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HOW INDIVIDUALS WITH PARKINSON'S DISEASE MODIFY THEIR SPEECH
IN A REPETITION FOR CLARIFICATION

by

Lynn Watkins

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 Audiology and Speech-Language Pathology

Brigham Young University

December 2005

BRIGHAM YOUNG UNIVERSITY

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ABSTRACT

HOW INDIVIDUALS WITH PARKINSON'S DISEASE MODIFY THEIR SPEECH IN A REPETITION FOR CLARIFICATION

Lynn Watkins

Department of Audiology and Speech-Language Pathology

Master of Science

The speech of individuals with Parkinson's disease (PD) is typically characterized as lacking in proper prosody because of its monopitch and monoloud quality, in addition to its reduced intensity. These qualities make it difficult for others to understand speakers with PD. The purpose of the current study was to identify what individuals with PD would do vocally, if anything at all, to improve speech production following a simulated misunderstanding of what they had just said. The study evaluated the performance of 5 individuals with PD and compared their performance to 5 age- and sex-matched controls. Specifically, measures of vocal intensity (loudness), fundamental frequency (pitch), and utterance duration were made for repetitions of a 'misheard' phrase. In one experimental condition noise was presented through headphones to induce the Lombard effect.

Both individuals with PD and controls used increased duration as a means of enhancing clarity in a repetition. Fundamental frequency (F0) and sound pressure level (SPL) were not consistently modified in repetitions for clarification. Under most speaking conditions, individuals with PD and controls had similar F0 and SPL. Individuals with PD, like the controls, responded to the presentation of masking noise by increasing their fundamental frequency and their intensity. Therefore, not all individuals with PD exhibit difficulty using prosody.

ACKNOWLEDGMENTS

Mother Teresa once said, “We can do no great things, only small things with great love.” I recognize my thesis is a small contribution to the world, but it was done with great love for my mother, grandmother and others who suffer from Parkinson’s disease. It would not have been possible if I had not received direction and confidence from many people. I want to thank to my committee chair, Dr. Christopher Dromey for his steady support. Dr. Dromey’s positive outlook kept me motivated. I want to thank the committee members for their help and guidance. I could not have finished my thesis without their assistance. My gratitude goes to my husband who I thank for always standing by my side, and helping me whenever possible. I thank those individuals willing to devote of their time to participate in this study; we could not further education and knowledge about how disorders affect the human body without the willingness of participants. To my friends, family and classmates, thank you for inspiring me with encouraging acts and words all along the way.

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Introduction

Parkinson's Disease

In 1817 James Parkinson first described the illness of Parkinson's disease (PD; Goetz, 1992). It is now evident how widespread PD is, with over 1.5 million people affected in the United States alone (Barlow, Iacono, Paseman, Biswas, & D'Antonio, 1998). Tanner, Goldman, and Ross (2002) raised the concern that the prevalence of PD could triple in the next 50 years, since its incidence increases with age and the population of this country is aging.

Although the cause of PD is unknown, years of research have helped clarify some details of the pathophysiology. PD is a neurodegenerative disorder, which affects the basal ganglia and the substantia nigra (Hurtig, 2000). As PD progresses, the dopamine producing neurons progressively degenerate (Hurtig, 2000). This causes a chemical imbalance of the neurotransmitters dopamine and acetylcholine, with acetylcholine being overactive and dopamine underactive (Goldmann & Horowitz, 2000).

Disease effects on limb and axial function. Dopamine is an inhibitory neurotransmitter that is involved in the regulation of skilled movements (Brookshire, 2003). The symptoms of PD only emerge after 60% of the dopamine-producing neurons have become necrotic. The most common of these symptoms are bradykinesia, resting tremor, rigidity, and postural instability. Bradykinesia is defined as "difficulty initiating volitional movements" (Brookshire, 2003, p. 528). Resting tremor is characterized as involuntary shaking movements of the body, which are present at rest and lessen or disappear during motion (Dewey, 2000). These tremors are low frequency (4-6 Hz) oscillations (Wichmann & Juncos, 1999). Rigidity is resistance to passive movement

(Ahlskog, 2000a), which is detected when an examiner moves a patient's limbs. Postural instability is present in the later stages of PD and involves severe shuffling, freezing, and falling (Hurtig, 2000). Postural instability occurs due to a loss of postural reflexes (Dewey, 2000). Falling is the most disabling feature of postural instability, which occurs because the lower body does not adjust to changes in the position of the upper body (Hurtig, 2000). Typically patients with PD also experience decreased ability in fine motor movements, as well as slowness and lack of power in limb movement (Adams, 1996).

Abnormal gait is another defining characteristic of PD (Ahlskog, 2000a). Festination is a gait difficulty which includes a short stride, and often a tendency to lean forward (Ahlskog, 2000a; Dewey, 2000). As the patient leans forward, his or her stride accelerates, as he or she takes smaller, shuffling steps (Dewey, 2000). A difficulty that arises with this shuffling step and advancing acceleration is that the patient is not able to lengthen his or her stride with the increased acceleration, and can fall (Dewey, 2000). Another gait difficulty is described as freezing (Dewey, 2000). This occurs when the patient experiences an unexpected stop in the forward movement of his or her walk (Dewey, 2000). It can become a frustrating experience as he or she may struggle to start steps again unsuccessfully (Dewey, 2000).

Disease effects on speech and voice. Sometimes the first symptom of a neurological disorder is a speech deficit (Duffy, 2000). "Speech is the most complex of innately acquired human motor skills," and requires the coordination of up to 100 muscle actions (Duffy, 2000, p. 35). Because speech is so complex, many areas are vulnerable to neurological damage. If a patient does exhibit speech deficits as one of the first

symptoms of a neurological disorder, his or her speech characteristics can be used as signs to localize or identify the pathophysiology of the underlying neurological condition (Duffy, 2000).

The motor speech disorder that accompanies PD is called hypokinetic dysarthria (Duffy, 1995, 2000). Of the different etiologies of hypokinetic dysarthria, PD is the most common (Brookshire, 2003; Duffy, 1995). Hypokinetic dysarthria can affect many of the muscles involved in speech, including those in the respiratory system, the larynx, the pharynx, the tongue, and the face. These muscles may be weak, their actions uncoordinated, or they may exhibit abnormal tone (Duffy, 2000). Hypokinetic dysarthria leads to speech changes which result in decreased intelligibility.

Voice is one of the first areas affected in hypokinetic dysarthria (Aronson, 1985). Logemann, Fisher, Boshes, and Blonsky (1978) stated that it is most common for patients with PD to experience voice deficits, observing that 89% experience some level of phonatory loss. PD speech is characterized as lacking emotion and expression because of its monopitch and monoloud quality (Duffy, 1995; Stemple, Glaze, & Klaben 2000). The most common voice deficits experienced by patients with PD are breathiness, hoarseness, roughness, and tremor (Logemann et al., 1978).

Another common voice deficit in PD is hypophonia, or reduced intensity in speech. A breathy and weak voice with some degree of vocal tremor is not uncommon in PD (Stemple et al., 2000). Brookshire (2003) suggested that reduced muscle coordination can lead to a breathy or strained voice. The vocal folds do not fully adduct because the muscles of the larynx are rigid and resist vibration. Duffy (1995) noted that many individuals will experience bowed folds or weak vocal fold approximation.

Another problem with voice in hypokinetic dysarthria is inadequate respiratory support. This lack of support may be linked to rigidity in the respiratory muscles (Brookshire, 2003). Insufficient vocal fold adduction and rigid laryngeal muscles in combination with weak respiratory function contribute to reduced vocal intensity (Brookshire, 2003).

Some patients with hypokinetic dysarthria also experience imprecise articulation, which may be a consequence of rigidity and restricted range of motion in the tongue, lips, and jaw (Brookshire, 2003). About 45% of patients with PD have an articulation disorder (Logemann et al., 1978). The severity of hypokinetic dysarthria typically increases with disease progression (Uitti, 2000), and it has been suggested by Logemann et al. (1978) that deterioration of articulation usually occurs later than voice disturbance.

Rate of speech is another vulnerable area in hypokinetic dysarthria, although it is not as frequently affected as voice, occurring in only about 20% of patients with PD (Logemann et al., 1978). Slow speech and rapid speech are both found in individuals with PD. Slow speech is commonly seen in many different types of dysarthria, whereas a rapid rate of speech is unique to hypokinetic dysarthria in PD (Darley, Aronson, & Brown, 1975).

Another fairly common characteristic of hypokinetic dysarthria in PD is repetitive speech or atypical dysfluency (Benke, Hohenstein, Poewe, & Butterworth, 2000; Koller, 1983). Dysfluency accompanying PD is different from developmental stuttering, which is hard, tense, and often accompanied by effortful blocks. A developmental stutterer often feels anxiety about speaking, and often has secondary characteristics like head nods

and eye blinks. Dysfluency in PD is effortless stuttering, which is usually void of secondary characteristics or anxiety (Koller, 1983).

Medical Treatments

Although there is no cure for PD, a variety of treatments are available. Several dopaminergic drugs are used for individuals with PD, which help restore the balance between dopamine and acetylcholine (Duffy, 1995; Goldmann & Horowitz, 2000). As noted by Brookshire (2003), one of the most common treatments for PD is levodopa, yet it works for only about two thirds of patients. The body is able to use levodopa as a precursor and convert it into dopamine. Levodopa is particularly useful for reducing tremor and slowing the deteriorating effects of PD upon the brain.

Another dopaminergic medication is deprenyl, which halts the breakdown of dopamine (Brookshire, 2003). Bromocriptine acts directly on the dopamine receptors (Duffy, 1995) and mimics the effects of dopamine (Brookshire, 2003). Anticholinergic drugs are often chosen to focus on treating resting tremor and akinesia (Duffy, 1995).

Dopaminergic drugs alleviate many of the symptoms experienced by individuals with PD, but they cannot relieve all symptoms. Another concern is that the disease continues to progress despite treatments (Ahlskog, 2000b). In fact, over time most patients build resistance to medications and will reach a point when the medications are no longer effective against the symptoms and signs of PD (Ahlskog, 2000b; Brookshire, 2003). After prolonged use of these drugs, patients will start to experience the so-called 'on-off' response (Duffy, 1995). This is manifest as a sudden, sharp decline in the drug's control of the symptoms in the time following its administration, rather than a subtle decline in its effectiveness before the next dose. The dopaminergic medications can have

serious side-effects, including dyskinesias, dystonias, and other involuntary movements, as well as confusion (Ahlskog, 2000b; Duffy, 1995).

During the last few years, renewed consideration has been given to neurosurgical approaches in the management of PD. The development of levodopa in the 1960s had led to a decline in surgical intervention. However, the realization that drugs eventually lose their effectiveness, and often result in debilitating side-effects, has brought about increased interest in alternative treatments. Recent advances in technology have led to improved surgical methods, which have also benefited from an increase in the understanding of the pathophysiology of PD.

One of the surgeries for PD is pallidotomy, in which destructive lesions are made to the parts of the globus pallidus, often by freezing or burning neural tissue. Tremor and rigidity have been reduced through pallidotomy surgery (Brookshire, 2003; Laitinen, Bergenheim, & Hariz, 1992b). One specific procedure is Leskel's stereotactic pallidotomy, where the lesion is made in the posteroventral part of the pallidum (Laitinen, Bergenheim, & Hariz, 1992a).

Many studies have shown that Leskel's pallidotomy is effective in reducing tremor as well as other Parkinson symptoms (De Bie et al., 2001; Dogali et al., 1995; Laitinen et al., 1992a). In the Dogali et al. (1995) study there was significant reduction of rigidity, bradykinesia, resting tremor, and ambulation difficulty, resulting in a less dramatic "off" stage. In addition they found that dyskinesias caused from levodopa type medications significantly subsided. Another study showed that unilateral pallidotomy, contralateral to the site of lesion, brought about significant improvements in tremor, akinesia, and rigidity during the "off" stages (De Bie et al., 2001). During the "on"

stages there were reductions in dyskinesias (De Bie et al., 2001). This study also showed that pallidotomy can be effective in eliminating gait disturbances and postural instability. Laitinen et al. (1992a) found that the results from pallidotomy were lasting. Some adverse effects can occur following pallidotomy including brain hemorrhage with hemiparesis, facial paralysis, as well as contralateral homonymous hemianopsia (De Bie et al., 2001; Laitinen, et al., 1992a).

Thalamotomy is a similar surgery, in which part of the thalamus is destroyed (Clinical Neuroscience, Inc., n.d.). The main goal in thalamotomy is to reduce tremor. As well as reducing tremor, thalamotomy has also been found helpful in eliminating rigidity (Tasker, DeCarvalho, Li, & Kestle, 1996). Tasker et al. (1996) were able to study patients who underwent bilateral and unilateral thalamotomies. Shortly after their thalamotomy, 83 of the 86 patients were experiencing a minimal tremor, and 59 of the 86 were experiencing minimal rigidity (Tasker et al., 1996).

Another surgery that can help to eliminate Parkinson symptoms is deep brain stimulation (DBS). During DBS a multipolar electrode is implanted into either the ventral thalamus, internal globus pallidus, or subthalamic nucleus (Clinical Neuroscience, Inc., n.d.). When the electrode is stimulated, tremor and other Parkinson symptoms decrease or disappear entirely. The stimulation comes from a permanently implanted device, much like a cardiac pacemaker, positioned below the clavicle. The patient can control this unit by turning it on or off with a magnet (Clinical Neuroscience, Inc., n.d.). When the unit is turned on it causes high frequency stimulation, above 50 Hz, which can provide the same type of response as a lesion to the brain, such as those from pallidotomy and thalamotomy (Benabid, Ni, Chabardes, Benazzouz, & Pollack, 2001). In fact,

benefits from DBS can even exceed those from lesion surgeries. Schuurman et al. (2000) found that DBS of the thalamus was more effective in reducing tremor than thalamotomy.

One of the benefits of DBS over pallidotomy and thalamotomy is its reversible or adjustable nature, which leaves the basal ganglia intact (Benabid et al., 1991). Benabid et al. (1991) found that even when there were adverse side effects to DBS of the thalamus, these side effects could be minimized by discontinuing stimulation. This is not an option for those who undergo thalamotomy or pallidotomy, where neural tissue is destroyed.

The results from DBS depend upon where the electrodes are placed within the brain. For example, when the electrodes stimulate the ventral intermediate nucleus of the thalamus there is effective reduction in tremor in individuals with PD (Benabid et al., 1991). DBS in the subthalamic nucleus (STN) reduced tremor in addition to rigidity during periods without levodopa medication (Krack et al., 2003). Even up to five years after the surgery there was still a significant reduction in tremor and rigidity (Krack et al., 2003). DBS of the STN was very effective in reducing dyskinesias while patients were taking levodopa medications (Krack et al., 2003). Parkinson's patients in this study also decreased the dosage of levodopa medications for five years post-operation (Krack et al., 2003). Benjjani et al. (2000) found that the optimal condition was with STN stimulation and levodopa therapy in concurrence, rather than either treatment individually.

Brookshire (2003) described another type of surgery, which is experimental and highly controversial. It is called fetal tissue transplant. The procedure of this surgery is to implant dopamine-producing tissue from human fetuses into the brains of patients with PD (Brookshire, 2003). The goal of this surgery is to increase dopamine production in the brain (The National Parkinson Foundation, 2003).

The Effects of Medical Treatments on Speech

The most commonly used treatment for reducing the symptoms of PD is oral levodopa (Cahill et al., 1998; Brookshire, 2003). However, the effect of dopaminergic medications upon speech remains unclear. Cahill et al. (1998) found that maximum lip pressure and labial fine force control improved in patients with PD when under the influence of oral levodopa during speech and non-speech tasks. Cahill et al. found that their study was consistent with others in reporting increased articulatory function after the use of levodopa. Not only did articulation improve, but also other studies have found that voice and overall intelligibility improved with levodopa therapy. For example, Adelman, Hoel, and Lassman (1970) found that articulatory function, intelligibility, loudness, ability to sustain phonation, pitch, and voice quality were all improved as a result of levodopa therapy. Wolfe, Garvin, Bacon, and Waldrop (1975) found significant improvements in voice quality, pitch change, and articulation with levodopa therapy. They also found a correlation between improved speech and improved overall physical status due to levodopa therapy. The Cahill et al. (1998) study found that a possible drawback to oral levodopa is that the effects on speech only lasted for a few hours post-treatment. Another drawback to levodopa therapy is that speech can manifest a sharp decline in performance for those who have the on-off fluctuations when taking dopaminergic medications.

