SPEECH PROSODY IN PARKINSON'S DISEASE

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ABSTRACT

This study investigated the relationship between perceived speech severity and the speech prosody in speakers with Parkinson's Disease (PD). Recordings from 39 males and females with PD and 33 healthy older adult males were analyzed. Results showed that there was not a significant relationship between perceptual severity and frequency (p= .970) and amplitude (p= .15) modulation, but that there was a positive relationship between perceived severity and amplitude and frequency modulation (p= <.001). Independent samples t-tests showed that there was an effect of sex on frequency modulation in speakers with PD (p=.002) and a difference in frequency modulation between male speakers with PD and male control speakers (p= .023). However, there was not an effect of age of onset on frequency (p= .608) and amplitude (p=.627) modulation and there was not an effect of sex on amplitude (p= .140) modulation. Also, there was not a difference in amplitude modulation between male speakers with PD and males control speakers (p= .758). This study shows the importance of considering factors like sex when working on speech with people with PD, and supports the need for further research on the effect of these and other variables on speech in people with PD.

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INTRODUCTION

Parkinson's Disease (PD) is a neurodegenerative disease that causes complex biochemical changes resulting in some or many of the following symptoms: tremor, rigid muscle tone, slow movement, cognitive dysfunction, depression, and hallucinations, among others (Bartels & Leenders, 2009). James Parkinson was the first neurologist to describe the disease in 1817. He first called what we now know as PD the "Shaking Palsy" or "Paralysis Agitans" because of the tremors and weakened muscles he saw in his patients. In 1912, Friedrich Heinrich Lewy described the abnormal protein clusters, now named after him (Lewy bodies), found in the degenerating neurons of people with PD. By the 1950s the link between the loss of dopamine in the basal ganglia and PD was discovered. More recently, PD has been thought to follow a specific sequence, starting with neuron cell degeneration and loss in the vagus nerve and the olfactory bulbs and nucleus, then the locus coeruleus, followed by loss in the substantia nigra pars compacta, and finally the less vulnerable nuclei and cortical areas (Bartels & Leenders, 2009).

Behind Alzheimer's disease, PD is the second most occurring neurodegenerative disease. Specifically, PD has about a 1 to 2 out of 1000 prevalence rate (Bartels & Leenders, 2009). The prevalence of PD varies by geographic location, ethnicity, and gender amongst the population. A study completed on the prevalence of PD among American Indians and Alaskan natives found the overall prevalence rate was 144 per 100,000 persons. This rate increased with aging and was higher in men than in women (Gordon et al., 2012). Per a study on the prevalence rates of PD among different ages, the incidence rate rapidly increases after age 60, with only 4% of cases of PD in patients under the age of 50. In addition, the rate for men was 91% higher than that for women (Van Den Eeden et al., 2002).

The early classification system of neurological diseases and disorders was based primarily on symptoms (Goetz, 2011). Based on work by Charcot, who determined which signs and symptoms of neurological disorders were unique to PD, numerous studies were then launched to determine the cause of PD. In the 1920s, studies completed on PD by professors and students at the French Neurologic School showed that damage to the substantia nigra and other parts of the midbrain were the cause of the motor impairments of PD. Additional studies on the involvement of the neural circuits in the development of PD showed that the key nuclei associated with the onset of the disease were located in the substantia nigra, globus pallidus, caudate nucleus, and the putamen, or striatum, parts of the brain (Goetz, 2011). Since these early theories on the cause of PD, new technologies have emerged and advanced studies have been performed, leading to the current, and still evolving, theories on the cause of PD.

