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THE EFFECTS OF INTENSIVE LOWER LIMB TRAINING ON THE SPEECH OF
PATIENTS WITH PARKINSON'S DISEASE

by

Lauren Fjeldsted Allen

A thesis submitted to the faculty of

Brigham Young University

in partial fulfillment of the requirements for the degree of

Master of Science

Department of Communication Disorders

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BRIGHAM YOUNG UNIVERSITY

GRADUATE COMMITTEE APPROVAL

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ABSTRACT

THE EFFECTS OF INTENSIVE LOWER LIMB TRAINING ON THE SPEECH OF PATIENTS WITH PARKINSON'S DISEASE

Lauren Fjeldsted Allen

Department of Communication Disorders

Master of Science

Research has documented the positive effects of physical therapy as a treatment for limb motor symptoms associated with Parkinson's disease (PD). Previous studies have shown that speech and voice measures can be reflective of overall cardiovascular health and fitness in young, middle-aged, and older adults. In healthy individuals, increased respiratory drive has been found to influence vocal function and speech articulation. The rationale for the present study was that improved lower limb function might lead to improvements in overall fitness, which then may influence speech in individuals with Parkinson's disease. To investigate this premise, 10 participants diagnosed with mild to moderate idiopathic PD were involved in an intensive lower limb training program called Resistance Exercise via Negative-Eccentric Work (RENEW).

The speech of the participants was recorded before and after the RENEW treatment in both medication-on and medication-off conditions. Following treatment there was a statistically significant increase in spirantization of the stop gap in DDK repetition in the medication-on condition and a decrease in diphthong duration in the medication-off condition. After treatment in the medication-on condition there was an increase in spirantization and a reduction in first and second formant transitions for the diphthong /ɔɪ/ compared to the medication-off condition. These results represented a slight worsening of articulatory precision and movement following treatment in response to PD medication. Overall, the present study found that intensive lower limb training did not influence the acoustic measures of speech articulation examined in the present study.

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TABLE OF CONTENTS

	Page
LIST OF TABLES	viii
LIST OF FIGURES	ix
Introduction.....	1
Nature and Characteristics of Parkinson's Disease	1
Treatment of Parkinson's Disease	5
The Present Study	13
Method	13
Participants.....	13
Treatment	14
Speaking Tasks	14
Instrumentation	15
Acoustic Analysis	15
Statistical Analysis.....	19
Results.....	22
Pretreatment Versus Posttreatment	22
Posttreatment Medication-on Versus Medication-off Conditions	22
Discussion.....	25
Changes in Speech Acoustic Measures.....	25
General Discussion	28
Future Research Directions.....	29
References.....	31

LIST OF TABLES

Table	Page
1. Means and SDs of Acoustic Variables for All Speakers in the Medication-off Condition.....	23
2. Means and SDs of Acoustic Variables for All Speakers in the Medication-on Condition	24

LIST OF FIGURES

Figure	Page
1. The Praat analysis display for the sentence <i>The boot on top is packed to keep</i>	16
2. The Praat analysis display for the formant tracks of the diphthong /ɔɪ/.	18
3. Praat analysis display for a normal stop gap and VOT segmentation from the DDK task.	20
4. Praat display for a fully spirantized stop from the DDK task.....	21

Introduction

Dr. James Parkinson was the first to separate the symptoms of Parkinson's disease (PD) from other causes of tremor and weakness. Dr. Parkinson wrote an essay entitled *An Essay on the Shaking Palsy* in 1817 to describe the disease. PD is the second most common neurodegenerative disorder (de Lau & Breteler, 2006), with an estimated 50 of every 100,000 people over the age of 50 affected by the disease (Duffy, 2005). Although no cure has been found, many treatment options are available to help manage patient symptoms. Research has documented the positive effects of physical therapy as a possible treatment for symptoms associated with PD (de Goede, Keus, Kwakkel, & Wagenaar, 2001; Hirsch, Toole, Maitland, & Rider, 2003; Morris, 2000). The success of the speech therapy technique, Lee Silverman Voice Treatment (LSVT), has also been documented for speech and voice symptoms associated with PD (Ramig & Dromey, 1996; Ramig et al., 2001; Sapir, Spielman, Ramig, Story, & Fox, 2007). The effect of physical therapy treatment on speech and voice characteristics of patients with PD has yet to be examined. The purpose of the present study was to determine the potential impact of intensive lower limb exercise on speech by analyzing a number of measures reflecting the rate and extent of tongue movement, spread of acoustic energy, and coordination of the articulators during speech. These included an index of vowel articulation, formant transition extent and slope for diphthongs, long-term average spectrum, voice onset time, stop closure duration, and an index of spirantization. These acoustic measures were used to draw inferences about vocal tract movements during speech.

Nature and Characteristics of Parkinson's Disease

PD is a progressive neurological disease that results in abnormal motor control. It is characterized by depletion of the neurotransmitter dopamine, which is produced in the

substantia nigra in the basal ganglia. The circuits of the basal ganglia function to facilitate volitional motor activity and inhibit involuntary movements (Webster, 1999).

According to both Meara and Koller (2000) and Playfer and Hindle (2001), the globus pallidus interna (GPi) and the ventrolateral thalamus (VL) play an important role in controlling movement. The VL facilitates movement by sending excitatory output to the motor cortex. The GPi and part of the substantia nigra (SN) send inhibitory output to the VL, thus increased output from the GPi/SN inhibits movements. The GPi/SN receives input from the putamen through two pathways, direct and indirect. The direct pathway inhibits the function of the GPi/SN, thus it releases the inhibition of movement caused by the GPi/SN. The indirect pathway has an excitatory effect on the GPi/SN; therefore, it causes an increase in the inhibitory function of the GPi/SN. Dopamine produced by the SN controls the activity levels of both pathways; the direct pathway is excited and the indirect pathway is inhibited. In the normal system this results in a low braking effect from the GPi/SN to the VL (Playfer & Hindle, 2001).

In PD, there is a loss of cells that produce dopamine. With the decrease in dopamine, the direct pathway is understimulated and the indirect pathway is underinhibited. This results in increased braking output from the GPi/SN to the VL and a decrease in the excitatory output from the VL to the motor cortex (Playfer & Hindle, 2001). This disruption results in PD symptoms of akinesia, rigidity, and in many cases tremor (Meara & Koller, 2000).

General motor characteristics. Akinesia, one of the main diagnostic components of PD, is a symptom complex comprised of multiple features (Playfer & Hindle, 2001). One feature, bradykinesia, is characterized by slowness in the performance of movement sequences (Morris, 2000). Early fatigue as tasks are repeated may also contribute to slow

movement (Playfer & Hindle, 2001). Another feature, hypokinesia, is poverty or lack of movement. Other characteristics of akinesia include reduced spontaneous voluntary motor activity and impairment of sequential and concurrent motor acts (Playfer & Hindle, 2001). Recognizable signs such as lack of facial expression, reduced blink rate, reduced arm swing while walking, and micrographia are indicators of akinesia.

