Correlational Analysis Between Speech and Gaitt in Parkinson's Disease

by

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ABSTRACT

Parkinson's Disease is one of the most complicated and abundant neurodegenerative diseases in the world. Previous analysis of Parkinson's disease has identified both speech and gait deficits throughout progression of the disease. There has been minimal research looking into the correlation between both the speech and gait deficits in those diagnosed with Parkinson's. There is high indication that there is a correlation between the two given the similar pathology and origins of both deficits. This exploratory study aims to establish correlation between both the gait and speech deficits in those diagnosed with Parkinson's disease. Using previously identified motor and speech measurements and tasks, I conducted a correlational study of individuals with Parkinson's disease at baseline. There were correlations between multiple speech and gait variability outcomes. The expected correlations ranged from average harmonics-to-noise ratio values against anticipatory postural adjustments-lateral peak distance to average shimmer values against anticipatory postural adjustments-lateral peak distance. There were also unexpected outcomes that ranged from F2 variability against the average number of steps in a turn to intensity variability against step duration variability. I also analyzed the speech changes over 1 year as a secondary outcome of the study. Finally, I found that averages and variabilities increased over 1 year regarding speech primary outcomes. This study serves as a basis for further treatment that may be able to simultaneously treat both speech and gait deficits in those diagnosed with Parkinson's. The exploratory study also indicates multiple targets for further investigation to better understand cohesive and compensatory mechanisms.

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Introduction

Pd Neuropathology and Background

Basal ganglia dysfunction can disrupt motor control and actions, especially in both speech and gait. Dysfunction in the basal ganglia is described as the key mechanism behind automatic execution of learnt motor actions as well as inducing Parkinsonian motor symptoms[1].

Previous definitions of Parkinson's disease have been centered around signs of motor dysfunction, such as bradykinesia or hypokinesia, postural instability, or resting tremor. There are familial forms of Parkinson disease as well as signs occurring following infection, intoxication, or head trauma. Following Lewy body pathology, protein $\boldsymbol{\alpha}$-synuclein deposits have previously been identified in vulnerable neuronal types and cortical areas creating the abnormal buildup of these protein masses, associated with the signs and symptoms of Parkinsons' disease[2]. These protein deposits have been associated with muscle rigidity, shuffling walk, bradykinetic, or hypokinetic movements. This pathology has also been known to cause cognitive problems, such as memory or attention difficulties[3]. As seen in Table 1, progression of Parkinson's disease has been described in stages, specifically 6 according to Braak, with each stage affecting either sensory or motor systems. Motor symptoms are primarily affected in the later stages of Parkinson's disease, specifically stages 5 and 6 as the Lewy pathology compounded with Lewy neurites and Lewy bodies affects the autonomic, limbic, and somatomotor symptoms within the neocortex and basal ganglia[2].

Sensory centers	Olfactory: early and severe involvement Nociceptive: early involvement Somatosensory: mostly intact Viscerosensory: mostly intact Auditory and visual: uninvolved
Motor centers	Visceromotor: early and severe involvement Somatomotor: partial involvement Limbic: severe involvement
Stage 1	Anterior olfactory nucleus Olfactory bulb, olfactory tract Dorsal motor nucleus of the vagal nerve Intermediate reticular zone
Stage 2	(Plexuses of Meissner and Auerbach) Lower raphe nuclei Magnocellular reticular nuclei Coeruleus-subcoeruleus complex
Stage 3	(Spinal cord lamina I) Central subnucleus of the amygdala Olfactory tubercle, piriform cortex (olfactory system) Periamygdalear cortex (olfactory system) Medial entorhinal region (olfactory system) Substantia nigra, pars compacta Paranigral nucleus
Stage 4	Interstitial nucleus of the terminal stria Cortical and basolateral amygdala Thalamic intralaminar nuclei Thalamic midline nuclei Anteromedial temporal mesocortex Ammon's horn, second sector (CA2) Insular and subgenual cortex Anterior cingulate cortex Ventral claustrum

Stage 5	High order sensory association neocortex Prefrontal neocortex Entorhinal region, CA1 and CA3 sectors First order sensory association neocortex Premotor neocortex Primary sensory areas
Stage 6	Primary motor field

Table 1. This table shows the different stages identified within the progression of what was defined as sporadic Parkinson's disease.

Gait in Parkinson's

In the simplest form, gait is initiated in the spinal cord where central pattern generators (CpG's) induce rhythmic activation of the complementary flexor and extensor muscles. At a more in depth level, the output targets of the basal ganglia that contribute significantly to the CpG circuits are the mesencephalic locomotor region (MLR) which in turn contains the pedunculopontine nucleus. All of these lower-order processing centers work in a highly connected state to create the automaticity of gait. Secondary to the primary motor cortex in initiating gait, areas such as the supplementary motor area, premotor cortex, and posterior parietal cortex are also involved in the organization of movement. More specifically, the basal ganglia circuitry assists with sensory information integration to the primary motor cortex via the thalamus, which has been implicated in automatic execution of gait. Measurable aberrations of gait in individuals have previously been defined terms of changes to a number of conceptual moments. The first concept represents mean values, such as changes in gait speed and stride length. The second concept is defined as variability in relation to overall changes in stride dynamics(i.e. gait speed, increased cadence, turn velocity, etc.) with previous studies

showing a positive correlation between variability of gait on a "stride-to-stride" basis and Parkinson's disease progression[4]. Previous motor signs and symptoms of Parkinson's disease have included rigidity, decreased gait speed, increased truncal instability, and increased postural instability, all of these affecting normal locomotion and gait.

Postural Instability/Gait Difficulty Phenotype

Given the symptoms of postural instability, one of the major phenotypes of Parkinson's disease is postural instability/gait difficulty (PIGD). This has been one of the cornerstone phenotypes to be involved in studying gait dysfunctions and also their relation to other motor symptoms. The alternative phenotype identified includes tremor dominant, in which a resting tremor is the dominant sign. Both phenotypes have been used in previous studies for evaluation of correlation to speech symptoms.

Freezing of Gait

Dysfunction of motor symptoms can progress to even more severe abnormalities such as freezing of gait, or FoG. Previously characterized as brief episodic absence or reduction in initiation of gait, typically, however not exclusively, occurring following festination[5]. Multiple indications are used to identify FoG episodes including:

1. The foot not leaving or barely coming off the ground.
2. Alternating trembling of lower extremities at approximately 3-8 Hz.
3. Increased cadence in walking, typically occurring prior to the FoG episode.

4. A feeling of feet stuck like glue to the floor resulting in freezing.
5. Episodes are consistently preceded by prior cues
6. It can be asymmetrical affecting only one foot, or expressing difficulty turning a particular direction

Previous structural imaging have shown decreased gray matter tracts, or gray matter atrophy, in deeper posterior cortical regions and brain areas suggestive of executive-visuospatial dysfunction and in a small portion of MLR and thalamus[6]. This offers a closer look into the decreased gait actions of Parkinson's.

Dysrhythmia of Gait

Parkinson's has previously been described with a dysfunction in a rhythmic component, commonly manifesting with "dysrhythmia" of motor movements. Symptoms correlate with the frontal and striatal regions necessary for optimal motor timing of movements. Described early in Parkinson's disease, patients are typically unable to coordinate alternating motor movements and experience more significant manifestations as the disease progresses. Previous studies have shown such manifestations in the motor subtypes of Parkinson's disease such as postural instability gait disorder[7].

