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Role of Therapeutic Devices in Enhancing Speech Intelligibility and Vocal Intensity in an Individual with Parkinson's Disease

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A thesis

presented to

the faculty of the Department of Audiology and Speech-Language Pathology

East Tennessee State University

In partial fulfillment

of the requirements for the degree

Master of Science in Communicative Disorders

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by

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August 2012

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Keywords: Parkinson's Disease, Behavioral Speech Therapy, Speech Intelligibility, Average Intensity, Ambulatory Phonation Monitor, Auditory Masker

ABSTRACT

Role of Therapeutic Devices in Enhancing Speech Intelligibility and Vocal Intensity in an Individual with Parkinson's Disease

by

Swetha Swaminathan

The prevailing speech therapy techniques for treating hypokinetic dysarthria in individuals with Parkinson's disease (PD) yields improvements within the clinical setting, however, maintenance and generalization of acquired behaviors continue to be a challenge. The purpose of this study was to investigate the effects of portable therapeutic devices including Ambulatory Phonation Monitor with biofeedback (APM) and auditory masker in maintenance and carryover of improved speech. Our participant was an individual diagnosed with PD for the past 25 years who continued to display speech disturbances despite undergoing several behavioral speech therapy programs and neurosurgical procedures. Speech intelligibility and average intensity measures under automatic, elicited, and spontaneous speech tasks were recorded pre- and postusage of APM and auditory masker for a period of 1 week each. Preliminary findings showed no significant difference in the measures between means ($P>0.05$) across all tasks for both the devices. Suggestions for future research on therapeutic devices are discussed.

DEDICATION

Thank you, God, for being very kind to me and bestowing me with wisdom and perseverance needed for my pursuit. I take this opportunity to express my sincere love and gratitude to my parents – Swaminathan and Geetha and my brother Amar. The ongoing encouragement and unflinching support you have provided throughout my life has helped me achieve the goals I once only dreamed of. I would also like to express my gratefulness to Appana Rao mama and Sarala mami. I couldn't have made it through this process without your prayers and blessings. I love you all!

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CHAPTER 1

INTRODUCTION

Nature and Purpose of the Study

"Hypokinetic dysarthria is a perceptually distinctive motor speech disorder associated with basal ganglia circuit pathology" (Duffy, 2005). Speech in Parkinson's disease (PD), which is a prototype of hypokinetic dysarthria, is characterized by perceptual features such as reduced loudness (hypophonia), reduced prosodic pitch inflection (monotone), hoarse voice, imprecise articulation, and festination (acceleration of words at the end of sentences) (Darley, Aronson, & Brown, 1975; Duffy 2005). These speech abnormalities may adversely affect the patient's social, economic, and psychological wellbeing (Oxtoby, 1982; Pitcairn, Clemie, Gray, & Pentland, 1990; Ramig et al., 2001). The treatment to alleviate motor and speech symptoms exhibited by individuals with PD includes pharmacological and surgical treatments and speech therapy. Despite availability of many treatment alternatives, the management of speech and voice disorders in PD has been challenging for both medical and rehabilitation practitioners.

Medical treatments consisting of neuropharmacological and neurosurgical approaches for the treatment of PD have had consistent positive outcomes and effects on the motor limb functions, but their effects on the associated speech and voice disorders have been insignificant and less compelling (Baker, Ramig, Luschei, & Smith, 1997; Kleinow, Smith, & Ramig, 2001). Some examples of medical treatments that have produced positive outcomes on limb function and insignificant changes in speech include the treatment with levodopa (Louis, 2001; Rigrodsky & Morrison, 1970; Thanvi, Lo, & Robinson, 2007; Wolfe, Garvin, Bacon, & Waldrop, 1975), the treatment with fetal dopamine transplant (Baker et al., 1997); bilateral thalamotomy and pallidotomy surgeries (Ghika et al., 1999; Schulz, Peterson, Sapienza, Greer, & Friedman, 1999);

and Deep Brain Stimulation (DBS), which has shown worsening of hypokinetic dysarthria post-surgery (Iulianella, Adams, & Gow, 2008; Tripoliti et al., 2011).

Traditional behavioral speech therapy techniques focusing on articulation, rate, and prosody involve conscious training to coordinate the respiratory, phonatory, and articulatory system and have proven to be beneficial within the treatment setting, although generalization and carryover of the treatment effects have been questionable (Fox, Morrison, Ramig, & Sapir, 2002; Johnson & Pring, 1990; Ramig et al., 2001; Weiner & Singer, 1989). However, research over the past 20 years including a series of randomized control trials have established Lee Silverman Voice Treatment (LSVT®) as an efficacious behavioral treatment that improves vocal fold adduction and overall voice and speech production in individuals with PD (Pinto et al., 2004; Ramig et al., 2001; Yorkston, Spencer, & Duffy, 2003). The LSVT® is an alternative, intensive, behavioral speech treatment that emphasizes high-effort, repetitive, loud phonations to improve respiratory, laryngeal, and articulatory functions during speech (Ramig, Countryman, Thompson, & Horii, 1995; Ramig & Dromey, 1996). To overcome the sensory mismatch between perceived vocal effort and vocal output in individuals with PD, LSVT® also accentuates simultaneous sensory awareness training, achieved by cueing and consistently asking individuals to "speak loud" (Fox et al., 2002). Though studies on LSVT® for individuals with PD have produced positive long-term (2-year) treatment outcomes for group data in a controlled clinic environment, successful maintenance of treatment effects to nonclinical environment offers challenge (Fox et al., 2002). There is a questionable transfer and generalization of the improved speech characteristics during conversational speech in natural setting following LSVT® treatment (Adams & Dykstra, 2009; Bourdreaux, 2011).

Another alternative treatment for individuals with PD with decreased loudness is the use therapeutic devices that include loudness biofeedback devices (e.g., APM), vocal amplifiers, Altered Auditory Feedback (AAF) devices including Delayed Auditory Feedback (DAF) devices, and auditory maskers. A biofeedback device is used in individuals who speak at a normal intensity level with cues but lack insight into maintenance of vocal intensity (Rubow & Swift, 1985), a condition that is commonly observed in individuals with PD. The Ambulatory Phonation Monitor (APM) (Cheyne, Hanson, Genereux, Stevens, & Hillman, 2003), a biofeedback device, offers long-term continuous tracking of vocal parameters and also provides feedback to the user when the target phonatory behaviors, such as increased or decreased intensity are not maintained (Hillman, 2004). Most studies conducted on the use of biofeedback treatment have concluded that it has a potential to impact speech production and communicative effectiveness, on the other hand there is a lack of efficacy data for use of APM with biofeedback (Yorkston, Spencer, & Duffy, 2003). Auditory masker, another alternative treatment option to behavioral speech techniques similar to biofeedback devices, has been used to improve loudness in individuals with PD (Adam & Lang, 1992). Masking works on a well-known phenomenon, Lombard effect, first described by Etienne Lombard in 1911 as the spontaneous tendency of speakers to increase their vocal intensity when talking under the presence of noise. The Lombard effect helps enhance speech output in individuals with PD (Gryczka et al., 2011). Studies done on small group of individuals with PD have shown that the use of white noise masking in clinical settings for individuals with Parkinson's disease results in an increase in vocal intensity ranging from 2.1 to 7.5dB (Adam & Lang, 1992). However, there has been no study till date measuring the changes in vocal intensity after using portable white noise masker in a nonclinical setting.

The primary purpose of this study was to examine and document the changes in vocal intensity and speech intelligibility with the use of two kinds of feedback devices, (a) APM with biofeedback relying on tactile feedback, and (b) the auditory masker relying on auditory feedback. The participant for the study was an individual with PD, who had not benefitted by LSVT® and other traditional behavioral therapy techniques. Additionally, the aim of this study was to investigate the possible use of the devices in everyday life and to determine patient satisfaction on the use of the devices.

Need for the Study

Current literature on behavioral speech techniques and LSVT® indicates improvement in speech characteristics within clinical settings; however, carryover and maintenance of the improved speech characteristics to nonclinical environment has been a challenge (Adams & Dykstra, 2009; Allan, 1970; Bourdreaux, 2011; Sarno, 1968; Johnson & Pring, 1990; Weiner & Singer, 1989; Yorkston, Spencer, & Duffy, 2003). This indicates the need to investigate alternate treatment methods using therapeutic devices and their role in helping individuals with PD generalize their improved speech characteristics to a variety of speech tasks in both clinical and natural settings. This case study aimed at elucidating the everyday use of portable therapeutic devices such as APM and auditory masker (AM) for maintaining and generalizing the improved vocal loudness and speech intelligibility in an individual with PD. The specific research questions were:

1. Are therapeutic devices helpful in facilitating improvement in vocal intensity and intelligibility during spontaneous, elicited, and automatic speech tasks in an individual with PD?

2. Which of the two devices, APM and AM, is perceived to be more comfortable for long-term everyday use in natural settings?

Conditionally, it is hypothesized that the present study will aid in identifying and promoting the use of APM with biofeedback and auditory maskers for individuals with PD who have not been observed to have long-term improvements in vocal loudness and intelligibility with LSVT and traditional speech therapy techniques.

CHAPTER 2

REVIEW OF THE LITERATURE

Parkinson's disease (PD), a neurodegenerative disorder with progressive impairment in motor functions and cognition, is the most common movement disorder in the world (Albin, 2006; de Lau & Breteler, 2006). According to National Institute of Neurological Disorders and Stroke (2012) incidence of PD increases with age, with an average onset age of 60 years, although 5% to 10% of the individuals with PD experience 'early onset' with symptoms beginning before the age of 50. It is estimated that in the United States more than 500,000 people suffer from this disease, with 50% more prevalence in men compared to women (NINDS, 2012). PD is attributed to the depletion of dopaminergic neurons in the substantia niagra of the basal ganglia (Spencers et al., 2010), which reduces secretion of dopamine resulting in a loss of ability to execute smooth, controlled movements (Baker et al., 1997). Recent studies have indicated that PD may also be attributed to the loss of nerve endings that produce the neurotransmitter norepinephrine, thus causing deficiency of norepinephrine. Norepinephrine functions as a neurotransmitter, and also a stress hormone controlling many automatic functions of the body, such as pulse and blood pressure. In consequence, the loss of norepinephrine in individuals with PD is also believed to contribute to the nonmotor features observed, including fatigue, abnormalities of blood pressure regulation, and emotional disorders (NINDS, 2012).