Rigidity, another general motor characteristic of PD, is an increase in resistance to passive movement of joints in the fully relaxed limb (Meara & Koller, 2000; Playfer & Hindle, 2001). Two types of rigidity are found in PD: lead-pipe and/or cog-wheel. Lead pipe is smooth motion, while cog-wheel is ratchet type movement with fluctuating resistance (Meara & Koller, 2000; Playfer & Hindle, 2001).

Another characteristic, resting tremor, is one of the most recognizable signs of PD. It presents as a tremor that has a 4-6 Hz oscillation and is most apparent when the patient is at rest. Resting tremor can be found in the majority of idiopathic PD cases; however, it is not typically found in other forms of parkinsonism (Meara & Koller, 2000). These characteristics of PD may be accompanied by festination (Duffy, 2005), postural instability and gait disturbance (Playfer & Hindle, 2001).

Speech characteristics. It is important to understand the speech and voice characteristics associated with PD because of the communication deficit they create. An understanding of these characteristics is also important because they have the potential to provide helpful information for the diagnosis of PD. Logemann, Fisher, Boshes, and Blonsky (1978) studied the frequency and occurrence of vocal tract disorders in 200 patients with PD. Eighty-nine percent of these individuals displayed voice disorders such as breathiness, hoarseness, roughness, and tremulousness. Forty-five percent of the participants displayed

articulation disorders. Oguz et al. (2006) found that people with Parkinson's disease who did not display voice symptoms still had significantly different acoustic values when compared to age- and sex-matched controls. Participants with PD had higher jitter values, lower harmonics to noise ratios, and lower intensity (dB) levels when compared to the control group (Oguz et al., 2006). Some voice characteristics of PD change as the disease progresses (Holmes, Oates, Phylard, & Hughes, 2000). Fundamental frequency has been investigated as a potential biomarker for the progression and onset of PD (Harel, Cannizzaro, Cohen, Reilly, & Snyder, 2004; Harel, Cannizzaro, & Snyder, 2004). The voice production system may be more sensitive to the onset of PD because of the coordination of the respiratory, phonatory, and articulatory systems involved in speech (Harel et al., 2004). One study found fundamental frequency variability to be significantly lower in patients with PD at the approximate time of diagnosis (Harel, Cannizzaro, Cohen et al., 2004).

Hypokinetic dysarthria is a motor speech disorder associated with disruption in basal ganglia control and is typically seen in PD (Duffy, 2005). The speech characteristics of hypokinetic dysarthria include weak breathy voice, monotone and monoloud speech, rate disturbances, and imprecise movements of the articulators (Dromey, 2003). A reduction in facial expression often accompanies these characteristics (Spielman, Borod, & Ramig, 2003).

Patients with PD may have bowing of the vocal folds (Blumin, Pcolinsky, & Atkins, 2004). This contributes to a significantly large glottic gap, which allows more air to pass through the vocal folds, which leads to breathiness and weak phonation (Blumin et al., 2004). Hypokinesia and bradykinesia may influence laryngeal function, as demonstrated by a study that found a reduction in the activity levels of the thyroarytenoid muscles in patients with PD (Baker, Ramig, Luschei, & Smith, 1998).

Patients with PD have been found to have reduced precision in the articulation of consonants. They may display incomplete occlusion or undershooting of the articulatory targets. This results in spirantization, which is the replacement of a stop gap with low-intensity frication (Tjaden & Watling, 2003). Patients may display either slowed or accelerated alternating motion rates of the articulators (Tjaden & Watling, 2003). Articulatory range of movement is also typically reduced, which may result in a smaller acoustic working space for vowels in PD. One study found patients with PD to have a more restrictive acoustic vowel space area (Weismer, Jeng, Laures, Kent, & Kent, 2001). Reduction in vowel space area can have a negative effect on intelligibility (Bradlow, Torretta, & Pisoni, 1996).

The physiologic mechanism underlying many of the speech and voice symptoms associated with PD is not known. Rigidity and bradykinesia are commonly thought to be the cause of these symptoms. Another explanation has to do with the perceptual and sensory processing deficits displayed by many patients with PD (Fox, Morrison, Ramig, & Sapir, 2002; Ramig, Fox, & Sapir, 2004; Trail et al., 2005). Patients fail to recognize that their loudness level is reduced compared to normal speakers. This suggests a breakdown in the processing of auditory and proprioceptive feedback while speaking (Trail et al., 2005).

Treatment of Parkinson's Disease

Pharmacological treatments. The goal of pharmacological treatment for PD is to control symptoms while minimizing side effects. The medications typically used either enhance (dopamine agonist) or replace (levodopa) dopamine in the brain (Schulz & Grant, 2000).

Dopamine agonists do not cause as many long-term motor complications as levodopa because they are more neuroprotective. However, they do not provide as much symptomatic relief and cause more side effects than levodopa treatment. Dopamine agonists can be used to delay the introduction of levodopa and may decrease the likelihood of long-term levodopa side-effects (Playfer & Hindle, 2001). Dopamine agonists can be taken during the *off* periods of levodopa to enhance the supply of dopamine or to prolong the effect of levodopa.

In combination with carbidopa, which prevents levodopa from being absorbed too quickly, levodopa is the principal medication used for patients with PD (Meara & Koller, 2000; Schulz & Grant, 2000). Its side effects include dyskinesic and involuntary movements, hypotension, and nausea (Meara & Koller, 2000; Playfer & Hindle, 2001; Schulz & Grant, 2000). Over time levodopa loses effectiveness and leads to the appearance and development of dyskinesias (Fabbrini, Brotchie, Grandas, Nomoto, & Goetz, 2007). After several years of levodopa treatment, PD patients commonly experience an *on/off* phenomenon. The *on* periods in which the motor movements improve become shorter and the *off* periods in which the patient becomes disabled due to the return of PD symptoms become longer (Schulz & Grant, 2000). Smaller doses and adjunctive treatments are used to combat the *on/off* effect (Playfer & Hindle, 2001; Schulz & Grant, 2000).

Phonatory characteristics have been studied during *on* and *off* states (Goberman, Coelho, & Robb, 2002; Schulz & Grant, 2000). No significant differences were found in groups while comparing *on* and *off* states; however, differences were found in individuals. Pharmacological treatment for PD has highly variable effects on the speech and voice of patients (Trail et al., 2005). Currently, pharmacological treatment alone is not sufficient for managing hypokinetic dysarthria associated with PD (Trail et al., 2005).

Surgical treatments. The speech effects of surgical treatments for PD such as thalamotomy, pallidotomy, deep brain stimulation (DBS), and fetal cell transplantation (FCT) have not been extensively studied; however, the studies that have been done do not show significant improvements in speech (Farrell, Theodoros, Ward, Hall, & Silburn, 2005; Schulz & Grant, 2000; Trail et al., 2005).