Treatment

Quantitative measurement techniques range from wearable markers(opals along the joints to measure and outline movement via computerized program) to laboratory gait analysis (complex program environments dedicated to optical, contact pressure, and electromyogram measurements).

Physical therapy methods involve multiple different aspects addressing motor skills and gait via cueing strategies, motor-strategy training, and resistance/aerobic exercises. Cognitively, reducing the amount of anxiety that the individual has is important due to the correlation with falls, and cognitive training addressing frontal/temporal lobe(addressing working memory, attention-shifting, and executive function).

Levodopa for Gait Dysfunction

Main function of pharmacological treatment is to improve symptoms of bradykinesia and rigidity, and the most efficient alleviation has been through Levodopa treatments. The concept behind pharmacological treatment is replacing dopamine deficiency in the synaptic cleft of the nigrostriatal pathway communicating voluntary movements to the basal ganglia. The main effects of dopaminergic replacement therapy are less straightforward in regards to gait dysfunction. Previous studies have shown consistent improvement in gait velocity and stride length across studies while in the ON(participant has been taking regular dose of Levodopa, consistent with efficient dose) state using levodopa compared to the OFF(participant has not been taking regular dose consistent with efficient dose) state however, stride duration and cadence have been shown to be unaffected[5].

Deep Brain Stimulation of the Subthalamic Nucleus (STN-DBS) Treatments for Gait Dysfunction

Deep brain stimulation has been gradually introduced and increasing in application as a therapy to improve the “cardinal” symptoms of Parkinson’s(i.e. bradykinesia, tremor, and rigidity). Routinely, the target area for stimulation has been the

subthalamic nucleus which has shown mixed results. Previous studies have shown anywhere from no improvement in gait parameters to a degree of improvement in those parameters such as FoG[8]. There have also been improvements in gait speed associated with STN-DBS[9].

Speech in Parkinson's

The pathophysiology of speech dysarthria is complex, including dopaminergic deficiency, basal ganglia dysfunction, and hypokinesia, bradykinesia manifestation. Dopamine deficiency does not fully explain the dysfunction of the muscle groups involved in speech[10].

Dysarthria is able to affect all subsystems of speech production with increase in severity in the later stages. Articulation is decreased in individuals with PD, correlating with reduced intelligibility. The articulatory parameters of speech have previously been evaluated for bradykinesia and hypokinesia by looking at the upper lip, lower lip and jaw, velar, and thyroarytenoid muscle[11]. Studies utilizing phonatory measures report distorted vowel production with reduced formant range, shallower formant slopes (i.e. rate of formant change), restricted acoustic vowel space, and longer voice onset times (VOT). Previous studies to establish the basic parameters of articulators in Parkinson's have shown decreased amplitude, decreased velocity of opening and closing of the lower lip, and again decreased F2 formant slopes[11]. There has been previous evidence of reduced velocity and amplitude as a result of improper function of the actual articulators (i.e. lip, tongue, and jaw movements) and rigidity of the vocal tract. Studies show

distorted vowel production, shallower formant slopes, restricted acoustic vowel space, and longer voice onset times.

Prosodic deficits have been previously defined as variable speech timing, reduction in intonation, altered speech intensity, and disfluencies[10]. Previous examinations of F0 intensity and variability have been decreased in individuals diagnosed with Parkinson's Disease, more specifically, when completing sentence production tasks. Overall, individuals diagnosed with Parkinson's Disease have shown a decrease in articulation and prosody.

Dysrhythmia of Speech

Previous papers have examined the potential of disruption in rhythmic fluency of speech of those diagnosed with Parkinson's. Relative analysis of this subject has shown presence of FoG was associated with older age and more severe rate/prosody impairment. Linear regression analysis showed more severe FoG correlates with more impaired rate/prosody of speech. Ultimately, data suggests a deficit in the rhythmic component of speech is more common in those patients with FoG. Association of FoG and speech impairments was influenced by older age and disease stage (severity) but not disease duration[12]. Overall, disproving the complete conclusion that the disruptions in speech are strictly a rhythmic component.

Treatment for Speech Deficits

Deep Brain Stimulation of the Subthalamic Nucleus (STN-DBS) Treatments for Speech Dysfunction

Subthalamic nucleus deep brain stimulation (STN-DBS) seems to show alleviation of symptoms of speech stuttering. Severity of dysarthria prior to any treatment are critical determinants in this form of treatment. In contrast, STN-DBS has previously shown deterioration in subjective ratings. For example, a previous study, done by Astrom and colleagues, evaluating speech under high amplitude stimulation described the participants as breathy and hyper functional resulting in imprecise articulation[13]. There have also been previous indications that while the speech deficits may be reduced, gait dysfunction may increase and vice versa depending on severity of disease.

Levodopa for Speech Dysfunction

Most reports surrounding pharmacological treatment of speech deficit symptoms, via Levodopa, have shown mixed results or no resolve of speech deficits[10]. Most of the time, Levodopa has been associated with relief of dysrhythmia symptoms.

Speech and Gait in Parkinson's

Correlations between Speech and Gait

Cantiniaux and colleagues previously examined the correlations between speech and gait under treatment conditions such as Levodopa or STN-DBS. Findings were primarily centered around the speech velocity by dividing it into two sub categories: interpause speech duration and the speech index of rhythmicity. Gait parameters were centered around walking length, step cadence, and walking velocity. Comparison of these parameters under treatment conditions have shown a significant positive correlation between step length and ISD was also found. Confirms that step length and ISD decrease are manifestations of both gait and speech akinesia, that they are both responsive to

levodopa and STN-DBS, and that just as akinesia is the main gait deficit, speech akinesia may be the main speech deficit in PD[1]. Previous evaluations of FoG individuals have also given better insight into the correlation between speech and gait as well. Considering the degradation of the motor pathways occur later in the staging of Parkinson's, freezing individuals have shown correlations in degradations of speech and degradations of gait, specifically changes in gait kinematics and delay of speech initiation, number of repetitions in a sentence, and a decreased speech rate[14]. A previous study evaluating correlations between speech disturbances and gait dysfunction in the specific phenotype showed increased articulatory dysfunction and severe speech impairment in line with those diagnosed with PIGD[15].

Cognitive Effects

Individuals diagnosed with PD have shown more difficulties with executive functioning and more generally cognitive function. These include, however not limited to, effortful processing, use of internal attentional cues, cognitive set-shifting, and self-directed strategy formation. Suggested that executive dysfunction can account for all cognitive deficits in non-demented PD. PD in terms of cognitive impairment, in relation to motor speech, have been thoroughly researched and studied showing frequent disruption of working memory, language processing, and comprehension. However, there are limited studies examining more mild cases of cognitive impairment and further research is needed on these subjects to fully examine speech production. Speech production is clearly related to motor control and working memory, however these need to be examined at a more in depth scale. While gait is mainly centered around the CpG's,

basal ganglia function, and integration in the spinal cord, further research has shown that higher- level, cortical structures also are essential for functional gait[16].