CLINICAL CHARACTERISTICS

Although the studies and information we have today on the signs and symptoms of PD are rather elaborate, initially this information was less accurate. In James Parkinson's essay on Shaking Palsy, the definition of this disorder was described as, "Involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported; with a propensity to bend the trunk forwards, and to pass from a walking to a running pace: the senses and intellects being uninjured" (Parkinson, 1817). Initially, one with Shaking Palsy may experience signs of weakness, specifically in the hands and arms. As the weakness progresses, one is found to have a less upright body position that can be seen when sitting or standing, but weakness is most easily seen when one is walking. In addition, as the disease progresses, the tremors in one's body cause sleepless nights and violent outbreaks. As one hits the last stage of the Shaking Palsy,

one's trunk is permanently bowed, one's muscular power has diminished, and one's tremors continue to be violent. In terms of other bodily functions, one's words are no longer intelligible and one's ability to eat has diminished by the end stages of Shaking Palsy (Parkinson, 1817). Because of the research and studies completed on Parkinson's Disease between 1817 and now, we have more information on the signs and symptoms associated with the disease.

There are four main motor symptoms of PD: tremors, rigidity, bradykinesia, and postural instability (Jankovic, 2007 & Politis et al., 2010). A rest tremor is the most common and most easily recognized symptom of PD. Tremors can occur at a frequency as low as 4 Hz and as high as 6 Hz and usually occur in the distal part of a limb. Rigidity is an increased resistance in muscles, specifically during the passive movement of a limb. Rigidity can also cause additional stiffness in the joints, creating pain for the patient. Bradykinesia is the slowing of bodily movements. Postural instability usually occurs in the later stages of PD, after the presence of other symptoms. One of the biggest risks associated with postural instability is falling. When one falls he or she risks fracturing or breaking a hip, arm, shoulder, or even back, causing more physical pain and instability (Jankovic, 2007). In addition to these common motor symptoms, there are also multiple non-motor symptoms that can be present in people with PD.

Three of the most common non-motor symptoms associated with PD are autonomic dysfunction, cognitive or neurobehavioral disorders, and sensory abnormalities (Jankovic, 2007). Some common autonomic dysfunctions include orthostatic hypotension, sweating dysfunction, and erectile dysfunction (Jankovic, 2007 & Politis et al., 2010). Per the results of The Sydney Multicenter Study of PD, "84% of patients showed cognitive decline and 48% met the diagnostic criteria for dementia after 15 years of follow-up" (Jankovic, 2007). In addition, depression, apathy, anxiety, and hallucinations are common neuropsychiatric symptoms in people with PD

(Jankovic, 2007). Some of the common sensory abnormalities experienced by people with PD include olfactory dysfunction, pain, paresthesia, akathisia, oral pain, and genital pain (Jankovic, 2007 & Politis et al., 2010).

It is difficult to define the differential effects of early onset of PD compared to late onset of PD because of the discrepancy in what age ranges are considered early onset versus late onset of PD (Wickremaratchi, Ben-Shlomo &Morris, 2009 and Wickremaratchi et al., 2011). Typically, early onset of PD is considered to be onset before 40 years of age and late onset of PD is considered to be onset after 70 years of age (Wickremaratchi, Ben-Shlomo &Morris, 2009). However, despite the age range differences, age of onset has been proven to impact prosody in those with PD (Wickremaratchi, Ben-Shlomo & Morris, 2009 & Wickremaratchi et al., 2011). People with early onset of PD tend to have slower disease progression, dystonia at onset, and early dyskinesia (Wickremaratchi, Ben-Shlomo & Morris, 2009).

COMMUNICATION IMPAIRMENTS IN PD

About 50 to 90% of those diagnosed with idiopathic PD will develop a speech and / or voice disorder throughout the course of their illness (Ramig, Fox & Sapir, 2004). Specifically, about 89% experience voice disorders, 45% experience articulatory impairment, and 20% experience problems with fluency (Ho et al., 1999). The most common signs of these disorders include reduced loudness, reduced prosodic pitch, hoarseness of voice, and imprecise articulation (Ramig, Fox & Sapir, 2004). Speech and voice disorders associated with PD have been collectively termed hypokinetic dysarthria, which is usually characterized by reduced intelligibility of speech. Typically, voice disorders occur first with the addition of speech disorders occurring at a later onset (Ramig, Fox, & Sapir, 2004 & Ho et al., 1999).