Thalamotomy is performed by lesioning the ventrolateral nucleus of the thalamus. It results in a reduction of severe tremor associated with PD (Rosenbaum, 2006). The ventralis intermedius portion of the ventrolateral nucleus has been found to have cells firing at resting tremor frequencies (Playfer & Hindle, 2001). Pallidotomy involves lesioning the GPi of the basal ganglia. This results in a decrease of the inhibitory influence on the motor thalamus. Pallidotomy decreases dyskinesia caused by drug therapies and decreases all major PD symptoms to some degree. Bilateral thalamotomy and pallidotomy have significant negative effects on speech and voice, while unilateral thalamotomy and pallidotomy have variable effects on speech and voice (Trail et al., 2005).

DBS sends electrical stimulation to the thalamus, subthalamic nucleus, or GPi through an electrode that has been surgically implanted in the brain. It results in the same effects as thalamotomy and pallidotomy, but without the risks of ablative surgery, in that the stimulation is adjustable. Patients can also have an external device which allows them to turn the stimulation on or off at will (Playfer & Hindle, 2001). FCT involves the placement of fetal dopaminergic cells within the caudate or putamen of the basal ganglia. Although this procedure is still considered experimental, benefits such as improved flexibility, reduced hypokinesia, a reduction of levodopa dosage, and reduction of *off* time have been found. The effects of DBS and FCT on speech have yet to be investigated thoroughly; however, the

studies that have been done show greater improvements in limb motor function than in speech production (Trail et al., 2005).

Physical therapy. As previously discussed, motor control is impaired in patients with PD. Patients have difficulty initiating and maintaining voluntary movements, as well as transitioning from one movement to another. They may also exhibit involuntary movement and impaired balance (Meara & Koller, 2000). Although medical and surgical treatments ameliorate symptoms temporarily, symptoms may return or side-effects may become debilitating (Fabbrini et al., 2007). In recent years, studies have found physical therapy to be effective in the treatment of symptoms in patients with PD (de Goede et al., 2001; Morris, 2000).

Physical therapy has been found to slow the degenerative process in PD (Schallert, Fleming, Leasure, Tillerson, & Bland, 2000). It also protects the brain, providing greater resilience to damage. (Carro, Trejo, Busiguina, & Torres-Aleman, 2001). Other benefits include attenuation of neurochemical deficits (Tillerson, Caudle, Reveron, & Miller, 2003) and restoration of function in the basal ganglia (Fisher et al., 2004). Multiple animal studies have added to the support of the neuroprotective effect of physical therapy in PD. For example, Tillerson et al. (2001) found forced-limb use, after the depletion of striatal dopamine, decreased the extent of striatal dopamine neuron degeneration in rats after unilateral exposure to the striatal dopamine neurotoxin, 6-hydroxydopamine. This neurotoxin induces PD symptoms (Tillerson et al., 2001). Furthermore, Tillerson et al. (2002) found forced nonuse of the affected limb exacerbated injury in rats after unilateral exposure to 6-hydroxydopamine. This suggests that decreased activity is not only a symptom of PD but also may promote degeneration (Tillerson et al., 2002). General exercise has been found to

provide neuroprotective effects in reducing PD risk (Cohen, Tillerson, Smith, Schallert, & Zigmond, 2003). Physical therapy was also found to improve motor performance and cognitive functional ability (de Goede et al., 2001; Farley & Koshland, 2005).

Physical therapy, specifically high-intensity resistance training, improves muscle strength and balance (Hirsch et al., 2003). It also leads to muscle hypertrophy, increases strength, and improves mobility in patients with PD (Dibble, Hale, Marcus, Droge et al., 2006). This training is a safe and feasible therapy option for patients with PD (Dibble, Hale, Marcus, Gerber, & Lastayo, 2006). Although more research is needed, the resistive exercise interventions currently available provide a general increase in muscle strength and function (Falvo, Schilling, & Earhart, 2008).

Physical therapy, or exercise, affects the overall fitness of individuals. Fitness is reflected in the cardiovascular, muscular, skeletal, neurological, and respiratory systems of the body. As noted by Ramig and Ringel (1983), studies have found the production of the voice to be dependent upon the function of these systems. Ramig and Ringel studied the relationship between physical wellness of individuals and characteristics of the voice. Their study found acoustic differences in the vocal production of individuals in poor physical condition and those in good physical condition (Ramig & Ringel, 1983). Participants in poor physical condition produced vowels with significantly more jitter and shimmer than the participants in good physical condition. Also, participants in good physical condition had significantly larger phonation ranges than the participants in poor physical condition.

Speech intervention. As previously discussed, pharmacological and surgical interventions provide little or no benefit to disordered speech in PD; some have even been found to have a negative effect. Due to the variability and lack of positive results from

surgical and medical remediation, behavioral approaches to the remediation of speech and voice impairment associated with PD remain the treatment of choice (Farrell et al., 2005; Trail et al., 2005).

The speech characteristics of reduced loudness and rapid rate have been the focus of speech therapy for individuals with PD. Collagen injection into the vocal folds has proven beneficial, as it decreases the glottal gap and improves maximum phonation time and phonation threshold pressure in patients with PD and hypophonia (Sewall, Jiang, & Ford, 2006). Pacing boards and delayed auditory feedback (DAF) devices can be used to help patients slow down their speech rate (Duffy, 2005). To promote increased speech loudness, devices such as vocal intensity monitors or feedback and masking devices have been used, as well as amplifiers (Duffy, 2005; Schulz & Grant, 2000). Longer lasting results and distributed effects across speech subsystems have been found to accompany intensive, focused speech therapy for patients with PD (Baumgartner, Sapir, & Ramig, 2001; Fox et al., 2002; Fox et al., 2006; Ramig & Dromey, 1996; Ramig et al., 2004; Ramig et al., 2001; Sapir et al., 2002; Trail et al., 2005; Zhang, Jiang, & Rahn, 2005).

The Lee Silverman Voice Treatment (LSVT), created by Ramig and Mead in 1987, is the current treatment of choice for the speech and voice symptoms associated with PD (Ramig et al., 2004; Trail et al., 2005). Duffy (2005) suggests that LSVT deserves attention because it applies basic principles of motor learning and has reliable evidence supporting its effectiveness.

Many studies have documented the positive effects of LSVT on voice and speech symptoms associated with PD. LSVT has been found to increase maximum flow declination rate (MFDR), which is an indication of greater vocal fold adduction, and increase subglottal

pressure (Dromey, Ramig, & Johnson, 1995; Ramig & Dromey, 1996). Perceptual improvements in hoarseness and breathiness after LSVT are consistent with acoustic data (Baumgartner et al., 2001). Increased intelligibility, possibly due to the upward spread of acoustic energy, has also been found after LSVT (Cannito et al., 2006). LSVT has been found to positively influence not only speech (Ramig et al., 2001; Sapir et al., 2002), but also non-speech activity, such as facial expression and swallowing (Fox et al., 2002; McFarland & Tremblay, 2006; Sapir et al., 2007; Spielman et al., 2003). LSVT is consistent with principles of skill acquisition and motor learning from the field of exercise science. These principles include high levels of physical effort, multiple repetitions, intensity, and simplicity (Fox et al., 2002). These principles have been shown to promote neural plasticity (Fox et al., 2006) and brain reorganization in animal models of PD (Fisher et al., 2004).