Gap in the Literature

The literature noted above indicates that there is an established relationship between speech and gait in those diagnosed with Parkinson's disease. The literature also provided the most similar and high linked parameters given the specific situations. However some gaps in the literature still remain. For example, previous studies have only included tremor dominant and postural instability/gait dysfunction phenotypes of gait outcomes. Given the increased rate of disease progression documented in those with PIGD, it would be beneficial to really focus on this discrete phenotype. While studies have shown a correlation between speech and gait in those afflicted with FoG, these freezing episodes are random and hard to elicit. Ultimately, the literature previously noted have always evaluated speech and gait in brief, episodic periods. Further research into the correlation between speech and gait in Parkinson's needs to be conducted on gait dysfunction with associated postural instability for the longitudinal aspect without treatment, in order to investigate which specific speech phenotypes are present and correlated with PIGD-PD throughout disease progression.

Study Aim/Hypothesis

The aim of this exploratory study is to evaluate the correlation between speech and gait in individuals diagnosed with Parkinson's Disease. For the purposes of continuing the more proven aspect of a deficit in motor control rather than a deficit in rhythmicity, this study will evaluate more the prosodic, articulatory, phonological and kinematic aspects of

speech. In regards to gait, the kinematic parameters will be analyzed which would include walking cadence, walking speed, postural stability, walking stability, and walking velocity.

Hypothesis

Hypothesize that postural instability and gait dysfunction at baseline in those diagnosed correlate with phonatory and articulatory dysfunctions of speech, prior to any form of treatment, in individuals diagnosed with Parkinson's disease . Additionally, the aim is to establish the degradation of speech in those diagnosed with Parkinson's disease prior to any treatment over the course of 1 year.

Methods

Participants

Participants were required to be 18-90 years of age and had a current diagnosis of Parkinson's disease. Patients were also required to be able to read and comprehend English. All exclusion criteria included any previously diagnosed neurological conditions other than Parkinson's disease (PD) or any previous orthopedic injury impacting balance or gait.

Recruitment

Participants were recruited through physician referral and through posting fliers in clinical offices. All participants received a neurological diagnosis of PD.

Number of Participants

Collected data from 9 participants with idiopathic PD, with the ability to complete balance and voice tasks, and interest in participating in the study.

Study Timeline

Each participant completed 1 visit to the laboratory lasting between 2 and 3 hours. Patients were also invited to complete a second, optional visit to the laboratory, 1 year later, to repeat all assessments. Participants who opt to come back 1 year later will be invited to complete at home surveys over this time period.

Data Collection

There was 3 ways in which individuals could participate in the study, addressing progressively more sophisticated research questions:

i. One, in-lab data collection, where there were collected clinical assessments (balance mobility) and voice characteristics

ii. Visit 1 (above), followed by a second session, approximately 12 months later, where the same two domains of data (mobility and, voice characteristics) was collected

iii. Visits 1 and 2 (noted above), plus, in the 1 year interval, participants will complete voice assessments and a falls questionnaire (electronically) every other week. For this path, participants were contacted by email every other week to complete 1) a brief falls questionnaire (attached), as well as a voice collection, which they completed via their mobile device.

Specific Data Collected

Clinical assessment for Parkinsons included:

1. Movement Disorders Society Unified Parkinson's Disease Rating Scale: MDS-UPDRS was used to conduct clinical

assessment to characterize parkinsonian symptoms. This assessment was videotaped to facilitate consistent scoring.

Speech Task

For the Speech and language elicitation task, participants went through a series of computerized and paper-based speech elicitation tasks. These tasks include reading standardized material, providing sustained phonations, repeating sounds, and spontaneous speech samples. At-Home Speech and language elicitation task had participants complete a truncated version of the in-lab language task noted above as part of the longitudinal aspect of speech dysfunction.

Motor Task

The Mini BEST and Berg Balance Tests are balance assessments. Each assessment lasted between 5 and 10 minutes, and was completed. The Functional Reach Test consisted of the participant leaning forward with arms outstretched. This assessment probed participants' ability to lean forward toward their limits of stability. As noted below, during all balance assessments, participants wore a gait-belt, and were spotted by 1-2 trained testers.

In Lab Set up and Procedures

For in-lab visits, the participant will come to the lab in the morning ~8 am-10 am, after overnight fasting (water allowed) and off anti-Parkinson medication (PD patients) for at least 12 hrs. Participants will then complete several questionnaires which ask about disease symptoms, clinical balance assessments (listed below), and a voice collection analysis (time permitted).

Data Analysis

Primary speech outcomes of this study included formant values F0(pitch), F1 formant value indicating the height of each vowel, F2 formant value which indicates how far back the vowel is occurring in the mouth(i.e. the more the vowel is toward the front of the mouth, the higher the F2 value), and intensity values(amplitude of the voice). Other primary outcomes included jitter, shimmer, noise-to-harmonics ratio, and harmonics-to-noise ratio (HNR). Again, all indicating some sort of dysfunction with the vocal folds in turn showing articulation dysfunction. Table 2 fully defines each specific outcome along with the gait outcomes as well.

Primary outcomes of the gait aspect of this study included gait speed, gait cadence, lateral step variability, step duration, stride length, turn duration, turn velocity, the amount of steps it takes to turn, postural sway on both a foam and firm surface, and anticipatory postural adjustments(APA).

Outcomes(units)	Primary gait and speech outcomes of the study Definition
Anticipatory Postural Adjustment (APA)(m/s^2)	The control of the position of the center of mass of the body by activating the trunk and leg muscles prior to a balance challenge, minimizing the risk of losing equilibrium. These are divided into two separate measures including peak forward and lateral distance. Larger values are typically considered better.
Gait - Double Support(%GCT)	Period of time when both feet are in contact with the ground as a percentage of gait cycle. Larger values are typically considered indicative of worse gait.
Gait speed(m/s)	Velocity of gait
Lateral step variability(cm)	Indicates stride variability and elevation of the step mid swing. Larger values are typically considered worse.

Step duration(s)	Time between two consecutive heel strikes of opposite feet. Larger values are typically considered worse.
Stride length Gait- Turn duration(s)	Length between two consecutive heel strikes of the same foot Time for the participant to make a 180 degree turn during natural gait. Larger values are typically considered worse.
Turn velocity(degrees/s)	Velocity of the 180 degree turn during natural gait. Larger values are typically considered worse
Gait - Turn-Number of steps(# of steps)	Amount of steps taken within the 180 degree turn. Larger values are typically considered worse.
Postural sway-Sway area (m^2/s^4)	Movement of the center of mass during quiet stance (i.e., when the participant is attempting to be as still as possible). Larger values are typically considered worse
Postural sway - Jerk (m^2/s^5)	The 3rd derivative of the center of mass position during quiet stance. Larger values are typically considered worse
Postural sway - Mean velocity(m/s)	The first derivative of center of mass position during quiet stance. Larger values are typically considered worse
Postural sway-Path length(m/s^2)	Distance that the center of mass moves over a set timeframe during a quiet stance. Larger values are typically considered worse.
F0(Hz)	Pitch of the individual
F1(Hz)	Represents the height of the tongue, to which a high frequency is equivalent to a low vowel and a low frequency is equivalent to a high vowel. This measurement is more so related to the oral and pharyngeal constrictions.
F2(Hz)	Measures, to a degree, the relative backness or depth of the vowel. The higher the frequency, the more towards the front the tongue and vowel is and vice versa. Increased F2 values indicate inappropriate or slow movements when positioned for vowels.
Intensity(dB)	Vocal intensity of the participants voice and sound

Local Jitter(Hz)	The variability of the fundamental frequency of speech from one cycle to the next
Shimmer(Hz)	The sequence of maximum extent of the signal amplitude within each vocal cycle
Harmonics-To-Noise Ratio (HNR)	The amplitude of tonal relative to noise components

Table 2. This table shows the primary speech and gait outcomes and their respective definitions.