Characteristics

The diagnosis of PD is based on the symptoms exhibited by the individual due to the lack of a current definitive test (Jankovic, 2007). The clinical criteria for the diagnosis, or the cardinal features of PD, can be grouped under the acronym TRAP: (1) Tremor at rest- stereotyped, rhythmic involuntary movement; (2) Rigidity - abnormal muscle tone and increased resistance;

(3) Akinesia or bradykinesia - slowness or no movement; and (4) Postural instability - the impairment of mechanisms responsible for maintenance of upright posture during standing or walking (Jankovic, 2007).

Most of the above listed cardinal features make an impact on the respiratory, phonatory, articulatory, and/or resonatory subsystems of speech production, affecting the speech in individuals with PD (Swigert, 1997). Reports indicate that around 75%-89% of individuals with PD experience voice and speech disorders (Fox et al., 2002). The dysarthric characteristics of individuals with PD can be analyzed and documented by kinematic, spirometric, perceptual, acoustic, aerodynamic, videostroboscopic, and electromyographic measures of the various subsystems (Baker et al., 1998; Darling & Huber, 2011; Dromey et al., 1995; Gerratt & Ward, 1984; Johnson & Pring, 1990; Moore & Scudder, 1989; Ramig, 1992; Ramig et al., 1994, 1995; Smith et al., 1995; Solomon & Hixon., 1993; Tjaden, 2011).

Respiratory System

Patients with PD display reduced vital capacity and reduced expiratory drive, resulting in increased breathing rate coupled with irregularities in breathing pattern (deep breathing and hyperventilation) (Solomon & Hixon, 1993; Stewart, 2000). These respiratory irregularities make an impact in production of speech, which might include decreased loudness, production of fewer words, faster interpause speech rate (more rapid speech rate between pauses), and/or longer and more frequent pauses (Hammem & Yorkston, 1996; Metter & Hanson, 1986; Pitcairn et al., 1990; Solomon & Hixon, 1993). Specifically, individuals with PD exhibit reduced intra-oral pressures during /p/ production in a syllable repetition task (Netsell et al., 1975; Solomon & Hizon, 1993) and decreased sustenance of prolonged vowel phonation (Boshes, 1966; Canter,

1965; Mueler, 1971). Reports indicate that individuals with PD might also display higher lung volume initiation and termination (increased breathing rate) during extemporaneous speech than in reading (Huber & Darling, 2011).

Phonatory System

Hypokinetic dysarthria also affects the phonatory system. Phonatory characteristics of individuals with PD analyzed by perceptual measures display reduced loudness, monotonous pitch and loudness, reduced stress, variable rate of speech, short rushes of speech, and hoarseness (Darley et al., 1969a; Ramig et al., 2004). Acoustic measures demonstrate higher fundamental frequency, reduced maximum phonation time, higher jitter-shimmer percentage, and increased voice onset time (VOT) in individuals with PD (Canter et al., 1965b; Dogan et al., 2008; Forrest et al., 1989; Ramig et al., 1988). Slightly more pronounced phonatory disturbances are displayed by individuals with PD when compared to articulatory disturbances under clinical-perceptual ratings (Ackerman & Ziegler, 1991; Logemann et al., 1978; Logemann & Fisher, 1981). The two explanations for the more prominent phonatory disturbances can be accounted by (1) progressive involvement of speech organs beginning at the laryngeal level and proceeding in the oral direction; and (2) the increased vulnerability of the laryngeal apparatus to the pathophysiological processes underlying PD (Ackerman & Ziegler, 1991; Logemann & Fisher, 1981). Physiological and neuropathological mechanisms attributing to the disordered phonatory characteristics in individuals with PD include (1) reduction in speech motor output (2) deficiency in sensory perception, and (3) abnormal structural changes in the larynx. Reduced speech motor output, caused by reduced TA muscle amplitude (Baker, Ramig, Luschei & Smith, 1998) may lead to decrease in neural drive to the muscles of the speech mechanism. This in turn might result in

reduced vocal loudness (hypophonia), reduced pitch inflection (hypoprosodia), and also reduced range of articulatory movements (hypokinetic articulation) (Albin, Young, & Penny, 1989; Penny & Young, 1983; Ramig et al., 2004). The reduced speech motor output observed in individuals with PD may also be explained by basal ganglia dysfunction, age-related muscle atrophy, or a combination of these conditions (Duffy, 2005). Secondly, disordered phonatory characteristics in individuals with PD may likewise be attributed to deficiency in sensory perception that prevents them from accurately regulating (internal cueing or scaling) the optimal amount of effort to produce adequate loudness (Demirci, Grill, McShane, & Hallet, 1995). Reduced vocal loudness levels in voluntary tasks (Canter, 1965a), and inability to reflexively regulate their volume in conversational speech without explicit volume instructions (Ho et al., 1999; Schulz & Grant, 2000) support this view of individuals with PD having deficiency in sensory perception. Thirdly, few studies have documented structural changes in the larynx through videoendoscopic and videostroboscopic studies that can attribute specifically to reduced loudness exhibited by individuals with PD. Laryngeal abnormalities in the form of bowed vocal cords, and an abnormally large glottic aperture have been observed, which results in incomplete approximation of the vocal cords and thus decreasing vocal loudness during speech (Hanson et al., 1984; Smith et al., 1995).

Articulatory System

Individuals with PD exhibit disordered production of consonants and vowels, disfluencies, and variable rates of speech. Kinematic and acoustic studies have revealed that individuals with PD display ‘undershooting’ of articulatory gestures (Ackermann & Ziegler, 1991; Forrest et al., 1989). Notably, there is reduction of articulatory precision in stop consonants (/t/, /d/, /k/, and /g/) that can be attributed to consistent reductions in both peak

velocity and amplitude of mandibular and labial openings (Ackermann & Ziegler, 1991; Forrest et al., 1989; Logemann & Fisher, 1981). Individuals with PD also exhibit misarticulations during production of affricates /tʃ/ and /ʤ/, and fricatives /s/, /z/, /ʃ/, and /f/ that have been attributed to inadequate tongue elevation to achieve complete closure while producing affricates, and close constriction of the airway in the production of lingual fricatives (Logemann & Fisher, 1981). Various other explanations are available to explain the phenomenon of misarticulation in individuals with PD. It has been suggested that the weakness exhibited as smaller Muscle Action Potential (MAP) under EMG studies during the production of stop consonants is probably of neurogenic origin as opposed to muscle contractile weakness, muscular fatigue, or deficits at the myoneural juncture (Netsell et al., 1975). The combination of the neurogenic weakness and the acceleration phenomenon combine to produce "articulatory undershoot" in individuals with PD (Logemann & Fisher, 1981; Netsell et al., 1975). Individuals with PD exhibit impaired vowel articulation, characterized by reduced vowel articulation index, reduced formant transitions, and restricted acoustic vowel space (Ackermann & Ziegler, 1991). This can also be contributed by reduced movement of the articulators (Forrest et al., 1989; Skodda, Visser, & Schlegel, 2011; Tjaden et al., 2005). Imprecise articulation of consonants and vowels in individuals with PD impacts the ability to perform diadochokinetic tasks such as Alternate Motion Rate (AMR) and Sequential Motion Rate (SMR) that involve rapid movements of the lips, tongue tip, and back of the tongue required, for e.g., repetition of /papapa/ or /pataka/ respectively (Canter, 1965b; Connor et al., 1989; Hirose et al., 1981). The reduction in rate of movement has been attributed to increased levels of tonic resting and background activity (Leanderson et al., 1971; Moore & Scudder, 1989; Netsell et al., 1975) and also due to loss of reciprocity between agonist and antagonistic muscles (Leanderson et al., 1971). In addition to disordered productions of

consonants and vowels, 15% to 45% of individuals with PD also exhibit stuttering like speech disfluencies typically at the beginning of the utterance or after a pause characterized by rapid and blurred phoneme repetitions (Logemann et al., 1973; Sapir et al., 2001). Moreover, prosodic deficits and disordered rates of speech have also been consistently reported in individuals with PD. Typical characteristics of the prosodic deficits in hypokinetic dysarthria include monoloudness, reduction of stress, and monopitch; however, significantly higher pitch levels and reduced pitch range have been documented in individuals with PD (Canter, 1963,1965a). There is high variability in rate of speech exhibited by individuals with PD, with 6% to 13% of the population exhibiting rapid rate or short rushes of speech (Canter 1965a; Canter 1965b; Logemann et al., 1978) and some reports with evidence supporting presence of speech rates slower than normal rates (Canter 1963). It has been concluded that the hypokinetic dysarthria is by no means homogenous with respect to speech rate (Ackermann & Ziegler, 1991).

Resonatory System

Resonatory system is also affected in individuals with PD. Hypernasality is a perceptual quality associated with excessive nasal air emission due to velopharyngeal insufficiency that may be caused by paresis or paralysis of levator veli palitini and superior constrictor muscles of the pharynx or inappropriately timed closure and opening of the port (Darley, Aronson, & Brown, 1969a; McWilliams, Morris, & Shelton, 1990). Hypernasality may be seen in some individuals with PD (Logemann et al., 1978). Aerodynamic and kinematic studies have indicated reduced velopharyngeal (VP) movements that can be positively attributed to the severity of the disease (Hoodin & Gilbert, 1989).

Co-Occurring Neurological Deficits

The patterns of hypokinetic dysarthria and the extent of the involvement of each speech subsystem in individuals with PD are highly variable. The disease severity, dysarthria severity, task type, coexisting conditions, and/or specific neurological substrate affected are some of the factors assumed to influence the variability (Schulz & Grant, 2000). The existence of other co-occurring neurological deficits such as dementia (Aarsland et al., 2007), cognitive deterioration (Hely et al., 2005), sensory processing deficits (Stamey et al., 2007; Tinnazi et al., 2006), and psychiatric and sleep disturbances (Gjerstad et al., 2006) may also account for the variability observed in the speech and voice exhibited by the individuals with PD (Schulz & Grant, 2000).

Treatment

Management of the motor and speech symptoms observed in individuals with PD is multi-fold, including medical, surgical, and behavioral therapy.