LSVT focuses primarily on the voice, in contrast to the traditional voice disorder treatment approaches that focus on other aspects of speech. According to Ramig (1995), the primary goal in LSVT is to increase loudness and decrease breathiness by increasing vocal fold adduction. Other goals of therapy include improving intonation by increasing cricothyroid activity and improving voice quality by increasing stability of vocal fold vibration (Ramig, 1995). It differs from other treatments in that it focuses on voice production that is intensive, and it habituates the patient to a high effort level.

Dromey and Ramig (1998) studied the effect of changes in sound pressure level (SPL) on the respiratory, phonatory, and articulatory subsystems of speech. Increased SPL is associated with increased loudness. They found that as SPL increased, a higher lung volume was used to initiate the louder speech (Dromey & Ramig, 1998). This study supports the

practice of LSVT. By focusing therapy on amplitude training, such as loudness, therapy can have distributed effects across motor systems.

The concept of LSVT, specifically training amplitude, has been applied to the limb motor system through a therapy known as Training BIG. Farley and Koshland (2005) found this to be a simple but effective intervention for improving speed-amplitude associations across the upper and lower limbs in patients with PD. After training, participants increased the amplitude of limb and body movements, which generalized to improved speed and balance (Farley & Koshland, 2005). Other pilot studies have been presented at conferences that integrate LSVT and Training BIG. This integrated approach is called Training BIG and LOUD. After receiving this treatment, participants with PD demonstrated increased vocal loudness and increased stride length. These findings suggest that amplitude training may facilitate organization across divergent motor systems (Fox et al., 2006). Increased complexity of the task may also promote neural plasticity (Fox et al., 2006). Fox et al. (2006) researched the potential of a single therapy target to encourage cross-system improvements, such as Training BIG and LOUD. The distributed effects across motor systems may be due to common underlying neural mechanisms or the stimulation of phylogenetically older neural systems.

Many PD patients have been found to have cognitive deficits (Mahler & Cummings, 1990). LSVT provides a therapy well-suited for those with cognitive decline due to the disease; goals and procedures are simple, redundant, and intensive (Fox et al., 2002). Single target treatments that cause cross-system improvements would be beneficial to PD patients because of their cognitive deficits.

The Present Study

Physical therapy can be a beneficial treatment for PD patients. The purpose of the present study is to evaluate whether there are spillover effects from lower limb training that influence speech and voice production of patients with PD. This study is part of a larger study that examines the influences of lower limb training on other motor activity.

As previously mentioned, Ramig and Ringel (1983) found that voice production is influenced by physical condition. Participants in better physical condition displayed less perturbation in voice production. Dromey and Ramig (1998) found a positive relationship between lung volume and SPL. Physical therapy improves physical condition, as reflected by several systems of the body, including the respiratory system. Increased physical exercise may increase lung function which may have an impact on voice production. After participation in the lower limb training, patients may improve in their cardiovascular health, which may be reflected in improved voice and even supraglottic speech performance.

Method

Participants

Six women with a mean age of 71 years and 4 men with a mean age of 57 years participated in the study. All patients were diagnosed by a neurologist as having mild to moderate idiopathic Parkinson's disease, with the time since diagnosis ranging from 3-15 years and Hoehn & Yahr stages ranging from 2-3. Participants in the current study also took part in a larger study of limb function conducted by Dr. Lee Dibble PT at the University of Utah. All participants volunteered to be in the present study and signed an informed consent document approved by the University of Utah Institutional Review Board.

Treatment

The lower limb training conducted by Dr. Dibble involved a high force and low metabolic resistance program called Resistance Exercise via Negative-Eccentric Work (RENEW). A seated stepping ergometer, which is a device that measures the work performed by exercising, was used for the RENEW training. Participants performed exercise on the ergometer 3 days a week for 12 weeks. Exercise time gradually increased over the first three weeks from 5-20 minutes and then remained at that duration for the remaining 9 weeks.

The RENEW training took place at the University of Utah Rehabilitation and Wellness Clinic. Speech recordings were made for each participant prior to the commencement and after the completion of training, under the direction of Dr. Dibble in the patients' medication-off and medication-on conditions. The recordings included the speaking tasks described below. These recordings were subsequently sent to Brigham Young University to be analyzed.

Speaking Tasks

Participants were instructed to take a deep breath and then sustain /a/ for five seconds. Participants read the sentence *The boot on top is packed to keep* to elicit productions of the corner vowels /i/, /a/, /u/, and /æ/ in a consonant-vowel-consonant syllable context.

This sentence was selected because it had a stress pattern that matches that of natural speech, and each word containing a vowel of interest received stress. The sentence *The boy gave a shout at the sight of the cake* was also read to elicit the diphthongs /ɔɪ/, /eɪ/, /aʊ/, and /aɪ/.

Each participant repeated both sentences five times and then read the first six sentences of the Rainbow Passage. Diadochokinetic (DDK) syllable repetition was also recorded for each

participant. Finally, participants were instructed to take deep breath and then sustain /a/ for as long as possible.

Instrumentation

During each of these tasks, the acoustic signal was recorded into a Macintosh computer via a USB headset microphone (Logitech) with a mouth-to-microphone distance of approximately 5 cm.

Acoustic Analysis

From the digital recordings of the sentence *The boot on top is packed to keep*, the first and second formant frequencies of the corner vowels were measured using Praat 5.0.47 and MATLAB 7.6.0.324 (R2008a). Figure 1 shows the Praat display for this analysis.

After the mean formant frequencies at the vowel midpoint were computed with Praat and MATLAB, the vowel articulation index (VAI) was calculated. A low value on this index reflects greater vowel neutralization, and a higher value reflects larger articulatory excursions. VAI was calculated with the formula $(F2i + F2æ + F1æ + F1α)/(F1i + F1u + F2u + F2α)$ for each condition (Roy, Nissen, Dromey, & Sapir, 2009). Thus, as the tongue moves more during articulation, the numerator terms would increase and the denominator terms decrease, thus raising the numeric value of the VAI.