There are some who have found that levodopa does not help speech. In a double-blind placebo-controlled design, Wang, Kompoliti, Jiang, and Goetz (2000) did not find significant improvements in speech and voice under dopaminergic treatment. Levodopa is not found to help repetitions in repetitive speech phenomenon (Koller, 1983), nor does

it significantly modify rate (Wolfe et al., 1975). It is also not as effective as other treatments for modifying voice. Goberman, Coelho, and Robb (2002) found that there were no significant group changes in measures of speech and voice between medicated and non-medicated states, yet there were a few individual patient differences. Perhaps this is why one can find both evidence supporting and opposing levodopa effects on speech; changes may happen to some individuals, but group effects are not always seen.

Surgery is another option for improving the speech of patients with PD, yet individuals do not frequently seek surgery to alleviate speech deficits alone. The outcome of surgeries on speech varies as well. Although pallidotomy is often effective in improving limb and axial movements, Barlow et al. (1998) did not find consistent or significant speech changes in their study. The only improvements were self-reported or observed anecdotally by examiners. The patients who did experience improvements in speech reported that it was easier to move their lips for speech and they were observed to have relaxed faces. In addition, improvements were seen in eating and drinking as well as in measurements of laryngeal resistance (Barlow et al., 1998). In contrast to these modest improvements, Theodoros, Ward, and Murdoch (2000) found that with both unilateral and bilateral pallidotomy speech worsened in intelligibility post-surgery.

Some researchers have found improvement in speech after pallidotomy. For example, Laitinen et al. (1992b) conducted a study of 48 patients with PD. Post-pallidotomy there were three who had severe deficits in speech intensity and eight who were moderate, while the rest had a mild or no speech dysfunction. An average of 30 months after ventroposterolateral pallidotomy surgery, measures were made again of speech intensity. At this point, there were none who had severe hypophonia, and only

five who had a moderate reduction in speech intensity. At the follow-up, the remainder had mild or no speech dysfunction; the number of those with no speech dysfunction increased by six.

Thalamotomy may not improve speech and has even been found to make speech worse (Tasker et al., 1996). Tasker et al. found that increased dysarthria was the most frequent complication of bilateral thalamotomy, with 27% experiencing increased dysarthria post-surgery in their study. Another study by Linhares and Tasker (2000) noted that only 20% of patients had any speech improvements after thalamotomy, while 47.5% had worse speech. There is, therefore, not much evidence to suggest that thalamotomy leads to improvements in speech.

The effects of DBS on speech still need further research, because the published data so far include substantial variability in the outcomes. Some research reports little effect of DBS on speech (Bejjani et al., 2000; Krack et al., 2003). Krack et al. (2003), using the Unified Parkinson's Disease Rating Scale (UPDRS), found significant improvements in all motor functions with exception of speech. Speech did not improve during the off-medication stages in individuals after DBS of the STN (Krack et al., 2003). Another study by Bejjani et al. (2000) found that there were some mild improvements for patients with PD on their speech scores on the UPDRS after DBS stimulation (either with or without levodopa medication), yet these scores were still similar to scores seen with levodopa medication alone.

Other research has found that there can be improvements in speech after DBS. After STN DBS, significant improvements were reported in speech measures on the UPDRS, maximum voluntary force of the lips and tongue, and as well as significant

improvements in other articulatory measures (Gentil, Garcia-Ruiz, Pollack, & Benabid, 1999). Another study showed that the speech score on the UPDRS was improved after STN stimulation in DBS (Gentil, Chauvin, Pinto, Pollack, & Benabid, 2001). In this same study, the DBS patients were compared with individuals with PD who had not undergone DBS. The DBS patients received better scores on some voice and speech measures. For example, DBS patients had longer maximum phonation times, and had faster diadokokinetic rate (on the sequence /pʌtʌkʌ/).

Another intervention, which is used to specifically treat the voice of individuals with PD, involves the injection of collagen into the vocal folds (Goldmann & Horowitz, 2000). These injections can reduce hypophonia by making the vocal folds thicker, and more easily able to meet at midline for phonation. This treatment was found to be effective for patients with PD who have hypophonia in two recent studies (Berke, Gerratt, Kreiman, & Jackson, 1999; Hill, Jankovic, Vuong, & Donovan, 2003). In the Hill et al. (2003) study 17 patients with PD who had severe hypophonia were given an average of 3.0 collagen injections with an average of 13.8 weeks between injections. On a post-treatment questionnaire each patient reported improvements on all six voice quality questions including parameters such as loudness, clarity, intelligibility, and quality. In addition, these patients showed significant improvements in their scores on the Glasgow Benefit Inventory, which uses 18 questions to assess health quality after otorhinolaryngological treatment.

A further treatment option is speech therapy. Yorkston (1996) concluded that individuals with dysarthria benefit from speech language pathology services, based on her survey of both scientific and clinical reports. There are, however, limitations in the

amount of change that results from behavioral intervention. Goldmann and Horowitz (2000) found that speech therapy can help maintain vocal function, but that large improvements seldom occur. This implies that soon after a person is diagnosed with PD it would be wise to enroll in SLP services so that he or she can maintain the current voice, rather than enrolling later and trying to reverse the effects of PD on communication.

The areas that speech therapists often address include the following: control of volume, articulation, self-monitoring, proprioception, relaxation, breath control, pacing, intonation, prosody, respiration, voice, intelligibility, pitch, loudness, and phonation (Yorkston, 1996). These areas of focus are used in many different combinations. Yorkston's (1996) studies have found that therapies that focus on voice and respiration, such as the Lee Silverman Voice Treatment, are more effective than those that focus on respiration alone. Portable pacing boards and delayed auditory feedback devices are therapy techniques that can help compensate for some of the dysarthric deficits in speech, but they do not make speech normal. The biggest problem in speech therapy for individuals with PD is they may show significant improvements in performance in the therapy room, but the effects are not generalized beyond the clinic (Yorkston, 1996). This is perhaps another reason why Goldmann and Horowitz (2000) concluded that large changes did not frequently occur after speech therapy.

Impaired Sensory Function

In PD research there has been a primary emphasis on the motor deficits resulting from this condition. However, basal ganglia dysfunction may also influence sensory processing. Some research into sensory processing dysfunction has focused on

conditions of external cueing versus self-cueing. An external cue would be something beyond one's internal senses, a source from outside of the body that allows the individual to make judgments about his or her performance. For example, individuals with PD who have disordered gait have used external cueing to assist them. Two types of disruption to gait are seen; some patients experience freezing resulting in a slow cadence, while others experience festination which leads to reduced stride length and increased cadence.

Dewey (2000) describes external cues used by individuals with PD to assist them out of a "freezing" moment. He said they are more successful at attempting to initiate their walk again if they do things like "counting a walking cadence out loud, visualizing marching in their mind, or stepping over a nearby object on the floor" (Dewey, 2000, p. 76). In fact a modified walking stick has been created which allows the patient to place a visual platter on the floor and maintain a more normal gait by stepping over the platter (Dewey, 2000). This walking stick with a platter is an external cue. It gives the individual something to assist them in judging the stride of their steps. Individuals with unimpaired gait rely upon self-cueing to assist them in determining their stride. They use and process sensory information about their velocity, the terrain they are walking upon, objects in the way, and other factors to make modifications to their gait.

The phenomenon of external cues assisting those with gait disturbance was documented by Howe et al. (2003). They found that individuals with PD whose gait and cadence was inhibited by PD responded to auditory cues. After finding the patient's preferred cadence they presented an auditory metronome signal at 85%, 92.5%, 107.5% and 115% of the patient's preferred cadence. Patient cadence and velocity measures were significantly altered by increasing the presented rate at 107.5% and 115% or by

decreasing to 85%. Under self-cueing conditions, Parkinson patients experienced impaired ability to walk with “normal” gait. Yet, when given an external cue that assisted them in counting a cadence they were able to alter their gait.

Other disordered movements experienced in PD have shown improvement under conditions of external cueing. Mak and Hui-Chan (2004) found that individuals with PD were able to improve their sit-to-stand ability with audio-visual cues. In this study the participants were required to go from sitting to standing under self-regulated circumstances, and then when accompanied by audio or visual preparatory cues. Under self-regulated sit-to-stand conditions the patients with PD showed slower movement times, decreased velocities, and more impaired measures of hip and knee movement in comparison to the controls. The group with PD showed improved scores when provided with audio or visual cues. They had shorter total movement time, increased their vertical velocities, and showed less impaired measures of hip and knee movements.

Majsak, Kaminski, Gentile, and Flanagan (1998) found that bradykinesia decreased in arm reaching movements under conditions with more external cueing. This study compared conditions of self-determined reaching movements and movements under visuotemporal external cueing. The self-determined condition involved reaching for a fixed ball as quickly as possible. The visuotemporal cueing entailed reaching for a ball moving down a track, where the participant had 400 ms of viewing and preparation time before reaching for the ball in a designated zone. The experimenters measured the speed and accuracy under both conditions. The controls experienced no differences between the self-determined and visuotemporal cueing conditions. The participants with PD, however, showed slower speed with the self-determined condition, yet under the

visuotemporal condition they significantly increased their speed, matching that of the controls. Some accuracy measures did not vary between patients and controls; however if accuracy had been determined by a successful ball grasp, then under the moving ball condition, the PD participants would have shown some impairment.

Schneider, Diamond, and Markham (1987) found that individuals with PD had difficulty with sensorimotor integration and proprioception in the areas of their hands and arms. The individuals with PD were asked to perform nine proprioception and movement tasks which were dependent on sensory processing. Two of the tasks involved moving a finger up and down in response to verbal cues, and determining if and in what direction their finger had been moved with a rod by an experimenter. The participants with PD had significantly more errors than controls. The mean number of errors in Parkinson's patients was 20.0, while for controls it was 6.73. Additionally, there were some tasks like sustained pointing, where the participants were required to perform the tasks with their eyes open and also with their eyes closed. When performing this task with their eyes open there was no difference in performance between patients and controls, yet with their eyes closed the Parkinson's patient's performance had significantly more errors. When the patients had their eyes closed they had fewer cues to rely upon, whereas when they had their eyes open they had visual cues which helped increase their accuracy.

These studies illustrate that individuals with PD can experience reduced ability in using sensory cues to modify their actions. Yet, when they are assisted with external cues, their movements become less disordered. Understanding a patient's responsiveness to external cues can be important in the treatment of an individual with PD.

Impact of Impaired Sensory Function on Speech.

As stated earlier, over 100 muscles are used to produce speech (Duffy, 1995). The system relies on somatosensory input to coordinate and plan these muscle movements. If sensory deficits contribute to disordered limb and axial movements in individuals with PD, it could be reasoned that similar sensory deficits might affect the muscle movements needed for speech.

Research in the field of communication disorders is consistent with studies of limb and axial movements, indicating some sensory problems and better performance under conditions of external cueing (Schneider, Diamond, & Markham, 1986). Schneider et al. (1986) found through tests of motor function, sensory function, and sensory motor integration that individuals with PD had difficulty encoding and using sensory information for orofacial movement. Some of the sensory tests were aimed at determining if participants were aware of orofacial movements or aware of the location of tactile stimulation of orofacial structures. The sensorimotor integration tests evaluated the accuracy of the motor response, which was dependent on correct coding and use of the sensory information. Patients with PD made significantly more total errors on all of the tasks than controls. The greatest numbers of errors were in the tests of sensory function and sensorimotor integration.

An important question for consideration is “How might sensory deficits contribute to disordered speech in PD?” One suggestion is that individuals with PD do not recognize the severity of their dysarthria. They often have family members or communicators who complain of not being able to understand them or communicate well with them (Adams, 1996; Duffy, 1999; Ramig, 1995). If the person has hypophonia, the

family member will often request louder speech. When patients with PD produce louder speech they often complain that they feel like they are yelling (Dromey & Adams, 2000). These communication difficulties are frustrating for the family members and for the individual with PD. Critchley (1981) found that when it becomes difficult to communicate effectively, it is common for patients with PD to withdraw from social situations because of their frustration.

Some studies show that individuals with PD have difficulty rating their dysarthric characteristics (Ho, Bradshaw, & Iansek, 2000; Lloyd, 1999; Scott, Caird, & Williams, 1984; Yorkston, Bombardier, & Hammen 1994). In the Yorkston et al. (1994) study, individuals with dysarthria filled out questionnaires about their dysarthria. One Parkinson's patient with mild dysarthria overrated her disability and reported more difficulty in conversational situations than did the group with severe dysarthria. Another patient with PD who had moderate dysarthria indicated far fewer conversational difficulties than the group with mild dysarthria. Not being able to correctly assess one's dysarthria could lead to a failure to recognize the need to modify speech.

Ho et al. (2000) examined the performance of patients with PD in the immediate perceptual rating of vocal intensity, rather than on the overall perception of dysarthria. The individuals were asked to read or converse and immediately afterwards were asked to rate their speech intensity using a volume control. Next, they were to rate their speech intensity after it was played back to them. In both of these measures patients consistently overestimated their vocal intensity. The patients had quieter speech than controls, but rated their vocal intensity level as greater than the controls.

Scott et al. (1984) found that PD can affect the ability to use and recognize prosody and facial expressions. In this study individuals with PD were not able to create sentences using proper prosody for anger or for a question. Individuals with PD performed worse than controls on tasks of identifying prosody in speech samples, except for identifying neutral speech. Lloyd (1999) made measures of discrimination and comprehension of prosody, and found that patients with PD were significantly impaired when compared to controls. Both studies emphasize the sensory and cognitive difficulties in PD rather than only the motor impairment.

Some reports have found that individuals with PD were able to detect that they had speech difficulties or volume differences in speech (Dromey & Adams, 2000; Fox & Ramig, 1997). In the Fox and Ramig (1997) study, although the patients recognized their speech deficits, they also reported that they did not have confidence in their voice and would be less likely to participate in social situations. To Fox and Ramig this indicated some negative stigmas regarding speech and conversation. This observation led them to speculate that these individuals might have rated their speech disabilities accurately because they had been made aware of their speech deficits by social partners who informed them of their disability through requests for more clear speech, or an inability to understand.

Dromey and Adams (2000) found no difference between controls and patients with PD in their perception of loudness. The participants rated loudness of warbled tones. They were asked to sustain phonation at random loudness levels, which they could monitor by keeping a biofeedback indicator centered on a display. They were asked to produce comfortable phonation, and then asked to produce phonation at different levels

in relation to their comfortable phonation. These results led the authors to believe that there is something beyond the simple perception of vocal intensity that makes patients with PD feel like they are shouting when speaking at a comfortable listening level for conversational partners.

A difficulty with the Dromey and Adams (2000) study was it may have used too many external cues on the vocal intensity tasks. For example, in the second portion of the experiment a biofeedback needle was an external cue. An external cue in the third portion of the experiment was when the experimenter verbally requested certain levels of loudness in phonation. Individuals with PD can respond to cues from requests to change vocal intensity. For example, individuals with PD have less disordered speech in the therapy room (Yorkston, 1996) where immediate feedback is available, through the therapist and, at times, via biofeedback devices. However, outside of therapy, when the only indications that speech intelligibility has improved comes from the patient's own judgment, they do not generalize these gains to their everyday speech (Yorkston, 1996). In order to examine this sensory deficit more clearly, an experiment needs to use subtle cueing, which allows the patients to modify behavior based upon their sensory recognition and integration.

Ho, Ianssek, and Bradshaw (1999) conducted a study that used more naturalistic speech measures, which relied on internal cueing. This study focused on measuring vocal intensity. Participants were presented speech samples at different distances and were asked to rate the speech intensity of the sample. After rating a speech sample they were to converse with the experimenter in either rote speech tasks or natural conversation to "prepare" for the next listening task. Valuable data were collected during the

“preparation” periods because there was more control for the Hawthorne effect. The Hawthorne effect is seen when participants change their behavior because they know that they are participating in a study. During these preparation periods the participant was not under the impression they were contributing to experimental data. The experimenter would stop and converse at different distances from the speaker, and the participants were left to their own sensory judgment to identify how to change their vocal intensity with these changes in interlocutor distance. This provided a more natural setting where measures were made in order to determine if the patients would increase vocal intensity with greater interlocutor distance.

Several interesting things are found in the results of the Ho et al. (1999) study. Although both groups perceived the presented speech to be quieter with greater distance, the patients with PD did not perceive as great a decrease in the speech intensity as the controls did. Another striking result from this study was that the sequential speech and conversational speech data taken during the “preparation” periods did increase as the interlocutor distance increased, indicating that the individuals with PD did understand the sensory cue to increase speech effort as the conversational partner increased in distance from the speaker. It is interesting to note that although the group with PD did increase their vocal intensity, they were still significantly quieter than the control group. Ho et al. noted that there was a reduction in the gain of vocal intensity as interlocutor distance increased. Ho et al. proposed this reduction in gain could be due to decreased awareness of pragmatic rules. It is possible that the patients were so focused on the content of their speech that they did not increase vocal intensity to be heard or understood.