Pharmacological Treatment

Medications developed and prescribed to treat PD include those that replace dopamine (Levadopa/L-dopa), and those that enhance dopamine levels (dopamine agonists) (Schulz & Grant, 2000). L-dopa is a frequently prescribed and widely used drug that emulates the effects of natural dopamine. L-dopa is always combined with carbidopa that produces Sinemet, the principle medication for treating PD (Marsden & Parkes, 1977). Carbidopa also prevents conversion of L-dopa to dopamine before crossing the blood brain barrier and hence increases cerebral levodopa bioavailability (Rao et al., 2006). With respect to improvement in speech characteristics, long-term effects of L-dopa seem to be far less consistent (De Letter, Santens, & Borsel, 2005). There have been reports of subjective short-term improvements in L-dopa therapy

that include improved voice quality, pitch variation, and articulation and improved rate, pause and rhythm during oral reading (Critchley, 1981; Rigrodsky & Morrison, 1970; Wolfe et al., 1975). Some studies have documented positive effects on fundamental frequency (Sanabria et al., 2001) and significant improvement of word intelligibility, posttreatment with L-dopa (De Letter et al., 2005). Labial pressure as measured by nonspeech and speech tasks has shown improvement following L-dopa administration (Nakano, Zubick, & Tyler, 1973). However, it has to be noted that no obvious and consistent speech improvement has been recorded when compared to dramatic improvement in limb symptoms with L-dopa treatment (Rigrodsky & Morrison, 1970). Several other studies have not found significant subjective improvement in speech (Quaglieri & Celesia, 1977), changes in oral function (Gentil, Tournier, Pollack, & Benabid, 1999), acoustic measures of vowels (Poluha, Teulings, & Brookshire, 1998), or speech breathing (Solomon & Hixon, 1993) post L-dopa treatment. On the contrary, worsening of speech with exacerbation of disfluencies due to L-dopa treatment has been documented (Louis, 2001). Discrepancies in speech and voice functions observed in individuals with PD undergoing treatment with L-dopa can also be due to patient-related differences of severity of dysarthria, dosage levels, etc., across the studies conducted (Schulz & Grant, 2000). Having stated that L-dopa is particularly effective at controlling bradykinesia and rigidity (Goestz et al., 2004), certain studies have testified that motor complications such as hypokinesia, dyskinesia, and dystonia associated with long-term levodopa treatment in Parkinson's disease are common and they can be more disabling than the disease itself (Thanvi et al., 2007). After 5 years of levodopa therapy, nearly 50% of patients develop motor complications and after 10 years nearly 100% of patients are affected by them (Verhagen & Metman, 2002). Motor complications are significantly more common with levodopa therapy compared with monotherapy with dopamine agonists. As the

disease progresses, the individuals with PD on L-dopa may also experience a "wearing-off" effect characterized by a shorter duration of benefit from each levodopa dose, hence causing the motor symptoms to re-emerge. This can be attributed to L-dopa's relavitely short half-life of ~1.5 hours. This "on-off" effect is characterized by unpredictable abrupt fluctuations in motor state from when the medication is effective and symptoms are controlled ("on") and when parkinsonian symptoms worsen ("off") (Rao et al., 2006). The resulting motor complications can be treated by adding a dopamine agonist (dopamine level enhancer), monoamine oxidase-B (MAO-B) inhibitor, or catechol *O*-methyltransferase (COMT) inhibitor (Rao et al., 2006).

Dopamine agonists including apomorphine, bromocriptine (Parlodel), lisuride, pergolide (Permax), cabergoline, quinpirole, ropinirole (Requip), and pramipexole (Mirapex) enhance the dopamine levels in the brain (Schulz & Grant, 2000). Dopamine agonists are shown to reduce the effects of "off" time and worsening of motor impairments, reducing the need for L-dopa and also prolong the effect of dopamine (Goetz et al., 2005; Tolosa & Valldeoriola, 1994). COMT inhibitors such as tolcapone (Tasmar) also aid in decreasing the degradation of L-dopa, extending its half-life and thus reducing the "off" time (Jankovic & Marsden, 1993).

MAO-B inhibitors such as selegiline (Deprenyl) aid in inhibiting the degradation of dopamine and also prolong the anti-Parkinsonian action of L-dopa (Shea et al., 1993). Improvement in measures of rate and range of oral motor diadochokinesis and in measures of vital capacity and words per exhalation were observed during speech reading in individuals under selegilline (Shea et al., 1993). However, dopamine agonists, COMT inhibitors, and MAO-B inhibitors may not be well tolerated by frail elderly patients and those with cognitive impairment. They are also associated with excessive daytime sleepiness (Verhagen & Metman, 2002).

Despite abrupt fluctuations in motor state and possible motor complications with prolonged use, L-dopa remains to be most effective in treating the symptoms of Parkinson's disease. Recent studies have established that after an initial period of dramatic benefit with the use of L-dopa several limitations that include fluctuations, dyskinesias, and dystonias that can be very disabling and difficult to treat become apparent (Thanvi et al., 2007). Even though dopamine agonists and MAO-B inhibitors help in reducing the "off-time" with progression of Parkinson's disease, there is often a need to add L-dopa when dopamine agonists alone fails to improve symptoms (Allain et al., 2000), which again results in associated motor complications such as dyskinesia, dystonia, and hypokinesia. In addition to these problems, long-term use of the drugs can cause confusion, dementia, hallucinations, and delusions (Calne, 1995). These factors may indicate the need for surgical intervention to aid in long-term improvement of motor functions.

Surgical Treatment

Neurosurgery is generally recommended for patients experiencing increased severity of motor fluctuations or disabling dyskinesia due to long-term use of PD drugs (Weaver et al., 2005). There are two major surgical approaches to PD: (1) Ablative surgery (i.e. thalamotomy and pallidotomy); and (2) deep brain stimulation (DBS) of the thalamus, internal globus pallidus (GPi), and subthalamic nucleus (STN).

Ablative Surgery. Ablative surgery can be of two types. They are as follows:

Thalamotomy. It is a surgical procedure of lesioning the ventralis intermedius (VIM) of the ventrolateral thalamus (Grossman & Hamilton, 1993) that interrupts the increased excitatory outflow from the thalamus (Marsden & Obeso, 1994). This is accomplished with a technique known as stereotactic surgery in which "a thin probe is delicately inserted into the brain through

a hole in the skull" (Stern & Lees, 1990). Lesions in the ventral intermediate nucleus are highly effective in the alleviation of parkinsonian tremor in more than 85% of patients (Jankovic et al., 1995; Kelly & Gillingham, 1980). This method is used to treat severe drug-resistant Parkinsonian tremor and also for unilateral or asymmetric PD where tremor predominates (Eskandar et al., 2001; Tasker et al., 1983). Speech has not been shown to improve postoperatively after VIM thalamotomy, but indeed a deterioration of speech is observed after the procedure and as PD progresses (Tasker et al., 1983). Unilateral operations of the thalamus in the dominant hemisphere produces speech disturbances such as dysarthria, monotonous voice, slow speech (Jenkins 1968), decreased vocal loudness, and articulation difficulties (Allan et al., 1966), than in nondominant hemispheres. Bilateral talamotomy is performed to relieve bilateral tremor and rigidity (Grossman & Hamilton, 1993). However, speech problems resulting from bilateral thalamotomy include persistent worsening of dysarthria (Tasker et al., 1983). Additionally, bilateral thalamotomies result in excessively high rate of cognitive and speech problems (Mastumoto et al., 1976) that prevents the use of this procedure for most patients with Parkinson's disease. For the many ill-effects post thalamotomy, many of the other surgical options are considered for treating individuals with PD.

Pallidotomy. This procedure involves lesioning the globus pallidus internus (GPi) of the basal ganglia, which interrupts the increased inhibitory outflow from the globus pallidus (Marsden & Obeso, 1994). Dopamine is found in high concentrations in the corpus striatum under normal circumstances, whereas for persons with PD dopamine input into the corpus striatum is depleted, resulting in over activity of the GPi, which is inhibitory to the thalamus and brainstem (Eller & Dan, 1997). Lesioning the GPi thus causes the release of inhibition to the thalamic and brainstem motor centers. This lesion may improve all major Parkinsonian

symptoms, including bradykinesia, contralateral tremor, rigidity, and dyskinesias (Grossman & Hamilton, 1993; Laitinen et al., 1992). Pallidotomy for Parkinson's disease has been largely restricted to unilateral procedures because of reports of significant hypophonia, dysarthria, and worsening cognitive and neuropsychiatric function after bilateral pallidotomy (Intemann et al., 2001). Studies have indicated that mildly dysarthric Parkinson's patients may benefit most from unilateral pallidotomy, perhaps due to less overall destruction of the basal ganglia sensorimotor control circuits involved in oral facial functions, thus increasing the chances to observe improvements on vocal intensity and articulatory measures postsurgery (Schulz & Grant, 2000).

Deep Brain Stimulation (DBS). It is a procedure that refers to the electrical stimulation of the thalamus, the subthalamic nucleus (STN), or the GPi for treatment of Parkinsonian symptoms. It involves placing a small quadripolar electrode in the ventral intermediate nucleus (VIM) of the thalamus, the subthalamic nucleus (STN), and/or GPi with continuous stimulation to the areas at frequencies below 100 hertz (Grossman & Hamilton, 1993). In contrast to thalamotomy or DBS of the VIM, DBS of the GPi and STN has reliably alleviated all the cardinal motor symptoms of Parkinson's disease including akinesia and bradykinesia, rigidity, tremor, and gait (Ghika et al., 1998; Kumar et al., 2000). However, most studies examining the effects of DBS have shown worsening of hypokinetic dysarthria postsurgery (Iulianella et al., 2008; Tripoliti et al., 2011). Individuals with PD postbilateral STN stimulation displayed reduced intelligibility during reading and spontaneous speech (Rousseauax et al., 2004). Deterioration in both acoustic and perceptual measures for an individual during stimulation-on vs. stimulation-off conditions were also reported (Narayana et al., 2004). Stimulation of the ventral-oral nucleus of the thalamus produced silencing and slowing of speech (Schaltenbrand, 1975). On the contrary, some of the recent literature examining deep brain stimulation of the

subthalamic nucleus (STN-DBS) for management of PD symptoms have reported positive effects of this surgery on velopharyngeal control during syllable production (Hammer et al., 2011), acoustic voice variables (Dromey et al., 2000), stuttering (Walker et al., 2009), and glottic tremor (Klostermann et al., 2008). These changes are considered to be insignificant clinical changes, moreover many of these studies have relied on the Unified Parkinson's Disease Rating Scale (UPDRS) speech item (item 18) as a means of measuring functional speech improvement. Item 18 in UPDRS classifies speech as normal or unintelligible on a scale of 0 to 4, which may be insufficiently sensitive for measuring changes in voice and speech (Rousseaux et al., 2004). The reasons for the disparate responses of speech, nonspeech, and limb function to STN DBS can be attributed to the apparent differences that exist in the neural innervation, motor origins and motor organization between motor-speech and motor-limb systems. The neural mechanisms contributing to speech, voice, and swallowing disorders associated with PD are not generally understood (Fox et al., 2002).