Statistics

To determine the relationship between speech dysfunction and gait dysfunction at baseline, we ran a nonparametric bivariate correlation between the two sets of primary outcomes to see any established relationships. The correlation statistic used was the Spearman correlation statistic due to the non-normality and small sample size of participants.

Results

Speech and Gait Analysis

Out of the 178 different comparisons made, there were only 25 significant correlations that included both positive and negative relationships. All data and outcomes of the correlational matrix are shown in both Table 4 and Table 5.

Positive Correlations

Correlations were conducted between the primary outcomes of both speech and gait analysis. Results showed multiple direct correlations between the articulators of speech and gait. There were established direct correlations between the HNR and average gait speed (α-level = 0.05, r =0.68), as well as HNR and lateral step variability (α-level =

0.05, r =0.71). There was also a significant positive correlation between stride length variability and vocal intensity variability (**α**-level = 0.05, r =-0.77). Lastly, there was a significant correlation between the average number of steps participants took to turn and the average variability of pitch (**α**-level = 0.05, r-squared=0.75), and the average variability of F2 (**α**-level = 0.05, r-squared=0.76).

Negative Correlation

Multiple instances of inverse relationships were observed. The first of which being between APA-forward peak and the average variability of F1 (**α**-level = 0.05, r =-0.66). In relation to the APA-lateral peak, there seemed to be an inverse correlation with average local jitter (**α**-level = 0.01, r = -0.92) and average shimmer (**α**-level = 0.01, r = -0.85). From the perspective of gait speed, there was an inverse relation with average local jitter (**α**-level = 0.01, r = -0.89). Lateral step variability (**α**-level = 0.05, r-squared= -0.71) and stride length (**α**-level = 0.05, r-squared= -0.89) seemed to show a significant inverse relation with average local jitter. Step duration inversely correlated with the average F2 (**α**-level = 0.05, r-squared= -0.89) as well as an inverse relationship between postural sway upon a firm surface and the average variability of intensity (**α**-level = 0.01, r-squared= -0.86). The variability of the duration of the participants turns showed a significant negative correlation with the average variability of F1 (**α**-level = 0.05, r =-0.67). Lastly, there was a significant inverse correlation between turn velocity variability and average shimmer (**α**-level = 0.01, r-squared= -0.67), as well as a significant negative correlation between the variability of turn velocity and average local jitter (**α**-level = 0.01, r =-0.82).

Articulatory measures (variability measures)	Gait averages
F1	Gait double support
F2	Gait speed
	Gait lateral step variability
	Gait step duration
	Gait stride length
	Turn duration
	Turn velocity
	Steps in turn

Table 3. This table lists the appropriate voluntary measurements and the comparison between articulatory measures and gait average measures. These comparisons are considered appropriate given the voluntary nature of each measure.

Articulatory measures	Gait - Lower Limb - Double Support (%GCT) [mean]	Gait - Lower Limb - Gait Speed (m/s) [mean]	Gait - Lower Limb - Lateral Step Variability (mean) (cm)	Gait - Lower Limb - Step Duration (s) [mean]	Gait - Lower Limb - Stride Length (m) [mean]	Turns - Duration (s) [mean]	Turns - Turn Velocity (degrees/s) [mean]	Turns - Steps in Turn (#) [mean]
Mean STD F1 *	-0.107	-0.178	-0.357	-0.428	-0.1785	0.2857	-0.357	0.414
Mean STD F2 *	0.214	-0.07142	0.0357	-0.5	-0.071	0.607	-0.286	***0.919**

Table 4. This table shows the correlation matrix from the aspect of articulatory measures in comparison to voluntary gait measures. The bolded value is the indicated significant value at an alpha level of 0.01.

Phonatory measures (variability measures)	Gait standard deviations, postural velocities, and distances
F0	APA-Forward peak
Intensity	APA-Lateral peak
Jitter	Gait double support
Shimmer	Gait speed
Harmonics to Noise ratio (HNR)	Gait step duration
	Gait stride length
	Turn duration
	Turn velocity
	Steps in turn
	Sway area
	Postural sway jerk

	Postural sway path length
	Postural sway mean velocity

Table 5. This table lists the appropriate automaticity comparison between phonatory measures and gait variability measures. These comparisons are considered given the automatic natures of each measure.

Phonatary measures	Gait - Double Support[std]	Gait - Gait Speed (m/s) [std]	Gait - Step Duration (s) [std]	Gait - Stride Length (m) [std]	Turns - Duration (s) [std]	Turns - Turn Velocity (degrees/s) [std]	Turns - Steps in Turn (#) [std]	Postural Sway - Sway Area (m^2/s^4)	Postural Sway - Jerk (m^2/s^5)	Postural Sway - Mean Velocity (m/s)Firm Surface	Postural Sway - Path Length (m/s^2) Firm Surface	Postural Sway - Sway Area (m^2/s^4)Foam Surface	Postural Sway - Jerk (m^2/s^5)Foam Surface	Postural Sway - Mean Velocity (m/s) Foam Surface	Postural Sway - Path Length (m/s^2) Foam Surface	Lateral APA Peak (m/s^2)	Forward APA Peak (m/s^2)
Mean STD F0	0.285	0.374	0.316	0.239	-0.571	0.392	0.074	-0.178	0.035	-0.357	0.393	0.357	0.035	-0.321	0.393	0.214	-0.19
Mean STD Int	0.428	0.168	**0.632	**0.776	-0.107	0.357	0.370	-0.285	**-0.857	-0.535	-0.357	0.107	-0.643	0.607	-0.107	0.286	-0.21
Average local jitter	0.392	0.355	0.158	0.298	-0.071	**-0.821	0.481	0.321	0.5	-0.035	0.464	-0.214	0.428	0.25	0.25	**-0.928	-0.50
Average shimmer	0.142	0.074	0.158	0.537	-0.214	-0.392	0.555	-0.178	-0.035	-0.5	-0.107	-0.535	-0.25	0.607	-0.25	**-0.857	-0.48
Average harmonics-to-noise	-0.142	-0.205	0	-0.298	0.142	0.607	-0.555	-0.107	-0.428	0.178	-0.321	0.392	-0.214	-0.25	-0.035	**1	0.55

Table 6. This table shows the correlation matrix from the aspect of phonatory measures in comparison to involuntary gait measures. The bolded values are the indicated significant values.*alpha level=0.05 **alpha level=0.01

Speech Analysis over 1 Year

Change in speech dysfunction over 1 year was also analyzed. The overall pitch variability of the sample size decreased (worsened) over the year($\sigma_{baseline} = 7.00, \sigma_{1-year} = 6.88$), as well as an increased pitch average from 146.00 Hz at baseline, to 146.32 Hz after 1 year. F1 showed a increase in average value from 380.59 Hz to 407.95 Hz in the following year, and increased variability over the year ($\sigma_{baseline} = 61.49, \sigma_{1-year} = 88.47$). The following outcome, F2 variability, showed a slight increase in the overall average from 1576.93 Hz to 1588.44 Hz along with an increased

variability ($\sigma_{baseline} = 151.72, \sigma_{1-year} = 187.78$). Lastly, I analyzed the relative intensity of each participant over the course of 1 year and showed an increase in average from 62.31 dB to 63.25 dB. There was also a slight increase in variability as well ($\sigma_{baseline} = 1.23, \sigma_{1-year} = 2.59$).