Research about vocal changes with interlocutor distance leads to questions about behavior of patients with PD in other situations where an increase in vocal intensity could be beneficial. One situation that could benefit from an increase in vocal intensity is during a conversational misunderstanding. The survey in the Yorkston et al. (1994) study included a section on compensatory strategies used by individuals with dysarthria. After a misunderstanding, dysarthric individuals indicated that “improved production” was the best compensatory strategy. In a preliminary study Clark, Lubker, and Hunnicutt (1987) found that healthy individuals increase duration, fundamental frequency, and intensity in a repetition for clarification. Since individuals with PD have demonstrated difficulty appropriately using prosody and vocal intensity, the focus of the current study was to identify what individuals with PD do vocally, if they do anything at all, to improve speech production following a misunderstanding (Ho et al. 1999; Scott et al. 1984). Do patients with PD identify and use sensory cues to change vocal parameters, particularly duration, fundamental frequency and vocal intensity, when listeners misunderstand them? Additionally, since the presentation of masking noise has been found to increase intensity in individuals with PD, the present study aimed to identify whether inducing the Lombard effect would influence vocal modifications made by individuals with PD in a repetition for clarification. The results will be relevant to our understanding of the nature of hypokinetic dysarthria, and may also have important clinical implications.

Method

Participants

The study involved 5 individuals with PD between the ages of 54-72 years, with a mean of 63.8 years (Table 1). All patients were diagnosed by a neurologist as having idiopathic PD. All patients were taking at least one anti-Parkinson medication including dopamine, Miraplex, Sentimet, Carbadopa/Levodopa, and Eldepryl. The participants with Parkinson's disease were tested one to two hours after the most recent dose. None of them had received any speech-language therapy.

The time since diagnosis ranged from 3.5 years to 14 years with a mean of 9.1 years. To determine the severity of dysarthria, a perceptual rating was performed by five individuals with training in Speech-Language Pathology. They listened to recordings of five sentences from each participant. The sentences were randomly ordered and no identification of the participant was given with a sentence. The listeners rated them on a visual analog scale with 100 representing the most severely disordered speech and zero being normal. To allow a calculation of intra-rater reliability, each listener was asked to listen to and perceptually rate the samples on two occasions separated by at least one day. Data were used from the three raters with the highest intra-rater reliability correlation coefficients. These ranged from .89 to .87 with a mean of .88. The three raters had an intraclass correlation coefficient of .608 ($F = 2.553, p = .003$), indicating good agreement. The mean perceptual severity ratings of the patients from these three listeners ranged from 4.7 to 36.6 with a mean of 20.7.

These participants were matched for sex and age (within 10 months) with 5 healthy control participants. The range of age difference between participants with PD

Table 1

Demographic Data from Participants with Parkinson's Disease

Participant	Gender	Age	Years Post Diagnosis	Dysarthria Severity Rating	Medications
P1	M	72	9.0	4.7	Dopamine agonist
P2	M	63	14.0	12.2	Carbadopa/Levodopa, Miraplex, Sentimet, Eldepryl
P3	M	68	9.0	14.3	Dopamine agonist
P4	M	54	3.5	35.7	Sentimet
P5	M	62	10.0	36.6	Eldepryl, Sentimet Carbadopa/Levodopa

and their controls was from 2 months to 10 months, with a mean of 5.6 months. The controls had an age range of 54-71 and mean age of 63.4 years (Table 2).

For all participants an interview was conducted prior to testing in order to determine that none of them had a history of any neurological, speech, language, or hearing disorders beyond PD. Any participants who reported visual impairment were required to wear their prescribed lenses during testing. None of the participants wore hearing aids. All participants had an audiometric screening to establish no significant hearing loss at frequencies of 250, 500, 1000, 2000, and 4000 Hz. In each group there were some individuals with hearing loss (Table 3). Participants P1 and P2 failed the screening at 20 dB, but P2 passed a screening at 35dB, and P1 passed a screening in both ears at 45 dB in all frequencies except at 4000 Hz. Each participant signed an informed consent before participating in the experiment (see Appendix A).

Instrumentation

All testing was conducted in a sound-attenuating booth. A head-mounted condenser microphone (AKG C-420) was used to collect the audio signal at a constant distance of 8 cm from the participant's lips. Speech intensity was sensed with a sound-level meter (Larson-Davis 712), located 50 cm from the speaker's lips. A two channel digital audio tape (DAT) recorder (Panasonic SV-3800) was used to record these two signals. The signals were subsequently digitized into a Kay Elemetrics Computerized Speech Lab (CSL 4400) system at 25 kHz.

Stimuli

Stimulus sentences from a series of studies by Cutler and Butterfield (1990, 1991) were used in the present experiment (see Appendix B). These sentences were created in

Table 2

*Demographic Data from Control**Participants*

Participant	Gender	Age
C1	M	71
C2	M	62
C3	M	62
C4	M	54
C5	M	68

pairs that were phonetically similar, whereby one could be ‘misheard’ as the other. For example, the participant might read, “It’s color of eyes that counts,” and the phonetically similar counterpart would be, “A skull will revise the count.” For 10 of these sentences additional mishearing stimuli were created. By creating more ‘mishearings’ for 10 Cutler Butterfield (1990, 1991) sentences then there could be 2 repetitions for 10 sentences (Appendix B).

Additional stimuli were created with sentences that had only one word different from the original sentence (see Appendix D). Both types of sentences pairs were used for deliberate ‘mishearings’ as described below.

Procedures

The procedures were modeled after those used by Clark et al. (1987), as well as by Cutler and Butterfield (1990, 1991). There were three experimental conditions. In condition 1 the participants were presented 20 sentences from the Cutler and Butterfield (1990 and 1991) studies (see Appendix B) and 20 sentences created for this study (see Appendix C). The sentences were randomly presented on a computer screen in front of the participant in the sound booth using PowerPoint slides in *Arial* 96 point font. Each participant was asked to read the sentences in his or her typical voice. This was recorded as the “normal” or baseline speech condition.

In condition 2 the participants were told that an individual listening outside the booth would type what he or she heard the speaker say. They were informed that this listener would hear their speech through a ‘distorting filter,’ which would make their speech harder to understand, and thus be more realistic in representing typically noisy communication environments. A second computer screen presented the words that the

listener supposedly heard. At random intervals a 'mishearing' was presented on the second computer screen. Either an entire phonetically paired sentence was presented or one word was changed from the original sentence. The participants were instructed to repeat the sentence if the response from the listener was incorrect. The participants read a total of 40 sentences. There were five sentence pairs where the "mishearings" occurred twice, five sentence pairs where the mishearing occurred once, and ten where one word was misheard once.

In condition 3 the participants were asked to read the sentences while speech spectrum noise was presented binaurally through the headphones at 70 dB HL. The purpose of presenting the speech spectrum noise was to induce the Lombard effect. The Lombard effect has been documented as an increase of vocal intensity when an individual is presented with masking noise. The theoretical explanation is that individuals compensate for the background noise by speaking more loudly. Individuals with PD have been documented to show a significant increase in vocal intensity under the masking noise condition (Adams & Lang, 1992). The goal of using this condition was to determine whether additional effort increases would result from the simulated mishearing of a subset of the stimulus items. In condition 3, the participants were told there was a listener outside the booth who would transcribe what they said. The participants were informed that the noise might make it difficult for the listener to hear them and that they should re-read a sentence if the listener's response was incorrect. Again at random intervals a 'mishearing' was presented on the second computer screen of either an entire phonetically paired sentence or a sentence with one word that was different.

Condition 1 was always presented first to get the most unbiased measures for the baseline condition. The order of conditions 2 and 3 was alternated between participants to reduce potential sequencing effects.

Conditions 2 and 3 were included to provide data about a speaker's spontaneous behavior when an utterance had been misheard. When the individual repeated a sentence after a 'mishearing,' it allowed a determination of whether individuals with PD use the miscommunication cues and change prosodic stress similarly to controls upon repetitions. Individuals with PD are commonly identified as being monotone and having a limited fundamental frequency (F0) range, which may alter their ability to employ normal prosodic mechanisms.

Data Analysis

The analysis included measures of sentence or word duration. This helped determine if upon the second repetition the participant read the sentence or word more slowly to increase clarity or prosodic stress.

The signals from the microphone and from the sound level meter were both digitized at 25 kHz using the Kay Elemetrics CSL system. F0 and amplitude contours were generated. From these, the average increases in sound pressure level (SPL) and F0 due to the Lombard effect were measured. The data were analyzed for changes in F0 and SPL in repeated sentences following a miscommunication. This analysis focused on changes in F0 or vocal intensity either for the whole sentence, or for the one misheard word in comparison to first utterance.

The statistical analysis involved a repeated measures ANOVA design. The within participants factor was condition (1, 2, or 3) and the between participants factor was group (PD or control).

Results

It is difficult to draw clearly generalizable inferences from the results of this study because the number of participants was so small. Additionally, the participants with PD demonstrated variability in the severity of their dysarthria, as judged from the perceptual ratings. Three of them were only very mildly dysarthric. Inferential statistics were nevertheless computed for the patient and control groups to compare the original and repeated utterances to evaluate the significance of any changes in the dependent variables. Descriptive statistics for measures of duration, F0, and intensity at the sentence level are presented in Tables 4, 5, and 6.

Duration

Sentence. The ANOVA comparing mean duration for conditions 1, 2, and 3 was not significant. Therefore, the overall mean sentence duration was not different for the baseline and the first reading in the other two experimental conditions (with or without masking noise). In condition 2 the comparison was made between the first reading of a sentence, the first repetition, and the second repetition. This test was also not significant. However, in condition 3, there was a statistically significant difference, $F = 7.551$, $p = .019$ [Huynh-Feldt], $df = 1.159, 9.269$. The change demonstrated by the error bars in Figure 1 indicates that the individuals with PD remained fairly constant in sentence duration over the multiple readings. The controls increased the duration of the sentence from the first reading to the first repetition, and from the first repetition to the second repetition. Although a slight group difference is seen in the error bars, there was no significant group interaction.

Table 4

Descriptive Statistics of Sentence and Word Duration Measures

	Group	<i>M</i>	<i>SD</i>	N
Duration	PD	2.08	0.17	5
Condition 1	control	2.03	0.18	5
First reading	Total	2.06	0.17	10
Duration	PD	4.34	4.47	5
Condition 2	control	4.04	2.13	5
First reading	Total	4.19	3.30	10
Duration	PD	5.92	4.26	5
Condition 2	control	5.11	2.66	5
First repeat	Total	5.51	3.38	10
Duration	PD	5.23	4.45	5
Condition 2	control	6.59	3.65	5
Second repeat	Total	5.91	3.90	10
Duration	PD	5.64	5.89	5
Condition 3	control	3.53	1.40	5
First reading	Total	4.58	4.19	10
Duration	PD	5.73	5.58	5
Condition 3	control	4.48	1.99	5
First repeat	Total	5.11	4.00	10
Duration	PD	6.11	5.03	5
Condition 3	control	6.04	3.19	5
2nd repeat	Total	6.08	3.97	10
Word duration	PD	0.43	0.22	5
Condition 2	control	0.50	0.18	5
First reading	Total	0.47	0.19	10

Table 4 (*continued*)*Descriptive Statistics of Sentence and Word Duration Measures*

	Group	<i>M</i>	<i>SD</i>	N
Word duration	PD	0.59	0.29	5
Condition 2	control	0.68	0.28	5
First repeat	Total	0.63	0.27	10
Word Duration	PD	0.53	0.21	5
Condition 3	control	0.50	0.13	5
First reading	Total	0.52	0.17	10
Word duration	PD	0.64	0.29	5
Condition 3	control	0.57	0.17	5
First repeat	Total	0.60	0.23	10

Table 5

Descriptive Statistics of Sentence and Word F0 Measures

	Group	<i>M</i>	<i>SD</i>	N
F0	PD	120.63	14.56	5
Condition 1	Control	116.03	13.48	5
First reading	Total	118.33	13.45	10
F0	PD	140.68	30.03	5
Condition 2	Control	120.54	15.56	5
First reading	Total	130.61	24.92	10
F0	PD	140.61	30.82	5
Condition 2	Control	117.41	14.47	5
First repeat	Total	129.01	25.78	10
F0	PD	147.12	32.76	5
Condition 2	Control	118.91	15.34	5
Second repeat	Total	133.02	28.33	10
F0	PD	150.57	24.34	5
Condition 3	Control	136.30	18.95	5
First reading	Total	143.43	21.90	10
F0	PD	149.76	25.60	5
Condition 3	Control	131.59	17.79	5
First repeat	Total	140.68	22.88	10
F0	PD	154.21	29.73	5
Condition 3	Control	134.77	17.60	5
2nd repeat	Total	144.49	25.21	10
Word F0	PD	150.01	34.02	5
Condition 2	Control	131.03	20.17	5
First reading	Total	140.52	28.20	10

Table 5 (continued)

Descriptive Statistics of Sentence and Word F0 Measures

	Group	<i>M</i>	<i>SD</i>	N
Word F0	PD	148.84	32.21	5
Condition 2	Control	124.26	16.00	5
First repeat	Total	136.55	27.25	10
Word F0	PD	149.70	29.42	5
Condition 3	Control	138.27	21.47	5
First reading	Total	143.98	25.02	10
Word F0	PD	153.39	32.68	5
Condition 3	Control	136.71	18.86	5
First repeat	Total	145.05	26.65	10

Table 6

Descriptive Statistics of Sentence and Word Intensity Measures

	Group	<i>M</i>	<i>SD</i>	N
SPL	PD	63.44	2.57	5
Condition 1	control	63.54	3.84	5
First reading	Total	63.49	3.08	10
SPL	PD	68.00	4.88	5
Condition 2	control	65.37	4.22	5
First reading	Total	66.68	4.52	10
SPL	PD	67.85	5.85	5
Condition 2	control	63.78	3.47	5
First repeat	Total	65.81	5.02	10
SPL	PD	67.72	6.76	5
Condition 2	control	63.59	3.82	5
Second repeat	Total	65.65	5.62	10
SPL	PD	72.40	4.25	5
Condition 3	control	70.00	2.40	5
First reading	Total	71.20	3.49	10
SPL	PD	72.11	4.48	5
Condition 3	control	69.06	2.96	5
First repeat	Total	70.59	3.92	10
SPL	PD	72.32	5.02	5
Condition 3	control	69.39	3.43	5
2nd repeat	Total	70.86	4.34	10
Word SPL	PD	71.47	4.52	5
Condition 2	control	68.69	4.50	5
First reading	Total	70.08	4.50	10

Table 6 (*continued*)*Descriptive Statistics of Sentence and Word Intensity Measures*

	Group	<i>M</i>	<i>SD</i>	N
Word SPL	PD	69.32	7.30	5
Condition 2	control	65.05	5.90	5
First repeat	Total	67.18	6.65	10
Word SPL	PD	70.04	3.58	5
Condition 3	control	70.75	2.93	5
First reading	Total	70.40	3.11	10
Word SPL	PD	70.41	4.95	5
Condition 3	control	65.90	5.59	5
First repeat	Total	68.15	5.51	10

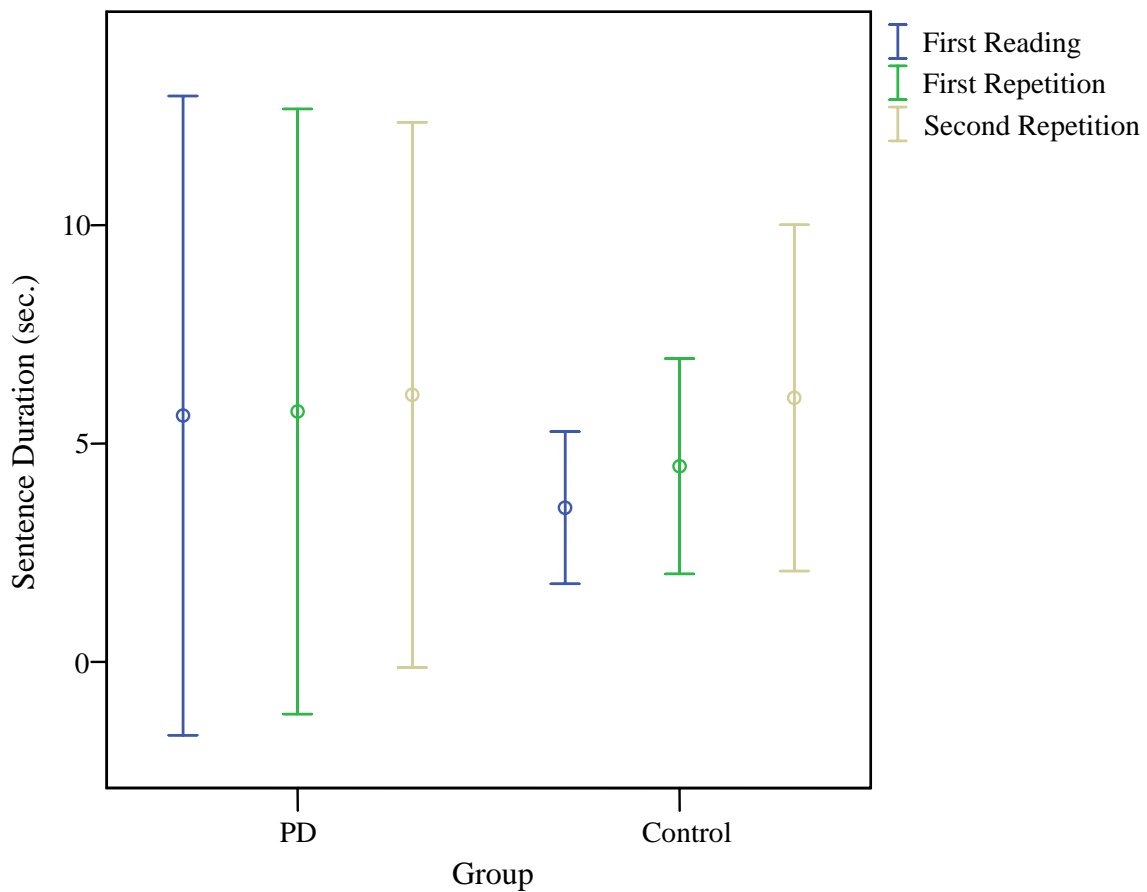


Figure 1. Confidence intervals (95%) of sentence duration measures in condition 3.

