THE EFFECT OF BOLUS VOLUME ON HYOLARYNGEAL MUSCLE ACTIVITY IN PARKINSON'S DISEASE

by

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Submitted to the Graduate Faculty of
The Harris College of Nursing & Health Sciences
Texas Christian University
In partial fulfillment of the requirements for the degree of

Masters of Science in Speech Language Pathology
May 2018

APPROVAL PAGE

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A Thesis for the Degree Master of Science

by

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ABSTRACT

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Purpose: The purpose of this study was to investigate the differences in submandibular hyolaryngeal muscles activation during swallowing in individuals with PD and healthy older adults (HOA) without PD. The study also investigated effects of bolus volume on submandibular hyolaryngeal during swallowing for people with PD and HOAs.

Methods: Participants included 14 participants with PD and 10 HOAs who served as controls. Four swallow tasks were conducted. Three liquid swallow trials at 5ml, 10ml, and 15ml of water, and three trials of 1tsp of pudding was presented as the maximum voluntary contraction swallow. The sEMG signals were recorded during each swallow and were saved as separate files. This resulted in a total of 12 sEMG files for each participant (3 swallows of 5 mL of water, 3 swallows of 10 mL of water, 3 swallows of 15 mL of water, 3 swallows of 1 tbsp. of pudding).

Results: The results from the MANOVA determined that 1) contraction amplitude of submandibular muscles differed for individuals with PD and healthy controls but contraction duration did not, and 2) bolus volume had no significant effect of contraction amplitude and contraction duration in either PD or HOA participants and 3) there is no group X condition interactional effect on either contraction amplitude or contraction duration.

Conclusion: Main findings from this study concluded contraction amplitude of submandibular muscles differed for individuals with PD and HOAs and bolus volume had no significant effect on contraction amplitude or contraction duration in both PD and HOA groups. Contraction duration was not found significantly different but descriptive statistics demonstrated a shorter duration than HOAs. This study gives us a better understanding about what is happening to the muscular physiology of the submandibular muscles. People with PD with subclinical dysphagia may be utilizing compensatory strategies to effect contraction timing. The findings from this study may support the theory of compensatory neuromuscular adaptions utilized by people with PD during the earlier stages of disease severity. Further studies should be completed to verify and expand on these findings.

ACKNOWLEDGEMENT

I would like to express many thanks to the people and organizations that have supported and encouraged me. I am deeply appreciative towards Dr. Christopher Watts, who played the role of my advising professor during this process. Dr. Watts graciously guided, supported, and dedicated his time and efforts towards this project. I would also like to express my appreciation to the members of my committee, Ms. Laurel Lynch and Dr. Philip Esposito. They each supported and provided direction through this process.

To the families, caregivers, and people who took part in my study, I would like to extend an appreciation. Specifically to Bedford Parkinson's support group, the Texas Harris Parkinson's support group, and TCU's staff. Because of the generosity of our participants, this whole study was made possible. The time and efforts freely given is the reason why we are able to gain ample information on how to best benefit people with Parkinson's Disease.

Finally, I would like to extend a sincere appreciation to those who have been my constant support and encouragement. My roommates and my community in Fort Worth, who have joyfully walked through this season of graduate school with me, and to my parents, who gave me the wonderful opportunity to pursue higher education at TCU and always encouraged me to strive for excellence in all I do.

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TABLE OF CONTENTS

LIST OF FIGURES

FIGURE **Page**

LIST OF TABLES

Table **Page**

INTRODUCTION

In 1817, a neurologist named James Parkinson first named a disease 'Shaking Palsy' and described it to involve "*tremors with lessened muscular power, a tendency to bend the trunk forward, an increase of pace when walking, and yet the sense and intellect remaining intact*" (Bartels & Leenders, 2009). About 100 years later, Friedrich Heinrich Lewy first discovered abnormal protein deposits within neurons of individuals with Parkinson's disease. Theses protein deposits are now named after him as "Lewy bodies" and are seen as a hallmark of idiopathic Parkinson's Disease (PD) (Lewy, 1912). Then in 1957, Carlsson and his colleagues deemed dopamine as the alleged neurotransmitter linked to cell death in neurons associated with PD (Suttrup & Warnecke, 2016). Currently, we know that PD is a progressive neurological disorder that results from widespread neuronal cell death from the brainstem to the cerebral cortex and the peripheral nervous system (Jankovic, 2008). Specifically, there is death of dopamine neurons that have cell bodies in substantria nigra and project to the striatum of the basal ganglia. Lewy bodies are found in these degenerating neurons and it is unclear whether they are causative or symptomatic in PD pathogenesis (Bartels & Leenders, 2009). Substantial degeneration of dopamine neurons is linked with the emergence of classical signs of the disease, such as loss of sensorimotor control, balance, gait, autonomic function, communication, swallowing and depression (Bartels & Leenders, 2009).

Prevalence & Incidence

PD is the 2nd most common neurological disorder after Alzheimer's disease (Pringsheim, Jette, Frolkis & Steeves, 2014) It affects 0.3% of the general population

worldwide, occurring in about 1% of individuals aged 60 years or older and increasing to about 4-5% to those aged 85 and older (WS Coriolano et al., 2012). It is estimated that by 2020, more than 40 million people worldwide will be diagnosed with PD (Suttrup & Warnecke, 2016). Onset for most cases is in the late 60s or early 70s, and young-onset disease (e.g., in the 30s) and juvenile-onset disease (before age 20) account for a small proportion of cases (Bartels & Leenders, 2009). PD is more common in men than women (1.5:1), and although the diseases occurs in all races the prevalence of PD is higher in Hispanic/Latino population than the Non-Hispanic white populations (WS Coriolano et al., 2012).

Clinical Features

There has been increasing evidence to support premotor PD symptoms, or symptoms that develop prior to the onset of the significant motor impairments that leads to the initial diagnosis of PD. The evidence reveals that olfactory dysfunction, sleep abnormalities, cardiac sympathetic denervation, intestinal dysmotility, and depression may predate the onset of the clinical motor symptoms (Obeso et al., 2010). Among the motor impairments of PD, the resting tremor is the most commonly recognized symptom. It occurs in approximately 75% of patients and is often the first motor manifestation, typically emerging in a hand or arm (Pallone, 2007). Other motor symptoms include bradykinesia (slowness of movement), rigidity, and postural instability, the latter of which leads to impaired balance. Motor symptoms have an asymmetric onset and progression, and usually will respond to dopaminergic treatment (Rizek, Kumar & Jog,

2016). Definitive diagnosis of PD is only possible after postmortem pathologic findings (Suttrup & Warnecke, 2016).