Following the Wilcoxon signed rank test, there were no significant differences between the baseline and post 1-year values. Pitch values showed a p-value of 0.86, F1 values showed a p-value of 1, F2 values showed a p-value of 0.31, and intensity values showed a p-value of 0.49, all of this shown in Table 7.

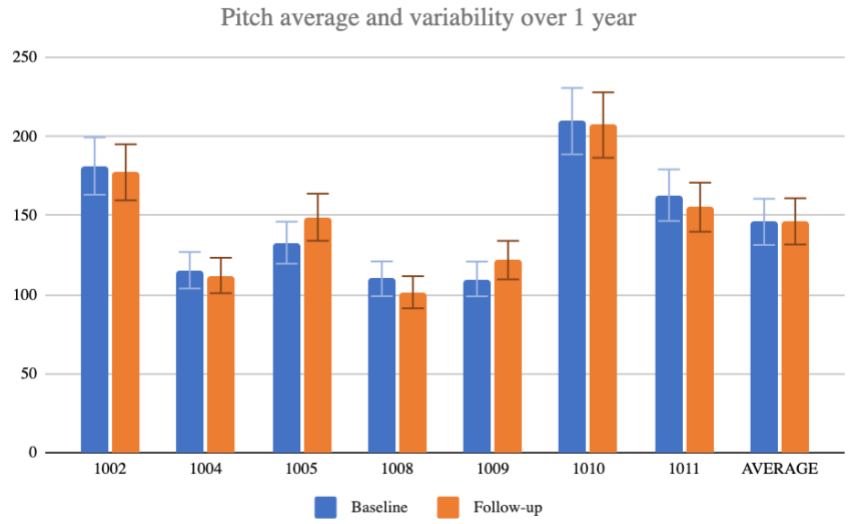

Figure 1. This graph illustrates the increase in pitch average, however a decrease in variability over the last year.

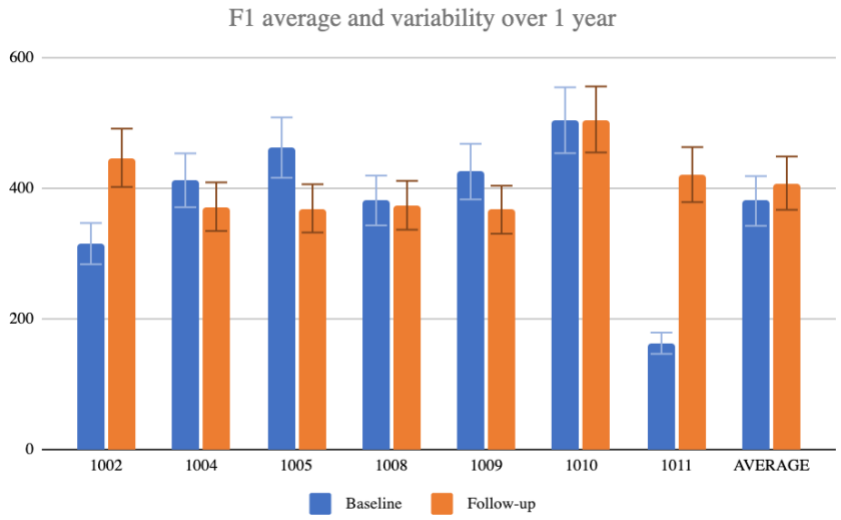

Figure 2. This graph illustrates the increase in both average and variability of the F1 average over 1 year.

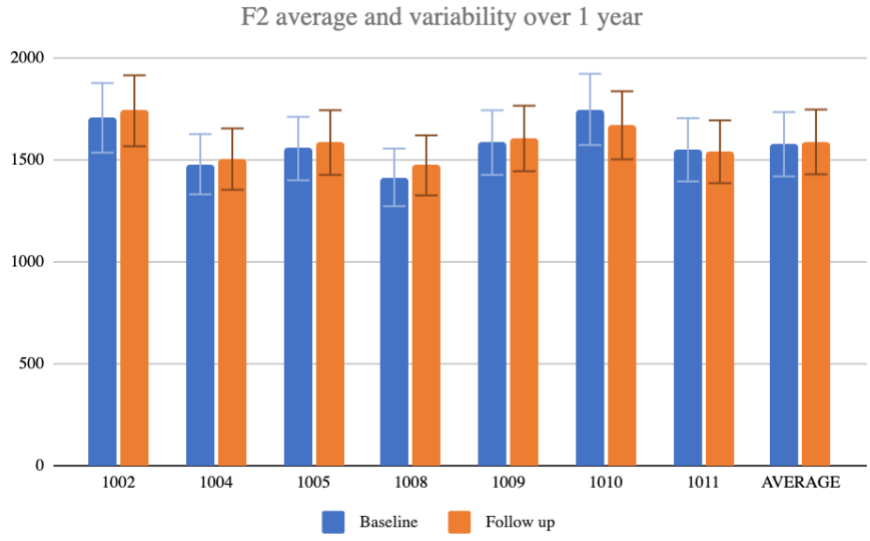

Figure 3. The graph illustrates the increase in both averages and variability of the F2 frequency over 1 year.

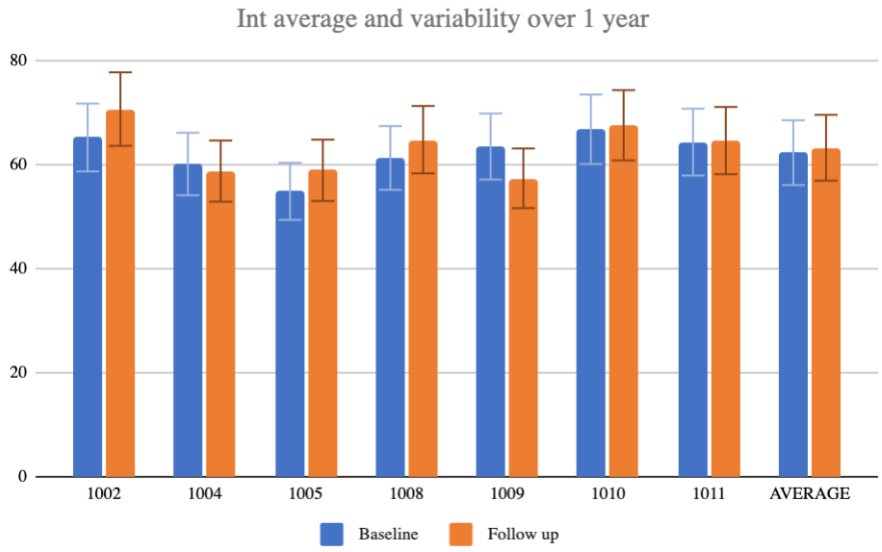

Figure 4. The graph illustrates the increase in both averages and variability of the intensity over 1 year.

Means, variabilities, and p-values for secondary speech outcomes

	pre	post	p-val
Pitch	146.00(39.36)	146.32(37.72)	0.86
F1	380.59(113.04)	407.95(52.89)	1
F2	1576.92(117.76)	1588.44(93.85)	0.31
Intensity	62.31(3.98)	63.25(5.02)	0.49
	mean(std)	mean(std)	

Table 7. This table displays both baseline and post 1 year average and standard deviation values of the secondary speech outcomes. The table also shows the relative p-values of each outcome.