To examine the rate and extent of tongue movement in the productions of diphthongs, the sounds /ɔɪ/, /eɪ/, /aʊ/, and /aɪ/ were extracted from five repetitions of the sentence *The boy gave a shout at the sight of the cake* using Praat. The first and second formants were

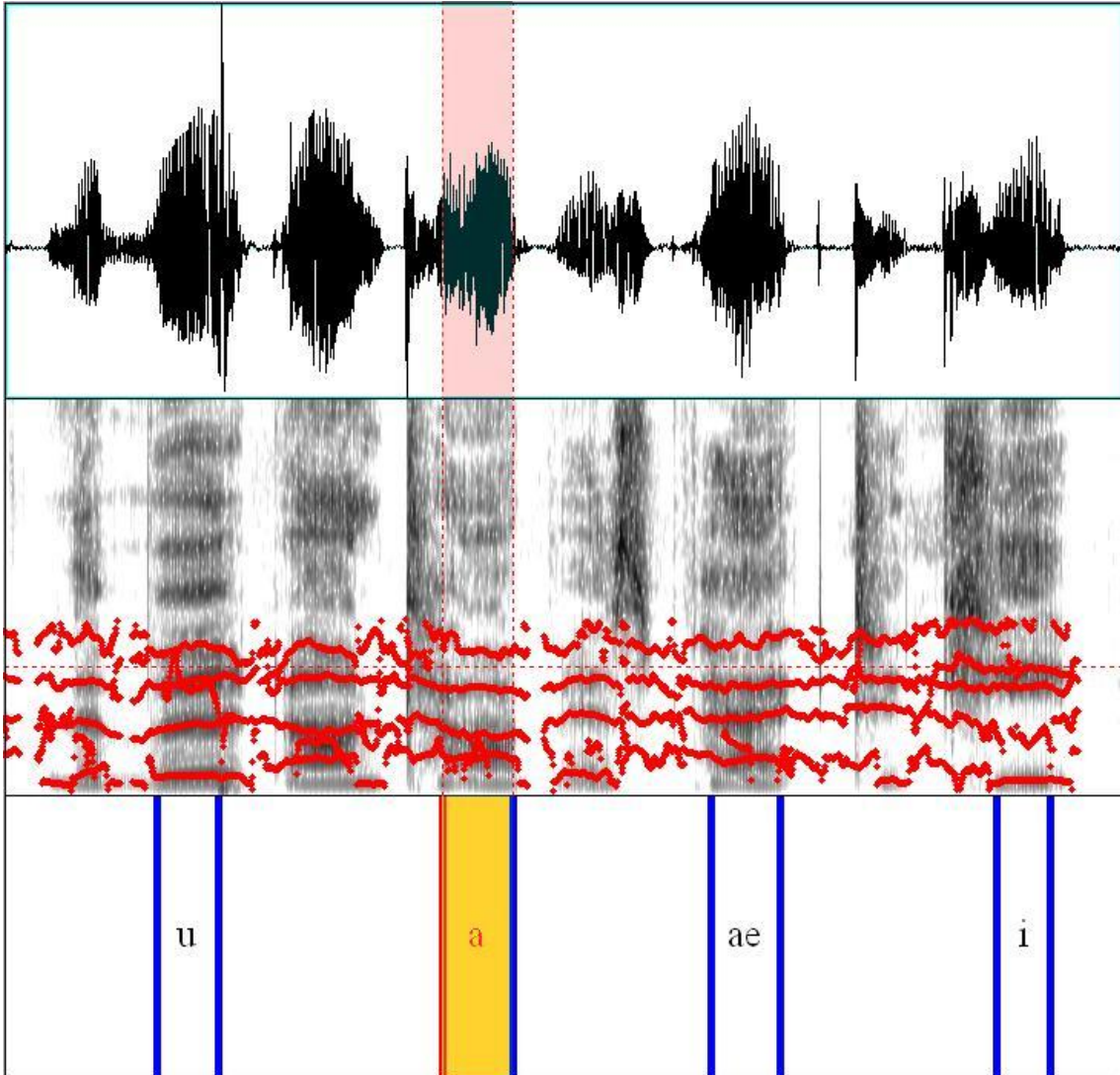


Figure 1. The Praat analysis display for the sentence *The boot on top is packed to keep.*

calculated using the same method as Dromey, Nissen, Roy, and Merrill (2008). Briefly, these formants were automatically tracked by Praat and were visually inspected for tracking accuracy and, where necessary, were hand-corrected prior to statistical analysis. For three individual vowel tokens and four individual diphthongs from the entire data set the first and second formants could not be tracked due to signal processing problems, thus they were not included in the analysis. Using values from the extracted formant tracks, average F1 and F2 frequencies were calculated for eight equally spaced and non-overlapping measurement intervals throughout each vowel's overall duration (t1-t8). Thus, t1 was an average of the formant values in the initial 12.5% of the vowel's duration. Onset and offset values for the diphthongs were calculated at 25% (t2-t3) and 75% (t6-t7), respectively. It was reasoned that the influence of the consonantal context would be limited by computing the onset and offset at these points. The transition slope of the diphthongs was determined by the frequency difference between the onset and offset values as a function of time. Figure 2 shows the analysis of formant tracks for the diphthong /ɔɪ/ in Praat.

Using the TF32 program (Milenkovic, 2000) the long term average spectrum (LTAS) was calculated for the Rainbow Passage that was read by each participant. This measure is of particular interest because Dromey (2003) has shown that statistical measures of the LTAS shape, referred to as *spectral moments* of the long term average spectrum, are sensitive to changes in voice quality associated with speakers with hypokinetic dysarthria. The first two spectral moments (mean and standard deviation) of the LTAS were used to indirectly assess the voice quality of participants in each condition.

Stop closure duration and voice onset time (VOT) were calculated using Praat to segment the desired portions of the signal from the DDK syllable repetition task. Voice onset

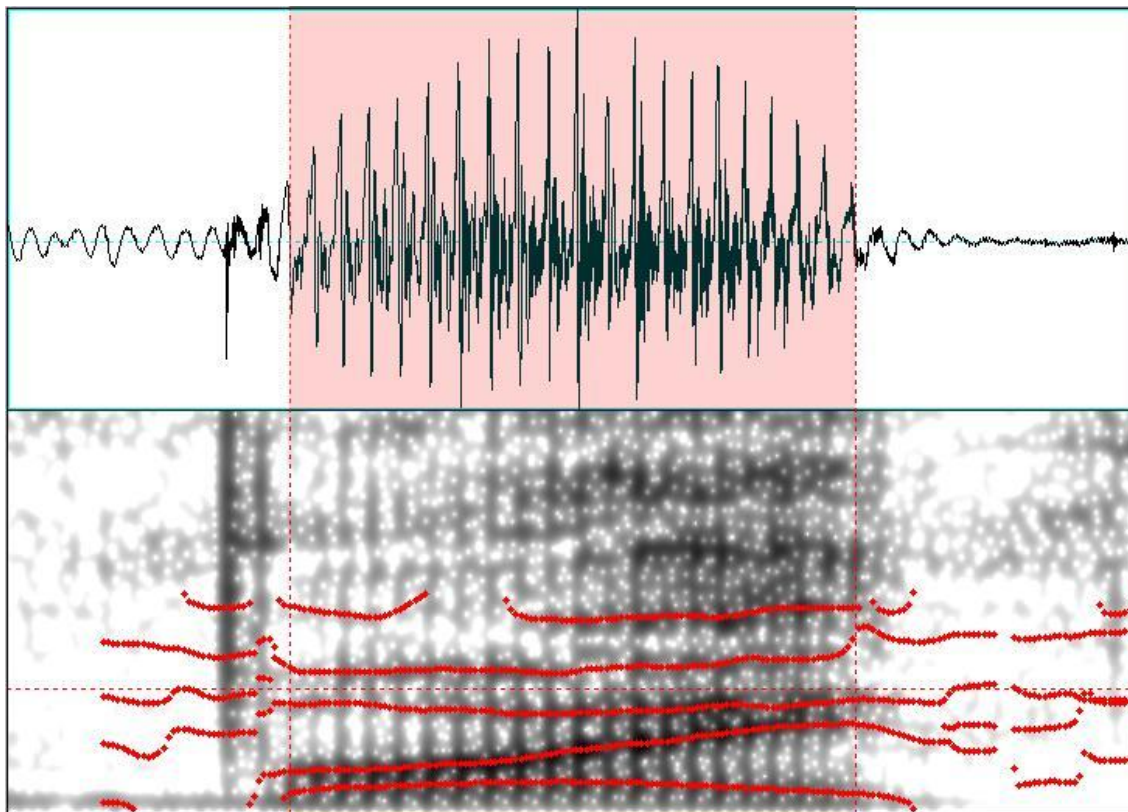


Figure 2. The Praat analysis display for the formant tracks of the diphthong /ɔɪ/.