Word. The measures of word duration were not significantly different between first readings in conditions 2 and 3. Word duration increased significantly between the first reading and the repetition in condition 2, $F = 26.646$, $p = .001$, $df = 1, 8$, and condition 3, $F = 9.804$, $p = .014$, $df = 1, 8$. There was no significant group interaction in either condition. Figures 2 and 3 show the increased duration upon the first repetition for both groups.

Fundamental Frequency

Sentence. F0 increased significantly across the three experimental conditions, $F = 22.689$, $p < .001$, $df = 2, 16$. Figure 4 shows this trend graphically. The F0 measures increased from condition 1 to condition 2, and from condition 2 to condition 3. The change was similar among the groups and there was no group interaction. The change in F0 measures for condition 2 between the first reading and the first and second repetitions was significant, $F = 6.015$, $p = .011$, $df = 2, 16$. The F0 measurements decreased slightly on the first repetition and then increased on the second repetition. There was also a significant group interaction, $F = 6.121$, $p = .011$, $df = 2, 16$. The F0 measures of the PD participants increased, while those of the control participants decreased for the second repetition, as demonstrated by the error bars in Figure 5. Comparison of the two repetitions relative to the first reading in condition 3 were significant, $F = 5.207$, $p = .018$, $df = 2, 16$. As seen in Figure 6, there is indication that both groups decreased F0 upon the first repetition, and increased F0 on the second repetition.

The standard deviation (*SD*) measures of F0 reflected the variability in pitch within a sentence. F0 variability did not change upon the first or second repetition of a sentence, nor in the three different conditions. Additionally, there was no statistically

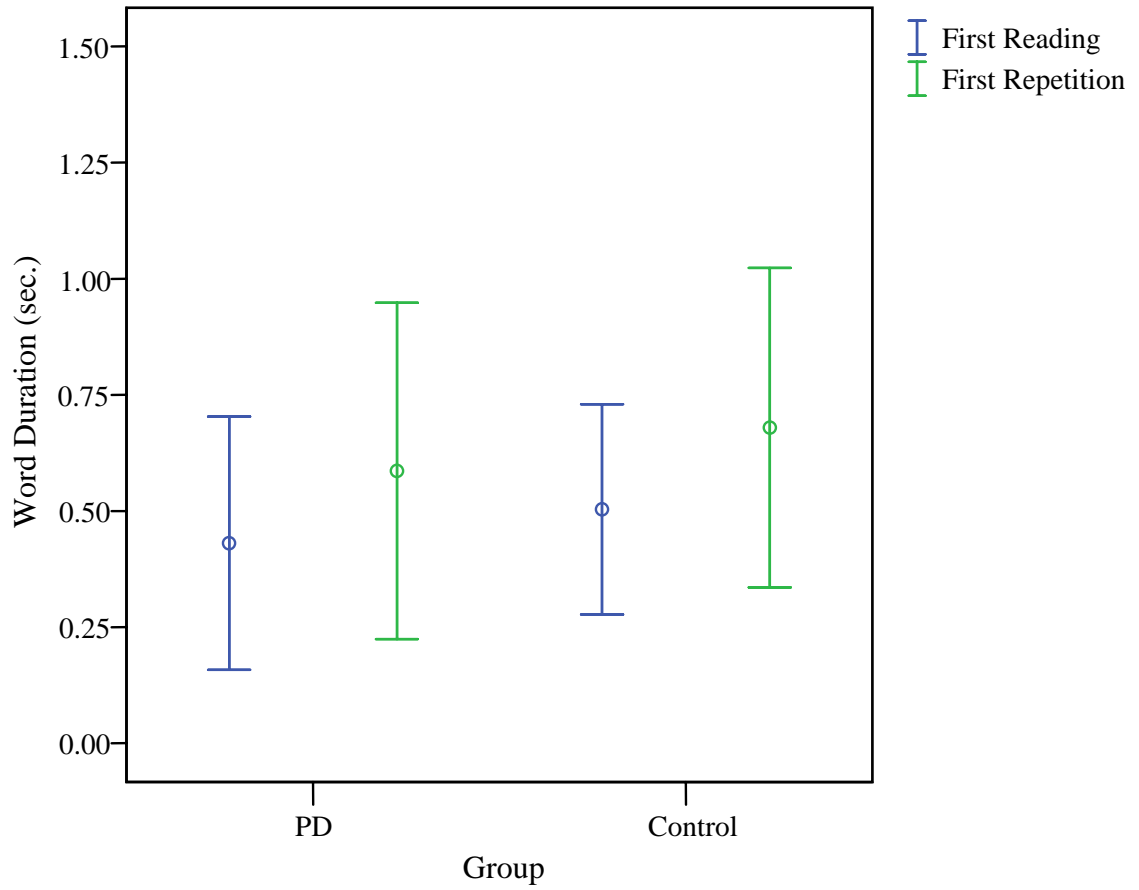


Figure 2. Confidence intervals (95%) for word duration measures in condition 2.

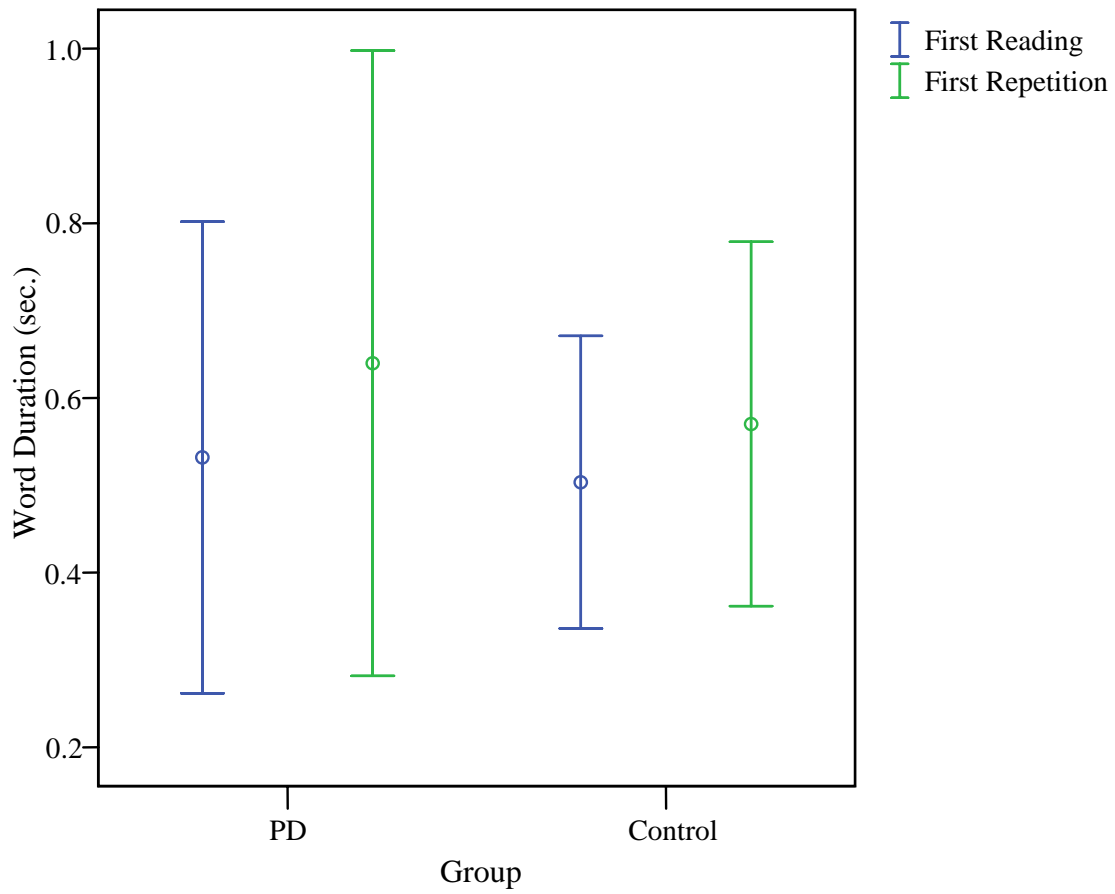


Figure 3. Confidence intervals (95%) for word duration measures in condition 3.

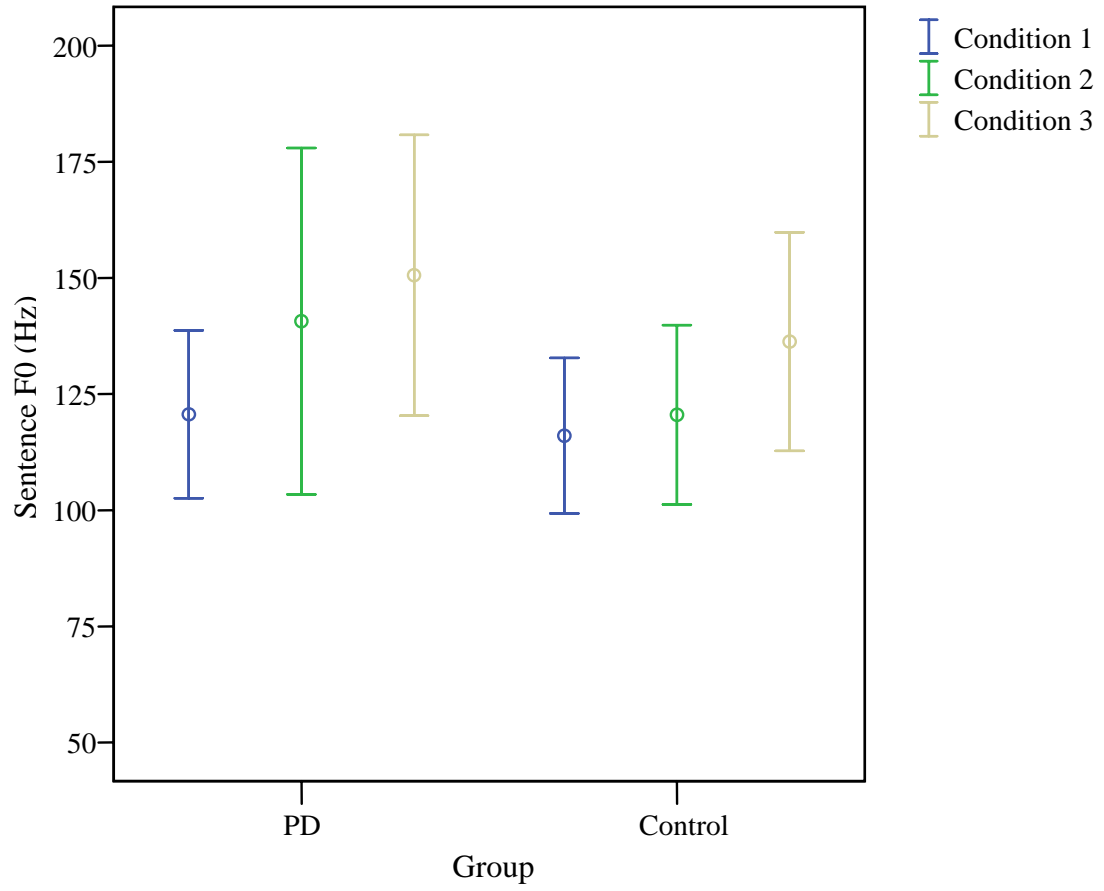


Figure 4. Confidence intervals (95%) of sentence mean F0 for the first reading across conditions 1, 2, and 3.

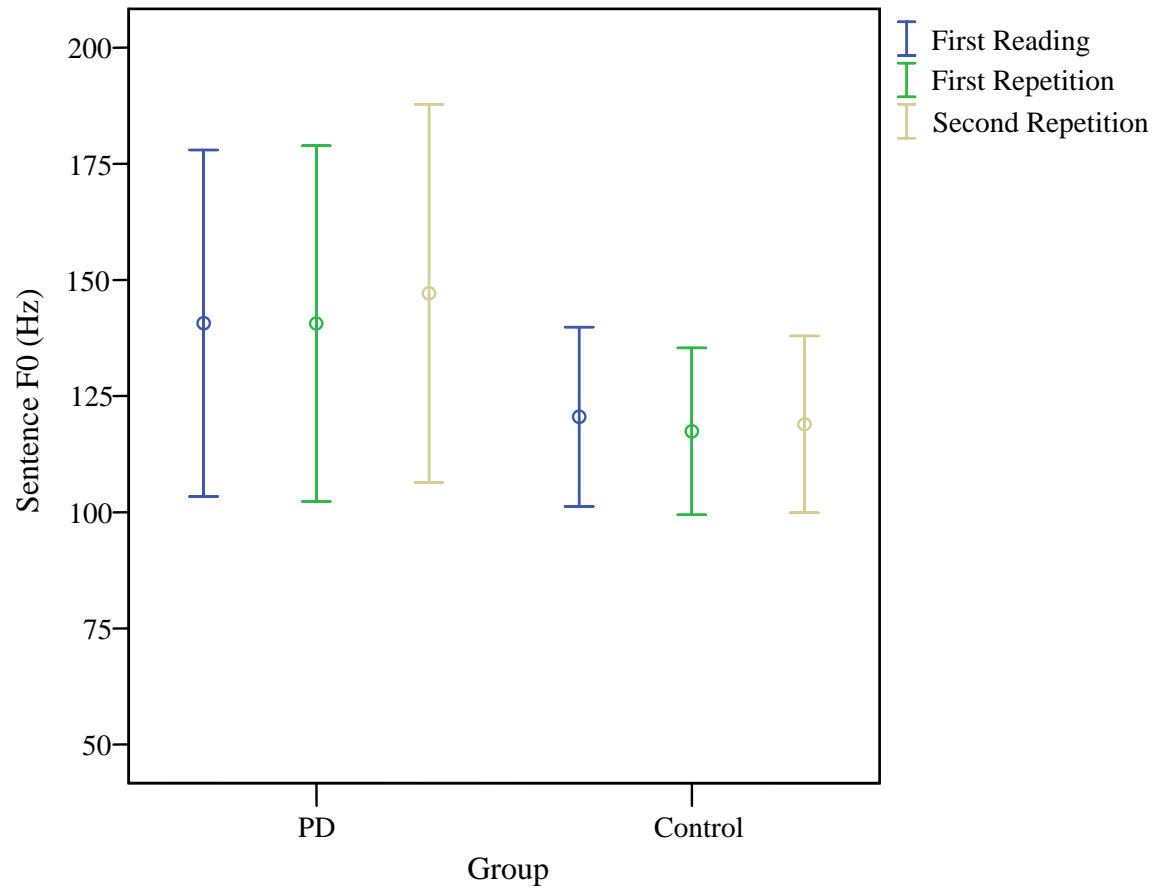


Figure 5. Confidence intervals (95%) of sentence mean F0 in condition 2.