All of the neurosurgical procedures have shown consistent desirable effects on motor-limb characteristics of PD but not on motor-speech characteristics (Baker et al., 1997, Kleinow et al., 2001). This has necessitated the need for supplementation with behaviorally-based techniques addressing speech and voice issues in individuals with PD.

Speech Therapy

Even though speech impairments which occur in around 75%-89% of individuals diagnosed with PD appear to be obvious incentives for speech therapy, only 3%-4% receive treatment (Fox et al., 2002). Explanations for this discrepancy include that (1) Because speech treatment has previously not been successful for individuals with PD, physicians do not refer them for therapy, (2) Individual performs well with the help of external cues in the quiet

examination room of the physician during follow-up visits, (3) Compensatory techniques adapted by the individual during the initial stages might make the caregivers unaware of the problem (Ramig, Fox & Sapir, 2007). On the contrary, those individuals with PD receiving treatment for dysarthria tend to show improvement in their speech intelligibility compared to patients who have not received speech therapy (Johnson & Pring, 1990, Robertson & Thompson, 1984; Scott & Caird, 1983). Speech therapy involves using behavioral therapy techniques focusing on training to control rate of speech, prosody, clear speech (articulation), and loudness; and/or using therapeutic devices such as biofeedfack devices, auditory masking, and DAF. The choice of therapy is based on the patient's need (Stewart, 2000).

Speaking Rate. Speech rate is often considered as a powerful modifiable variable for improving the intelligibility of dysarthric speech, but the correlation between rate and intelligibility is unknown (Duffy, 2005; Marshall & Karrow, 2002; Yorkston et al., 1992). Some individuals with PD exhibit faster rates of speech than individuals without PD (Hammen & Yorkston, 1996). Rate control in the form of a slower-than-typical rate has long been used as a clinical technique for improving intelligibility in dysarthria (Yorkston, Hakel, Beukelman, & Fager, 2007). Rate control has been achieved by using traditional therapy of increasing pauses and/or stretching out articulation and also with the use of external pacing devices that include DAF, pacing board, metronome, computer software such as PACER (Hammen & Yorkston, 1996), behavioral instructions, and biofeedback (Duffy, 1995; Yorkston, Beukelman, Strand, & Bell, 1999; Yorkston et al., 2007). Reports suggest that slowed articulatory rates in dysarthric individuals are associated with articulatory displacements and vocal tract shapes that more closely approximate those of healthy speakers (Adams, 1994; Caliguiri, 1989; Turner, Tjaden, & Weismer, 1995). As articulatory rate is slowed, articulatory displacements tend to increase,

resulting in an expanded acoustic working space and phonetic events that are more acoustically distinct (Tjaden & Welding, 2004). While using PACER, a computer pacing software, it has been found that individuals with PD demonstrated shorter speech duration, frequent pauses, and more time per pause than the control group (Hammen & Yorkston, 1996). It has also been identified that when individuals with PD are paced at 60% of habitual reading rate, their speech duration, i.e. the duration of pauses, moves towards a more normal value (Hammen & Yorkston, 1996). Fifty percent of 27 speakers with various neurological diagnoses and dysarthrias exhibited a significant 20% improvement in scaled intelligibility when using rate reduction methods such as pacing boards, alphabet board, and delayed auditory feedback with delays of 50ms, 100ms, and 150ms (Van Nuffelen et al., 2010). No significant differences in intelligibility measures or articulation rate (AR) or speaking rate (SR) between DAF50ms, DAF100ms, or DAF150ms has been identified, nor has the ideal delay for DAF. However, combining DAF and prolonged speech caused increased intelligibility scores in one of three subjects when compared to using DAF only (Dagenais, Southwood, & Lee, 1998). Studies have shown that speakers with dysarthria can voluntarily reduce overall articulation rate for sentence-level material or a reading passage (Lowit, Brendel, Dobinson, & Howell, 2006; McRae, Tjaden, & Schoonings, 2002; Turner & Weismer, 1993). It was also established that speaking slower on demand is a more naturalistic rate control method as compared to assisted techniques like delayed auditory feedback, alphabet supplementation or pacing board; however, speaking slower on demand seemed to be the least efficient rate control method in conversational speech (Van Nuffelen et al., 2010). Also, factors predicting those individuals who will benefit from therapeutic techniques aimed at reducing speech rate are poorly understood, although the type of dysarthria, habitual speaking rate, and overall speech severity did not differentiate individuals who did and did not

experience improved intelligibility when using rate reduction (Van Nuffelen et al. 2010). Moreover, knowledge of how speakers with dysarthria voluntarily adjust pause location, pause time, and articulation time to accomplish an overall reduced speech rate is incomplete (Van Nuffelen et al. 2010). In contrast to positive results on improved intelligibility postrate reduction, reports also suggest that rate control might have an inverse effect on intelligibility even though a significant reduction in articulation rate (AR) and speaking rate (SR) have been reported (Van Nuffelen et al., 2009). Overall, it can be concluded with the help of recent studies that rate reduction can help in improving intelligibility of speech in most individuals with PD.

Clear Speech. Clear speech has been elicited with instructions to speak as clearly and precisely as possible (Picheny et al., 1985; Schum, 1996), and it has been found to increase intelligibility when compared to conversational speech in individuals with and without PD (Bradlow, Kraus, & Hayes, 2003; Goberman & Elmer, 2005; Hargus Ferguson & Kewley-Port, 2002; Helfer, 1997; Picheny et al., 1985; Schum, 1996). This increase in intelligibility with clear speech production has been found to be independent of both listener factors and speaker factors (Bradlow et al., 2003; Picheny et al., 1985; Schum, 1996). In the past, clear speech production has been studied as a strategy for increasing the intelligibility of speech produced for listeners with hearing impairments (Ferguson & Kewley-Port, 2007). Acoustic analyses conducted have revealed decreased articulation rate, increased frequency and length of pauses, increased fundamental frequency (Fo), increased variability of speaking Fo, and increased intensity of certain consonants with clear speech in neurologically normal individuals (Bradlow et al., 2003; Picheny et al., 1986). The few published studies investigating clear speech in dysarthria associated with PD suggest that relative to habitual conversational speech clear or hyperarticulate speech is associated with reduced articulatory rate, increased mean fundamental frequency, and

increased speaking fundamental frequency variability in both reading and monologue tasks (Dromey, 2000; Goberman & Elmer, 2004). However, there is a certain need for further studies focusing on determining whether or not the production of clear speech improves the perceptual characteristics or intelligibility of speech in PD and also whether the improvements would generalize outside of clinical setting (Goberman & Elmer, 2005; Tjaden & Welding, 2011).

Prosody Therapy. Prosody is defined as that aspect of spoken language encompassing the rhythm, intonation, and stress conveying form and meaning and emotional state of the speaker (Monrad-Krohn, 1957). It is responsible for conveying subtle changes of meaning independent of words or grammatical order and also makes a major contribution to the emotional content of speech (Monrad-Krohn, 1957; Scott & Caird, 1983). Prosodic abnormalities in speech attributes to the 'excess/equal stress' patterning noted in individuals with PD (Monrad-Krohn, 1957; Yorkston et al., 2007). Effects of variety of treatment approaches have been studied, which include the use of behavioral instruction and biofeedback devices. Speech therapy focusing on increasing awareness of the prosodic problems and practicing more normal patterns of intonation in conversational speech in addition to intonational exercises have resulted in improvement in prosodic characteristics of speech in individuals with PD (Scott & Caird, 1983). In addition to therapy techniques, use of visual feedback device resulted in 25% more improvement in prosodic characteristics than using prosodic exercises alone (Scott & Caird, 1983). Individuals with PD showed significant improvement post prosody-focused therapy with visual aid (Visispeech), the Frenchay Dysarthria Scale, and also in several other secondary speech measures including increased volume, fundamental frequency, and pitch range when compared to individuals who had not received therapy (Johnson & Pring, 1990). Another case study described the positive long-term effects of using computer assisted auditory and visual feedback (SpeechViewer) in

attaining the target F_0 and speaking rate (LeDorze, 1992). The participant was instructed to model the desirable speech behavior during sentence reading with the help of a real-time display of F_0 and intensity against time spread over 25 therapy sessions. The improved prosody and intelligibility of speech was also found to have maintained 10 weeks posttreatment (LeDorze, 1992). It should be noted that biofeedback was found to be effective in many studies involving individuals with PD (LeDorze, 1992; Scott & Caird, 1983) and this implies the need for further studies on utility of therapeutic biofeedback devices in long-term everyday use.

