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The Effect of Increased Vocal Intensity on Interarticulator Timing in Speakers with Parkinson's Disease: A Preliminary Analysis

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Abstract

Purpose—The purpose of the current study was to investigate the effect of increased vocal intensity on interarticulator timing in individuals with Parkinson's disease (PD).

Methods—Ten individuals with mild to moderate hypophonia, secondary to PD, were selected for study. Over an 8-week treatment period, multi-talker babble noise was presented monaurally to the individuals with PD during everyday communication contexts to elicit increased vocal intensity (Lombard effect). Outcome measures included sound pressure level (SPL), voice onset time (VOT), VOT ratio, percent voicing, and speech intelligibility.

Results—Group and individual participant responses to the treatment are reported and discussed. Speakers with PD were shown to significantly increase SPL in response to treatment. Six of the ten speakers showed improved temporal coordination between the laryngeal and supralaryngeal mechanisms (interarticulator timing) in response to treatment. Four of the ten speakers, however, showed reduced laryngeal-supralaryngeal timing at the end of treatment. Group speech intelligibility scores were significantly higher post-treatment as compared to pre-treatment.

Conclusions—Voice treatment during everyday communication resulted in improved temporal coordination across the laryngeal and supralaryngeal mechanisms for the majority of speakers with PD and made them easier to understand. Further investigations are planned to explore individual differences in response to treatment. The identification of speaker-specific voicing and devoicing strategies is consistent with the heterogeneous nature of PD.

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Keywords

voice onset time; interarticulator timing; Parkinson's disease; Lombard effect

1.1 INTRODUCTION

Idiopathic Parkinson's disease (PD) is a degenerative neuromuscular disorder caused by a loss of dopamine in a region of the midbrain known as the substantia nigra (Brodal, 1998; Lang & Lozano, 1998). Dopamine is a neurotransmitter that modulates basal ganglia function, and thus plays an essential role in maintaining coordinated and smooth motor activity (Volkow et al., 1998). As a result of changes in the brain, individuals with PD often exhibit a range of deficits across the speech mechanism.

To date, a number of studies of individuals with PD have provided evidence of reduced respiratory support (Huber & Darling, 2011; Huber, Stathopoulos, Ramig, & Lancaster, 2003; Sadagopan & Huber, 2007; Solomon & Hixon, 1993), maladaptive changes to laryngeal structure and function (Canter, 1963; Duffy, 2005; Gallena, Smith, Zeffiro, & Ludlow, 2001; Goberman, Coelho, & Robb, 2005; Hanson, Gerratt, & Ward, 1984; Perez, Ramig, Smith, & Dromey, 1996), changes in vocal quality (Doyle, Raade, St. Pierre, & Desai, 1995; Goberman et al., 2002; Hertrich & Ackermann, 1995), and articulation deficits (Ackermann & Ziegler, 1991; Canter, 1965; Darley, Aronson, & Brown, 1969, 1975; R.D. Kent, J.F. Kent, Duffy, & Weismer, 1998; Logemann & Fisher, 1981). More specifically, the speech and voice impairments reported to date include reduced speaking volume (hypophonia), breathiness, hoarseness, consonant imprecision, and variable speech rates (Darley, Aronson, & Brown, 1969, 1975). Little information is currently available, however, on the relative speech timing patterns in individuals with PD and how these timing patterns may change in response to voice treatment. The focus of the present study, therefore, was to examine changes in the relative timing of laryngeal and supralaryngeal events, in individuals with PD, following completion of a behavioral voice treatment program.

1.1.1 Behavioral treatment for speech and voice disorders in individuals with PD

Current behavioral voice treatment programs aim to remediate speech and voice impairments in individuals with PD by targeting increased respiratory and vocal effort (e.g., the Lee Silverman Voice Treatment LSVT® program). Studies of normal and disordered speech production have shown that the use of a single treatment target (i.e. increased vocal effort) can have global treatment benefits (Dromey & Ramig, 1998a, 1998b; Sapir, Spielman, Ramig, Story, & Fox, 2007; Spielman, Ramig, Story, & Fox, 2000). For example, when individuals with PD are cued to speak louder, improvements extend beyond loud phonation to include improvements in voice quality, prosody, articulation, and speech intelligibility (Baumgartner, Sapir, & Ramig, 2001; Darling & Huber, 2011; Dromey et al., 1995; Ramig, Countryman, O'Brien, Hoehn, & Thompson, 1996). Fox and colleagues (2002) postulated that the system-wide treatment effects observed in individuals with PD may reflect an increase in motor output across the speech mechanism (Fox, Morrison, Ramig, & Sapir, 2002). Although the physiologic mechanism(s) responsible for this multi-system interaction cannot be viewed directly, acoustic and kinematic data have provided