Discussion

Although 25 significant correlations were established, Table 3 and Table 5 indicate the more appropriate and pertinent comparisons within the study. Out of the 25

significant correlations established, only 8 correlations proved to be pertinent based on previous data and established studies. When stating the pertinent comparisons, these comparisons are defined as relationships that actually display information of use. The other noted significant correlations do not display any information of use. One of the previous pertinent comparisons noted in Table 3 was the articulatory speech measures (i.e. F1 and F2 variability) against the gait outcome averages, rather than variability. The F1 and F2 frequencies are more related to voluntary mandible and other articulator controls, while the averages of the gait primary outcomes show more so voluntary control of the gait aspect of the individual. The other previous pertinent comparison, as seen in Table 5, include phonatory measures(i.e. F0, intensity, jitter, shimmer, and HNR) against the variability of the primary gait outcomes, as well as the outcomes related to postural sway. These phonatory measures are more related to the involuntary and automatic control of speech, similarly to how the variability of gait outcomes and regulation of posture are related to the automaticity of gait. In the following sections, we will discuss the 8 significant correlations between gait and speech. Specifically, we will discuss them with respect to those that supported our hypotheses (i.e., showed that better speech related to better gait), and those that did not support our hypothesis (i.e., showed that there was an indirect relationship between speech and gait).

Expected Outcomes

Phonatory Measures Vs. Gait Automaticity

The first significant and pertinent correlation showed to be between HNR and APA-lateral peak with a correlation value of 0.71. As apparent in Graph 1, as the HNR

values increased so did the APA-lateral peak. As previous data would suggest, decreases in HNR values have been heavily associated with PD[10], similarly, decreases in APA-lateral movements in response to perturbations have been associated with PD and in most cases FOG type individuals[18]. It can be inferred that treating one of these aspects may improve the counterpart. Given that these measurements are both indications of automaticity, it can be inferred that treatments of these simultaneously would be significantly beneficial for involuntary movements of the respective muscle groups and neuronal pathways. These would prove to be targets that could be simultaneously improved via treatment. However, this would only be a secondary target in comparison against intensity variability.

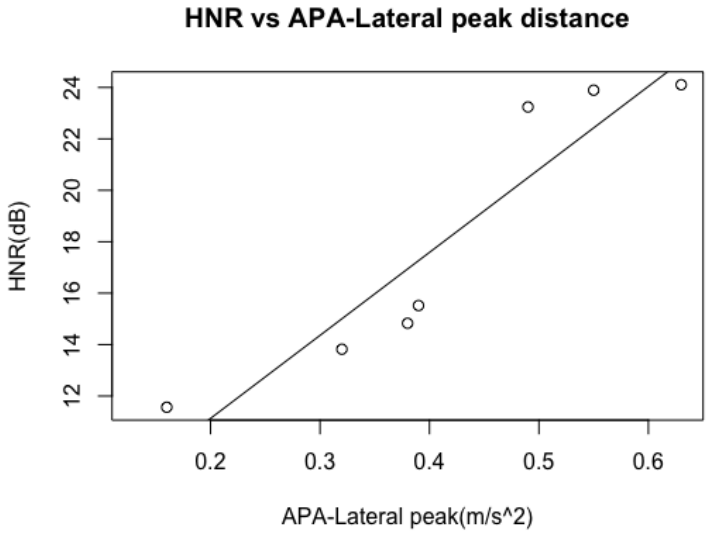

Figure 5. This graph illustrates the expected outcome of increased HNR, as lateral peak also increases. This appropriately shows that as the HNR increases, APA-lateral peak also increases, indicating that these aspects of speech and gait may work under the same mechanism.

Intensity variability and stride length variability seemed to show a significant correlation when comparing the two components, this being a key finding within this study. This comparison showed a correlation value of 0.77 indicating a strong correlation between the two measurements. Previous data suggests that increased intensity variability and increased gait variability in the form of stride length have shown to be associated with individuals with PD[10, 4] . Shown in Graph 2, higher intensity variability values were correlated with the higher stride length variability values. It could be reasonably inferred that a physical therapy treatment, or other form of treatment, could attend to both aspects to improve these measurements.

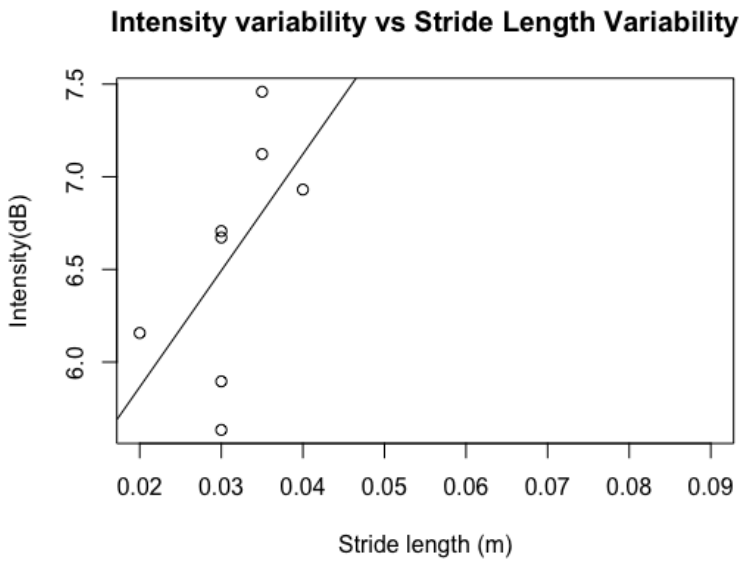

Figure 6. This graph illustrates the expected outcome of how the higher intensity variability values correlate with higher stride length variability values, similarly with low variability values for each respective measurement. This indicates a potential similarity in the mechanisms used for both aspects.

Average local jitter seemed to show significant correlations with two separate gait outcomes. The first being against turn velocity variability with a correlation value of -0.82. Previous studies have shown how higher average jitter values have been associated with those diagnosed with PD, while decreased turning velocities and increased turn velocity variabilities have also been linked to PD[10,5]. Local jitter also showed a strong correlation with APA-lateral peak with a correlation value of -0.92. Given this information, a physical therapy method that targets both improving the lateral peak and turning velocity of the individual may potentially also improve the local jitter of the individual.

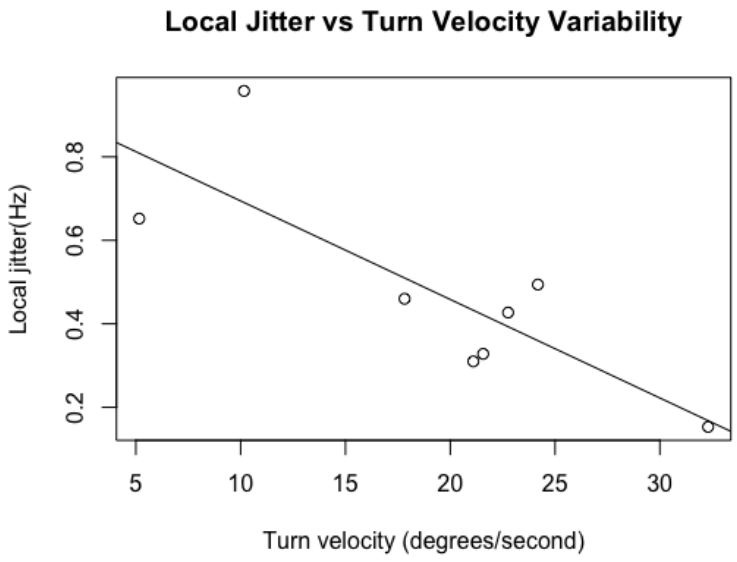

Figure 7. This graph illustrates the expected outcome of both average local jitter and turn velocity variability values. Showing how lower local jitter values seem to correlate with higher turn velocity variabilities.