Non-motor features of PD include anxiety, apathy, fatigue, some degree of executive function impairment (slow thought process, memory problems, and difficulty with abstract thought), and sensation deficits (smell, visual processing, balance) (Chaudhuri, Healy & Schapira 2006). Autonomic dysfunctions in PD include intestinal dysmotility, urinary urgency, swallowing dysfunction, orthostatic hypotension, seborrheic dermatitis, increased sweating (hyperhidrosis), drooling, and sexual dysfunction (WS Coriolano et al., 2012).

Etiopathology

No single cause of PD has been discovered. Although, there are some forms that have been genetically linked, although most cases are idiopathic. First-degree relatives of PD patients have a two-to-threefold increased risk of developing the disease (Gasser, 1998). A limited number of genes have been associated with genetically linked PD. However, there have been limitations in genetic studies, due to the difficulty in estimating concordance rates based on clinical information alone. A generally accepted hypothesis is PD is the result of an interaction between genetic predisposition and environmental factors. According to this theory, this interaction induces mitochondrial respiratory failure and oxidative stress within nigral neurons, leading to cell death (Bartels & Leenders, 2009). Environmentally, the only agent that has been found to produce levodopa-responsive Parkinsonism in humans is 1-methyl-4-phenyl-1, 2, 3, 6, -tetrahydropyridine, which is a by-product of unregulated or illegal production of an analog of opioid meperidine (Bartels & Leenders, 2009).

Researchers have suggested the pathology of the disorder follows a specific sequence: starting at the dorsal motor nucleus of the vagus nerve and the olfactory bulbs and nucleus, followed by the locus coeruleus, then the death of cells in substantria nigra pars compacta (SNc), to the less vulnerable nuclei and cortical areas (Braak and Braak, 2000). This route of progression starts in the lower brainstem and heads rostrally towards the midbrain and cortex. Although it is still unknown if Lewy bodies are symptomatic or causative in the PD pathogenesis, they are still a marker in PD pathology and are found in the degenerating neurons consisting of α-synuclein (Bartels & Leenders, 2009).

Progression

Studies show that the rate of decline in motor function in PD is not linear. Kuramoto et al., (2013) collected longitudinal Positron Emission Tomography (PET) measurements of the brain on 78 PD patients, from 4 different subregions on each side of the brain. The results showed a non-linear progression in PD patients; this was revealed by the exponential function depending on two patient related characteristics 1) duration since symptom onset 2) age at symptom onset. This study found the earlier age of disease onset is associated with greater degree of denervation prior to initial symptom onset of PD (Kuramoto et al., 2013). This means younger patients had considerably more dopamine nerve terminals before the clinical manifestation of PD, suggesting younger patients have a greater compensatory mechanism and were able to tolerate greater degrees of denervation prior to PD symptom onset (Kuramoto et al., 2013). With that said, age plays a big factor in predicting PD progression rate and it remains the most prominent risk factor for developing the disease (Bartels & Leenders, 2009). Extending

this study further, Kuramoto et al. (2013) also discovered that over the course of the illness the degree of damage was significantly greater in the posterior putamen than the anterior putamen.

When looking at severity of the clinical symptoms, the disease has been found to progress faster in patients with a more mild motor impairment than those with marked impairments at the initial evaluation (Jankovic, 2008). Cognitive impairment is also more common and emerges earlier in patients who are older (> 70 years) at the onset of the disease (Hely, Morris, Reid & Trafficante, 2005). The disease progresses through stages; the latter stages of the disease an individual may be non-ambulatory and require assistance to complete activities of daily living.

Voice

Voice disorders are present in 92% of patients with PD and are considered a component of the larger speech impairment of PD, called hypokinetic dysarthria (Robbins & Logemann, 1986). The voice component of hypokinetic dysarthria includes dysphonia characterized by hoarseness, decreased volume, and monotonous tone. It has been reported that dysphonic symptoms occur in the early periods of the disease with relatively high incidence (Ikui et al., 2015). In 1978, Logemann, Fisher, and Boshes perceptually examined 200 cases of unmedicated PD patients. Voice characteristics found included breathiness (15%), hoarseness (45%), and roughness (29%). These authors concluded a progression of speech dysfunction in PD; beginning with laryngeal abnormalities and advancing to lingual and labial neuromuscular defects (Logemann, Fisher, Boshes, 1978).

Breathing and breath support are directly related to the ability to produce voice. Speech breathing studies signify persons with PD produce less efficient and variable movements of the chest wall during speech when compared to neurologically normal controls (Tjaden, 2008). The inefficient usage of the mechanism by speakers with PD is further supported by reports of decreased lung volume during initiation of speech and a tendency to speak below end expiratory level (Tjaden, 2008). Additionally, vocal fold bowing causing incomplete vocal fold closure during phonation is commonly observed in patients with PD. It is unclear if the inadequate closure of vocal folds is caused by reduced muscle activation, weakness, and reduced effort or excessive laryngeal muscle activity and muscular rigidity in the larynx (Tjaden, 2008). Other measurements such as, vocal intensity and vocal frequency have been observed through various studies. Vocal intensity is found to be around 2-4 dB lower in the PD population than normal controls across a number of different types of speech task and voice tasks (Fox & Ramig, 1997). Fox and Ramig (1997) reported that even in early stage, PD patients had softer voices than what would be expected for normal subjects based on their data of mean loudness ratings. Fundamental frequency is also found to be significantly lower in PD patients than their age-matched normal controls (Ikui et al., 2015).

Articulation

Prior research has demonstrated that around 70-90% of people with PD have a distinct alteration of their speech associated with hypokinetic dysarthria (Logemann et al., 1978). Hypokinetic dysarthria is a multidimensional impairment that affects various speech aspects such as respiration, phonation, articulation and prosody (Darley et al.,

1969). Abnormal perceptual characteristics of hypokinetic dysarthria associated with articulation include brief rushes of speech, short phrases, variable rate, inappropriate silences, repeated phonemes, decreased stress, and inaccurate consonant production (Tjaden, 2008). Vowel errors are a frequently occurring deficit associated with hypokinetic dysarthria and may be considered a possible early marker of PD (Rusz et al., 2013). Acoustically, the findings reveal that people with PD tend to have reduced vowel formant transition, a collapsed vowel acoustic working space, and a reduced trend on consonant spectral distinctiveness (Tjaden, 2008). Moreover, a study reported that PD speakers have abnormalities in their formant frequency across sentence repetition, passage reading, and monologue, when compared to their norms (Rusz et al., 2013). These abnormalities support the reduced range in the displacement and velocities of the oral articulators (Tjaden, 2008). As articulation errors increase a person's intelligibility decreases. One study evaluating the intelligibility of speech in PD revealed that 69.6% of people with PD fell below the control mean of perceived disordered speech by unfamiliar listeners. Although overall speech intelligibility was reduced in people with PD, it was not dependent on disease severity or motor phenotype of disease duration (Miller et al., 2007).