time was defined as the time from the stop release to the end of the frication and aspiration of the stop. Repetitions two through six of the stop consonants /p/, /t/, and /k/ were averaged together to obtain a mean stop closure duration and VOT for each participant in each condition. Both the spectrographic display and the waveform were used to determine the boundaries of the stop closure.

Spirantization is defined as replacement of the stop gap with low-intensity frication (Duffy, 2005). It is indicative of incomplete occlusion or undershooting of the articulatory targets (Duffy, 2005; Kent, Weismer, Kent, Vorperian, & Duffy, 1999). Spirantization during the DDK task was quantified by computing a vowel stop closure index, hereafter referred to as the spirantization index (SI). This index was derived by using Praat to extract the average amplitude of the signal during stop closure and the average amplitude of the vowel. The difference between the two amplitude measures (in dB) was calculated. The procedure was repeated for repetitions 2-6 of /pə/, /tə/, and /kə/ produced by each participant. Figure 3 shows an example of a normal stop gap and figure 4 shows an example of a fully spirantized stop.

Statistical Analysis

SPSS 16 was used to test for differences in the dependent variables between the pretreatment and posttreatment and the on and off medication recordings. Repeated measures ANOVAs were performed for the vowel articulation index, formant slopes, long-term average spectrum, voice onset time, stop closure duration, and the vowel stop (spirantization) index.

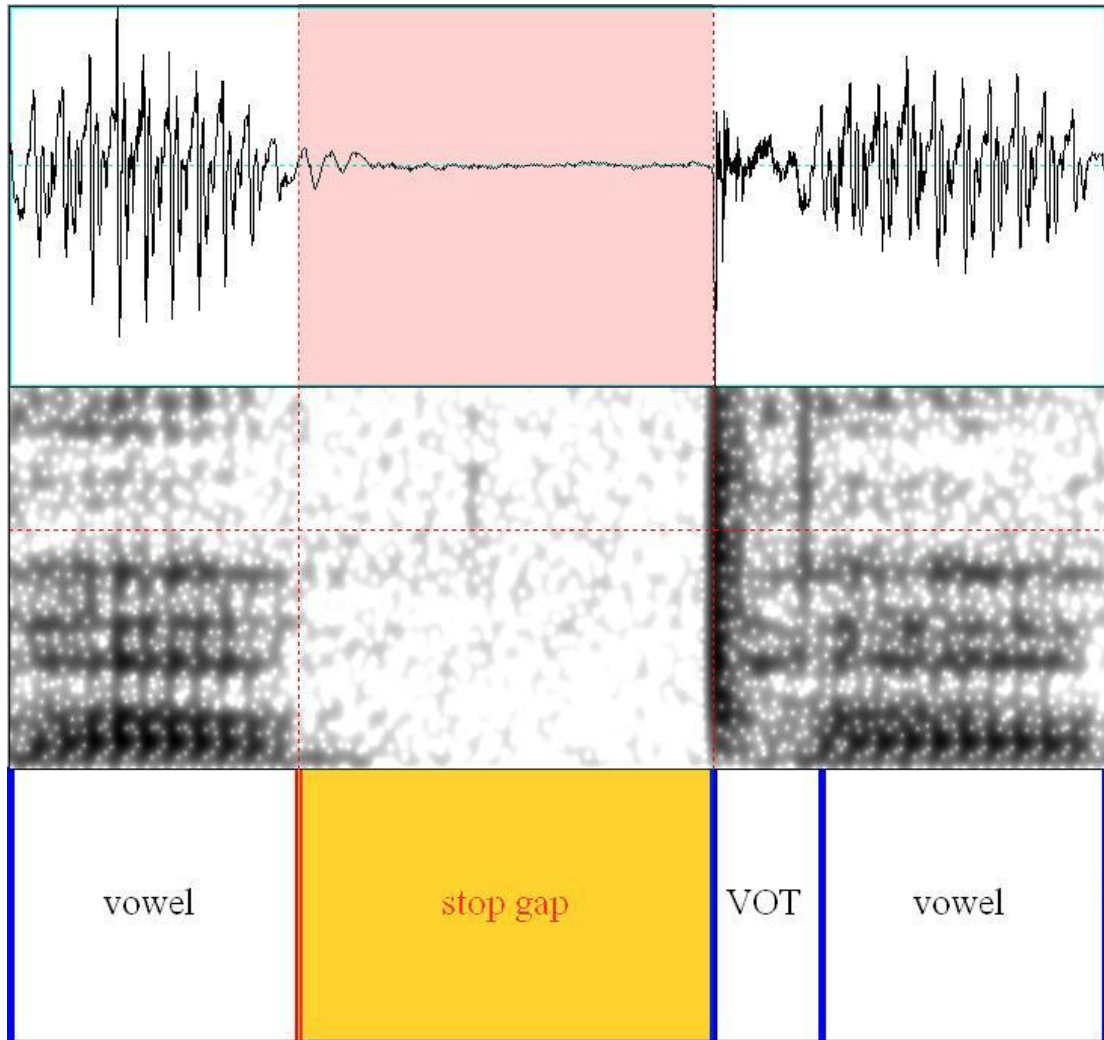


Figure 3. Praat analysis display for a normal stop gap and VOT segmentation from the DDK task.