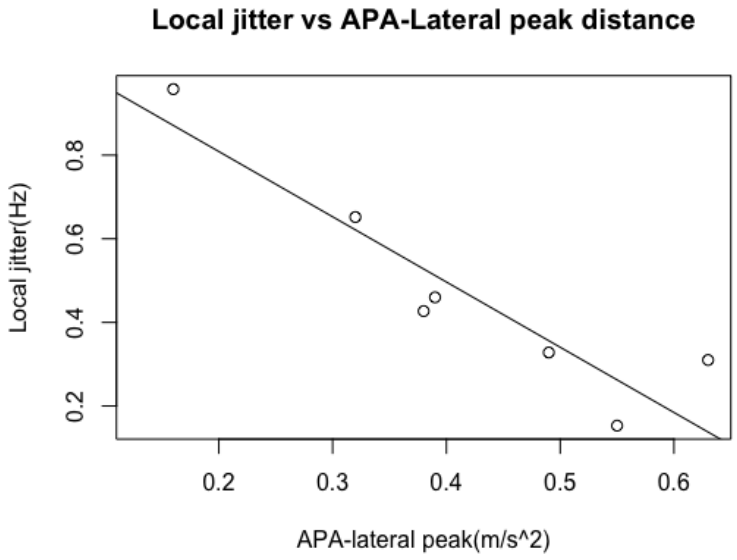

Figure 8. This graph appropriately illustrates the expected outcome of decreasing jitter as lateral peak increases. As decreased lateral peak and increased jitter are main indications of PD, this graph illustrates how the mechanisms underlying these may work cohesively to improve symptoms.

Lastly, the last expected outcome that came from this study was the comparison of shimmer against APA-lateral peak with a correlation value of -0.85, indicating a strong negative correlation, as seen in Graph 5. As previous data suggests, increased shimmer values have been associated with PD, therefore it can be reasonably inferred that attributing a treatment towards increasing the anticipatory lateral shift and decreasing the shimmer values would benefit the individual[10].

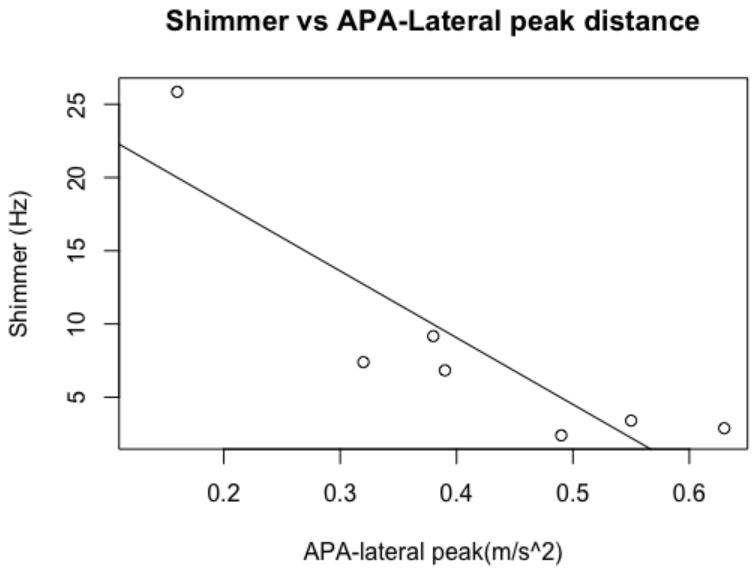

Figure 9. This graph illustrates the expected outcome of average shimmer values in comparison to lateral peaks. That is that as higher lateral peak values correlate with lower average shimmer values. As previous data would suggest, increased shimmer and decreased APA-lateral peak are main outcomes of those diagnosed with PD.

Unexpected Outcomes

Articulatory Measures Vs Gait Averages

There were few unexpected outcomes as a result of the correlation between the speech and gait outcomes. The first unexpected outcome found was between F2 variability and the average number of steps taken within a turn. The correlation between the two outcomes produced a correlation value of 0.91 indicating a strong correlation between the two outcomes. As previously mentioned, low F2 variability values indicate a smaller vowel space area for the individual and is a main indicator of PD, while, similarly to symptoms experienced for FOG, increased step counts are main indicators of PD. Therefore, it is interesting that individuals with larger (better) F2 variability, would also

take more steps during turning (a worse turning outcome). The rationale for this observed relationship is not entirely clear. However it may be related to participants taking more turns to ensure safe turning, rather than fewer, less safe turns.

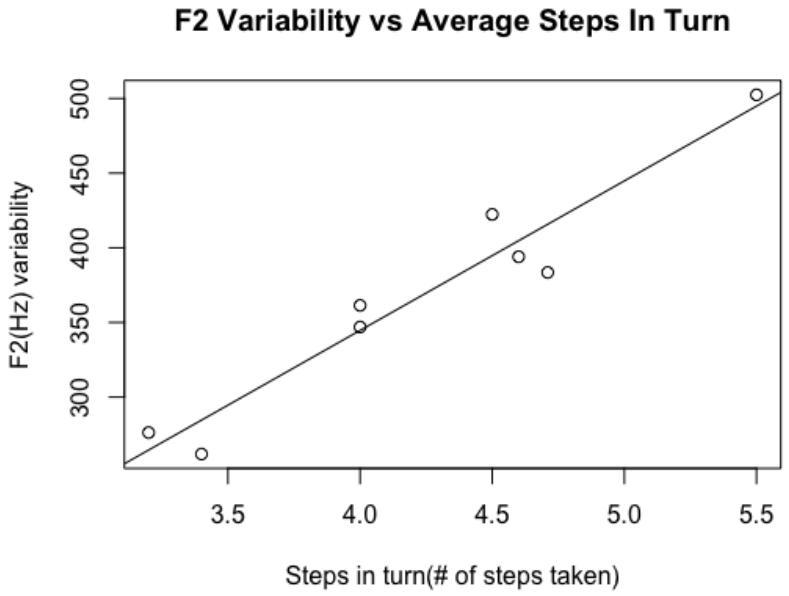

Figure 10. This graph illustrates the unexpected outcome that higher variability values seem to correlate with the higher number of steps taken. Previous data shows how decreased F2 values and shallow F2 slopes indicate slower tongue movements, therefore increases in variability are viewed as a deficit, similarly as increases in the amount of steps it takes to turn are viewed as deficits given that festination of gait is a strong predecessor of FOG.

Phonatory Measures Vs. Gait Automaticity

The next unexpected outcome that was found during this correlation was between intensity variability and postural sway-jerk. As Figure 12 shows, increased postural sway jerkiness values seem to correlate with lower intensity variability values. As previous

data would suggest, increased intensity range and variabilities are main indicators of PD, while increased postural instability, in this case increased postural jerk during sway, are also a symptom of progressive PD[17,4]. This information should be considered when thinking of potential treatments to treat both gait and speech deficits. Given this, there is further investigation needed to understand this relationship, and treatments should keep note of this when trying to attend to one of these measurements.