Loudness Therapy. Maximizing intelligibility is an important treatment goal for many patients with dysarthria. Based on the recent findings of increased fundamental frequency (F_0) variation in the loud condition relative to that produced in reduced speech rate and habitual condition in individuals with dysarthria, it has been concluded that therapeutic techniques focusing on increasing vocal loudness might be preferred to techniques focusing on rate reduction for maximizing intelligibility (Tjaden & Wilding, 2011). An increased vocal intensity is accompanied by a reduction in articulatory rate as well as enhanced F_0 variation in dysarthria (Yorkston et al., 2007), and increase in vocal intensity has direct association with a more precise articulation (Carrara et al., 1997; Countinho et al., 2009; Ramig et al., 1994). A popular speech therapy technique adapted for treating loudness issues related to hypokinetic dysarthria in PD is the Lee Silverman Voice Treatment (LSVT®). The LSVT® is a widely used behavioral therapy primarily focusing on increasing vocal loudness by increasing phonatory effort (Ramig et al., 1995). LSVT® is designed to address the issues of decreased speech motor output and deficiency in sensory perception associated with PD by its five essential concepts: (1) focus on voice, (2) improve sensory perception of effort, (3) administer treatment in high effort style, (4) treat intensively, and (5) quantify treatment related changes (Ramig et al., 2004). LSVT® emphasizes

on multiple repetitions of simple high effort vocal productions within the context of an intensive therapy regimen to improve respiratory, phonatory, and articulatory functions during speech (Fox et al., 2002; Ramig et al., 1994, 1995, 2001). Various studies have focused on establishing the efficacy of LSVT® through a wide range of outcome measures and study designs (Adams & Dykstra, 2009; Fox et al., 2002; Ramig et al., 2004). Research has indicated the effectiveness of LSVT® in attenuating respiratory and laryngeal function abnormalities associated with PD (Fox et al., 2002). Decrease in pretreatment hyperfunctional behavior (false vocal fold closure, laryngeal elevation) (Countryman & Ramig, 1993) and increased subglottal air pressure and maximum flow declination rate accompanying increased vocal SPL (Ramig & Dromey, 1996) have been documented following treatment with LSVT®. Articulation (Ramig et al., 2001), amplitude of articulatory movements (Fox et al., 2002), phonatory stability (Dromey et al., 1995), and orofacial expression (Fox et al., 2002) have all been shown to improve with LSVT®. Although LSVT® represents an uncommon and impressive effort at establishing efficacy evidence in the treatment of speech disorders in PD, it raises few concerns (Adams & Dysktra, 2009). LSVT® has a primary focus of treatment on the intensity (laryngeal), which can be too narrow to be applicable to most hypokinetic dysarthrias where non-laryngeal processes such as oral articulation, velopharyngeal control, repiratory and postural control may lead to reduced intensity levels (Adams & Dykstra, 2009). Another foremost concern is that most of the efficacy studies done on LSVT® have obtained measures from clinical settings. Although evidence for positive effects of LSVT® is strong comparative to other behavioral treatments, long-term maintenance of effects with and without ongoing treatment needs to be established (Yorkston et al., 2003). The vocal parameters (amplitude and fundamental frequency) have been measured in the laboratory, with limited information on the ability to generalize clinically achieved vocal

intensity to extemporaneous speech in natural setting (Bourdreaux, 2011). This implies a definite requisite for future studies focusing on innovative treatment options focusing on transfer and maintenance of improved speech characteristics to natural setting.

Therapeutic Devices. Recent research has focused on the role of therapeutic devices in the treatment of hypokinetic dysarthria. Devices include wearable intensity biofeedback device (Ambulatory Phonation Monitor-APM) and masking device.

Biofeedback Devices. According to Rubrow (1984), "Biofeedback is a process of transducing a physiological variable, transforming the signal to extract useful information and displaying that information to the subject in a format that will facilitate learning to regulate the physiological variable" (p. 1). Biofeedback devices can transduce and display the vocal parameters to the speakers and are hence well suited to aid in impairments resulting from the respiratory and phonatory systems such as vocal loudness (Yorkston et al., 2003). A portable microcomputer based biofeedback device was developed to generalize improved speech characteristics outside the clinic (Rubow & Swift 1985). Three sets of speech samples, one each in clinical setting and natural setting without feedback and one in natural setting with feedback, were obtained using the microcomputer. The microcomputer provided data for the measurement of treatment transfer, and it recorded the time of occurrence for each low-intensity alarm generated by decrease in speech intensity and the total speaking time between the alarms. It was found that the average alarm interval in the clinic increased and a substantial portion of that increase was retained outside the clinic while wearing the feedback device. There was significant improvement in perceptual dimensions of loudness, rate, and stress that include reduction in articulatory breakdown, imprecise consonants, monopitch, monoloudness, breathiness, and vowel distortions (8-9 parameters on 12), and improvement in acoustic measures with

spectrographic analyses revealing predominant periodic vibration with good formant structure and reduced noise component. These data suggests the utility of a microcomputer-based wearable device for assessing treatment effects as well as for improving transfer (Rubow & Swift 1985).

Another study compared the effectiveness of speech therapy with and without Vocalite, a voice-operated light source as visual feedback. The therapy involved intensive period of prosodic exercises aimed at improving loudness and pitch variations. Results indicated a positive 45% improvement on the ratings of speech prosody under visual reinforcement when compared to a 33% improvement without feedback (Scott & Caird, 1983). Studies have consistently proven that visual and auditory feedback assists in greater percentage of improvement in prosody and intelligibility, when compared to behavioral therapy without visual and auditory feedback (Johnson & Pring, 1990; LeDorze, 1992). In addition to auditory and visual feedback devices, effects of tactile feedback devices in improving speech characteristics in individuals with PD have also been investigated.

Ambulatory Phonation Monitor (APM) is a wearable monitor and a tactile biofeedback system for provision of long-term, continuous tracking of parameters of vocal function (Cheyne, Hanson, Genereux, Steven, & Hillman, 2003). In addition to collecting objective data on fundamental frequency (F_0), sound pressure level (SPL), phonation duration and periodicity via an accelerometer, which measures vibration of neck surface during phonation, the APM can gather data continuously for up to 10 hours approximately (Hillman, 2004). Hauser et al, 2005 determined the effect of using of APM with biofeedback when provided in conjunction with LSVT®, on maintaining the target loudness level. Baseline data were obtained from two participants with PD before the initiation of treatment with LSVT® and APM biofeedback.

Results indicated that the combined use of LSVT® and APM in individuals with PD did not demonstrate better maintenance of target loudness levels, as well no reports of consistent increase in vocal intensity over the course of LSVT therapy. The author attributed the reasons of technical and patient scheduling issues along with protocol violations and small number of participants for the unanticipated findings (Hauser et al., 2005). Boudreaux et al, 2011 used APM to determine objective differences in vocal parameters including mean fundamental frequency, mean amplitude, and total phonation time in 10 older individuals with and without PD. 93% of the participants found that the APM did not affect their speech in any way, and comfort in public was rated 4.67 out of 5, and 3.82 out of 5 for APM being a comfortable device. 7% of the participants who reported that the APM affected their speech were in the PD group. These participants also commented that the APM served as an external cue and reminded them to use the techniques learned in their previous sessions (Boudreaux et al, 2011), thereby insinuating the possibility of APM serving as a good fit for long-term every day wear in aiding individuals with PD.

It can be inferred from all the studies that biofeedback devices demonstrate possibilities of altering the physiological variables and perceptual speech characteristics, thus displaying positive potential to improve communication effectiveness in individuals with dysarthria (Yorkston et al., 2003).

Auditory Masker. Another well-known biological phenomenon that induces variations in loudness levels is the Lombard's effect (Adams & Lang, 1992). It is the decreased ability of the auditory system to detect one sound in the presence of another due to auditory masking (Gulick, Gescheider, & Frisina, 1989). In other words, Lombard's effect describes the predisposition to

increase the vocal loudness in the presence of noise. However, the underlying mechanism of this phenomenon is still unclear (Nonanka et al, 1997). The effect of white noise of 40, 70, and 90 dBSL during phonation tasks in individuals with PD, whose hearing thresholds were below 20dBHL was determined. It was established that the vocal utterance intensity and frequency was progressive and proportional to the increase in masking, thus resulting in improvement in vocal utterance stability (Gryczka et al., 2011; Quedas et al., 2007). Studies have shown that Lombard's effect resulted in an increase in vocal intensity ranging from 2.1-7.5dB, during a reading task when subjected to auditory masking at 90dBSPL through headphones, than that produced when the participants were instructed to speak at their maximum intensity level (Adams & Lang, 1992). Marked improvements of voice in terms of vocal utterance stability (intensity and fundamental frequency) were documented when the individuals with PD were subjected to binaural auditory masking of 100dBSPL through headphones when compared to conditions of 150ms delay in auditory feedback and habitual listening (Countinho et al., 2009). Similarly, it has also been determined that individuals with PD produce higher mean SPL under 70dBA of background multitalker noise than at a level they perceived to be 'comfortable' and 'twice as comfortable' (Darling & Huber, 2010). It can be concluded from these studies that auditory masking results in greater improvements in fundamental frequency and vocal intensity than when compared to conditions involving instructions to speak louder or using delayed auditory feedback. It also has to be noted that individuals with PD produced the most efficient respiratory patterns in the noise condition as compared to other loudness conditions (Sadagopan & Huber, 2007). This effective use of the respiratory system may have produced large enough gains in SPL to overcome the small mouth opening, which suggests that individuals with PD may use the respiratory system to a greater extent than articulatory system while speaking in the

presence of background noise than when under instructions to speak at a specific level (Darling & Huber, 2010; Sadagopan & Huber, 2007). While these studies have explored the immediate effects of background noise in the speech of individuals with PD in a clinical setting, no study has been testified the long-term effects of using an in-the-ear auditory masker during extemporaneous speech in a natural setting.

Regardless of the availability of variety of speech treatment options aimed and proven to improve loudness, intelligibility, and rate of speech in clinical settings, carryover and maintenance of these improved speech characteristics during spontaneous speech has been a challenge (Adams & Dykstra, 2009; Allan, 1970; Bourdreaux, 2011; Johnson & Pring, 1990; Sarno, 1968; Weiner & Singer, 1989; Yorkston, Spencer, & Duffy, 2003). This necessitates the need for other treatment options that possibly have the potential to help individuals with PD generalize their improved speech characteristics to a variety of speech tasks in both clinical and natural settings. In this case study the primary aim is to determine the effects of everyday use of portable therapeutic devices such as APM and auditory masker for improving vocal intensity and speech intelligibility in an individual with PD. The specific research questions are:

1. Are therapeutic devices helpful in facilitating improvement in vocal intensity and intelligibility on spontaneous, elicited, and automatic speech tasks in an individual with PD?
2. Which of the two devices, APM and AM, is perceived to be more comfortable for long-term everyday use in natural settings?

CHAPTER 3

METHOD

Design

This is a within-subject case study comparing treatment effectiveness of APM and AM in an individual with PD. Both treatments were administered for an equal period of 1 week. The dependent variables in this study were: (1) average amplitude (vocal intensity) and (2) perceived intelligibility of speech. Independent variables were therapeutic devices, (1) APM with the biofeedback device, and (2) AM.

Participant

A 74-year-old male, native speaker of English, with a 25-year diagnosis of PD, and a recent history of head injury served as our participant. His speech was characterized by monopitch, decreased loudness, imprecise consonants, hypernasal resonance and nasal air emission. He had previously received speech services using LSVT®, but weak voice, decreased loudness, and decreased intelligibility during conversational speech continued to persist. He also underwent deep brain stimulation in the past, which resulted in an attenuation of his motor-limb symptoms with no improvement in his speech characteristics. The participant was fluent in reading with the use of reading glasses, and his hearing seemed adequate to converse and follow verbal commands without issues. The participant was medicated throughout the treatment and testing, and no changes in medications were made during the study period.