Dysphagia

Swallowing is highly complex sensorimotor activity; it involves organized interactions between cortical, cerebellar, bulbar and peripheral systems (Bailey, 2004). The central nervous system (CNS) and the peripheral nervous system (PNS) are activated during swallowing. The CNS, specifically the corticobulbar and extrapyramidal pathways

send important information to the brainstem. Once brainstem receives from the corticobulbar and extrapyramidal pathways, it then excites peripheral sensory neurons and motor cranial neurons, respectively. Connections between specific sensory and motor neurons within the brainstem stimulate the sequential muscle activity of the pharyngeal swallow reflex (Bailey, 2004). The muscles involved with swallowing include facial muscles such as the mastication muscles (temporalis, masseter) and pterygoids, oropharyngeal muscles such as the tensor veli palatini and pharyngeal constrictors, the intrinsic laryngeal muscles such as the thyroarytenoid, and the suprahyoid laryngeal elevator muscles such as the mylohyoid and anterior belly of digastric (Bailey, 2004). These latter suprahyoid muscles are located in the submandibular region, connecting the mandible to the hyoid bone.

Normal swallowing is organized into four phases, 1) the oral preparatory phase where food is broken down into a single bolus consistency and held by the tongue in preparation for the swallow; 2) the oral phase, in which the tongue drives the bolus posteriorly towards the base of the tongue so that the swallow reflex will be initiated; 3) the pharyngeal phase, in which the bolus enters the pharynx; and 4) the esophageal stage, which the bolus travels through the esophagus to the stomach (Logemann, 1983).

During the pharyngeal stage, a number of neuromotor activities occur driving muscular action. These pharyngeal stage muscular events include: a) velopharyngeal closure b) contraction in the pharyngeal constrictors, c) tongue-base retraction; d) laryngeal elevation e) laryngeal closure and f) cricopharyngeal relaxation (Logemann, 1983). The swallowing reflex mediated in the brainstem triggers these events, working to move the bolus safely from the oropharynx to the esophagus. This entire pharyngeal stage

lasts approximately one second, regardless of food texture and factors of sex and age (Logemann, 1983) (Hamlet, Muz, Patterson & Jones, 1989) (Dodds, Stewart, & Logemann, 1990).

Dysphagia in persons with PD can manifest as various abnormalities in different swallowing stages. These abnormalities include the disruption of bolus flow through the mouth, pharynx and esophagus (Umay, Unlu, Saylam, Cakci, & Korkmaz, 2013). Dysphagia is classified into four categories that are dependent on the location of the swallowing impairment: oropharyngeal, esophageal, esophagogastric, and paraesophageal (Wolf, 1990). Subjective dysphagia occurs in one third of community-dwelling PD patients, while 4 out of 5 patients have objectively measured dysphagia detected with instrumentation (Belo et al., 2014). This suggests that dysphagia is common in PD and is potentially under diagnosed. Dysphagia in PD is not necessarily correlated with the severity of the disease based on body movements or clinical staging (Troche, Sapienza, & Rosenbek, 2008). The cause of dysphagia in PD patients has been linked to the lack of dopamine in the striatum, which may impair the brainstem and subcortical swallowing networks. This hypothesis is supported by the observation that some individuals with PD show a significant improvement of swallowing function after application L-Dopa (Suttrup & Warnecke, 2016).

Persons with PD experience abnormal movements of swallowing in the oral stage of swallowing. These abnormalities include presence of labial bolus leakage, deficient or hesitant mastication, lingual tremor, lingual pumping prolonged lingual elevation, and slow limited mandibular excursion (Troche et al., 2008). During the pharyngeal stage, abnormal swallowing movements include delayed contraction of the pharyngeal wall,

deficient epiglottitic retroversion, decreased epiglottic range of motion, slow laryngeal elevation and excursion, and upper esophageal sphincter incoordination. This may result in coating of the pharyngeal walls, stasis in the valleculae and/or pyriform sinuses, and penetration or aspiration (Troche et al., 2008).

The average volume per swallow is the volume that is comfortable to swallow sequentially while the dysphagia limit is the volume at which second or more swallows become necessary to swallow the whole amount of bolus (Ertekin et al., 1996). Persons with PD have shown to manifest a significantly lower dysphagia limit and lower average volume per swallow (Belo et al., 2014). Belo et al. (2014) demonstrated that both measurements of average volume per swallow and limit of dysphagia could be used as quick objective screeners to test for the early identification of dysphagia in persons with PD.

The characteristic of the bolus can also affect swallowing function in persons with PD. Research has shown that bolus consistency can significantly affect oral transit time; oral transit time is faster with thinner boluses (Troche et al., 2008). In addition, Troche's study indicated that less penetration-aspiration occurred for thicker consistencies than thinner, suggesting that thicker consistencies should yield safer swallows (2008). This study showed no significant difference in pharyngeal transit time between the two consistencies.

Electromyographic Studies of Swallowing

The muscle activity occurring during swallowing can be evaluated with intramuscular or surface electromyography techniques. Surface electromyography (sEMG) is a noninvasive, easily repeatable measure that can be used for swallowing

assessment and rehabilitation (WS Coriolano et al., 2012). sEMG has primarily been used to 1) identify the presence of swallowing activity 2) analyze swallowing function (amplitude and timing) 3) treat swallowing disorders using sEMG as a biofeedback strategy (Crary, Carnaby, & Groher, 2006). The limitation with the sEMG device is it cannot measure the movement of individual laryngeal muscles. However, it is able to evaluate groups of muscles that are associated with the pharyngeal stage of swallowing, including the submandibular mylohyoid, geniohyoid, and anterior digastric muscles, known as the suprahyoids Additionally, sEMG reveal the differences in muscle activity when bolus' are manipulated. Ueda et al., (2013) found an increase in bolus volume there will be greater submental muscle activity (Ueda et al., 2013).

One study observed 4 different muscle groups (orbicularis oris muscles, infrahyoid muscles, masseter muscles, submental suprahyoids) during normal swallowing (Vaiman, Eviatar, & Segal, 2004). This study found that the movement in submandibular muscle group had 30% to 50% higher amplitude than masseter muscle activity, and 40% to 65% higher amplitude than the infrahyoid activity (Vaiman et al., 2004). It can be concluded that the submandibular laryngeal elevators contribute a substantial amount of information to the sEMG signal during swallowing (Crary et al., 2006).

sEMG technology has been shown to be reliable in measuring the collective physiology of submandibular muscles during swallowing. Ertekin et al (1995, 1997) found that laryngeal elevation was significantly related to increased submental sEMG activity and decreased cricopharyngeal muscle activity. Additionally, Ding, Larson, Logemann, and Rademaker (2002) identified a strong temporal relationship between

laryngeal elevation and submental sEMG in both normal swallows and in swallows using the Mendelsohn maneuver (sustained laryngeal elevation during swallow).