Acoustic analyses of voice production have shown that the voice characteristics of individuals match the auditory-perceptual descriptions by listeners. Studies in the areas of sound pressure level, fundamental frequency variability, and short-term and long-term phonatory stability have shown results matching the signs and symptoms of voice and speech disorders in individuals with PD (Ramig, Fox, & Sapir, 2004). In terms of sound pressure level, individuals with PD were found to have a 40% decrease in loudness when compared to individuals without PD in the same age range. In addition to a lower sound pressure level, individuals with PD also had lower fundamental frequency variability compared to a non-disordered group. Lower fundamental frequency variation is what leads to one's voice sounding monotone. People with PD also showed an instability in both short-term and long-term phonatory stability. Short-term phonatory instability is assessed with classic measures such as jitter and shimmer, and is associated with perceptions of hoarse and breathy voice. Long-term phonatory instability can be associated with vocal tremor (Ramig, Fox, & Sapir, 2004).

One study on 200 patients with PD focused on classifying patients' overall speech function into five categories, based on severity (Ho et al., 1999). The five categories were: level 5 (80-100% functional production), level 4 (60-80% functional production), level 3 (40-60% functional production), level 2 (20-40% functional production), and level 1 (0-20% functional production). Voice, fluency, and articulation were measured when determining one's overall speech function. Of the 200 patients with PD sampled, 26% were not impaired, 18% were mildly impaired, 34% were moderately impaired, 19% were severely impaired, and 3% were profoundly impaired. In addition, only 1% had a purely articulation impairment and only 3.5% had a purely speech fluency impairment. These percentages show that articulation disorders rarely exist alone in patients with PD and confirm that voice impairments are the dominant disorder in people with

PD (Ho et al., 1999). Voice disorders in people with PD are initially characterized by a change in vocal quality and later lead to a reduction in speech volume (Ho et al., 1999; Ramig, Fox, & Sapir, 2004).

Prosodic features of speech include emphasis on syllables, change in tempo, or changes in pitch and intonation (Lloyd, 1999). These speech features help people convey semantic and paralinguistic information. Individuals with PD often lack normal prosodic variation, and their speech is often called dysprosodic. The speech of those with PD is described as flat, lacking rhythm and melody, and impaired in timing. The lack of these speech characteristics has been tied to dopamine depletion in the basal ganglia of the brain in people with PD. Dopamine depletion in the basal ganglia affects one's movements and therefore can disrupt speech (Lloyd, 1999).

Studies performed on prosody in people with PD have had mixed results. In terms of looking at prosody comprehension, more studies have found that people with PD have a deficit in prosody comprehension compared to those without PD (Lloyd, 1999). However, there have been a few studies that resulted in no evidence of a deficit in prosody comprehension in people with PD. In addition, multiple studies have reported that people with PD were poor at identifying and comprehending prosody, but that they were able to differentiate between sentence boundaries (Lloyd, 1999 & Darkins, Fromkin & Benson, 1988). The variation in results on prosody tests can be explained by the idea that only some people with PD are impaired in prosody, and the level of impairment is different on a case by case basis. Because there is significant evidence to support the idea that individuals with PD have impairments in prosodic comprehension and prosodic production, it can be theorized that prosodic impairments may be caused by a cognitive and motor deficit (Lloyd, 1999).

Another impairment in prosody associated with PD is the perception of emotional prosody. An impairment in the perception of emotional prosody could be caused by two things: a general cognitive deficit or an acoustic processing deficit (Breitenstein et al., 2001). It could be caused by a general cognitive deficit because people with PD often present selective dysfunctions of the central executive component of the working memory. On the other hand, an acoustic processing deficit could be caused by an inefficiency of the acoustic parameters needed for prosodic classifications in speech.