Procedure

Figure 1 displays the general experimental protocol followed during the study.

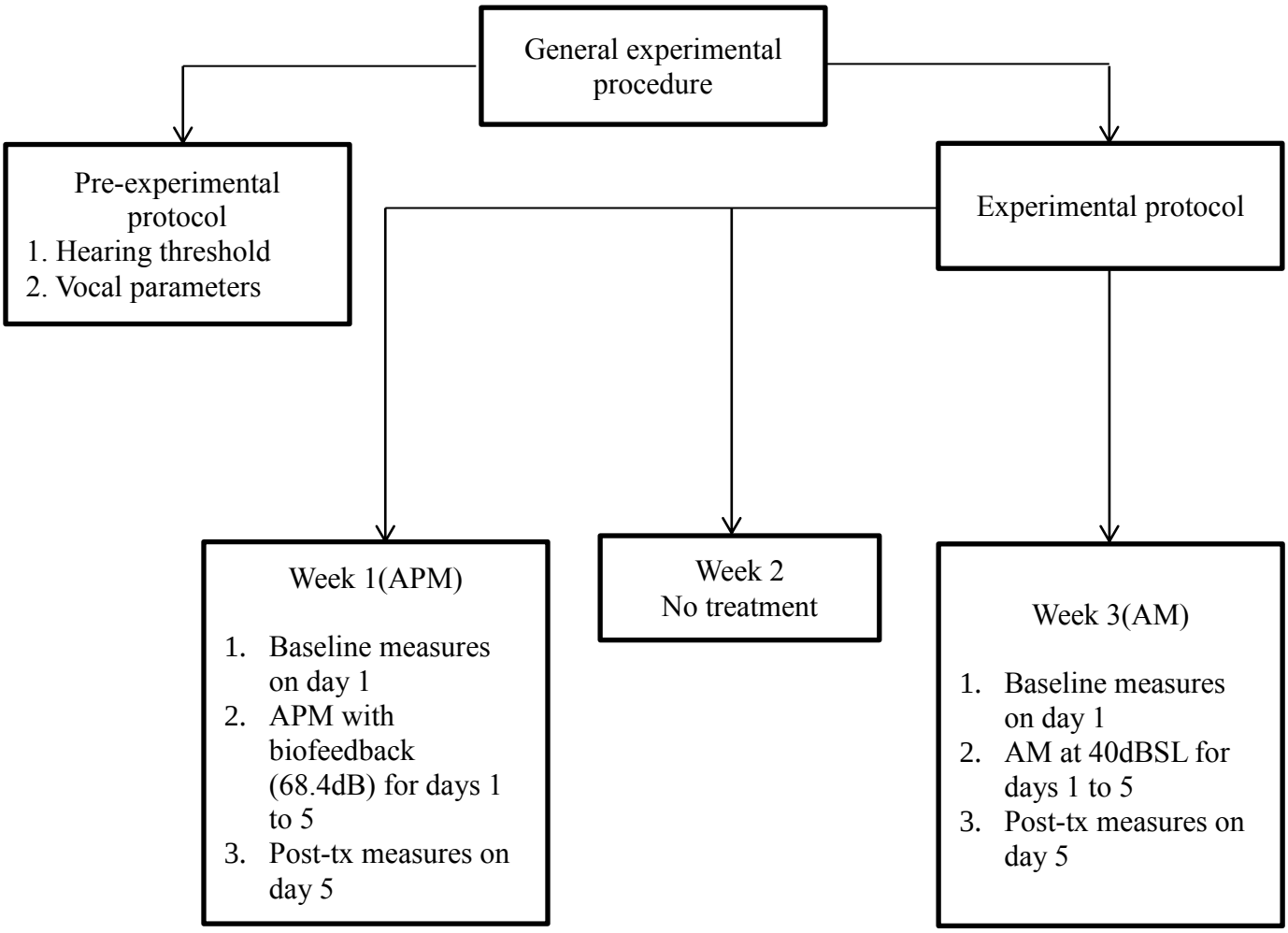

Figure 1. General Experimental Protocol

Pre-experimental Protocol. Prior to the actual treatment protocol, the participant's hearing thresholds were obtained at 500,1000, and 2000 Hz in a sound treated room using GSI-61 audiometer. The participant's pure tone average (PTA) was found to be 13.3 and 1.6; and threshold for white noise to be 0dB and -5dB, in his right and left ear respectively. The auditory maskers were programmed to produce noise at 40dBSL of the white noise thresholds, specifically at 40dBSPL and 35dBSPL for right and left ear respectively.

Baseline vocal parameters of average intensity and fundamental frequency over a typical 8-hour day were determined by fitting the participant with the APM. Below is an outline of the fitting and calibration process. (1) Before the fitting APM was connected to the computer and designated microphone using the company guidelines, (2) Following this, the accelerometer sensor was attached to the participant's throat precisely at midline in the hollow area above the sternal notch and below the larynx using the secure adhesive glue, (3) The wire was then fed down his shirt exiting at the waist, which was plugged into the APM, (4) As part of the calibration process, the participant was instructed to sustain phonation on the vowel /a/, beginning softly and increasing his volume to the loudest he can produce, (5) Having achieved adequate calibration, the clinician initiated the monitoring phase, disconnected the APM from the computer and the microphone, and placed the APM in the waist pouch. The participant was instructed to wear the device all day long and keep it safely away from water. Data were retrieved from the APM, at the participant's residence following a typical 8-hour day involving conversation with his spouse and family. Based on the average amplitude of 63.4dB from the baseline data, the biofeedback level for week 1 was determined to be 68.4dB, which is +5dB of the average amplitude.

Treatment Protocol. All voice recordings were obtained in a quiet room occupied by the participant and the primary examiner. The participant was seated in a chair and the data collection took approximately 20 minutes for every session. Treatment protocol followed the schedule described below.

Week 1 – APM with Biofeedback. Figure 2 presents the steps involved in week 1 of the experimental protocol

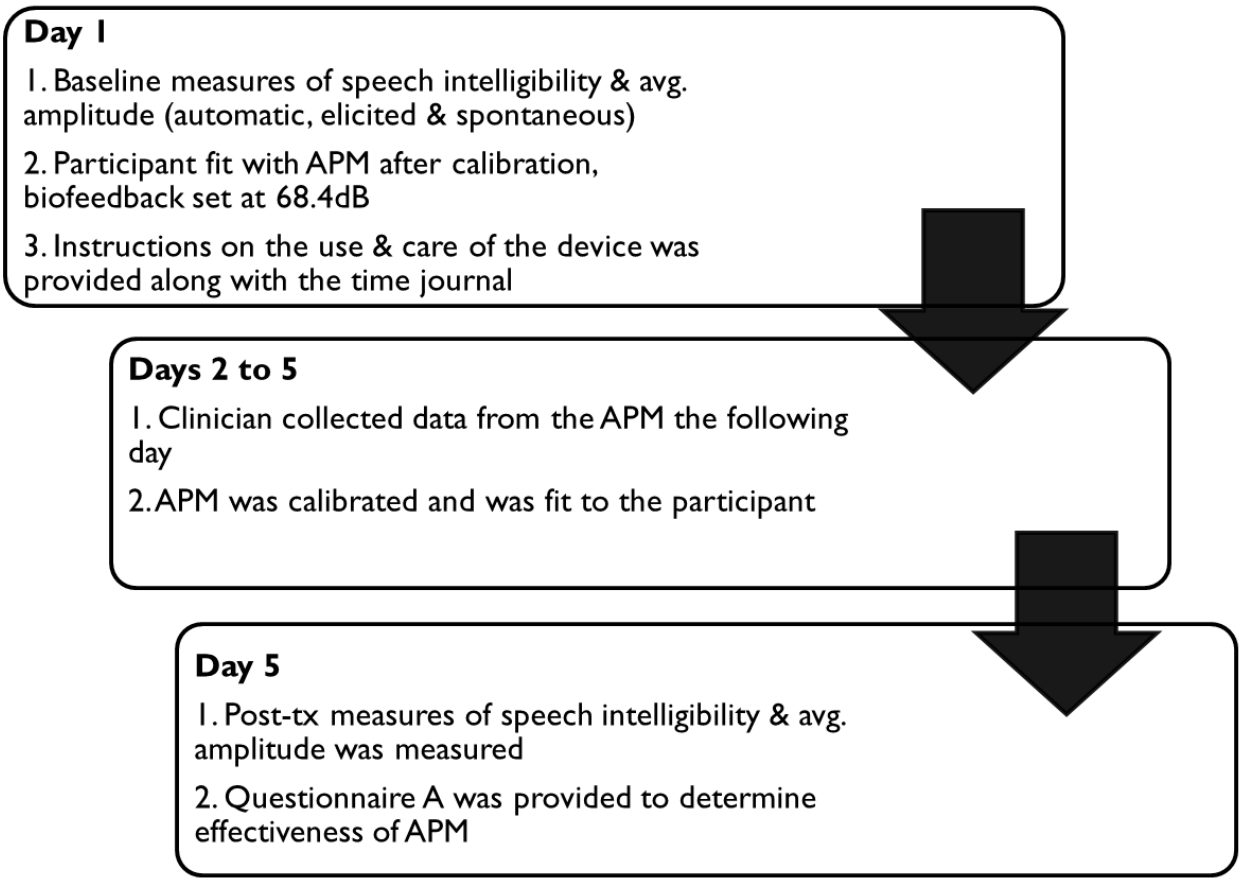

Figure 2. Week 1 – Treatment with APM with Biofeedback

Pretreatment Measures. On day 1, baseline (pretreatment) value of the participant's speech intelligibility and average amplitude (average loudness) was measured and documented using Computerized Speech Lab (CSL) Model 5400 (Kay Elemetrics Corp). The default calibration settings of the CSL were used, and the microphone was kept at a regulated distance of approximately 12 inches (30 cm) from the participant's mouth. Combination of automatic, elicited, and spontaneous speech tasks were chosen to assess the change in intelligibility and average amplitude, and the possible effects of the varying cognitive load in each task on the measured parameters. Intelligibility of speech was calculated for tasks including reading, counting, and conversational speech. The average amplitude logged during 'loud phonation' and

‘comfortable phonation’ in addition to the tasks of reading, counting and conversation was documented.