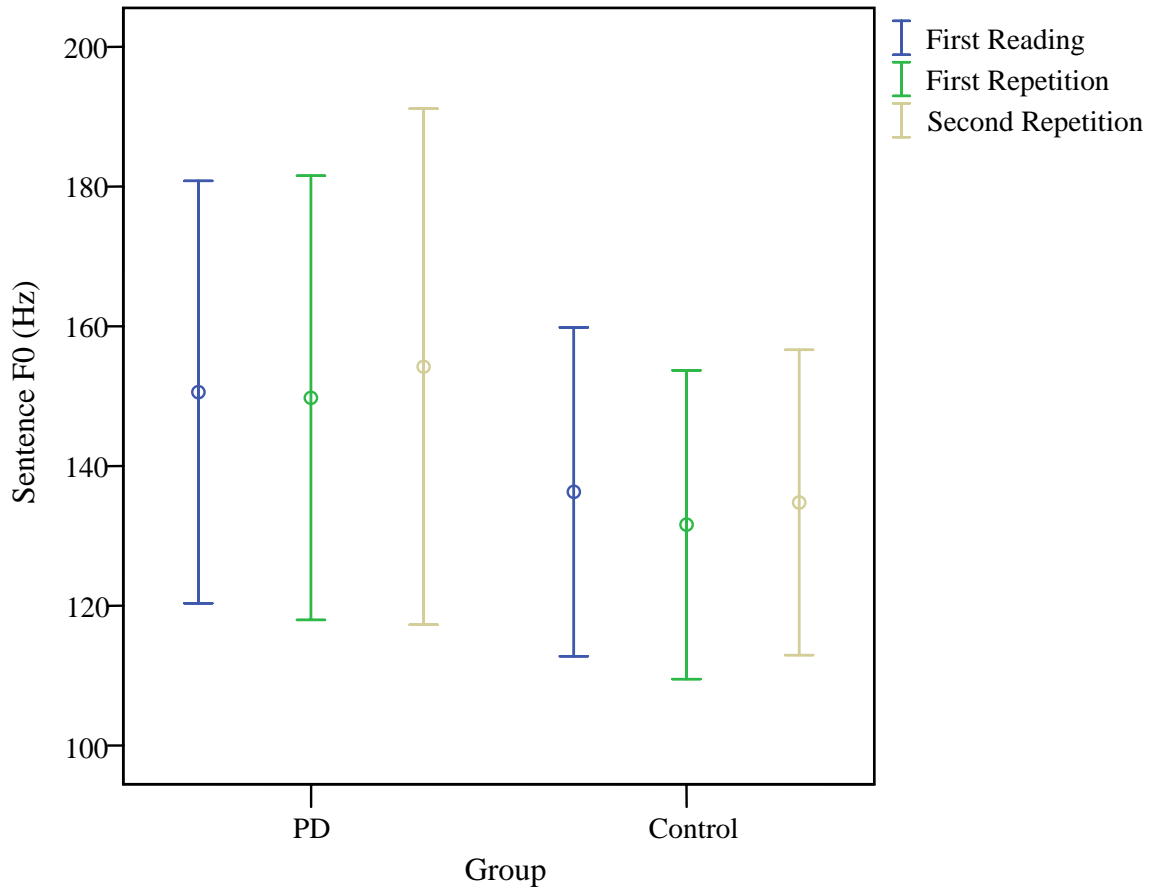


Figure 6. Confidence intervals (95%) of sentence mean F0 in condition 3.

Although not significant, it was observed that speakers with PD had lower *SD* in the baseline condition, but a larger *SD* than controls in the first reading in conditions 2 and 3 (Figure 7). Also in condition 2 the controls' *SD* of F0 decreased from the first reading the first and second repetitions, while individuals with PD had greater F0 *SD*, although the difference was not statistically significant (Figure 8).

Word. There was no significant change in word F0 from the first reading to the first repetition in conditions 2 or 3. Comparisons were made in the first repetition to determine if the word F0 was greater than the overall sentence F0. In condition 3 there was no significant difference; however, in condition 2 there was a significant difference, $F = 9.655, p = .015, df = 1, 8$. In condition 2 there was a significant difference between word and sentence F0 in the first reading as well, $F = 18.109, p = .003, df = 1, 8$, as shown in Figures 9 and 10. There was no group interaction in the comparisons of word and sentence F0 for either of the experimental conditions 2 or 3.

Intensity

Sentence. There was a significant change in SPL across conditions 1, 2 and 3, $F = 36.107, p < .001, df = 2, 16$. As Figure 11 reveals, there was no difference between participant groups. There was no significant change in intensity measures between the conditions 2 and 3, the SPL for participants with PD was actually higher than for the control group (see Figures 11 and 12), but not significantly so.

Word. The word SPL significantly decreased from the first reading to the first repetition in condition 2 ($F = 7.355, p = .027, df = 1, 8$). This decrease can be seen in Figure 13. In condition 3 there was a significant difference in SPL between the first reading and the subsequent repetition ($F = 6.647, p = .033, df = 1, 8$), as well as a

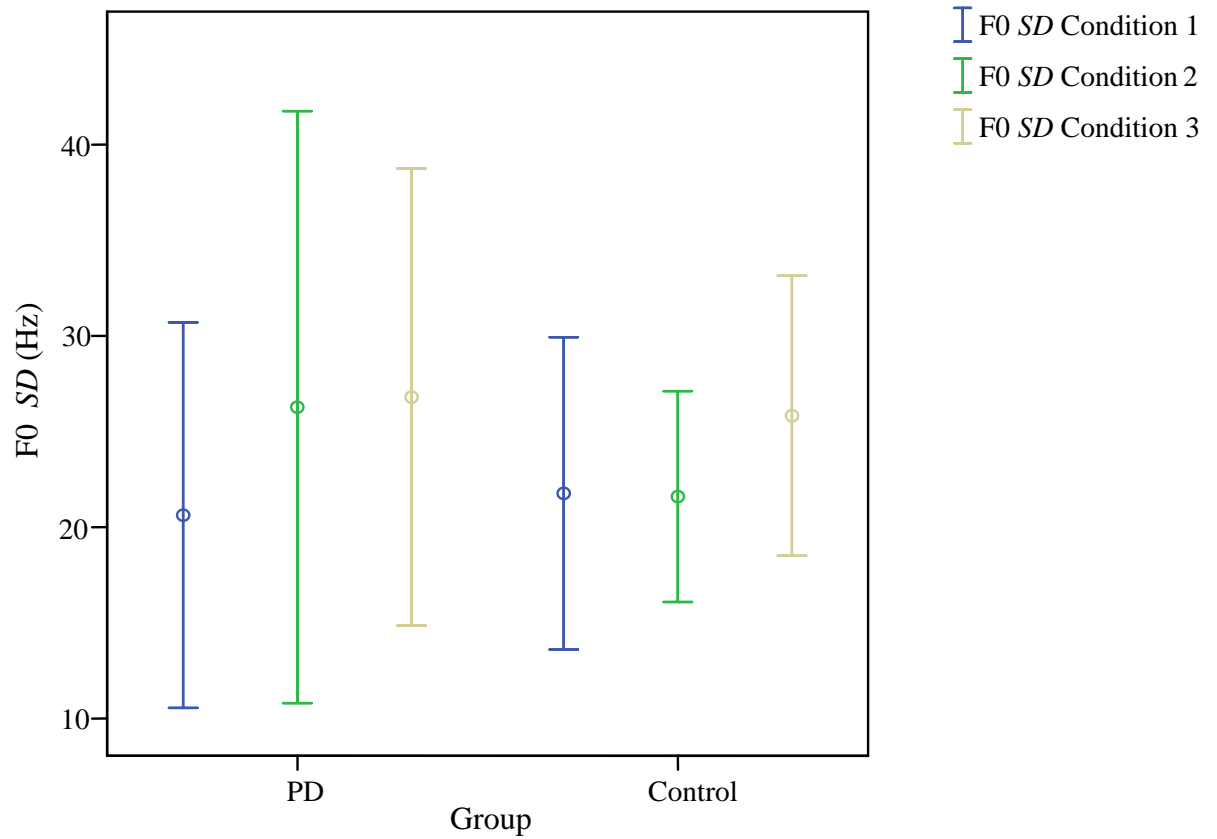


Figure 7. Confidence intervals (95%) for sentence F0 SD in first readings across conditions 1, 2, and 3.

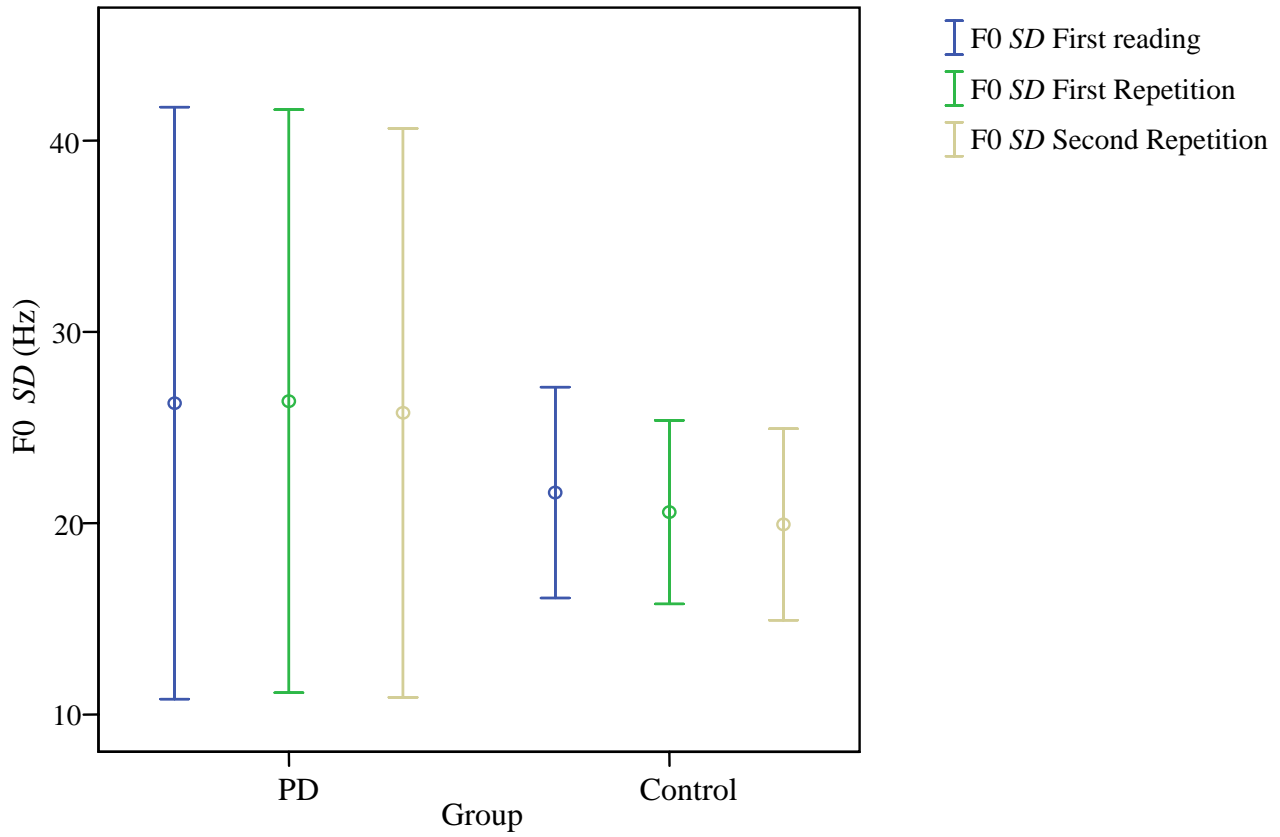


Figure 8. Confidence intervals (95%) for sentence F0 SD in condition 2.

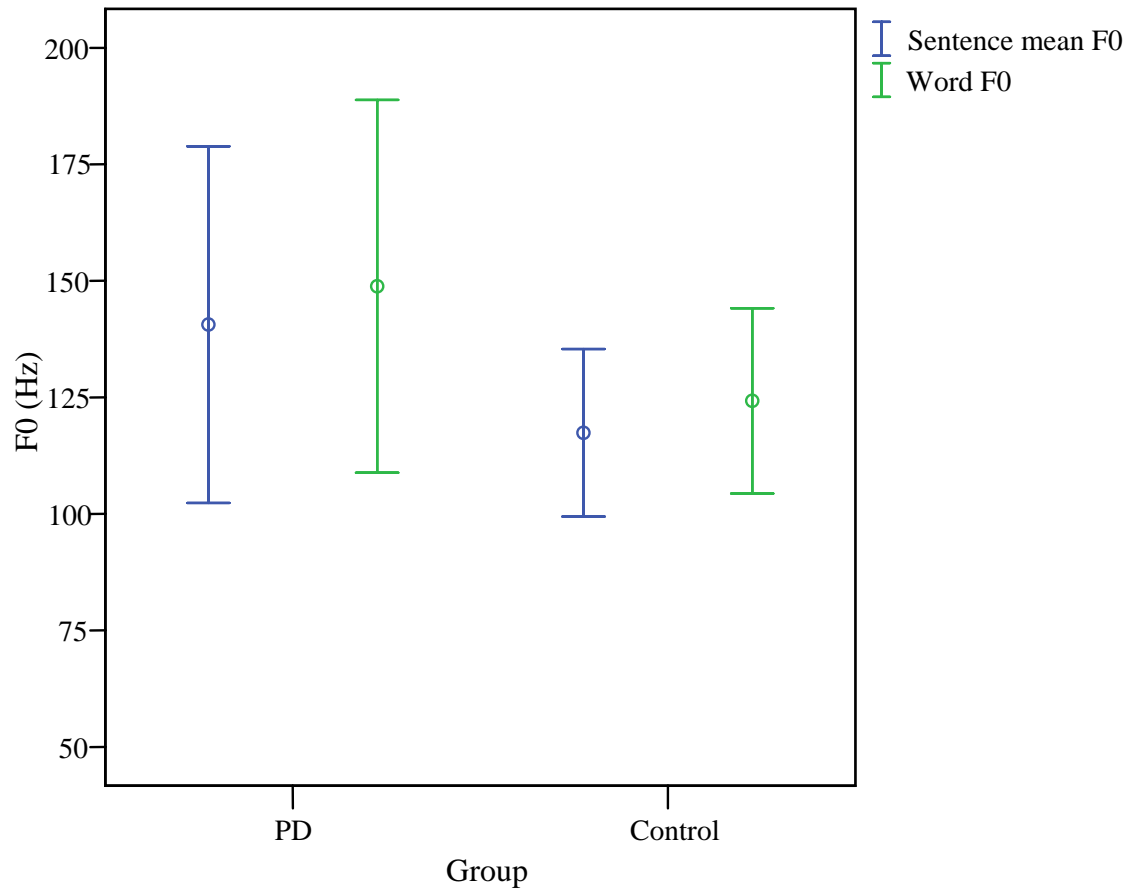


Figure 9. Confidence intervals (95%) for word F0 compared with sentence mean F0 in condition 2, first repetition.

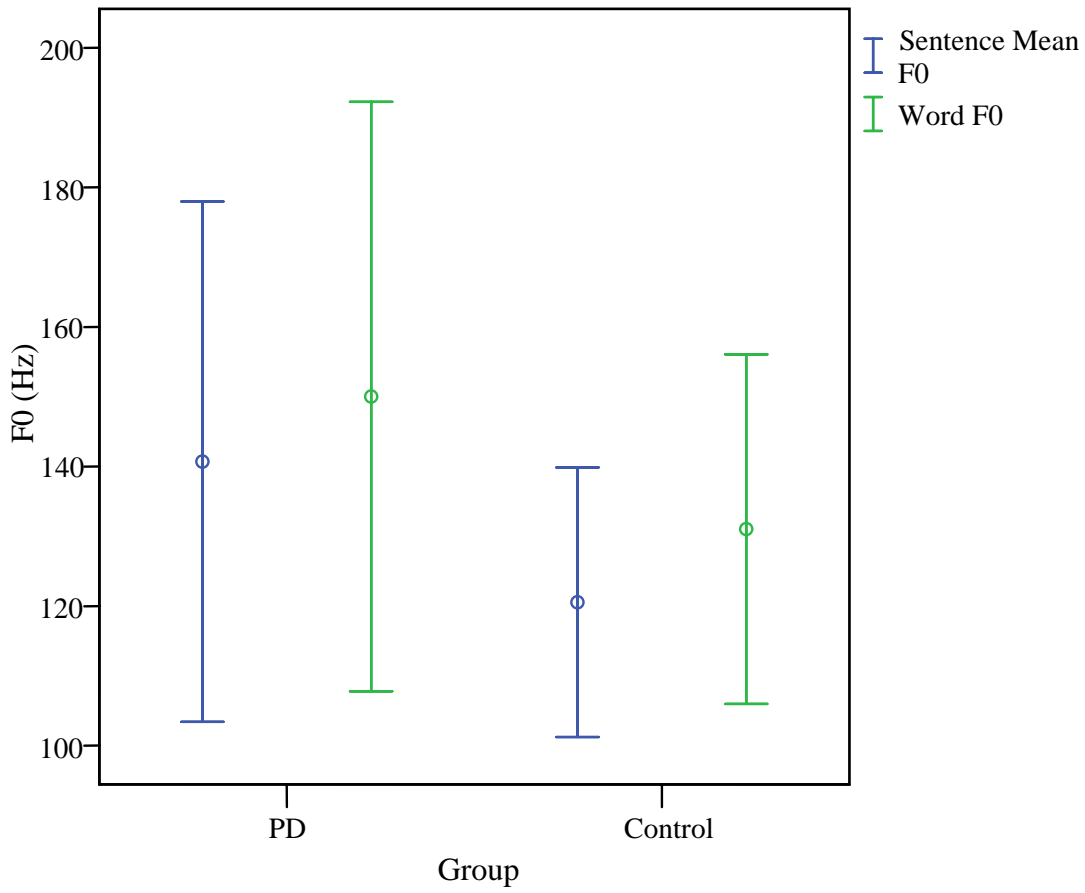


Figure 10. Confidence intervals (95%) for word F0 and sentence mean F0 in condition 2, first reading.