A recent study completed by Poorjavad and collogues (2017) investigated the usefulness of sEMG measurement tool in swallowing assessment and treatments. The study set out to establish reliability of the sEMG measures of the swallowing function of muscles during different swallowing conditions (in healthy young and old volunteers). Twenty-Four older adults and ten younger adults participated in this study. The activity of submental suprahyoid, infrahyoid, and masseter groups were measured using sEMG during three repetitive swallow tasks. Results showed the sEMG relative reliability calculation had significant agreements between mean and peak amplitude and average median frequency in both groups. The authors concluded that sEMG is a reliable measure for assessing swallow function in both younger and older adults (Poorjavad, Talebian, Ansari & Soleymani, 2017).

Another study completed by Crary & Groher (2006) looked at biomechanical events (hyoid elevation, pharyngeal constriction, and opening-closing of the pharyngoesophageal segment) while swallowing using simultaneous videofluroscopy and sEMG. Their findings indicated that all three biomechanical events have a strong correspondence to the sEMG signal, with hyoid elevation having the strongest association.

sEMG has also been used to observe the swallowing differences in people with PD and normal controls. The sEMG revealed the patients with PD took significantly more time and needed significantly more swallows to drink 100ml of water than normal controls. The significant differences in this study indicate that the sEMG can be a simple

tool to study and monitor swallowing in people who have PD (WS Coriolano et al., 2012).

Although there is limited information on specific biomechanical correlation of sEMG signal during swallowing, the few studies reporting data show the sEMG signal is a useful indicator of major biomechanical events in swallowing such as hyolaryngeal excursion (e.g., elevation), pharyngeal constriction and opening-closing of the pharyngoesophageal segment. The information within the sEMG signal may be important for clinical application. For instance, knowledge of the muscular activity during these biomechanical events during swallowing may help clinicians advance in a more effective treatment.

Effect of Bolus Volume on sEMG Activity:

Research has shown that the volume of the bolus effects submental muscle movement during swallowing (Ueda et al., 2013). The anterior and superior hyoid movement during a swallow is controlled with submental muscle movement. Ueda and his colleagues (2013) observed the maximum velocity of hyoid movement during normal swallowing in relation to bolus size and discovered that a larger bolus volume resulted in a greater maximum hyoid velocity. Therefore this study concluded that when bolus size increases in volume there is greater submental muscle movement (Ueda et al., 2013). Another study by Vaiman, Eviatar and Segal observed the amplitude of sEMG signals during normal swallows with persons in 6 different age ranges (18-30, 31-40, 41-50, 51-60, 61-70, 70+) for the following muscle groups: orbicularis oris, masseter, submental, and infrahyoid. They found that the range of sEMG activity in all muscle groups (expect

orbicularis oris) have a linear tendency to decrease with age. They discovered that elderly subjects (70+) had increased masseter muscle movement during a saliva swallow, which may indicate that elders have difficulties obtaining sufficient amounts of saliva to swallow. Additionally, they found that in the older generation (61+), the submental sEMG muscle activity increased as the bolus volume increased (Vaiman et al., 2004). Conversely, Zu et al. (2011) concluded that bolus volume had little to no effects on the extent of hyoid movement. Because of this incongruity in the literature, it is challenging to conclude the extent that bolus volume influences the muscles of the hyoid. It is probable that application of sEMG measurements to swallowing different bolus sizes could provide clarity on this discrepancy.

STATEMENT OF THE PROBLEM

Dysphagia is a common impairment occurring secondary to PD and has a substantial impact on quality of life. The dysphagia profile of individuals with PD includes pharyngeal stage problems, one of which is reduced hyolaryngeal excursion. This is thought to be caused by hypokinesia of the submandibular muscles. While sEMG is a less invasive technology than videofluroscopic studies or endoscopic studies for studying these muscles, little evidence exists for the use of sEMG to study submandibular hyolaryngeal muscle function during swallowing in PD. Such information might better inform our understanding of pharyngeal stage swallowing impairments secondary to PD, and thus clinical practice. The purpose of this study was to use sEMG to measure the activation in the submandibular hyolaryngeal muscles of individuals with PD when swallowing different bolus volumes, and to compare these measures to those of healthy older adults (HOA) without PD. The specific research questions to be addressed included: (1) Is the contraction amplitude and contraction duration of submandibular muscles during swallowing different for individuals with PD and healthy older controls? (2) Does bolus volume effect contraction amplitude and contraction duration of the submandibular muscles during swallowing differently for individuals with PD and healthy controls?

METHODOLOGY:

Participants: There were a total of 24 participants recruited for this study. Fourteen participants with PD were recruited from the volunteer database of Dr. Christopher Watts. PD participants consisted of six males and eight females, with mean age of 72.5 (SD: 6.20) and mean disease duration of 5 years (SD: 7.13). The inclusion criteria for the

PD participants included: 1) diagnosis of idiopathic PD by a neurologist, 2) no other diagnosed neurological conditions unrelated to PD, and 3) not presently being treated for dysphagia by a speech-language pathologist. Self-perceived symptoms of pharyngeal dysphagia using the PD Swallow Symptom Questionnaire (see appendix A), which was developed for a previous study, were collected for baseline information regarding swallowing complaints. Further information regarding participants with PD can be found in Table 1.

HOA participants were recruited from the local community and family members of volunteers. Inclusion criteria were as follows: 1) no self-reported history of swallow difficulty, 2) no history of chronic neurological illness, 3) no self-reported indication of any pharyngeal stage swallowing symptoms. HOA participants consisted of four males and six females, with mean age of 67 years (SD: 4.3) Further information regarding HOAs can be found in Table 1.

Table 1. Demographic information on the participants with PD and healthy older adults (HOA).