Protocol. Subsequently, the APM was calibrated, and a biofeedback level of 68.4dB (+5dB of the baseline average amplitude) was set. Tactile-vibratory feedback was provided by the device when the participant’s vocal intensity dropped below the preset level, in turn cueing him to speak louder. The participant was given instructions to wear the device all day long, keep it safely away from water, and simply to disconnect the sensor and the wire connecting the sensor to the APM unit before bed. To remove the throat sensor, the patient was provided with an adhesive remover aid with instructions to lift one edge of the sensor and gently peel away from skin. An alcohol wipe was also provided to remove residual adhesive that may have been left on the skin. The participant was instructed to place the sensor in the pouch provided along with the APM, which the clinician collected the following day at the participant’s residence. On each day the clinician retrieved the previous day’s data from the APM, calibrated and fitted the participant with the APM, and the biofeedback device (68.4dB) prior to the start of the day. During the days 1 through 5, when the participant conducted his usual daily activities, the APM collected data, analyzed it, and provided real time feedback when the voiced input was below threshold level, via a small belt-worn vibrator. Further, the participant was provided with a time journal (see appendix D) to document the estimated amount of talking time in minutes for every 2 hours in a typical 8-hour day, in addition to phonation time data from the APM, which is an index of total speaking time.

Posttreatment Measures. A posttreatment measure of intelligibility of speech and average amplitude was obtained without APM at the end of Day 5 by following similar protocol

implemented in obtaining pretreatment measures. The participant was then provided with questionnaire A (see appendix A) to gather data about the effectiveness and comfort of using APM with biofeedback.

Week 2. No treatment was administered.

Week 3 – AM. Figure 3 displays the steps involved in week 3 of the experimental protocol.

Pretreatment Measures. On day 1, baseline (pretreatment) value of the participant's speech intelligibility and average amplitude (average loudness) was measured and documented using the same protocol implemented during week 1.

Protocol. Based on the participant's white noise thresholds of 0dB and -5dB for right and left ear, the behind-the-ear (BTE) auditory maskers (AM) were programmed to produce noise at 40dBSL, specifically, 40dBSPL and 35dBSPL for right and left ear respectively. The participant was fitted with the maskers and was instructed to use them at home during functional communication on days 1 through 5. The clinician visited the participant at his residence every morning to document for participant-clinician contact period similar to the APM week. The main purpose of this was to ensure no clinician bias between the treatment weeks. During the visit the participant was fitted with the auditory maskers and was provided a time journal (see Appendix E) to document the estimated amount of talking time every 2 hours for a typical 8-hour day.

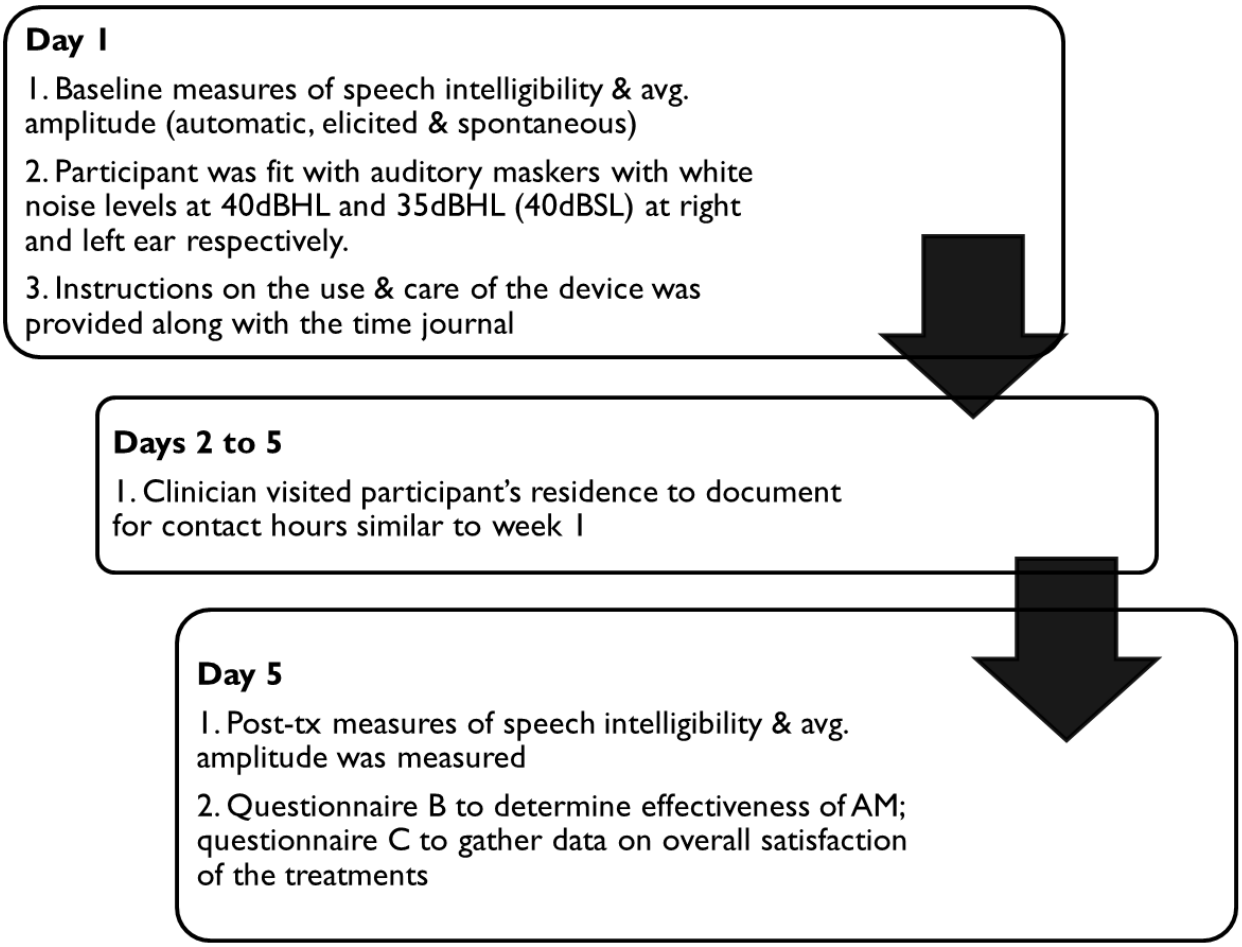

Figure 3. Week 3 – Treatment with AM

Posttreatment Measures. A posttreatment measure of intelligibility of speech and average amplitude was obtained without AM at the end of Day 5 by following similar protocol implemented in obtaining pretreatment measures. Following this, the participant was provided with two short questionnaires - questionnaire B (see Appendix B), to gather data about the effectiveness and comfort of using AM; and questionnaire C (see Appendix C) to gather data about overall satisfaction of the treatments undergone.

CHAPTER 4

RESULTS

Data Analysis

The purpose of this study was to determine changes in speech intelligibility and average amplitude in an individual with PD under two kinds of feedback devices, (a) APM that relies on tactile feedback, and (b) a white noise auditory masker (AM) that inhibits auditory feedback. Additionally, the study helped to investigate the immediate effects of each of the devices, possible use of the devices in everyday life, and patient satisfaction on the use of the devices. Data analyses primarily consisted of visual inspection and comparison of descriptive data and paired t-test looking for systemic and meaningful significance or changes related to speech intelligibility and average amplitude between the two treatment devices. It has to be noted that the participant missed wearing his palatal lift prosthesis during posttreatment AM measures, in exception to wearing the prosthesis for the other measurement intervals.

Speech Intelligibility

The PI rated intelligibility for all tasks. In addition, inter-rater reliability for intelligibility was established through the use of a graduate Speech-Language Pathology student listening to speech samples of different tasks in a quiet room. Intelligibility was determined by counting the number of clearly articulated words in various speech tasks including reading, conversation, and counting. Credit was given for singular or plural listening errors (e.g., if the subject said ‘‘coat’’ and the listener wrote ‘‘coats,’’ it was scored as correct) but for no other morphologically related words (e.g., swift for swiftly). Percentage intelligibility was calculated using the formula below:

$$\frac{\text{\# of intelligible words} \times 100}{\text{Total \# of words}}$$

Inter-rater reliability for intelligibility scores was calculated using Pearson product-moment correlation coefficient, a measure of the strength of a linear association between the ratings of the two listeners. Intelligibility scores demonstrated good inter-rater agreement, with *r*-=0.998 for pre- and post APM, and *r*=0.999 for pre- and post-AM. Because the Pearson product-moment correlation coefficients were high for both the devices, an average of the values for each individual task was determined to compare the differences pre- and posttreatment. Table 1 presents data for speech intelligibility pre- and post-APM and AM.

Table 1

Average Speech Intelligibility Scores – Pre- and Posttreatments

Task	Pre-APM	Post-APM	Pre-AM	Post-AM
Reading	97.04%	79.25%	92.84%	87.81%
Counting	95%	90%	95%	87.5%
Conversation	60.35%	61.85%	45.1%	45.71%

No positive changes in speech intelligibility were observed after using both the devices across three different speech tasks. On the contrary, a decline in speech intelligibility scores for the tasks of reading and counting after the use of APM and AM was noticed. A dependent t-test, also called as the paired t-test, was used to compare the means of the two different groups of pre- and post-APM; and pre- and post-AM. No significant difference between the means were observed ($P>0.05$).

Average Amplitude

Average amplitude for each task in decibels (dB) was obtained by the Kay CSL 5400 and results are shown in Table 2.

Table 2

Average Amplitude (dB) – Pre- and Posttreatments

Task	Pre-APM	Post-APM	Pre-AM	Post-AM
Reading	57.54	54.33	53.26	54.88
Counting	47.08	55.67	45.48	53.00
Conversation	49.70	40.90	46.48	49.26
Phonation-comfortable	59.95	55.94	61.87	62.50
Phonation-loud	78.28	81.63	82.35	83.47

Data from the table indicates an increase of 8.59dB and 7.52dB in average amplitude for the task of counting, post- APM and AM respectively. There is also an increase of 2.78dB during conversation post- AM. Additionally, an unpredicted decrease in average amplitude for the tasks of conversation and phonation-comfortable can be noted post- APM. Despite the positive and negative changes noticed postusage of the devices, paired t-test displayed no significant difference between means at $P>0.05$ across all tasks for APM and AM.