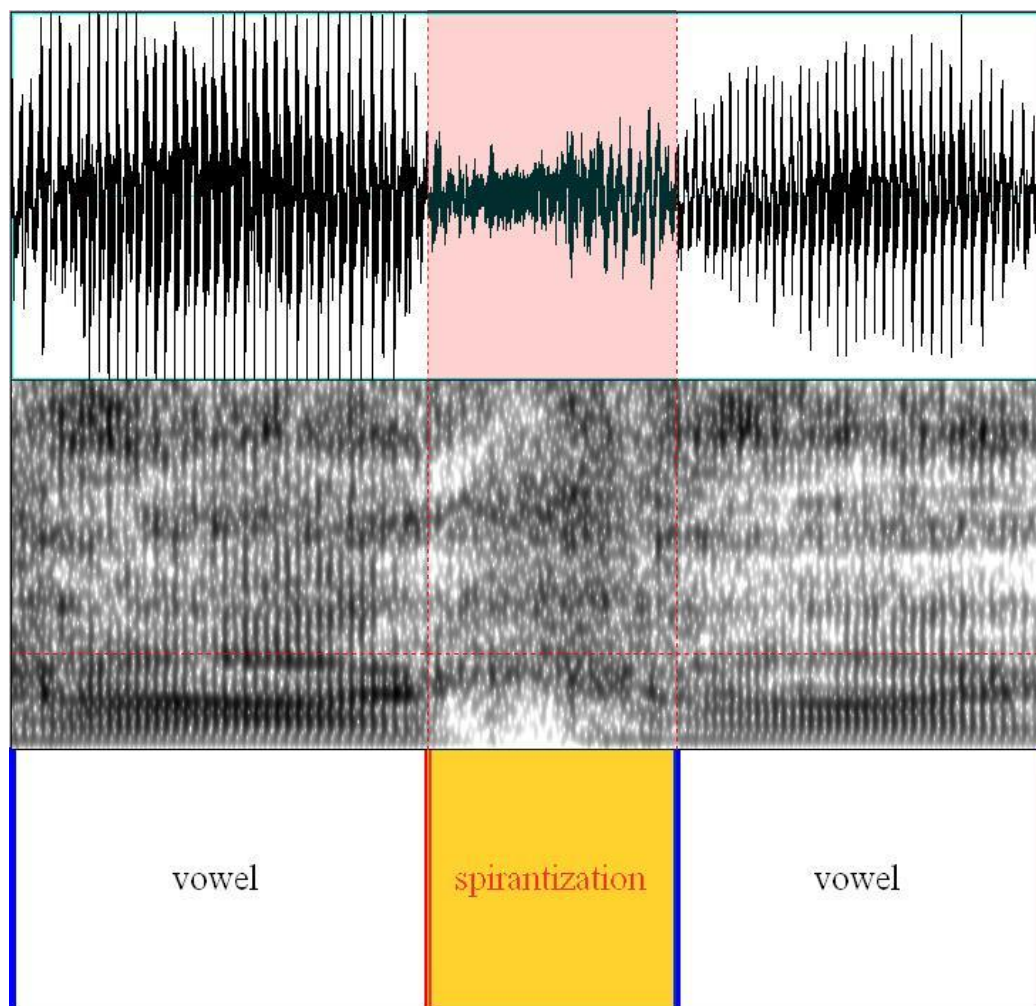


Figure 4. Praat display for a fully spirantized stop from the DDK task.

Results

Pretreatment Versus Posttreatment

Means and standard deviations for all variables before and after treatment are reported in Table 1 for the medication off condition and Table 2 for the medication on condition.

The ANOVA revealed a significant difference between pretreatment and posttreatment values of the spirantization index for the DDK repetition of /pə/ in the medication-on state, $F(1, 9) = 8.27, p = 0.02$. The speakers had lower values after treatment ($M = 14.22, SD = 2.59$) than before treatment ($M = 16.44, SD = 2.15$) for this syllable and condition, reflecting increased spirantization after the exercise program.

ANOVA testing revealed a significant difference between pretreatment and posttreatment values of the duration of the diphthong /aɪ/ in the medication-off condition, $F(1, 9) = 7.84, p = .02$. On average, the speakers had a slightly shorter diphthong duration after treatment ($M = 0.12, SD = 0.03$) than they did before treatment ($M = 0.13, SD = 0.03$).

Posttreatment Medication-on Versus Medication-off Conditions

The repeated measures ANOVA revealed a significant difference between medication-on and medication-off values of the spirantization index for the DDK repetition of /pə/ following treatment, $F(1, 9) = 7.41, p = .02$. As a group, the speakers had lower index values with the medication on ($M = 14.22, SD = 2.58$) than off ($M = 16.09, SD = 1.86$) for this syllable and condition, indicating increased spirantization when medicated.

Table 1

Means and SDs of Acoustic Variables for All Speakers in the Medication-off Condition

Variable	Context	Pre		Post	
		Mean	SD	Mean	SD
VAI		1.65	0.16	1.64	0.16
F1 slope	/ɔɪ/	-214.4	351.4	-307.7	264.5
	/aʊ/	151.1	680.6	210.6	662.0
	/aɪ/	-1150.1	717.9	-1114.1	732.3
	/eɪ/	-713.3	513.4	-662.3	468.1
F2 slope	/ɔɪ/	6363.3	1357.7	6299.9	1538.5
	/aʊ/	-2117.1	766.1	-2090.1	705.8
	/aɪ/	2903.3	1445.3	2908.5	1125.3
	/eɪ/	1487.0	929.2	1320.4	1016.1
LTAS	Spec mean	5.66	0.75	5.80	0.71
	Spec SD	2.66	0.35	2.56	0.34
Stop Gap	/p/	82.4	28.0	76.4	19.2
	/t/	63.8	13.9	60.9	14.2
	/k/	59.1	10.3	61.4	17.8
VOT	/p/	24.3	9.1	23.9	12.9
	/t/	31.2	9.0	28.7	7.4
	/k/	40.2	9.0	37.6	11.7
SI	/p/	17.7	3.0	16.1	1.9
	/t/	15.0	2.0	14.9	2.2
	/k/	17.6	2.6	17.3	2.9

Note. VAI = vowel articulation index; LTAS = long term average spectrum; VOT = voice onset time; SI = spirantization index.

Table 2

Means and SDs of Acoustic Variables for All Speakers in the Medication-on Condition

Variable	Context	Pre		Post	
		Mean	SD	Mean	SD
VAI		1.63	0.17	1.63	0.16
F1 slope	/ɔɪ/	-161.0	270.8	-190.5	295.8
	/aʊ/	218.4	618.9	371.2	649.0
	/aɪ/	-1082.0	666.4	-1053.0	705.5
	/eɪ/	-643.6	483.5	-702.3	429.8
F2 slope	/ɔɪ/	6154.6	1077.1	5880.1	1527.9
	/aʊ/	-2086.7	758.2	-2095.7	827.3
	/aɪ/	3056.6	1470.2	2693.4	1112.4
	/eɪ/	1366.7	743.7	1237.5	1004.0
LTAS	Spec mean	5.58	0.81	5.75	0.84
	Spec SD	2.65	0.43	2.59	0.34
Stop Gap	/p/	69.1	24.1	77.3	13.1
	/t/	62.8	16.9	58.4	12.8
	/k/	63.0	6.5	63.0	11.9
VOT	/p/	26.1	16.9	17.1	4.9
	/t/	35.5	9.8	32.4	11.0
	/k/	37.1	8.2	37.7	8.4
SI	/p/	16.5	2.2	14.2	2.6
	/t/	15.4	2.8	15.1	2.3
	/k/	15.9	3.9	15.8	3.4

Note. VAI = vowel articulation index; LTAS = long term average spectrum; VOT = voice onset time; SI = spirantization index.