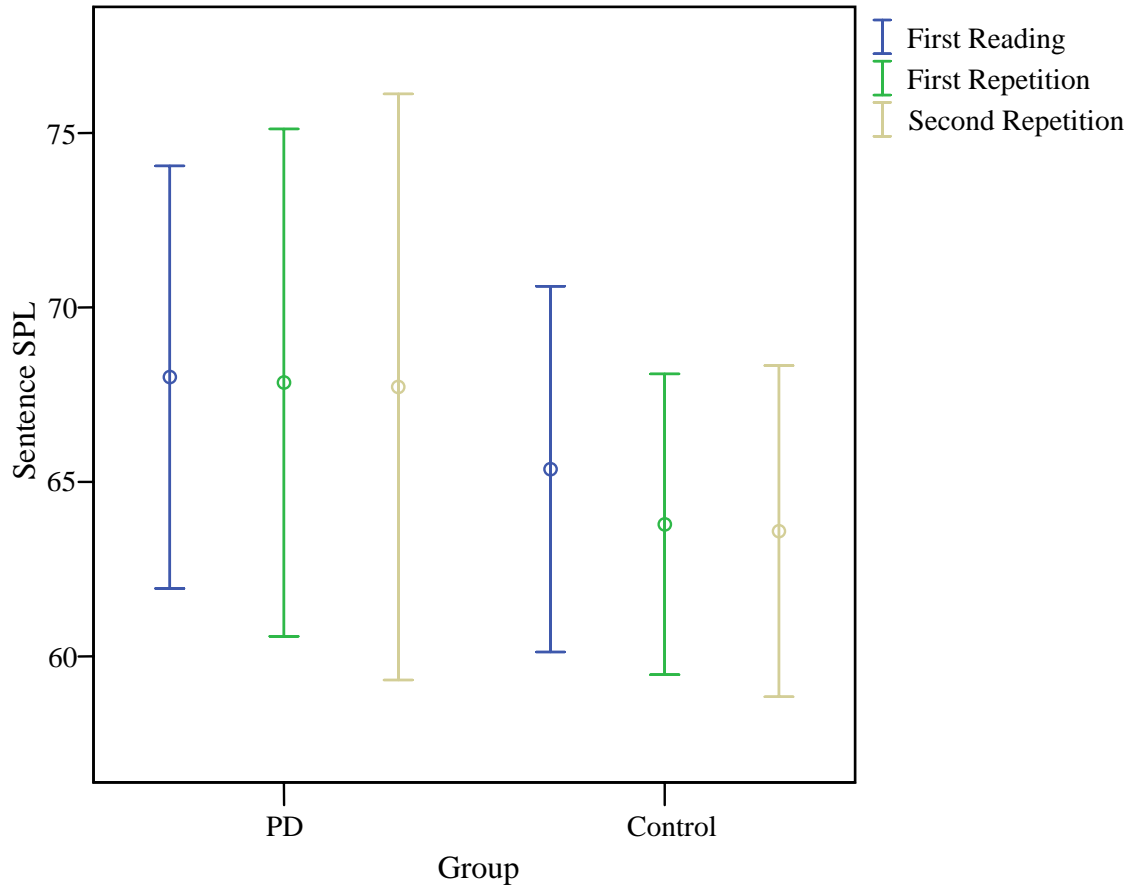


Figure 11. Confidence intervals (95%) for sentence SPL in condition 2.

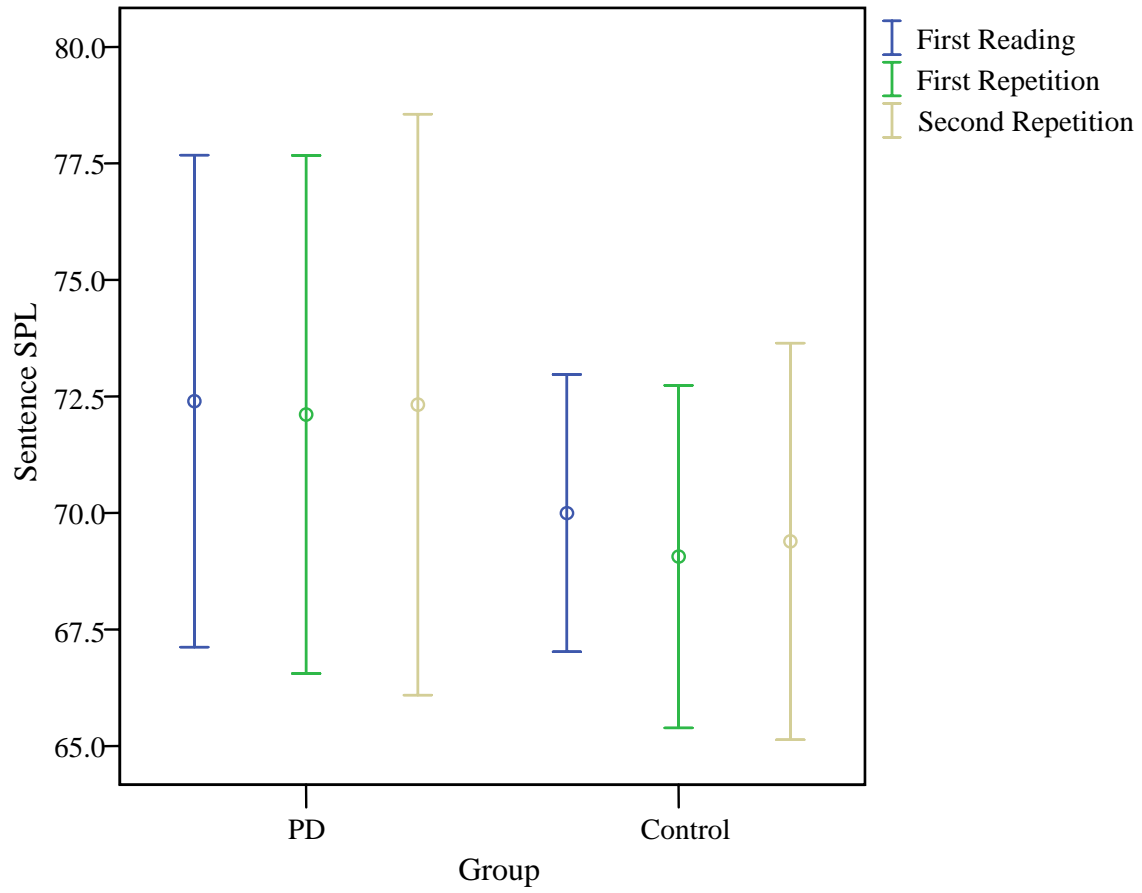


Figure 12. Confidence intervals (95%) for sentence SPL in condition 3.

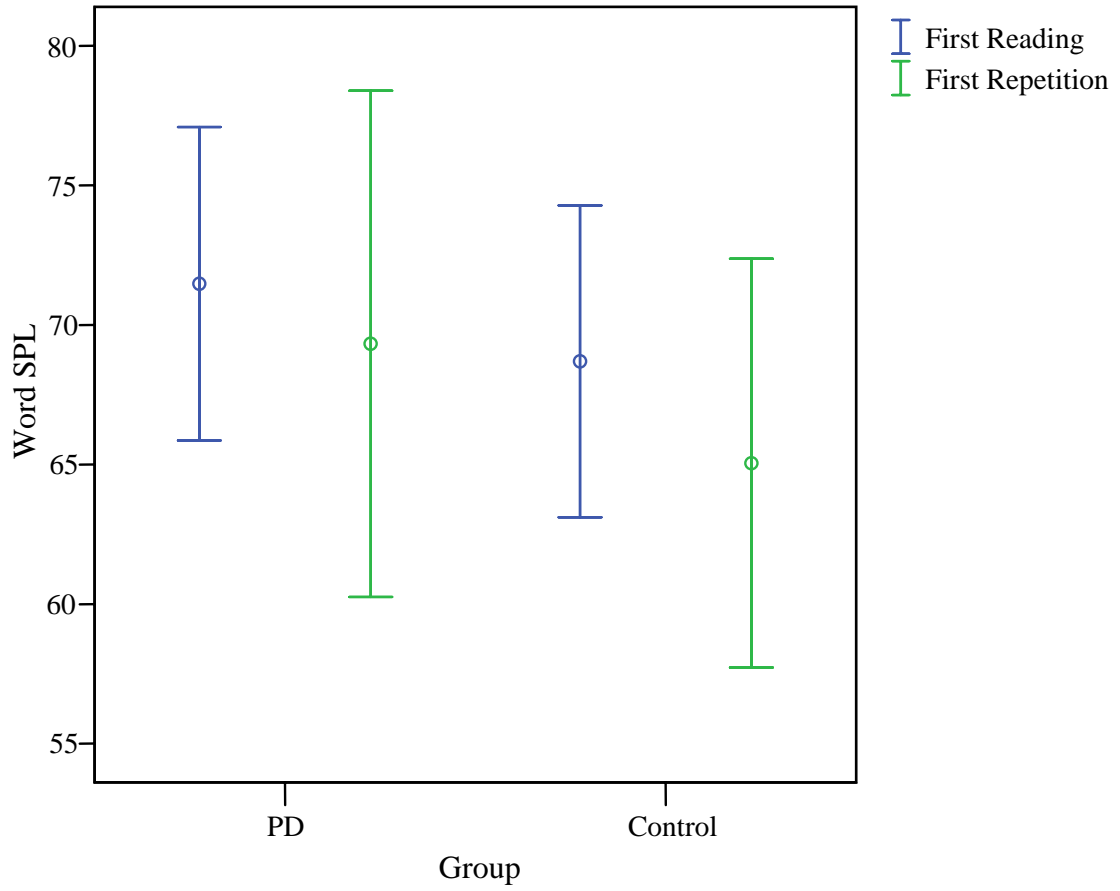


Figure 13. Confidence intervals (95%) for word SPL in condition 2.

significant group interaction ($F = 9.012, p = .017, df = 1, 8$). Figure 14 indicates that the control group decreased in SPL while the participants with PD remained more constant in measures of word SPL.

Word SPL was compared to its sentence SPL to identify whether the speakers emphasized a word by changing its SPL relative to the average level of the sentence. In condition 3 the word SPL was significantly lower than sentence SPL in the first repetition ($F = 10.065, p = .013, df = 1, 8$) (Figure 15). There were no other significant changes between word SPL and sentence SPL, indicating neither group consistently emphasized a word in a sentence by modifying the word SPL relative to the sentence SPL.

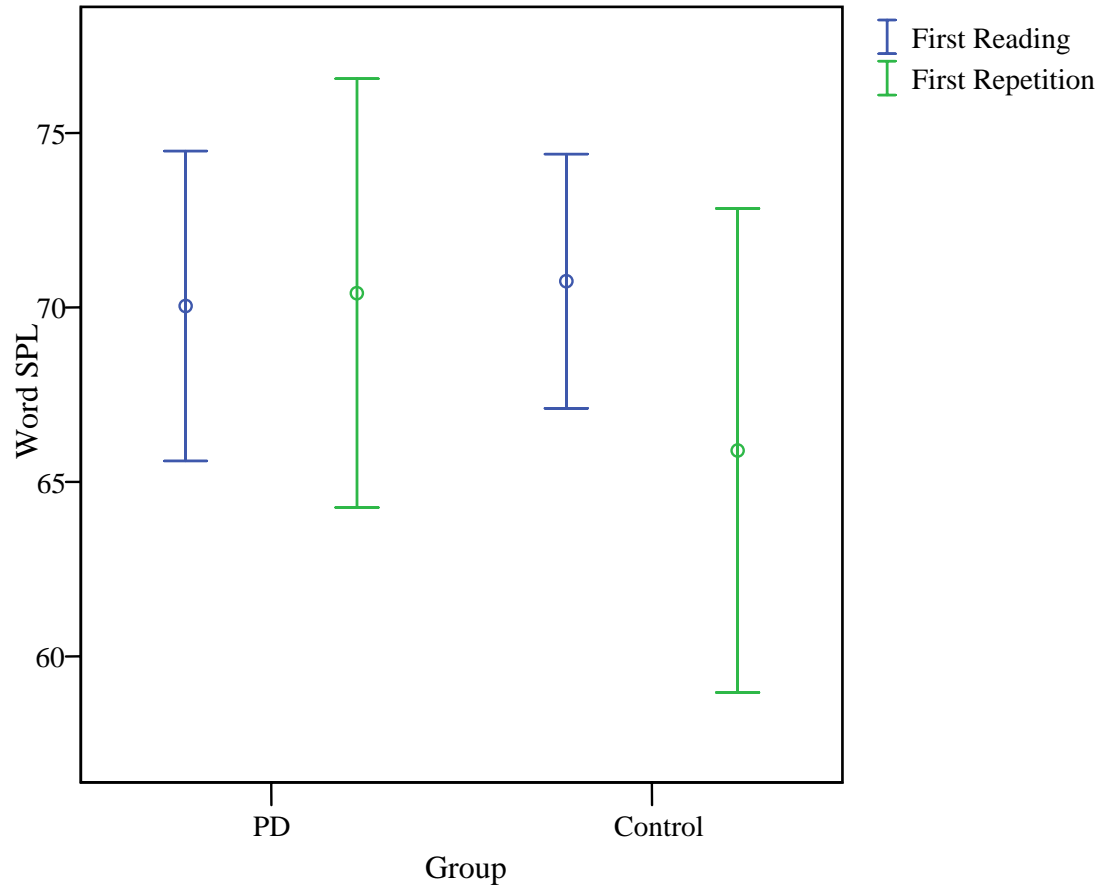


Figure 14. Confidence interval (95%) for word SPL in condition 3.

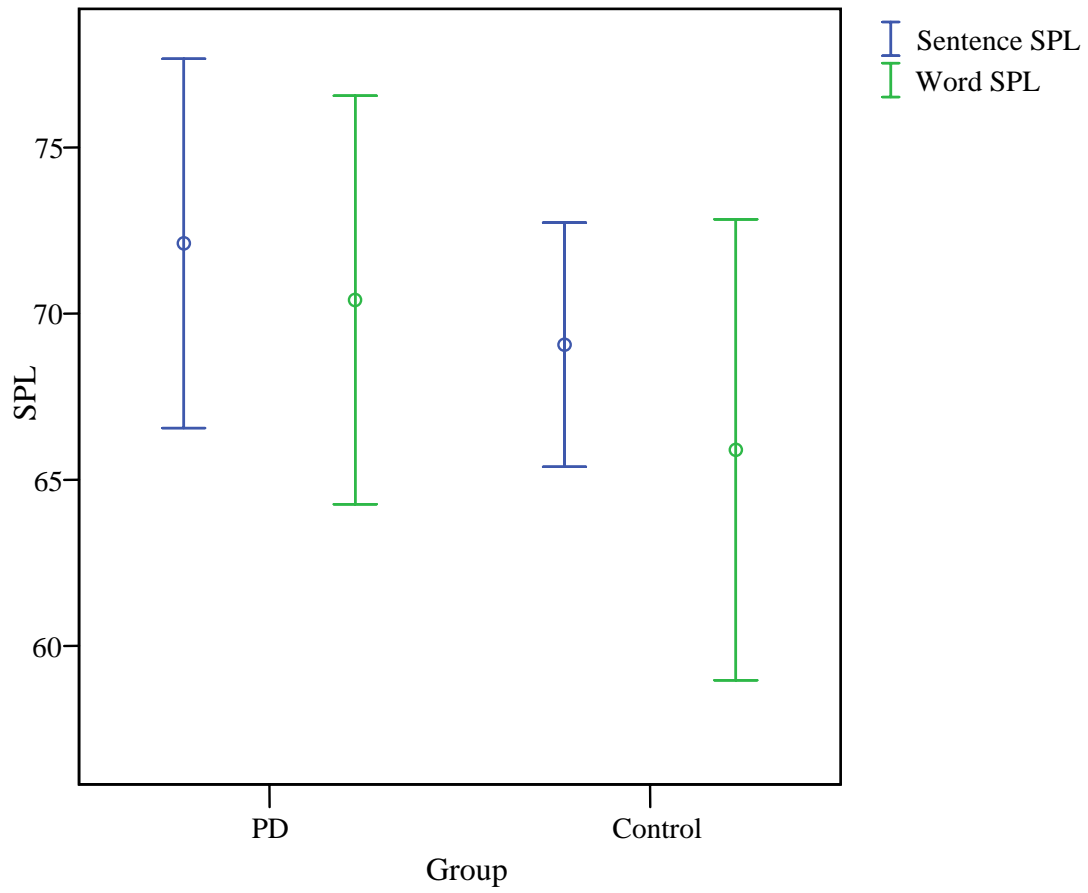


Figure 15. Confidence interval (95%) for word SPL and sentence SPL in condition 3, first repetition.