Data on intelligibility and average amplitude during the use of devices in a natural setting would have helped determine the immediate effects of the devices on the participant's speech.

Table 3 displays the retrospective data from APM (week 1) during functional communication at home, on average amplitude, %compliance, and # of times triggered, for days 1 through 5.

Table 3

Retrospective Data from APM during Functional Communication

Parameter	Day 1	Day 2	Day 3	Day 4	Day 5	Average
Average amplitude (dB)	65.11	65.31	68.93	58.32	62.61	64.05
% compliance	44.77%	37.14%	54.17%	12.98%	29.57%	35.72%
No. of times triggered	1138	606	444	377	516	616

It can be observed that there is a notable increase in average amplitude, with simultaneous decrease in the frequency of a trigger, demonstrating the effectiveness of the APM with biofeedback for days 1, 2, and 3. There is a drastic drop in average amplitude by 10.61dB on day 4, during which the participant had experienced a fall. As per the participant's report in his time journal, the accelerometer was detached from his neck around the same time of his fall, which is depicted by the decrease in the percent compliance and number of times triggered. Unfortunately, there is no retrospective data available during functional communication at home for AM to compare and account for the immediate effects of the device on the participant's speech characteristics in a natural setting.

Another purpose of the study was to determine if either of the devices is perceived to be more comfortable for potential long-term use. Questionnaire A and B were designed to rate APM and AM respectively on a scale of 1-5 (1 being extremely satisfied or comfortable and 5 being extremely dissatisfied or uncomfortable) (see Appendixes A and B). Questionnaire C was designed to establish the participant's satisfaction response comparing the two treatments, on a scale of 1-5 (1 being extremely satisfied or convenient and 5 being extremely dissatisfied or inconvenient) (see Appendix C). In questionnaires A and B, the participant rated the effectiveness of APM and AM on his speech to be 1 and 3, and comfort in public and convenience to wear to be 5 and 3 for APM and AM respectively. These ratings display the participant's perception of APM to be the more effective device on his speech, and AM to be the more comfortable and convenient device to wear. In contrast, in Questionnaire C, the participant rated his overall satisfaction with the treatment device to be 4 and 2 for APM and AM, suggesting he found AM to be the most effective and convenient therapeutic device towards the end of the research study.

CHAPTER 5

DISCUSSION

This study was an attempt to determine if there is improvement in speech intelligibility and vocal loudness by using portable therapeutic devices such as APM and AM in an individual with PD, who could not transfer or generalize vocal loudness learned through traditional speech therapy techniques and LSVT®.

Clinical Implications

The findings of this case study demonstrated no improvement in speech intelligibility and vocal loudness after using APM and AM individually for a period of 1 week. On the contrary, nominal drop in scores of intelligibility and average amplitude was observed subsequently after using the devices. Moreover, contrasting results were obtained from the participant satisfaction questionnaires between the effectiveness, convenience, and comfort of use of the two devices. The collective results from questionnaires A and B that rated the devices individually depicted that the participant found APM to be the more effective device on his speech, whereas results from questionnaire C that rated the participant's satisfaction of the treatments, described AM to be the most effective device. This discussion addresses some factors that could potentially have contributed to these unexpected and variable findings, as well as some recommendations for future work. Factors discussed are confounding effects, sample size, and study design.

The participant during the course of the study experienced unanticipated weak vocal health and decline in general health due to a fall during day 4 of week 1, and the participant's performance was determined to be subsiding since the beginning of week 2. This could have directed to the 15% decline in speech intelligibility during conversation from post-APM (week

1) to pre-AM (week 3) measures. However, it has to be noted that there is a 4.29dB increase in average amplitude on day 5 during week 1 (Table 3) indicating the participant's ability to adapt and perform based on the purpose of the APM device. Also during post- AM measures an average increase of 3dB was noticed across all tasks, consistent with the existing literature on positive effects of clinical masking (Adam & Lang, 1992; Countinho et al., 2009; Gryczka et al., 2011; Quedas et al., 2007), although, the participant forgot to wear his palatal lift prosthesis. However, by further spectral analyses it was determined that nasalence contributed to the concentration of energy in the low frequency, resulting in an improvement in average amplitude.

Due to these confounding factors, the true effects of AM on the participant could not be established. Even though this case study is one of the first studies to compare effects of two therapeutic devices on the speech of an individual with PD, the results obtained did not reveal an improvement in speech characteristics from the use of the deivces. Preliminary conclusion from these data should be approached with caution given the single case study, and other medical factors associated with the participant. Future studies involving larger sample size might reduce variability in the results and also aid in generalizing the outcomes for this population.

In this case study it is likely that the tactile and auditory feedback may not have been sufficiently perceptible to elicit a consistent and improved response from the participant. The idiosyncratic nature of PD compels the thought that individuals with PD will require different levels of feedback threshold in order to produce increases in speech intensity and intelligibility (Adam & Lang, 1992). Data on speech intelligibility and average amplitude during conversational speech while wearing the device would assist in presenting evidence on the immediate effects of the therapeutic devices, further determining the adequate level of feedback

and its influence on speech intelligibility and vocal amplitude. Future studies can implement multiple baseline measures with different feedback levels throughout the experimental procedure in order to document the influence of the device on the individual's speech in clinical as well as nonclinical environments. Upcoming studies on therapeutic devices can also analyze the speech samples at different time intervals rather than estimating the average value for vocal intensity and/or speech intelligibility. Such measures of analyzing a speech sample at different time points may enhance and provide information on speech performance, including, vocal amplitude and intelligibility during initiation and termination of speech. Supplementary qualitative data from the spouse or caregiver on the participant's speech, in addition to the questionnaires given to the participants, will help the researcher determine the effects of therapeutic devices from a listener's perspective, in nonclinical environments.

Conclusion

Though the results of this case study have not been favorable, it has opened avenues for further studies with larger sample size, multiple baseline measures, and qualitative data from spouse or caregiver to determine the effects of therapeutic devices in improving speech intelligibility and vocal loudness in individuals with PD. It may also prove useful to extend the testing period for each of the devices by a couple of weeks so that the participant is provided with reasonable amount of time to get adapted to respond to the cue in both clinical and natural environment.

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APPENDICES

Appendix A

Questionnaire – A (Adapted with permission from Boudreaux, 2011)

1. Rate your satisfaction on the effectiveness of treatment on your speech intelligibility. (1 = extremely satisfied; 5 = extremely dissatisfied)

   1 2 3 4 5

2. Rate your satisfaction on the effectiveness of treatment on your vocal loudness. (1 = extremely satisfied; 5 = extremely dissatisfied)

   1 2 3 4 5

3. How was it to use the Ambulatory Phonation Monitor as instructed? (1 = very convenient; 5 = very inconvenient)

   1 2 3 4 5

4. Was the Ambulatory Phonation Monitor a comfortable device to wear? (1 = very comfortable; 5 = not very comfortable)

   1 2 3 4 5

5. How comfortable were you wearing the Ambulatory Phonation Monitor in public? (1 = very comfortable; 5 = not very comfortable)

   1 2 3 4 5

6. Did you feel comfortable speaking while wearing the Ambulatory Phonation Monitor? (1 = very comfortable; 5 = not very comfortable)

   1 2 3 4 5

7. Do you feel that the using the Ambulatory Phonation Monitor affected your speech in any way?

   No

   Yes. How?

8. Did you experience any other difficulties with the Ambulatory Phonation Monitor?

Appendix B

Questionnaire – B (Adapted with permission from Boudreaux, 2011)

1. Rate your satisfaction on the effectiveness of treatment on your speech intelligibility. (1 = extremely satisfied; 5 = extremely dissatisfied)

   1 2 3 4 5

2. Rate your satisfaction on the effectiveness of treatment on your vocal loudness. (1 = extremely satisfied; 5 = extremely dissatisfied)

   1 2 3 4 5

3. How was it to use the auditory masker as instructed? (1 = very convenient; 5 = very inconvenient)

   1 2 3 4 5

4. Was the auditory masker a comfortable device to wear? (1 = very comfortable; 5 = very uncomfortable)

   1 2 3 4 5

5. How comfortable were you wearing the auditory masker in public? (1 = very comfortable; 5 = very uncomfortable)

   1 2 3 4 5

6. Did you feel comfortable speaking while wearing the auditory masker? (1 = very comfortable; 5 = very uncomfortable)

   1 2 3 4 5

7. Do you feel that using the auditory masker affected your speech in any way?

   No

   Yes. How?

8. Did you experience any difficulties with the auditory masker?

Appendix C

Questionnaire - C

Key: Instrument A – Ambulatory Phonation Monitor; Instrument B – Auditory masker

1. To what extent did you like the use of instrument A to improve your speech intelligibility and loudness? (1 = extremely; 5 = not at all)

   1 2 3 4 5

2. To what extent did you like the use of instrument B to improve your speech intelligibility and loudness? (1 = extremely; 5 = not at all)

   1 2 3 4 5

3. How convenient was it to use the instrument A? (1 = very convenient; 5 = very inconvenient)

   1 2 3 4 5

4. How convenient was it to use the instrument B? (1 = very convenient; 5 = very inconvenient)

   1 2 3 4 5

5. Taking all things into account, how satisfied or dissatisfied are you with the treatment with instrument A? (1 = extremely satisfied; 5 = extremely dissatisfied)

   1 2 3 4 5

6. Taking all things into account, how satisfied or dissatisfied are you with the treatment with instrument B? (1 = extremely satisfied; 5 = extremely dissatisfied)

   1 2 3 4 5

Appendix D

Time Journal - Ambulatory Phonation Monitor

(Adapted with permission from Boudreaux, 2011)

Please write down an approximation of how much you spoke in every 2-hour time slot.

Hours 1-2 (9.00-11.00)	
Hours 3-4 (11.00-1.00)	
Hours 5-6 (1.00-3.00)	
Hours 7-8 (3.00-5.00)	

Appendix E

Time Journal – Auditory Masker

Adapted with permission from Boudreaux, 2011

Please write down an approximation of how much you spoke in every 2-hour time slot.

Hours 1-2 (9.00-11.00)	
Hours 3-4 (11.00-1.00)	
Hours 5-6 (1.00-3.00)	
Hours 7-8 (3.00-5.00)	

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