A study on early-PD, moderate-PD, and normal controls was conducted to look at the presence of emotional prosody deficits in people with PD (Breitenstein et al., 2001). The people with moderate-PD were significantly impaired when it came to tasks that involved central executive functions like recalling the final words of someone talking. The performance of people with early-PD in the executive function tasks did not significantly differ from the control group of people who do not have PD. Overall, the results of this study showed that people with PD do not suffer from a generalized emotional processing deficit, but rather from a deficit in the nonverbal emotional domain (Breitenstein et al., 2001).

In another study looking at the possibility of an acoustic processing deficit, individuals with early-PD, moderate-PD, and controls were tested on two important acoustic parameters: frequency variability and duration (Breitenstein et al., 2001). The early-PD group and moderate-PD group results were combined due to the lack of differences between the two groups. The combined PD group showed a deficit in differentiating between emotional categories at fast and slow speech rates compared to controls. People with PD were significantly more likely to identify all of the fast and slow emotional stimuli as “frightened” than the control group people. In addition, the PD group of people were a lot less efficient then the control group of people in

using the temporal changes as a cue to identify the vocal emotion. Overall, the findings of this study support the idea that people with PD often have a deficit in speech time processing, particularly for the emotions, happy and sad (Breitenstein et al., 2001).

STATEMENT OF THE PROBLEM

It has been demonstrated that speech prosody is impaired in speakers with PD. This impairment includes changes in the frequency and amplitude variations which naturally occur in connected speech. While studies have shown that speakers with PD manifest problems perceiving prosodic features in speech, the association between their own speech prosody and the perceived severity of their own speech impairment is not well understood. The purpose of this study was to investigate the relationship between perceived speech severity and the prosodic manipulation of frequency and amplitude within connected speech by speakers with PD. In addition, this study investigated the effect of disease onset age and sex on frequency and intensity variability within connected speech of speakers with PD. The specific research questions addressed in this study were:

1. What is the degree of association between perceived speech severity and frequency modulation and amplitude modulation within connected speech of speakers with PD?
2. Is there an effect of age of onset on frequency modulation and amplitude modulation within connected speech of speakers with PD?
3. Do male PD speakers exhibit different frequency and amplitude modulation than female PD speakers?
4. Do male PD speakers exhibit different frequency and amplitude modulation than male control speakers?

Hypotheses were as follows:

1. Speakers with PD perceived as manifesting greater speech severity will exhibit decreased frequency and amplitude modulation in connected speech as evidenced by a strong correlation coefficient.

2. Speakers diagnosed at a younger age will manifest greater frequency and amplitude modulation in connected speech than those diagnosed at an older age.
3. Male speakers with PD will manifest greater frequency and amplitude modulation in connected speech than female speakers with PD.
4. Male control speakers will manifest greater frequency and amplitude modulation in connected speech than male speakers with PD.

METHODOLOGY

Participants

Data for this study was obtained from existing audio recordings and demographic data of speakers with PD. These individuals were recruited and recorded as part of a previous research study at TCU. Inclusion criteria for those participants required diagnosis of idiopathic PD by a neurologist but with absence of any other diagnosed neurological disease. Existing recordings from 39 speakers with PD were used. Recordings from 33 healthy older adult males, acquired during a previous study, were also available for analysis. To compare prosody as a function of PD versus no PD, new acoustic analyses of those recordings was conducted.

For the PD participants, perceptual assessment of speech severity was conducted by recruiting three naïve listeners. The listeners were recruited from the graduate population of speech language pathology majors at TCU. Listeners were required to pass a hearing screening with thresholds at 25dB for frequencies between 500Hz to 8000Hz.

Instruments /Tools

The Computerized Speech Lab (CSL - Pentax Medical, Montvale, NJ) was used for acoustic analyses and for presentation of the perceptual stimuli for the perceptual rating task. The Real-Time Pitch software of the CSL was used to analyze existing recordings of PD speakers to acquire measurements of frequency and amplitude modulation. The core CSL software presented audio recordings to listeners via a connected speaker.