The ANOVA revealed a significant difference between medication-on ($M = -18.50$, $SD = 33.80$) and medication-off ($M = -36.24$, $SD = 27.87$) values of the first formant transition of the diphthong /ɔɪ/, in the posttreatment condition, $F(1, 9) = 8.82$, $p = .02$. A significant difference between medication-on ($M = 731.70$, $SD = 89.11$) and medication-off ($M = 795.92$, $SD = 121.06$) conditions for the second formant was also found for this diphthong, $F(1, 9) = 5.78$, $p = .04$. The mean values for all speakers revealed more modest first and second formant transitions in the medication-on than the medication-off condition for this diphthong.

Discussion

The purpose of the current study was to investigate the effect of intensive lower limb training on the speech of participants with Parkinson's disease in medication-on and medication-off conditions. This involved the assessment of several speech acoustic measures.

Changes in Speech Acoustic Measures

Vowel articulation index. Tjaden and Wilding (2004) reported that reduced vowel space area is characteristic of individuals with PD as a result of smaller displacement of the articulators during speech. Vowel space area can be sensitive to inter-speaker differences in vocal tract anatomy, thus the VAI was used to evaluate any potential change in the production of the corner vowels in the sentence repetition context (Roy et al., 2009). Larger VAI values indicate greater articulatory movement. In the present study, it was hypothesized that intensive exercise, specifically lower limb training, would improve cardiovascular health, including more vigorous pulmonary function. It was hypothesized that such improvements might lead to increases in articulatory performance as reflected by the VAI

following treatment. However, there were no significant changes in the VAI following the exercise treatment. There were also no significant changes in this measure between the medication-on and medication-off conditions. These findings suggest that the exercise training program may not have resulted in increased pulmonary function, or that any respiratory changes were insufficient to promote cross-system effects from the lungs to the articulators.

Formant slopes. Forrest, Weismer, and Turner (1989) found that the formant transitions of speakers with PD were smaller than those of healthy geriatric individuals. Poluha et al. (1998) hypothesized that a reduction in rigidity and bradykinesia from PD patients' use of levodopa would permit faster articulatory changes and thus result in a greater F2 slope. The present study found significant changes between medication-on and medication-off conditions in the first and second formants of the diphthong /ɔɪ/ following treatment. However, the significant difference reflected generally *smaller* diphthong movements when the participants were in the medication-on state, which could be interpreted to represent poorer speech performance as a side effect of the medication. The present study did not find significant formant slope differences across the target diphthongs following the exercise program, thus there was no evidence of either increased or decreased articulatory movements in connection with the treatment.

Long term average spectrum. With regard to the spectral distribution of energy in speech, Dromey (2003) found that a low spectral mean and standard deviation (SD) in PD generally suggested a weak upper harmonic structure, with most of the energy in the lower frequencies of the voice. In contrast, normal speakers had a higher SD, indicating a wider spread of energy across the spectrum. Louder phonation is associated with a less steep decay

in energy from the lower harmonics to the upper harmonics (Dromey, 2003). Thus, a weak upper harmonic structure suggests a decrease in clarity and voice quality. In the current study we hypothesized that there might be an increase in respiratory activity as a result of the intensive lower limb training, which might result in louder speech and with it a wider spread of acoustic energy across all harmonics. However, the data revealed no statistically significant differences in the values of LTAS between the pretreatment and posttreatment or the medication-on and medication-off conditions. These results suggest that the anticipated improvements in pulmonary function either did not occur or were insufficient to influence speech production.

Voice onset time. Forrest et al. (1989) documented increased VOTs for Parkinson's disease patients when compared to normal geriatrics. The current study found no statistically significant VOT differences between pretreatment and posttreatment or medication-on and medication-off conditions, suggesting that neither factor was influential on this acoustic measure of laryngeal-articulatory coordination.

Stop gap duration. Kent and Read (2002) noted that a stop gap is "a region of reduced energy, typically between 50-150 ms in duration" (p. 144). Tjaden and Watling (2003) found that stop gap durations were shorter for individuals with PD when compared to healthy controls, but the difference was not significant. In the current study, there was no statistically significant difference in stop gap duration as a function of treatment or medication condition.

Spirantization. Several studies have documented the presence of spirantization in the speech of individuals with PD (Duffy, 2005; Kent et al., 1999). In the present study, a lower index value would suggest more severe spirantization. The speakers were found to have

lower spirantization index values in the medication-on condition after treatment for the DDK /pə/ syllable when compared to pretreatment values. Lower values were also found in the medication-on condition following treatment in the /pə/ syllable when compared to the medication-off condition. The lower spirantization index values suggest that the articulators were forming incomplete closures; this implies that the participants' articulatory function decreased in the medication-on condition and following treatment, which is the opposite of what would be expected in measures of limb performance.

General Discussion

The current study found no significant effects on acoustic measures of speech as a result of participating in the exercise regimen RENEW. The few significant differences were between medication-on and medication-off conditions following treatment. The results of the current study showed some variability in the effect of intensive lower limb exercise on speech in medication-on and medication-off conditions; in general, participants showed a slight worsening of speech after treatment and when medicated.

Participation in RENEW training did not seem to have a significant overall impact on the speech of those with PD. Thus, it is assumed that there were no significant cross-system effects, such as increased or decreased speech respiratory drive or articulatory precision. The findings of the current study suggest that the RENEW training program may not have sufficiently changed respiratory drive to impact speech. Thus, there was a lack of carry-over from the lower limbs to speech performance. Slightly more significant were the medication-on versus medication-off results. The current study found a slight worsening of speech following treatment and in the medication-on condition, which supports other studies that have found minimal effects of levopoda on the speech of individuals with PD (Goberman et

al., 2002; Schulz & Grant, 2000; Trail et al., 2005). Although the current study found a slight worsening of speech, there was substantial variability in the medication-on and medication-off results. Previous studies have found similar levels of variability (Schulz & Grant, 2000; Trail et al., 2005), thus further research appears warranted to examine the impact of medication on speech in PD.

Future Research Directions

One weakness of the current study was the limited number of participants. Although statistical analysis was performed on the data from 10 speakers, the likelihood of finding any effects from the training or medication would have been greater with a larger sample. Another limitation of the current study was that the participants were not recruited for participation on the basis of having a speech disorder. For future studies it would be beneficial to select a group of participants with moderate levels of hypokinetic dysarthria, since not all individuals with PD are dysarthric (Logemann et al., 1978). It would also be beneficial to recruit participants based on the number of years since PD diagnosis and the severity of PD.

The RENEW physical therapy program used in this study may not have been sufficient in intensity to lead to increases in the respiratory drive for speech. In future studies, it may be beneficial to select a physical therapy program with an increased number of training sessions per week and increased duration of each training session. Greater understanding of the impact of physical therapy on the speech of those with PD would further our understanding of cross-system effects. As rehabilitation professionals, such as physical therapists, occupational therapists, and speech therapists, work to help those with PD they could take advantage of any positive cross-system effects. This knowledge would

promote greater interaction between professionals and may lead to a more coordinated and effective intervention approach.

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