *Neuropsychopharmacology: the fifth generation of progress* (pp. 1781–1793). Philadelphia, PA: Lippincott Williams & Wilkins.