Procedures

After consenting, perceptual judges were instructed to listen and rate the speech severity of recorded sentences. A magnitude estimation task based on the procedures of Weismer (2002) was implemented. Prior to the task, judges were instructed to consider their ratings in the context of perceived prosody. The magnitude estimation task utilized a perceptual anchor consisting of a speaker with PD manifesting a moderate degree of prosodic impairment. This anchor was chosen by the faculty research mentor and a graduate assistant. The severity of the perceptual anchor was assigned a rating of 100 and represented a moderate degree of severity. Judges were asked to rank the rest of the speakers based on this scale. (E.g. If the speaker was twice as severe as the anchor, they were to give a score of 200; one-half severe, a rating of 50.) The 2nd and 3rd sentences of the Rainbow Passage from each recording were played in randomized order with the anchor played approximately every five samples for perceptual calibration (Fairbanks 1960).

For prosodic analysis of the recordings, the 2nd and 3rd sentences of the Rainbow Passage from each recording were analyzed using the Real-Time Pitch software. The software program automatically calculated the coefficient of fundamental frequency variation (FoV), a measure of frequency modulation that controls for natural inter-speaker differences in fundamental frequency, and the standard deviation of vocal amplitude (dBV), a measurement of amplitude modulation.

Analyses

The dependent variables in this study consisted of (a) speech severity ratings, (b) frequency modulation (FoV), and (c) amplitude modulation (dBV). The independent variables included (a) age of onset and (b) PD participant sex, and (c) PD vs. Control group. To answer the first research question, correlation analyses were applied to the speech severity ratings and (a) FoV data along with the (b) dBV data. To answer the second research question, the participant pool of 39 recorded speakers were separated into those with diagnosed onset at age 68 years or less, and those with diagnosed onset at age greater than 68 years. The existing recordings represented a natural separation at this age cutoff, with 19 speakers with PD at age 68 or less and 20 speakers with PD at age greater than 68. Separate independent samples t-tests were applied to the FoV and dBV data, with age of onset as the independent variable. To investigate the effect of sex on prosody in male and female speakers, an independent samples t-test was applied to the FoV and dBV data, with participant sex as the independent variable. To investigate the effect of PD on prosody in male speakers, independent samples t-tests were applied to the FoV and dBV data in the male PD and male control participants.

RESULTS

Perceptual severity ratings for prosody were available for 28 out of the 39 participants with PD. The average severity rating for these 28 speakers was 94.6 (standard deviation = 50.3, where a rating of 100 corresponded to a moderate degree of severity). The correlation coefficient comparing perceptual severity to vFo was non-significant ($r = - 0.008$, $p = 0.970$), as was the relationship between severity ratings and vAm ($r = 0.28$, $p = 0.15$). Although not a research question of this study there was a significant positive relationship between vFo and vAm ($r = 0.57$, $p =$ less than .001).

Table 1 displays the effect of age of onset on frequency modulation (vFo) and amplitude modulation (vAm) in males and females with PD. Males and females who were younger than 68 years old at age of onset were placed in the younger age of onset group, and males and females who were 68 or older at age of onset were placed in the older age of onset group. The mean age of onset for the younger group was approximately 60 years old while the mean age of onset for the older group was approximately 75 years old. The vFo for males and females with PD in the younger group was 19.47% with a standard deviation of 8.1%. The vFo for males and females with PD in the older group was 18.25% with a standard deviation of 6.1%. When looking at the variance of amplitude, the younger group had an average vAm of 8.71% dBV with a standard deviation of 1.3%. The older group had an average vAm of 9.0% dBV with a standard deviation of 2.1%.

Table 1: Results for vFo and vAm of males and females with PD divided by age of onset.

Age of Onset	vFo	Std. Deviation	vAm	Std. Deviation
Younger PD (<68 years old at age of onset)	**.1947**	**.0816**	**.0871**	**.0136**
Older PD (68 years old or older at age of onset)	**.1825**	**.0616**	**.0900**	**.0213**

Table 2 shows the effect of sex on vFo and vAm in males and females with PD. The average age of males with PD was approximately 67 years old and the average age of females with PD was approximately 71 years old. Males had an average vFo of 21.45% with a standard deviation of 7.5%. On the other hand, females had an average vFo 14.93% with a standard deviation of 3.8%. In terms of variance of amplitude, males had an average vAm of 9.23% dBV with a standard deviation of 1.9%. The females had an average vAm of 8.33% dBV with a standard deviation of 1.4%.