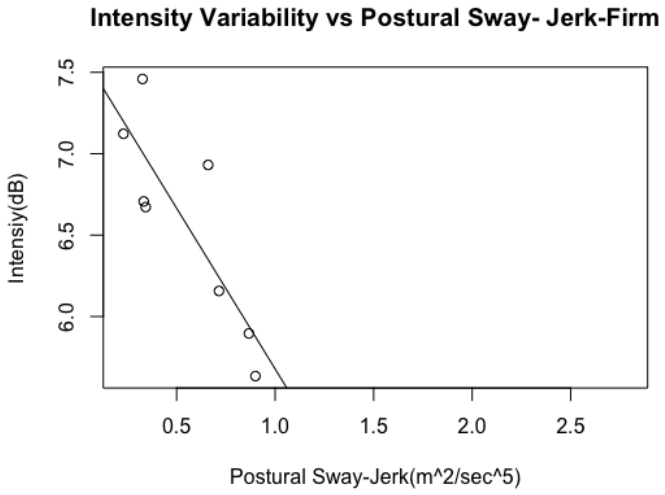

Figure 11. The figure illustrates how lower intensity variability values tend to correlate with higher postural jerk values for both a foam and firm surface, indicating this compensatory phenomenon.

Another unexpected outcome in relation to intensity variability was against step duration variability. As previously noted, increased step duration variability is a main indication of PD, especially in those afflicted by FOG. Similarly, increased intensity variability values are also indications of PD, as previously mentioned above[5]. Indicated by Graph 8, higher intensity variability values seem to correlate with higher step duration variability values. Given the multiple relationships that intensity has with other gait

values, developing a particular treatment to address the amplitude of the individual should keep these compensatory relationships in mind. Again, to truly understand this mechanism, further investigation is needed beyond the scope of this exploratory study.

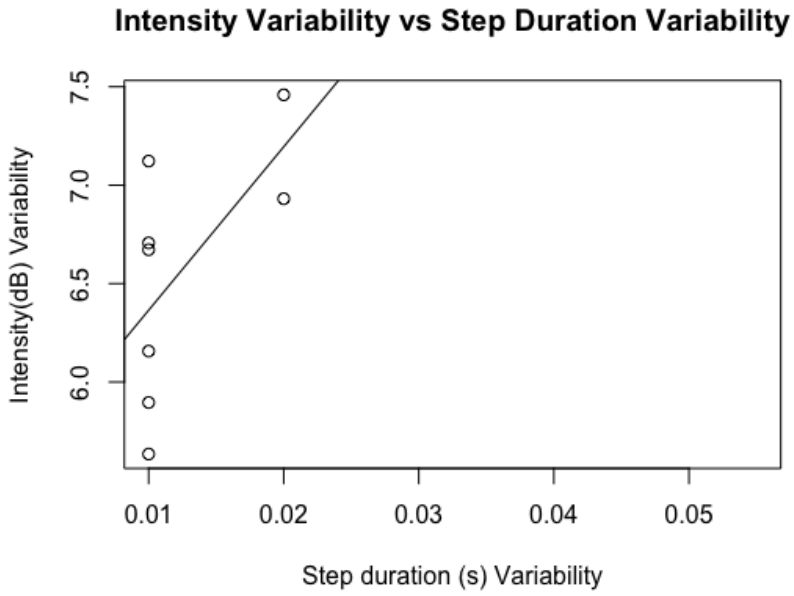

Figure 12. The graph illustrates the unexpected outcome of how higher intensity variability values correlate with higher step duration variability values. As increased step duration is a previous indication of dysfunction, specifically FOG, along with increased variability in intensity indicating speech dysfunction, this graph shows how these aspects may be connected.

Speech over 1 Year

After establishing a correlation between speech and gait, we were able to obtain even more speech data following 1 year. We evaluated the primary outcomes once again, and found slight increases in formant frequency average, while also showing an increase in formant frequency variability. For example, over all participants' completion of the

pertinent tasks, F1 average rose from 380.59 Hz at baseline, to 407.95 Hz after 1 year and showed an increased variability of 61.49 to 88.47, as shown in graph 2. For a frame of reference, increased F1 average and variability values would indicate a larger range of motion, showing this abnormality that has occurred over the last year. Just as well, F2 showed an increase in average from 1576.93 to 1588.44, along with an increase in variability from 151.72 to 187.78, as shown in graph 3. Again, similar to F1 variability and averages, increased F2 variability and average values indicate a larger range of motion within the articulators. This demonstrates the unexpected outcomes that occurred over the time period. Lastly, intensity followed this same pattern as the intensity averages increased from 62.31 dB to 63.25 dB, with an increase in variability from 1.23 to 2.59 following 1 year, as shown in graph 4. Although the slight increase in intensity average was unexpected, the increase in intensity variability is indicative of PD and is to be expected over a 1-year time period. Similarly, pitch averages for the participants did increase from 146.00 to 146.32, however variability did decrease from 7.00 to 6.88 following 1 year, as shown in graph 1.

Overall, there were no significant differences between both the baseline and post 1-year values. As seen in Table 7, the p-values displayed insignificant values which can be attributed to both a small sample size and the time period allotted to this study. The expected outcomes were to show worsening in each respective outcome, however, given the improved results this classifies under an unexpected outcome.

Limitations

There were some obvious limitations associated with this study. The first of which being the inability to fully analyze the correlation between speech and gait over 1 year given the inability to access the data. Even given just the baseline information, there is more data analysis required to fully understand the correlation between speech and gait characteristics. As well, when evaluating the speech outcomes, these statistics reflect the participants in the OFF state, however, within the year they were not required to hold off on taking their medication. Therefore, there is some pharmacological influence of Levodopa with these results, most likely reflected in the increase of averages across the primary outcomes. Lastly, a limitation experienced with the speech outcomes was evaluating the dynamic range of both F1 and F2. Unfortunately, the F2 variability values can be considered "contaminated" considering that the values do not account for one specific phoneme, and account for multiple phonemes across a sentence. Given this fact, the variability values are only analyzed across a range of phoneme targets rather than one specific one.

Conclusion

Ultimately, this study fully displayed a relationship between both speech and gait in multiple different aspects and furthermore confirmed the hypothesis that there is a significant relationship between both speech and gait deficits in those diagnosed with PD. This correlational and explorational study really showed multiple different avenues for studying the possible similarities between both aspects of deficits within PD.

The exploratory study seemed to confirm the more expected outcomes between the two aspects and gives multiple different targets for potential therapy methods, such

that better gait was related to better speech across the participants. The benefit to this study is that it helps clarify different target areas that may also treat the gait or speech counterpart simultaneously. These expected outcomes serve as target points to treat simultaneous aspects of the PD. With further investigation and research, possible treatment methods could be developed in order to improve measurements of both speech and gait.

The unexpected outcomes identified in this study are very important for the understanding of PD. Some of the compensatory mechanisms introduced in this study are pertinent towards the understanding of each individual aspect of PD. This also helps individualize different treatment methods to correctly treat one aspect while not hurting the other or causing further deficit.

Overall, these different expected and unexpected outcomes could lead to other possible treatment methods that could be the combination of treatment methods or new ones entirely in order to attend to both aspects of Parkinson's Disease.

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