PD Subjects	Age	Gender	Dx	Dysphagia Complaints
PD1	80	Male	2014	Yes
PD2	61	Male	2008	No
PD3	73	Female	2015	No
PD4	63	Female	2014	No
PD5	63	Female	2012	Yes
PD6	65	Female	2017	Yes
PD7	72	Male	2012	No
PD8	75	Female	2011	Yes
PD9	73	Female	1988	No
PD10	75	Male	2013	No
PD11	75	Male	2017	No
PD12	67	Male	2013	Yes

PD13	80	Female	2014	Yes
PD14	70	Male	2012	Yes
HOA Subjects	**Age**	**Gender**		
HOA1	70	Female		
HOA2	62	Female		
HOA3	66	Male		
HOA4	62	Male		
HOA5	70	Female		
HOA6	68	Male		
HOA7	69	Female		
HOA8	65	Female		
HOA9	62	Female		
HOA10	66	Male		

Instrumentation: sEMG signals were obtained using the Swallow Signals Lab (Pentax Medical, Montvale, NJ), set up in a similar method as Crary, Carnaby, and Groher (2006). Electrodes associated with this system were disposable self- adhesive patches, approximately 2.25 in. in diameter. Patches consisted of three electrodes organized in a triangle configuration approximately 0.25 in. from each other. Two of the electrodes served as recording electrodes and a third served as the ground. Signals from the electrodes were processed by the Swallowing Signals Lab, which was connected to a desktop PC. Signal processing consisted of digital sampling at 500Hz followed by band-pass filtering between 50-250Hz. Signals were then integrated with a time constant of 50ms and rectified. The resulting signal was a smoothed sEMG waveform displayed as amplitude over time.

Procedures: All participants underwent consenting procedures prior to their participation in the study. Each participant was recorded in the Laryngeal Function Laboratory at the Miller Speech and Hearing Clinic (MSHC) on the campus of Texas Christian University. Participants underwent one session where all the measurements were obtained. Following

consent for participants with PD, researcher asked "Have you noticed any signs or symptoms (s/s) of swallowing difficulty since you've been diagnosed with PD, including but not limited to greater coughing or throat clearing with food/drink, feeling of food/drink getting stuck, wet vocal quality when drinking etc.?" and gather information regarding their date of disease onset. Once eligibility was determined, the procedure for electrode placement took place. For HOA, swallowing questions were not asked and procedures for electrode placement were completed following consent. For electrode placement procedures, the skin surface was first cleaned with an alcohol wipe to remove any skin oils present. After, the sEMG electrode patch was placed on the skin surface above the submandibular hyolaryngeal muscles, approximately 1 cm below the chin. This placement was to ensure the electrodes recorded the submental hyolaryngeal activity, as shown in Figure 1. This placement has been shown to detect activation of the submandibular muscles as seen by the anatomical location of these muscles presented in Figure 2 (Crary 2015). Additionally, the placement has been validated by Crary and collogues in various studies (Crary et al., 2006) (Crary 2015). The wireless electrode heads communicated with the Swallowing Signals Lab software that plotted the sEMG signals on the computer. Each participant's signals were saved for later analysis.

Figure 1. Surface EMG electrode placement.

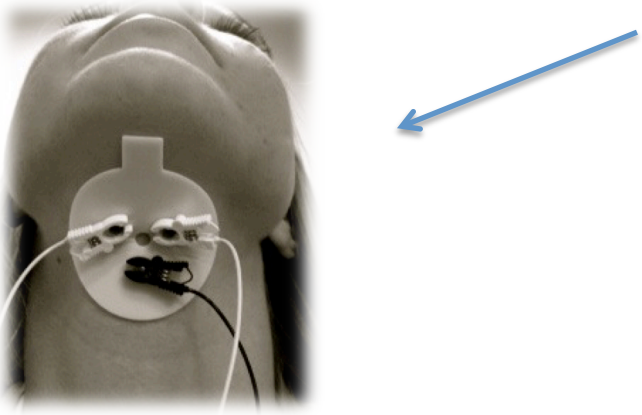
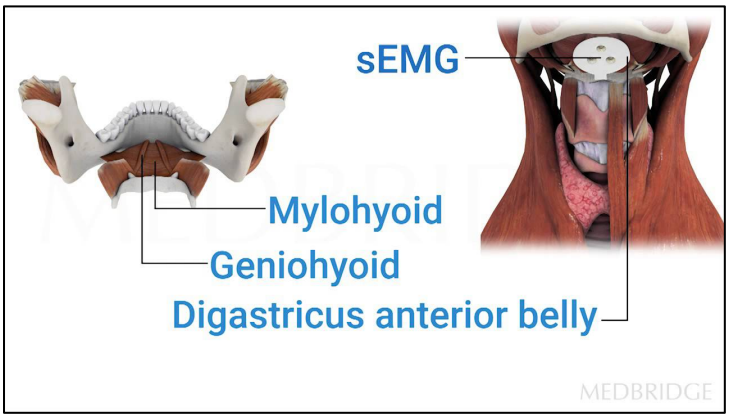


Figure 2. sEMG placement on submandibular muscles.



After electrode placement, each participant performed 4 tasks: Three trials of liquid swallow for the following volumes of water: 1) 5mL, 2) 10mL, and 3) 15mL; and 4) three trials of 1 tbsp. of pudding with maximum effort. For liquid trials, participants were instructed to "swallow all of the water/pudding presented with one gulp." These instructions were designed to elicit "typical" swallowing behavior. For the maximum effort task participants were instructed to "swallow as hard and as fast you can." This condition was used as the equivalent of a maximum voluntary contraction (MVC) against which measurements from the other three conditions would be compared to normalize sEMG amplitude measurements (raw sEMG amplitude measurements are highly variable between subjects due to natural variability in neurophysiology).

To control for bolus order effects, the bolus types were presented randomly across all participants. In addition, the MVC trials were always presented after the three liquid trials so that the instructions of "swallow as hard and as fast as you can" would not bias the typical swallow behavior. During maximum effort trials, a different consistency (pudding) was used because previous research has shown that a thicker consistency resulted in greater submental muscle contraction (Zu et al., 2011, Watts, & Kelly, 2015).

The sEMG signals were recorded during each swallow and were saved as separate files. This resulted in a total of 12 sEMG files for each participant (3 swallows of 5 mL of water, 3 swallows of 10 mL of water, 3 swallows of 15 mL of water, 3 swallows of 1 tbsp. of pudding).

Analyses:

The methodology of this study consisted of two independent variables (group [PD vs. HOA] and bolus volume [3mL, 5mL, 10mL]) and two dependent variables (contraction amplitude ratio and contraction duration). Contraction amplitude ratio corresponded to the normalized peak amplitude, in microvolts, of submandibular muscle activity during any single swallow. It was calculated by dividing the mean contraction amplitude from any liquid swallow trial by the maximum amplitude value from the three MVC trials. Specifically, contraction amplitude ratio was calculated through the following process:

A. Contraction amplitude was taken from the three swallow tasks in each volume conditions (3mL, 5mL, 10mL). A mean contraction amplitude was calculated for each volume condition per participant.
B. Contraction amplitude was calculated from the three maximum swallows of each patient.
C. Each mean amplitude was divided by the corresponding maximum amplitude (e.g., mean 5ml swallow amplitude / maximum amplitude of participant).
D. From the 3 amplitude ratios in each condition, a mean was calculated to represent the amplitude data point for a particular subject in a particular condition.