Table 2: Results for vFo and vAm of people with PD divided by sex.

Sex	vFo	Std. Deviation	vAm	Std. Deviation
Male PD	**.2145**	**.07595**	**.0923**	**.01950**
Female PD	**.1493**	**.03863**	**.0833**	**.01447**

Table 3 shows the effect of PD on vFo and vAm in PD and control males. The average age of males with PD was approximately 67 years old and the average age of the males in the control group was about 64 years old. The males with PD had an average vFo of 21.45% with a standard deviation of 7.6%. The males in the control group had an average vFo of 26.76% with a standard deviation of 8.5%. After testing variance of amplitude, the males with PD had an average vAm of 9.23% dBV with a standard deviation of 1.9%. The males in the control group had an average vAm of 9.36% dBV with a standard deviation of 1.3%.

Table 3: Results for vFo and vAm of males divided by those with PD and those without PD.

Group	vFo (Hz)	Std. Deviation	vAm (dB)	Std. Deviation
Control Group	**.2676**	**.08592**	**.0936**	**.01319**
Male PD Group	**.2145**	**.0760**	**.0923**	**.01950**

When the independent samples t-test was applied to the vFo and vAm data, with age of onset as the independent variable, the p-value for vFo was 0.608 and the p-value for vAm was 0.627. Because both p-values are greater than the alpha level of 0.05, the null hypothesis could not be rejected and there was not an effect of age of onset on vFo modulation and vAm modulation in people with PD.

When the independent samples t-test was applied to the vFo and vAm data, with sex as the independent variable, the p-value for vFo was 0.002 and the p-value for vAm was 0.140. Because the p-value for vFo (0.002) was less than the alpha level of 0.05, the null hypothesis could be rejected and there was an effect of sex on vFo modulation in people with PD. The p-value for vAm (0.140) was greater than the alpha level of 0.05 so the null hypothesis could not be rejected and there was not an effect of sex on vAm modulation in people with PD.

When the independent samples t-test was applied to the vFo and vAm data, with PD as the independent variable (PD male vs. Control), the p-value for vFo was 0.023 and the p-value for vAm was 0.758. Because the p-value for vFo (0.023) was less than the alpha level of 0.05, the null hypothesis could be rejected and there was an effect of PD on vFo modulation in males. The p-value for vAm (0.758) was greater than the alpha level of 0.05, so the null hypothesis could not be rejected and there was not an effect of PD on vAm modulation in males.

DISCUSSION

The purpose of this study was to investigate the relationship between perceived speech severity and the prosodic manipulation of frequency and amplitude within connected speech by speakers with PD. In addition, this study investigated the effect of disease onset age and the effect of sex on frequency and intensity variability within connected speech of speakers with PD.

The first research question asked: what was the degree of association between perceived speech severity and frequency modulation and amplitude modulation within connected speech of speakers with PD? Results indicated that there was not a significant relationship between perceptual severity and frequency modulation or amplitude modulation. Although not a specific question of this research study, results did suggest that there was a positive relationship between perceived severity and amplitude and frequency modulation, meaning that listeners who perceived a PD speaker as having lower amplitude modulation variability were likely to also perceive that the PD speaker's frequency modulation had lower variability or vice versa.

It is unclear why no relationship between the primary dependent variables (vFo and vAm) and perceptual speech severity was found. One explanation may be that perceptual judges focused on speech characteristics other than frequency and amplitude modulation when making

judgements. Another explanation may be that perceptual judges varied amongst themselves in how high of a rating they gave the speech samples which they thought were the most severe or how low of a rating they gave the speech samples they thought were the least severe. A rating of 100 was used to represent moderate severity and was based on an anchor speaker; however, no limit was put on how high or low the perceptual judges could rate the speakers in the group.