Discussion

The cumulative results of the present study indicated that individuals with PD responded similarly to controls in measures of duration, F0 and SPL, with slight variations. Individuals with PD had similar sentence and word duration to controls. Most measures of F0 and SPL were similar for individuals with PD and controls. However, word SPL was significantly higher for individuals with PD in condition 3. Finally, there were no differences between groups in F0 variability. This would indicate that the individuals with PD in the present study did not have more monopitch intonation than controls. In addition to the lack of clear differences between groups, there were also similar responses for both patient and control groups in repetitions for clarification, and when noise was presented through headphones.

If a participant was 'misheard' in the first reading, the subsequent repetitions allowed insight into how that person attempted clearer speech. Increased duration proved to be a consistent means for modifying speech in a repetition for clarification. Both groups increased word duration in repetitions in conditions 2 and 3. Additionally, they increased sentence duration for repetitions in condition 3. Both groups changed F0 in repetitions, but the type of change (increase or decrease) depended on the sequence of the repetition. In condition 3 both groups slightly decreased the mean sentence F0 of first repetitions and then increased mean sentence F0 of second repetitions. On the other hand, neither group changed sentence SPL for repetitions in conditions 2 or 3. However, for repetitions in condition 2, both groups decreased word SPL. Therefore, both groups were essentially similar in their use of increased duration, rather than changing F0 or SPL, for repetitions following a simulated mishearing.

Similarities were observed in the response of individuals with PD and controls to the presentation of noise through headphones. By analyzing the changes in the first readings in the three conditions, a predictable response to the presence of noise was documented. Both groups in condition 3 showed increased F0 and SPL as compared to condition 1. In contrast, neither group showed a change in duration when noise was present. Therefore, the introduction of noise through headphones had an influence upon F0 and SPL, but did not affect duration.

Duration

Comparison between participants with PD and controls. Overall, the individuals with PD had similar duration measures to controls. Other studies have measured rate of speech rather than duration and compared results of individuals with hypokinetic dysarthria and controls (Canter, 1963; Metter & Hanson, 1986). Rate of speech is comparable to duration measures because a rapid rate would suggest shorter sentence duration due to shorter speech segments and/or shorter intervals between these segments. The results of present study are consistent with those of Canter (1963), who found that median rate of speech did not differ between controls and participants with PD. There were three participants with PD who had either extremely slow or fast rate of speech in comparison to controls. The present study included one individual with PD who had particularly short mean sentence duration. In the baseline condition P5 had the shortest mean sentence duration of 1.78 seconds, while the overall mean duration for controls was 2.03 seconds, with a range of 1.87 to 2.32 seconds.

The speakers with PD in the present study may have been similar in their sentence duration to controls because of the relative mildness of their dysarthria. Metter and

Hanson (1986) found that individuals with severe hypokinetic dysarthria were more likely to exhibit a disordered rate of speech (either fast or slow). Since participants in the present study were perceptually rated as having mild or moderate dysarthria, they would not be predicted to differ substantially from the controls.

While the speakers with PD had longer mean durations for the first reading and repetitions in condition 3, changes between the first reading and the repetitions were minimal. This suggests that individuals with PD had a long duration the first time they read a sentence, but were not changing their duration for repetitions. On the other hand, the control group increased duration incrementally for the first and second repetitions. Individuals with PD did not increase sentence duration in the same way as the controls, but held a constant duration for repetitions. Therefore, in repetitions the individuals with PD were not actually increasing in duration as much as the controls. Comparable patient/control differences were observed in a study by Ho et al. (1999). They found that individuals with PD increased SPL with greater interlocutor distance, but that they did so to a lesser extent than the controls. In the present study, individuals with PD increased the duration of their utterances less substantially than the controls. Due to the modest sample size, it is difficult to determine whether the increased duration would be observed in other speakers with PD. Further research into durational changes during a repetition with a greater sample size could yield more reliable results.

Changes in repetitions for clarification. The present study found that individuals with PD, in similar fashion to the controls, used the strategy of lengthening the duration of their words and sentences to increase clarity in a repetition. In condition 3 both groups used longer duration upon first or second repetitions. Word duration increased

significantly for both groups from the first reading to the first repetition in both conditions 2 and 3. These data support the suggestion that increased duration is a reliable prosodic modification to improve speech clarity in normal and disordered speakers.

Increasing duration in a repetition for clarification is consistent with previously published accounts. The Clark et al. (1987) preliminary study found that healthy individuals used longer duration in a repetition for clarification. The Cutler and Butterfield (1990) study was mainly focused on durations of pauses at word boundaries and pre-word boundaries (syllable preceding the boundary), while the present study examined the total duration of a word or a sentence. The Cutler and Butterfield (1990) study indicated that healthy individuals increased the duration of segments prior to a word boundary or the duration of pauses at word boundaries in creating clearer speech in a repetition. When considered along with the present data, these findings indicate that increasing duration in a request for clarification is a frequently used approach for individuals with PD and healthy controls alike.

Changes due to the Lombard effect. In the present study there were no significant changes in duration associated with the presentation of noise through headphones. Increased duration was observed in the repetition of sentences in condition 3 and not in condition 2. However, an overall increase in duration for condition 3 was not found when comparing the first readings of the three conditions. This could indicate that the presentation of noise contributed to an increased duration when speakers believed they had been misheard. The lack of a consistent increase in speech duration in the presence of noise is consistent with Adams and Lang (1992), who found that the presentation of noise did not change duration.

Fundamental Frequency

Comparison between participants with PD and controls. The current investigation found that the F0 of individuals with PD was similar to, or slightly higher than controls. This finding is consistent with that of Metter and Hansen (1986), who reported that individuals with PD had the same F0 as controls. Other authors have reported that individuals with PD may have either lower or higher F0 than control speakers. For example, Dromey (2003) and Gamboa et al. (1997) found that individuals with PD had higher F0 than controls during a monologue task. On the other hand, Darley, Aronson, and Brown (1969a) indicated that individuals with PD can have low pitch. However, these authors did not specify low pitch as a primary characteristic of speech in Parkinson's disease. The participants with PD in our study did not exhibit low F0. This supports the idea that low F0 is not a key feature of hypokinetic dysarthria.

The apparently disparate reports in the literature, that speakers with PD can have lower, higher, or the same pitch as controls, may be at least partially explained by physiological changes that occur as the disease progresses. Research indicates that many individuals with PD have bowed or atrophied vocal folds (Hanson, Gerratt, & Ward, 1984). Bowed or atrophied vocal folds may lead to higher F0. However, these laryngeal signs are not present in all individuals with PD, since the disease affects individuals differently in terms of severity and clinical symptoms.

Participants with PD had a comparable sentence standard deviation of F0 to controls, and in condition 2 participants with PD had larger F0 *SD* compared to controls, although not significantly so. These data indicate that individuals with PD in the present study did not have the reduced F0 *SD* which is frequently associated with hypokinetic

dysarthria. These results, however, are inconsistent with those from other studies, which have perceptually identified monotone voice as a key characteristic of individuals with hypokinetic dysarthria (Darley et al., 1969a; Darley, Aronson & Brown, 1969b; Logemann et al., 1978). Likewise, other studies have identified reduced F0 *SD* through acoustic measures of speech in PD (Canter, 1965).

The results of the Metter and Hanson (1986) study offer some explanation for these conflicting results. They found that lack of variation in F0 was particularly apparent in individuals with more severe dysarthria. The present study did not have any participants who were perceptually rated as having severe dysarthria. In fact, the perceptual ratings indicate that only 2 of the participants were moderately dysarthric (having a rating above 25) and the other three PD participants had ratings of less than 25, indicating mild dysarthria. As Metter and Hanson (1986) noted, individuals with mild to moderate hypokinetic dysarthria may lack the speech characteristics that accompany more severe dysarthria, such as reduced F0 *SD*. Since the participants in the present study did not have severe dysarthria, they did not exhibit some of the more advanced dysarthric characteristics, such as reduced F0 *SD*.

Additionally, Zwirner, Murray, and Woodson (1991) found that individuals with PD had similar F0 *SD* to controls. In their study, individuals with PD and other neurologically impaired patients were examined acoustically and perceptually. Acoustically they found that there was no statistically significant difference between F0 *SD* in individuals with PD and controls (Zwirner et al., 1991). As in the present study, their participants were not severely dysarthric. As Metter and Hanson (1986) point out, participants with mild to moderate dysarthria are less likely to demonstrate reduced F0

SD. This suggests that not all research in hypokinetic dysarthria will find that individuals with PD have reduced F0 *SD*.

Changes in repetitions for clarification. Another parallel between individuals with PD and controls was observed in their behavior during repetitions for clarification. In condition 3 it was found that in the first repetition, mean sentence F0 decreased slightly. Then in the second repetition mean sentence F0 increased for both participant groups. Furthermore, neither group used changes in word F0 to emphasize a misheard word. Conclusions about F0 changes made in a repetition for clarification cannot be made since neither groups consistently changed F0 in repetitions. These inconsistent changes in F0 were nonetheless displayed by both participant groups.

It seems inconsistent that the mean F0 during repetitions slightly decreased for the first repetition and then increased for the second. However, there are other studies which identify similar discrepancies in F0 changes in repetitions. The Cutler and Butterfield (1991) study found that F0 of healthy individuals either slightly decreased, slightly increased, or remained unchanged in repetitions for clarification. The authors concluded that a slight increase in F0 is typical in speech which is intended to be clearer. Although the conclusions drawn from the Cutler and Butterfield study are dissimilar to those in the present study, the inconsistencies in mean F0 for repeated words are comparable. The results in the present investigation were inconsistent with another study which reported an increase in F0 in a repetition. Clark et al. (1987) reported that healthy individuals increased F0 upon a repetition for clarification.

The lack of increased F0 variability in repetitions is not consistent with similarly conducted research. Cutler and Butterfield (1991) found that individuals increased their

SD F0 for a repetition. The different units of measurement in the present study may partially explain this discrepancy in F0 *SD* data. Specifically, Cutler and Butterfield (1991) measured the F0 *SD* of individual words and syllables. The current study, however, measured F0 *SD* of entire sentences. A comparison of these two studies suggests that word F0 *SD* and sentence F0 *SD* may be affected differently in a repetition for clarification.

Changes due to the Lombard effect. While being presented noise through headphones, individuals with PD and controls modified F0 similarly. Neither group showed a difference in word F0 between experimental conditions. Yet both individuals with PD and controls increased sentence F0 incrementally from the baseline condition to the first readings in conditions 2 and 3. The greatest amount of change was seen in condition 3. Increase in F0 would be expected in condition 3, since inducing the Lombard effect increases F0 (Junqua & Anglade, 1990). Therefore, it would have been anticipated that speakers would increase their F0 upon presentation of noise through headphones.

While the mean F0 was expected to increase in the presence of noise, it was not anticipated that it would increase for the first readings in condition 2, where no noise was present. However, this occurred for both groups in condition 2. A possible explanation for this finding may lie in the speakers' anticipation of being misunderstood. It was observed by the experimenter that after being 'misunderstood' several times, individuals were subsequently trying to avoid being misheard during the remaining readings. For example, in the first sentences of the second condition, individuals had similar duration, F0, and SPL as they did in the baseline condition, but by the end of that condition their

effort had increased. The last sentences demonstrated increased duration, F0 and SPL compared to the first sentences in that condition. Table 7 reports the effects of this increased effort in the second condition in which speakers participated. It should be noted that the second administered condition could be either condition 2 or 3, since conditions 2 and 3 were randomly sequenced across speakers. The mean duration, F0, and SPL of the first five measured sentences versus the last five measured sentences of C2, C4, P3, and P5 are shown in Table 7. These data support the speculation that an increase in F0 may be due to awareness that a listener could be having difficulty understanding, and that subsequent miscommunication could be remedied with more deliberately clear speech.

The use of increased effort to avoid being misheard was also reported in research where participants communicated with an audience who demonstrated difficulty understanding (Cutler and Butterfield, 1991). These authors noted that individuals in their study may have started using increased effort on first readings because the participants knew their audience had a difficulty understanding. These findings and those from the present study suggest that the noise is not the only condition where individuals increase F0; they may also do so in a condition where they must communicate with a listener who has difficulty understanding.

Intensity

Comparison between participants with PD and controls. Not only were there many similarities between the two groups in duration and F0, but intensity was also frequently similar. Both groups increased SPL incrementally in the three conditions. Mean sentence SPL in repetitions for conditions 2 and 3 remained static for both groups.

Table 7

Comparison of the First Reading of the First Five Sentences and Last Five Sentences

in the Second Speaking Condition

Participant	Condition	Mean of First Five Sentences			Mean of Last Five Sentences		
		Duration	F0	SPL	Duration	F0	SPL
C2	2	2.41	123.00	68.38	1.76	136.05	74.02
C4	3	4.68	121.15	66.60	6.35	132.77	68.34
P3	2	8.96	177.17	72.65	14.56	193.65	76.72
P5	3	2.09	156.18	69.50	2.14	163.66	71.90

Although the difference was not significant, a slight variation was observed in conditions 2 and 3. The sentence SPL for participants with PD was actually higher than the control group. Additionally, the word SPL was significantly higher for the group with PD than the control group, under condition 3 from the first reading to the first repetition. Therefore individuals with PD overall had similar or elevated SPL in comparison to controls.

There is some discrepancy in the literature about the SPL of individuals with PD compared with controls. Perceptual and acoustic studies of hypokinetic speech have reported reduced intensity, along with a breathy and hoarse voice quality (Darley et al., 1969a; Ho et al., 1999; Logemann et al., 1978). However, three different studies using acoustic measurements to determine voice intensity found no differences between individuals with PD and controls (Canter, 1963; Dromey & Adams, 2000; Metter & Hanson, 1986). The results of the present study are consistent with the data from these latter studies, which identify no difference in SPL between individuals with PD and controls.

One possible explanation for the lack of reduced SPL in the present participants with PD may lie in the mildness of their dysarthria. They were rated as being only mildly to moderately affected. The Darley et al. (1969a), Ho et al. (1999), and Logemann et al. (1978) studies all had larger sample sizes (30, 12, and 200, respectively). A larger sample size would likely include more severely dysarthric participants. Therefore, the present study may have found little difference in SPL between participants with PD and controls because the sample size was so modest.

Another explanation for the similarities between intensity of individuals with PD and controls in the present study may be because the design included a task performed in isolation from other activities. Ho, Ianssek, and Bradshaw (2002) reported that individuals with PD can have reduced speech intensity and greater volume decay compared to controls when speaking and working on a simultaneous task. Individuals with PD may have less disordered behavior when participating in an isolated task, because they can devote greater attention to the task. Since the present study design included only a single task, this could have allowed the participants to focus on their speech and produce SPL comparable to controls.

Adams (1996) offered a possible explanation why acoustic and perceptual research do not always have consistent findings. The Darley et al. (1969a) and Logemann et al. (1978) reports were of perceptual studies, and they found that individuals with PD had reduced loudness (a perceptual rather than an acoustic characteristic). On the other hand, three acoustic studies reported that individuals with PD have similar intensity measures to controls (Canter, 1963; Dromey & Adams, 2000; Metter & Hanson, 1986). “The apparent lack of agreement between these acoustic and perceptual findings suggests that additional parameters of the acoustic speech signal may be contributing to the perception of reduced loudness in PD.” (Adams, 1996, p. 264). A recent study comparing acoustic measurements and perceptual severity ratings of the speech of individuals with PD found no clear association between the two (Dromey, 2003). These former observations along with the results of the present study support the notion that acoustic and perceptual findings do not always correlate.