E. Each participant had a mean 5mL, 10mL, and 15mL amplitude ratio.

For the purposes of this paper "contraction amplitude" will be used interchangeably with "contraction amplitude ratio" calculated using the process described above.

Contraction duration corresponded to the total time, in milliseconds, of submandibular muscle activity for any single swallow. These measurements were calculated using the Digital Swallow Workstation from visual inspection and marking of the sEMG waveforms representing activity during each swallow.

The statistical software SPSS was used to apply a multivariate analysis of variance (MANOVA) to the contraction amplitude and contraction duration data with group (PD vs HOA) and swallow condition (5ml, 10ml, 15ml) as factors along with amplitude and duration as separate variables in the statistical design. A significance level (α -level) of 0.05 was used for the analysis. A significant main effect for the MANOVA was followed up with separate F-tests for amplitude and duration data sets.

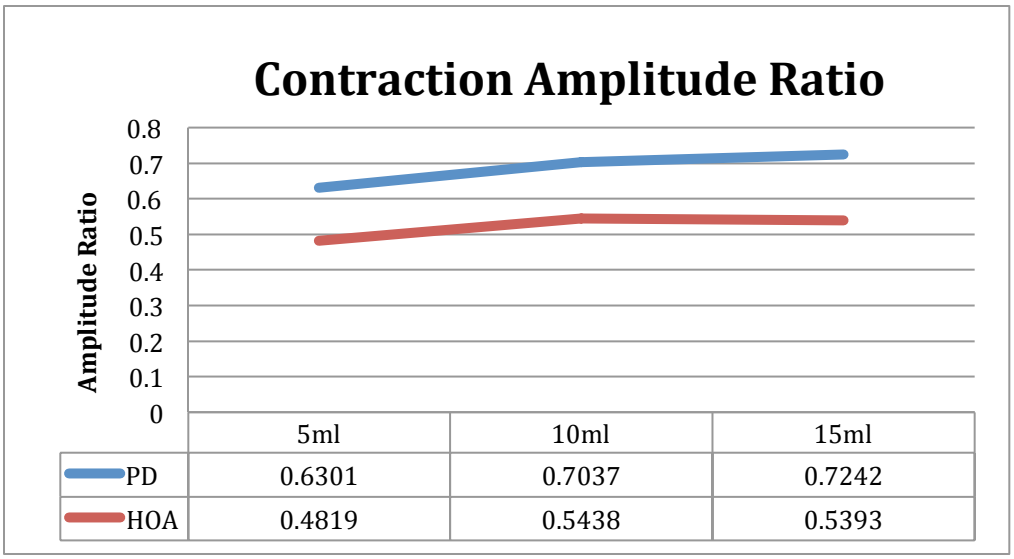
Intra-measurer reliability was assessed by re-measurement of 10% of the swallow files. Correlational analysis was used to assess the degree of relationship between initial and reliability measures. A correlation coefficient of equal to or greater than 0.90 was deemed sufficient for reliability purposes.

RESULTS:

Reliability: Intraobserver agreement measures were calculated for researcher procedural fidelity among the participants. Reliability regarding contraction duration measurements was r=0.99 and reliability regarding contraction amplitude was r=1.0. Both contraction duration and amplitude measurements were greater than the correlation coefficient of 0.90, deeming the reported measures adequate for reliability purposes.

Contraction Amplitude: The mean values of contraction amplitude ratios are shown in. The data trend for amplitude suggested that participants with PD consistently used a greater percentage of their maximum swallow contraction force during typical swallows across all volume conditions compared to HOA participants (Figure 3). Contraction amplitude ratios were slightly greater as bolus volume increased, a trend demonstrated by both groups.

Figure 3. Contraction amplitude ratios (in microvolts) for the PD and HOA participants in each bolus condition.



Contraction Duration: The mean values of contraction duration are shown in Figure 4. The range from the fastest mean time to the slowest mean time was 0.10 seconds. The data trend from the descriptive statistic shown in Figure 4 suggested that participants with PD exhibited faster muscle contractions than controls in the 5mL and 20mL conditions, and relatively similar durations in the 10mL condition.

Figure 4. Contraction Duration (in seconds) for the PD and HOA participants in each bolus condition.

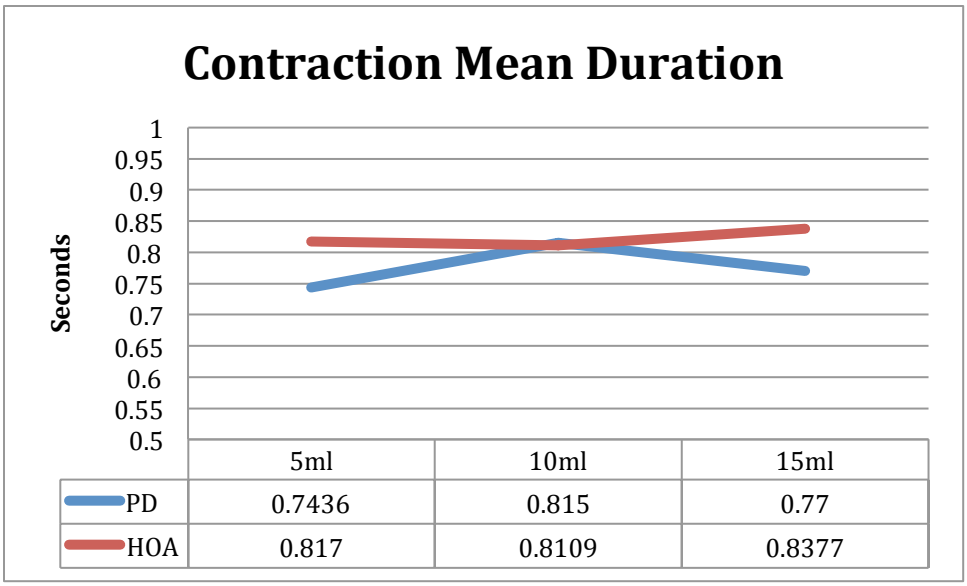


Table 2 displays the results from the MANOVA applied to the group (PD vs HOA) and condition (5ml, 10ml, 15ml) factors, along with the interaction effect of group X condition. These results revealed a significant difference in Group (PD vs HOA), however no significance regarding size of the bolus volume and interactional effects.