The second research question asked, was there an effect of age of onset on frequency modulation and amplitude modulation within connected speech of speakers with PD? Results indicated that there was no effect of age of onset on frequency or amplitude variability in male and female speakers with PD. This means that people with PD diagnosed at a younger age (68 years old or younger) and people with PD diagnosed at an older age (older than 68 years old) do not differ in their frequency and amplitude modulation.

It is possible that no effect of age of onset was found because speakers with PD in both younger and older groups manifested varied disease durations. Although the literature has been mixed, some studies have reported a stronger association between disease duration (e.g., time post-onset) and speech impairment than other patient-specific characteristics (Majdinasab et al., 2016). Another reason could be that the cut-off age of 68 was not high or low enough of an age to divide the group of PD speakers into early and late onset groups. The literature has shown that studies vary in the age ranges of when looking at early versus late onset of PD. Some studies consider an age of 60 or earlier to be the cut-off age between early and late onset of PD or divide ages into 3 groups (Diamond et al., 1989).

The third research question asked: did male PD speakers exhibit different frequency and amplitude modulation than female PD speakers? Results indicated that there was an effect of sex on frequency modulation; specifically, females with PD had less variation in frequency

modulation than males with PD. However, results indicated that there was not an effect of sex on amplitude modulation, meaning that there was not a significant difference in a male speaker with PD's amplitude modulation when compared to a female speaker with PD's amplitude modulation.

The finding that females with PD exhibited less frequency modulation than males with PD is novel. The literature is conflicting on this result, some studies showing an effect of gender on frequency and other voice characteristics in people with PD and some not showing an effect of gender on voice. A study on voice characteristics in PD showed that females with PD in the later stages of the disease had more limited pitch variability than females in the control group (Holmes et al., 1999). However, in another study on gender-related dysprosody, the female PD group showed a significant decrease in frequency standard deviation, frequency variance and pitch variation when compared to the control group, but no difference in mean frequency (Skodda et al., 2011).

The fourth research question asked: did male PD speakers exhibit different frequency and amplitude modulation than male control speakers? Results indicated that there was a difference between male PD speakers and male control speakers in frequency modulation. Male control speakers had more variance in frequency modulation than male PD speakers. However, results indicated that there was not a difference in amplitude modulation between male PD speakers and male control speakers.

This study's result that male PD speakers had less frequency modulation than male control speakers matches with previous studies. In a study on the progression of dysprosody over time amongst speakers with PD, frequency variation was significantly lower in male speakers with PD when compared to male control speakers, both in the initial testing and the testing after

7 months (Skodda & Rinsche, 2009). In addition, a study on gender-related patterns of dysprosody in PD showed that mean frequency and frequency standard deviation were significantly reduced in male PD speakers compared to male control speakers (Skodda et al., 2011).

LIMITATIONS OF THE STUDY

Although this study did successfully answer all research questions asked, it still had limitations. Because of location and convenience, participants with PD and participants in the control group were recruited for this study from the Dallas / Fort Worth area. Also, due to time limitations on this study, a female control group was unable to be obtained. A larger group of participants from both genders and from a variety of locations would further improve the accuracy and external validity of this study. In addition, this study did not control for or analyze for disease durations. Some studies have found that disease duration is related to speech impairment in PD, and future studies will need to investigate if prosody is related to that domain.

CONCLUSION

This study looked at the effect of PD on frequency and amplitude modulation and whether or not sex or age of onset affected frequency and amplitude modulation in speakers with PD. Data indicated that males with PD had reduced frequency modulation when compared to males in the control group and that females with PD had reduced frequency modulation when compared to males with PD. Data did not show that speakers with PD perceived as manifesting greater speech severity exhibited decreased frequency and amplitude modulation or that speakers at a younger age manifested greater frequency and amplitude modulation, as originally hypothesized. Future studies can expand on this data by looking at the frequency and amplitude modulation of different subgroups within the population including, but not limited to, groups based on age of onset or sex.

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