Changes in repetitions for clarification. The present study found that neither individuals with PD nor controls increased intensity to improve clarity in a repetition subsequent to a simulated misunderstanding. This is inconsistent with other literature which indicates that in a repetition for clarity, healthy individuals increase SPL (Clark et al., 1987; Cutler & Butterfield, 1991; Martin & Ross, 1994). In the present study neither controls nor individuals with PD resembled the participants in the previous studies by increasing SPL in a repetition for clarification.

Other research reports that individuals with PD increased SPL in a need for increased clarity in communication, although they did not increase SPL to the same extent as controls (Ho et al., 1999). In the present study the need for increased clarity was sparked by a misunderstanding, while in the Ho et al. study the need was due to increased interlocutor distance. The Ho et al. study revealed that although individuals with PD had reduced intensity compared to controls, they were able to increase their intensity with greater interlocutor distance. This would suggest that individuals with PD in the present study would increase SPL in repetition, but with reduced intensity compared to controls. Therefore the lack of change exhibited by both individuals with PD and controls is inconsistent with other research.

It is possible that the inconsistency between the present study and the Ho et al. (1999) study comes from their different designs. The Ho et al. study was designed to elicit natural speech samples. Speech samples in the present study were read from a screen and were not samples of natural conversational speech. Reading a sentence from a screen may cause a difference in prosodic stress unlike that found in conversational

speech. This suggests that increasing the naturalness of the speech sample in the present study might have led to increased intensity in a repetition for clarification.

It could be speculated that speakers in both groups did not increase SPL for repetitions because they may have been focusing on duration. As mentioned previously, increasing duration was a strategy for increasing clarity in repetitions. The participants in the present study may have concentrated on increasing duration because the sentences were designed to allow the measurement of word boundaries (Cutler & Butterfield, 1990, 1991). If word boundaries are emphasized in these sentences, this would increase duration, due to longer pauses between words. For example, if the participant said, “He called in to view it himself,” the paired mishearing is, “The cold interviewer was selfish.” Since the words “in to view” was heard as one word “interviewer” a participant could stress word boundaries by taking larger pauses between these three words to make a clearer repetition. Cutler and Butterfield (1990) found that individuals introduced longer pauses between words to mark word boundaries. Both groups in the present study significantly increased duration for their repetitions and also decreased SPL. The use of the Cutler and Butterfield sentences, which combined word boundaries, may have caused participants in the present study to put more emphasis on duration and word boundaries, and less emphasis on other prosodic adjustments.

Increased word duration measures in conditions 2 and 3 could provide an explanation why word SPL did not increase for repetitions. Word duration significantly increased for the repetitions in conditions 2 and 3 for both groups. Eight of the 20 repetition words had voiceless continuants such as (/s/ or /f/). The continuants are easily prolonged because they do not constrict the vocal tract to discontinue air flow. For

example, if the word 'fit' was said with increased duration, the easiest part of the word to extend would be the /f/. The prolongation of this voiceless sound would decrease the word's SPL because a large portion of the word is voiceless, and therefore does not contribute much to the overall word SPL. Thus, the increased word duration in conditions 2 and 3 may have led to the decreased word SPL.

Changes due to the Lombard effect. Comparing the change in SPL found in the first reading of conditions 1, 2, and 3 indicates that both participant groups increased SPL with the presentation of noise through headphones. This is consistent with the findings of Adams and Lang (1992), who reported that individuals with PD demonstrated a significant increase in SPL subsequent to the introduction of noise through headphones. The increased SPL in condition 3 of the present study thus follows the patterns established in the Adams and Lang (1992) study. The presentation of noise, therefore, induced the Lombard effect. However, similar to the increase in F0 measures, the SPL of the first reading increased from condition 1 to condition 2. As previously discussed, some participants may have started making all of their utterances with increased effort to avoid being misheard. This increased effort may be reflected in the increased SPL. For example, Table 7 shows that some participants increased the SPL of their sentences from the beginning of a condition to the end of a condition. This is an explanation for why SPL increased from condition 1 to condition 2.

The finding in the present study that both groups increased in SPL similarly with the presentation of noise is not consistent with a report by Ho, Bradshaw, Ianssek and Alfredson (2000). They found that although individuals with PD increased in SPL with the presentation of noise, they did so to a lesser degree than control speakers. The

participants in that study were all identified as being hypophonic. This disparity in findings may be attributable to the mildness of the speech deficit in the present study's participants. Had they been more severely dysarthric, they might have increased SPL with the presentation of noise in a similar manner to the participants in the Ho et al. study.

Conclusion

The findings in this study reveal that there is more to be learned about the vocal modifications individuals make when repeating utterances for clarification. Future research models that would be helpful to this end might incorporate similar methods as the present study, but with a larger sample size and a more dysarthric PD group. Both individuals with PD and controls increased duration for repetitions, yet they did not increase F0 or intensity as was suggested by the Clark et al. (1987) study. Perhaps the conditions of the present study could be modified to appropriately reveal the prosodic changes which occur in a repetition for clarification.

One aspect of the present study which could be modified to more accurately reflect vocal modifications would be to increase the naturalness of the experimental conditions. People may not behave naturally when they are participating in an experiment, because they are more conscious that their behaviors are being observed. A key to collecting natural behavior in a study is to disguise from the participants what is being observed in the experiment. The present study could have been influenced by the Hawthorne effect, since participants knew they were participating in an experiment which recorded their voice. This awareness of the experimental circumstances may have altered participant performance with regard to prosody.

Collecting speech samples in a manner similar to the Ho et al. (1999) study may be a way to increase the naturalness of the conditions. The Ho et al. study had “preparation” periods between experimental conditions, during which time the individuals conversed with the experimenter and were recorded, yet did not realize that this was part of the experiment. The individuals thought the conversation was simply to help clear their minds before the next experimental condition, and the resulting speech samples were more natural. A study more similar to the Ho et al. research may result in more natural use of prosodic stress.

Another potential method for eliciting more natural speech could be modeled after Brinton, Fujiki, and Sonnenberg (1988). Experimenters in this study made requests for clarification by saying, “Huh?”, “What?”, or “I didn’t understand that” during a spontaneous conversational language sample. Although they were examining the linguistic characteristics of conversational repair, perhaps a similar method could be used to evaluate more natural characteristics of speech of individuals with Parkinson’s in comparison to age and gender matched controls.

Studying natural speech is a somewhat difficult task. Conversational speech tasks are not uniform, which makes it difficult to obtain reliable and valid comparisons of performance. This study was designed to use the same initial and repeated sentences for all participants so that there would be reliability in the comparisons of duration, F0, and SPL from the first reading to the repetition. Additionally, the sentences were to be the same for all participants so there would be increased validity in the comparison of measurements of duration, F0, and SPL between groups. These comparisons would be difficult to make in conversational speech.

The goal of the present study was to investigate the ability of individuals with PD to appropriately modify speech. According to the survey published in the Yorkston et al. (1994) study, individuals with PD indicated they would use improved production as a way to modify speech after being misunderstood. Scott et al. (1984) found individuals with PD did not appropriately use prosody. The present study sought to identify whether individuals with PD use prosody, and how they do so after a communication misunderstanding. The overall results of this study show that individuals with PD modified their speech in a similar way to controls as they repeated utterances for clarification. Both groups increased their duration, but did not consistently modify intensity or F0 in repetitions. Likewise, both groups responded similarly to the presentation of noise, by increasing SPL and F0. Therefore, it was observed that individuals with PD did not differ frequently from controls in repetitions and in their response to the different experimental conditions.

The findings of the present study may suggest that not all individuals with PD have a deficit when they need to modify speech appropriately. However, the participants with PD in the present study were only mildly to moderately dysarthric. Metter and Hanson (1986) found that abnormal acoustic measures were more prevalent in individuals with more severe dysarthria. They indicated that the progression of the disease has little or no effect upon some dysarthria characteristics. This leads to the hypothesis that a significant factor in a reduced ability to modify prosody could be the severity of dysarthria.

This study also helped highlight the variability in patients with PD. Metter and Hanson (1986) similarly described the variability which they found in their participants

with PD. They speculated that the basal ganglia were affected in specific areas differently, and that certain areas of the basal ganglia could contribute more to dysarthria. If this suggestion is true, the individuals with PD in the present study may not have been greatly affected in those localized areas of the basal ganglia, and were therefore able to produce speech similarly to healthy controls. A larger sample size of individuals with PD would better represent the population.

One reason why individuals with PD might not make appropriate prosodic changes could be related to their potential sensory deficits. As was discussed earlier, individuals with PD have been described as having a variety of sensory deficits (Dewey, 2000; Howe et al., 2003; Majsak et al., 1998; Mak and Hui-Chan, 2004; Schneider et al., 1987). These can interfere with their ability to appropriately gauge their performance, which can lead to disordered behavior. Yet, when suitable cues are provided, the behavior can be modified and the deficit decreases. One example of this was found in individuals with PD who demonstrated gait disturbances (Howe et al., 2003). They were able to modify the pace of their stride and diminished some of their shuffling gait characteristics when visual external cues were offered (Howe et al., 2003). These sensory deficits have also been speculated to interfere with speech skills (Ho et al., 2000; Lloyd, 1999; Schneider et al., 1986; Scott et al., 1984; Yorkston et al., 1994). The goal of the present investigation was to identify whether feedback suggesting a mishearing was a self-cue that would allow the individual with PD to recognize the need for modification of their speech.

External cues assist individuals with PD focus on the task. The tasks of the present study avoided external cueing, but was designed to allow modifications in speech

to be determined through self-cuing (the use and processing of sensory information independently). Although there was no external cueing, individuals with PD may have been able to focus their attention because of the simplicity of the task in the present study. Dual-task studies can cause modifications in behavior due to multiple sensory demands. Skeletal motor performance has been identified as more disordered in individuals with PD during multiple tasks (Beneke, Rothwell, Dick, Day, & Marsden, 1986; Oliveria, Gurd, Nixon, Marshall, & Passingham, 1998). Divided attention tasks involving speech have been found to result in reduced speech volume, increased volume decay, and reduced rate of speech (Ho et al., 2002). Since the present study only tested one task, sensory deficits that lead to reduced speech abilities may not have been observed.

In conclusion, some reports have indicated that individuals with PD have difficulty modifying speech when there is a greater need for clarity. However, our participants with PD were able to modify their speech appropriately. These findings lead to two hypotheses. One possibility is that not all individuals with PD have a sensory deficit that causes them to be impaired in modifying their speech appropriately. Another explanation is that the experimental conditions in the present study were not demanding enough to expose any sensory deficits. Both hypotheses need to be researched further to determine the factors contributing to the behaviors of individuals with PD in a communication misunderstanding.

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Appendix A

Informed consent to participate in a research study

You are being asked to participate in a research study in the Department of Audiology and Speech Language Pathology at Brigham Young University. This research is being conducted by Lynn Watkins, a graduate student, under the direction of Christopher Dromey, Ph.D. This research project is designed to compare the speech characteristics of people with Parkinson's disease with the speech of those who do not. You have been invited to participate in the study because you are individual who has Parkinson's disease or you are neurologically healthy and match the age and sex of a study participant with Parkinson's disease.

For this research you will be asked to visit the speech research lab in the John Taylor Building at BYU for one session lasting approximately 60 minutes. First, your hearing will be tested by having you listen to quiet tones through headphones. You will then be asked to read some sentences from a computer screen. While you read, your speech will be recorded with a microphone. For part of the study you will be asked to wear headphones, which will produce a sound that covers up the sound of your own speech while you read sentences from a screen. You are free to ask questions at any time during testing and expect those questions to be answered.

Names of participants will be kept confidential by assigning control numbers to data in all computer and paper records. Participation in the study is a voluntary service and you are free to withdraw from the study at any time without any effect on your relationship with BYU.

If you have any questions regarding this research project you may contact Dr. Christopher Dromey, 133 TLRB, Brigham Young University, Provo, Utah 84602; phone (801) 422-6461. If you have any questions regarding your rights as a participant in a research project you may contact Dr. Renea Beckstrand, Chair of the Institutional Review Board, 422 SWKT, Brigham Young University, Provo, UT 84602; phone (801) 422-3873.

I agree to participate in the research study described above. I confirm that I have read the preceding information and my questions have been answered to my satisfaction. I have been offered a copy of this form. I hereby give my consent for participation as described.

Name: _____ Age: _____

Signature of Participant

Date

Appendix B

Stimulus sentence pairs with ‘mishearings’
taken from the Cutler and Butterfield study (1990; 1991).

1. Original: He called in to view it himself.
Mishearing: The cold interviewer was selfish.
2. Original: The waiter should serve us first.
Mishearing: Creating a nervous curse.
3. Original: The scene must be headed at night.
Mishearing: A queen was beheaded tonight.
4. Original: She tried to free her trapped dog.
Mishearing 1: The side of a rear trapdoor.
*Mishearing 2: The side of her trapped dog.
5. Original: A rich woman in furs met us.
Mishearing 1: Which one can infer some method.
*Mishearing 2: Which woman in furs met us.
6. Original: Park in a nearer place next year.
Mishearing: How can an ear replace six ears.
7. Original: There’s an even split in the party.
Mishearing: This uneven kitten departed.
8. Original: The berry soup could poison us too.
Mishearing: This very super poisonous tool.
9. Original: He speaks of the fallen in discreet terms.
Mishearing 1: The speakers are calling indiscreet words.
*Mishearing 2: He speaks of falling indiscreet words.
10. Original: We should view these paintings in different light.
Mishearing 1: The smoothies pain is indifferent tonight.
*Mishearing 2: We should view this is a different night.
11. Original: The new boss is excellent and patient too.
Mishearing 1: Two nurses select all the patients too.
*Mishearing 2: The new boss will select all the patients too.
12. Original: That chocolate is expensive but nice.
Mishearing 1: A lot isn’t sensibly priced.
*Mishearing 2: That chocolate isn’t sensibly priced.
13. Original: Why can’t every member pay a fee.
Mishearing 1: I can’t even remember paying, did he?
*Mishearing 2: Why can’t I remember paying a fee?
14. Original: A bust or memorial is planned.
Mishearing: The customer always plans.
15. Original: Attach the partition to the backing.
Mishearing: The cheaper tissue will do for packing.
16. Original: Lots of hour-long sessions are needed.
Mishearing: Dogs devour long sections of needles.
17. Original: The new employees are just fine.
Mishearing 1: Renewal lawyers adjust fines.

- *Mishearing 2: The new employees adjust fines.
18. Original: It's hard to buy decent clothes cheaply.
Mishearing 1: Its art divides even close people.
*Mishearing 2: It's hard to buy even close cheaply.
19. Original: They wondered if I was making the rules.
Mishearing 1: To under-divide is breaking rules.
*Mishearing 2: They wondered if I was breaking rules.
20. Original: It heats to over four hundred degrees.
Mishearing: The feast won't afford under-threes.

* The second "mishearing" pairs were not developed by Cutler and Butterfield (1990; 1991), but were specifically designed for this study.

Appendix C

Stimulus sentence pairs with one 'misheard' word

1. Original: I saw the red dog running.
Mishearing: I saw the head dog running.
2. Original: The girl had to yell to be noticed.
Mishearing: The girl had to smell to be noticed.
3. Original: The leader saved the day.
Mishearing: The leader paved the day.
4. Original: The sun shone brightly midday.
Mishearing: The gun shone brightly midday.
5. Original: The dress wouldn't fit anymore.
Mishearing: The dress wouldn't sit anymore.
6. Original: What I need is a cookie.
Mishearing: What I feed is a cookie.
7. Original: After the movie she cried.
Mishearing: After the movie she lied.
8. Original: Tonight I will take my pill.
Mishearing: Tonight I will take my bill.
9. Original: It was the best course offered.
Mishearing: It was the best horse offered.
10. Original: The dark house must have a ghost.
Mishearing: The dark house must have a roast.
11. Original: He would bend the metal bar.
Mishearing: He would mend the metal bar.
12. Original: Mother's face was beautiful.
Mishearing: Mother's vase was beautiful.
13. Original: She thought the grade was good.
Mishearing: She thought the trade was good.
14. Original: The airplane flew past the runway.
Mishearing: The airplane blew past the runway.
15. Original: The long shore was finally seen.
Mishearing: The long door was finally seen.
16. Original: The silver can was in the room.
Mishearing: The silver fan was in the room.
17. Original: She wants to make a blanket.
Mishearing: She wants to take a blanket.
18. Original: He always beats me in checkers.
Mishearing: He always cheats me in checkers.
19. Original: I always take a hook fishing.
Mishearing: I always take a book fishing.
20. Original: She shot the basketball quickly.
Mishearing: She caught the basketball quickly.