Post hoc F-tests were completed to further investigate the significant group effects on contraction amplitude and duration and to verify the lack of effect on bolus volume condition on contraction amplitude and duration. Further testing indicated a significant difference of group regarding contraction amplitude ratio, but no significant difference of group regarding contraction duration. Regarding bolus volume effects, findings indicated no significant differences between contraction amplitude or contraction duration across the three liquid bolus volumes. Results for post-hoc group effects are displayed in Table

3 and results for effect of bolus volumes on measures of contraction amplitude and duration are shown in Table 4.

Table 2. Effect of Group, Condition, Group X Condition

Effect	*Test*	*Value*	*P-value*
Group (PD vs HOA)	Pillai's Trace	.245	.000*
Condition (5ml, 10ml, 15ml)	Pillai's Trace	.055	.412
Group X Condition	Pillai's Trace	.005	.942

*Significant at 0.05

Table 3. Post-hoc Group Effects

Effect	*Test*	*Value*	*P-value*
Group (PD vs HOA) Amplitude Ratio	Post-hoc F-tests	F= 20.919	.000*
Group (PD vs HOA) Contraction Duration	Post-hoc F-tests	F=1.000	.321

*Significant at 0.05

Table 4. Effect of Bolus Volumes on Measures of Contraction Amplitude and Duration

Dependent Variable	***Condition (I)***	***Condition (J)***	***Mean Difference (I-J)***	***P-value***

Amplitude Ratio	5ml	10ml	-.068	.128
		15ml	-.076	.090
Amplitude Ratio	10ml	5ml	.068	.128
		15ml	-.008	.857
Amplitude Ratio	15ml	5ml	.076	.090
		10ml	.008	.857
Time Ratio	5ml	10ml	-.047	.608
		15ml	-.023	.803
Time Ratio	10ml	5ml	.047	.608
		15ml	.024	.792
Time Ratio	15ml	5ml	.023	.803
		10ml	-.024	.792

DISCUSSION

Summary of Findings

The purpose of this study was to investigate if (1) contraction amplitude and contraction duration of submandibular muscles during swallowing differ for individuals with PD and healthy controls, and (2) if bolus volume effected contraction amplitude and contraction duration of the submandibular muscles during swallowing differently for individuals with PD and healthy controls. The results from the MANOVA determined that 1) contraction amplitude of submandibular muscles differed for individuals with PD and healthy controls but contraction duration did not, and 2) bolus volume had no significant effect of contraction amplitude and contraction duration in either PD or HOA participants and 3) there is no group X condition interactional effect on either contraction amplitude or contraction duration.

Contraction Amplitude

Overall the contraction amplitude ratio was significantly different for people with PD when compared with healthy controls. People with PD used a greater percentage of their maximal swallow contraction force during their typical swallows (3ml, 5ml, 15ml) ($p < .000$). This finding suggested that participants with PD have a less efficient swallow than HOAs. It is hypothesized that our findings are linked to the increased rigidity in Parkinson's disease as described in a Videofluroscopic study by Robbins, Logemann, & Kirshner (1986). Additionally, the increased contraction amplitude ratios could be influenced by the impairments of motor amplitude scaling in PD. Together with previous research, findings of this study support a theory that rigidity and bradykinesia underlie the disordered oral and pharyngeal stages of swallowing in people with Parkinson's (Robbins, Logemann, & Kirshner, 1986).

Interestingly, participant reports on swallowing problems did not always match up with greater amplitude ratios as expected. The mean contraction amplitude ratio for HOAs was 0.47 (SD: 0.07) (Table 5). This meant HOAs were producing an average of 47% of their maximum swallow amplitude during their normal swallowing trials. The mean contraction amplitude ratio for participants with PD was 0.71 (SD: 0.17) (Table 5), revealing that participants with PD were using an average of 71% of their maximum swallow amplitude during their normal swallowing trials. Out of the total participants with PD, 50% (7/14) (Table 6) reported at least one s/s of swallowing problems. Of the 50%, a little less than half had a lower contraction amplitude ratio than the mean amplitude ratio for HOAs (.47). In other words, this implied that 42% of the participants who reported s/s had a more efficient swallow than the average swallow performance of

HOAs, as measured by the contraction amplitude ratio.

Furthermore, of the other 50% (7/14) (Table 6) that reported to have zero symptoms of swallowing problems, half had a greater contraction amplitude ratio than mean contraction amplitude ratio of the participants with PD (.71). This suggested 50% of the participants who related zero symptoms had a less efficient swallow than the average swallow performance of participants with PD. Our findings demonstrated and confirmed what Kalf and collogues discovered in their meta-analysis study completed in 2012. This study analyzed the prevalence of oropharyngeal dysphagia in people with PD. They found that only 1/3 of community-dwelling patients with PD reported s/s of dysphagia. However, when the community was objectively measured for dysphagia 4/5 of the patients were affected. This indicates that patients with PD do not always accurately report swallowing difficulties and that dysphagia is underreported. The clinical implication of this is that medical providers should take proactive clinical approaches towards treating dysphagia in this population (Kalf, de Swart, Bloem & Munneke, 2012).

Table 5. PD and HOA Contraction Amplitude Means and SDs

	Mean	SD
PD	0.71	0.177774725
HOA	0.47	0.072533748

Table 6. PD s/s of Swallow and Contraction Amplitude

s/s of swallowing issues reported	Amp Ratio
Yes	0.485919327
No	0.809082892
No	0.433049012
No	0.706314919
Yes	0.89823313
Yes	0.41351388
No	0.714407636

Yes	0.453818964
No	0.809863236
No	0.800880669
No	0.407574844
Yes	0.705415162
Yes	0.727321887
Yes	0.456091772

Contraction Duration

Overall contraction duration was calculated by the mean duration of three trials from each condition. The descriptive statistics suggested that people with PD swallowed faster than controls, however the difference was not statistically significant and no group differences for contraction duration could be noted. Although sEMG studies looking at swallowing duration are limited, previous studies that looked at bolus movement noted increased duration in the oral and pharyngeal swallowing stage for people with Parkinson's (Ertekin, et. al, 2002) (Nagaya, Kachi, Yamada & Igata, 1998). One sEMG study by WS Coriolano et al looked at the effects of bolus volume on swallowing duration in people with PD compared to normal adults. They reported increased swallowing duration in patients with PD compared to normals. However, the duration values reported in that study are judged to be outside of normal limits of what would be reasonable for pharyngeal stage swallow duration. Past studies have determined the average swallow time for the pharyngeal phase in normal individuals is less than 1.2 seconds (Hamlet, Muz, Patterson & Jones, 1989) (Dodds, Stewart, & Logemann, 1990). In WS Coriolano et al. study, the reported swallowing duration for normal participants ranged from 1.7-2.5 seconds and swallow duration for PD participants ranged from 2.2-4.0 seconds. These values are far greater than what is considered normal which questions

the significance of their results. Nevertheless, it is known that pharyngeal transit and reflex initiation time is significantly delayed in people with PD due to the bradykinesia present in the sequential muscle movements (Ertekin, et. al, 2002), which makes the lack of significant findings in this current study to be intriguing.

The lack of significant duration differences between PD and controls may be due to the severity of dysphagia in the participants with PD. Although 42% of the participants with PD reported one or more sign or symptoms (s/s) of swallowing difficultly, none of the participants had clinically been diagnosed with dysphagia from a medical healthcare professional. The participants were all concluded to have subclinical dysphagia because there was no definite medical diagnosis. Also, none of the participants with PD demonstrated overt s/s of dysphagia during their swallowing trials and were able to complete all 12 trials safely. Perhaps the degree of their swallowing difficulties was not severe enough to influence the duration of their swallowing. The participants with PD may have been able to compensate for the timing of their muscle contraction, even in the presence of having elevated amplitude. Moreover, the range in age of diagnosis of PD was 29 (Mean 5; SD: 7.13). However, one participant was a noted outlier, as he was diagnosed (dx) with PD 20 year greater than the next oldest dx participant. When this outlier was removed, the range decreased to 9 years (Mean: 5, SD: 2.4) representing a relatively newer dx PD group. The shorter dx period of our participant with PD may also have influenced the reduced severity of dysphagia. Increased severity of PD is likely to have greater threats to quality of life from PD-related dysphagia (Leow, Huckabee, Anderson, 2010). It is possible that the newer diagnosed participant group may have limited our study to people with subclinical dysphagia.

Effects of Bolus Volume on Contraction Amplitude and Duration

There was no influence on contraction amplitude and duration in regards to the different bolus volumes of 5ml, 10ml and 15ml given in this study. Past investigations reported effects of bolus volumes on the magnitude of the anterior and superior movement of the hyoid bone (Dodds et al., 1988) (Ueda et al., 2013). Conversely and similar to the findings from this current study, Zu et al. (2011) concluded that bolus volume had little to no effects on the extent of hyoid movement. Although bolus volume may not impact the contraction amplitude or duration, there has been promising evidence that bolus viscosity effects the hyoid movement (Zu et al., 2011) (Chi-Fishman & Sonies, 2002). Chi-Fishman & Sonies (2002) used ultrasonography to assess 31 healthy adults for effects of bolus viscosity on hyoid movements. She found that spoon-thick viscosity had the greatest amount of laryngeal displacement. Additionally, a study done by Ashida et al. (2010) analyzed the effects of bolus viscosity on the duration of the suprahyoid muscle activity through sEMG in 5 healthy young adults. This study assessed three different bolus textures defined by the amount of thickening agent, called Mousse-up (MU), added (3%, 6%, 9%). They discovered swallowing the 9% MU involved significantly longer duration than the 3% MU, concluding that the thicker bolus texture increases the duration of the sEMG swallow signal.

Limitations and Future directions

There are several limitations inherent in the methodology of this study that warrant guarded generalizations. First, although the same instrumentation and procedures were used in collecting recorded voice samples, patient's history of dysphagia was not

taken into consideration. The researchers did not consider severity or medical diagnosis of dysphagia as a criterion of the study and patient history of therapy of dysphagia was not considered. Additionally, all participants were able to take part of the study without overt sign/symptoms of dysphagia, which communicates that the severity of dysphagia, if present, wasn't severe enough to interfere with everyday swallowing. Interestingly, there was still a significant difference in contraction amplitude ratio. This could may mean the swallowing mechanism has notable differences in its efficiency of swallowing prior to impacting the participants' daily swallowing abilities. Second, the sample size was limited to only 14 individuals with PD and 10 HOAs. To confirm the findings of this study, a larger sample size should be considered. Future studies will obtain a larger sample size and better control for participant swallowing history through objective dysphagia screening measures like the Mann Assessment of Swallowing Ability (MASA) (Mann, 2002).

Considering future directions, our dataset manifested a similar trend for contraction duration in the descriptive statistics as past studies. Again, our contraction duration did not reach significance, however, descriptive statistics revealed *faster* contraction time in PD compared to HOAs. Like our study, Malloy et al. (2014) and Tawadros, et al. (2012) both reported no significance on overall sEMG durations during the pharyngeal stage of swallowing for participants with PD. Similarly, the descriptive statistics for Malloy et al. (2014) study indicated that trends in their data set suggested overall contraction duration was *shorter* in people with PD compared to control. People with PD exhibited faster contraction duration in the 5mL and 20mL conditions than controls (Malloy et al., 2014). As both studies utilized participants with subclinical

dysphagia, this may be an interesting area of future research. If people with PD who manifest subclinical dysphagia utilize involuntary compensatory strategies to swallow with greater contraction force, perhaps these same strategies influence contraction timing. When progression of the disease leads to clinical dysphagia, pharyngeal stage swallow timing could also be affected. This may support the theory of compensatory neuromuscular adaptions utilized by people with PD during earlier stages of disease severity.

CONCLUSION

Surface electromyography (sEMG) has been validated as a tool for measuring submandibular muscular activity associated with hyolaryngeal excursion during swallowing (Crary, Carnaby Mann, & Groher, 2006; Crary, Carnaby Mann, & Groher, 2007; Huckabee et al., 2005; Stepp, 2012; Wheeler-Hegland et al., 2008; Vaiman et al., 2004). Researchers utilized Surface Electromyography (sEMG) to noninvasively study the Duration and Amplitude of 24 participant's swallows (14 PDs & 10 HOAs). The goal of the study was to examine if bolus volume impacted the contraction amplitude and contraction duration for individuals with PD and healthy controls, and what difference if any were there between participants with PD and HOAs. Main findings from this study concluded contraction amplitude of submandibular muscles differed for individuals with PD and HOAs and bolus volume had no significant effect on contraction amplitude or contraction duration in both PD and HOA groups. Contraction duration was not found significantly different but descriptive statistics demonstrated a shorter duration than HOAs.

Dysphagia is highly prevalent in people with PD. This study gives us a better understanding about what is happening to the muscular physiology of the submandibular muscles. People with PD with subclinical dysphagia may be utilizing compensatory strategies to effect contraction timing. The findings from this study may support the theory of compensatory neuromuscular adaptions utilized by people with PD during the earlier stages of disease severity. Further studies should be completed to verify and expand on these findings.

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