evidence of a functional link between the speech subsystems. For example, perturbation studies have shown that disruption to the movement of a single articulator (i.e. the lips), leads to modification not only in the neighboring articulators, but also to laryngeal events (Folkins, Dromey, & Zimmerman, 1982; van Lieshout, Peters, & Bakker, 1997). Similarly, Geumann (2001) reported that speakers cued to produce loud speech have shown evidence of changes not only in sound pressure level and subglottal pressure, but also in fundamental frequency, first formant frequencies, jaw displacement, and consonant and vowel duration. If a single treatment target has the potential to influence system-wide improvements, therapy can be executed more efficiently and effectively.

1.1.2 Use of the Lombard effect to improve speech and voice characteristics in individuals with PD

To maximize voice treatment outcomes, individuals with PD must be able to accurately and consistently monitor their intensity levels and adjust their vocal amplitude in everyday communicative contexts (Ramig, Countryman, Thompson, & Horii, 1995; Ramig, Fox, & Sapis, 2008; Stemple, Glaze, & Gerdeman-Klaben, 2000). As a result of the sensory, cognitive, and behavioral impairments, however, that are often reported for individuals with PD (Ho et al., 2000; Rosenbeck & LaPointe, 1985; Zgaljardic et al., 2003), some patients may have difficulty monitoring and adjusting their vocal response during daily communication. To circumvent these compliance issues, it has been previously suggested that masking noise be investigated as a potential treatment for hypophonia (Adams & Lang, 1992; A. Dewar, A.D. Dewar, Austin, & Brash, 1979). When speaking in the presence of noise, individuals have been observed to “automatically” speak louder (Lane & Tranel, 1971; Lombard, 1911). Use of a reflexive response, like the Lombard effect, eliminates the need for conscious control of vocal intensity. The Lombard effect has been successfully used to elicit increased vocal intensity levels in both neurologically-healthy and neurologically-involved individuals (Adams & Lang, 1992; Adams, Moon, Dykstra, Abrams, Jenkins, & Jog, 2006; Darling & Huber, 2011; Lane & Tranel, 1971; Sadagopan & Huber, 2007; Stathopoulos et al., 2014). To date, however, these Lombard-elicited changes in vocal intensity have only been captured in a single application of noise exposure (Adams & Lang, 1992; Sadagopan & Huber, 2007; Shrivastav, Skowronski, Kopf, & Rakerd, 2014). The potential treatment benefit of using Lombard-elicited louder speech to condition the underlying musculature and improve speech and voice quality, however, remains unknown. The present study therefore, aimed to investigate Lombard-elicited changes in vocal intensity in individuals with PD over an extended treatment period.

1.1.3 Muscle training and detraining

Consistent with the principles of exercise science and motor learning, the present treatment paradigm aimed to improve underlying physiologic function using high intensity training (Brown, McCartney, & Sale, 1990; Duffy, 2005; Schmidt & Lee, 1999). Physiologic data have shown that improvements in muscle function can be achieved when skeletal muscles are overloaded beyond normal use (Cerny & Burton, 2001; Gonyea, 1980). For example, previously unconditioned muscles can ‘learn’ to recruit all of the motor units in the first few weeks of training, including the fastest motor units which were previously unstimulable (Cerny & Burton, 2001; Marieb, 1999). Neural recruitment, however, has been shown to

peak three to four weeks into muscle training, after which time muscle mass begins to increase, predominating as the major contributor to strength gains (Cerny & Burton, 2001; Marieb, 1999). In the present study, the muscle overload condition was targeted using a high effort speaking task (speaking-in-noise) over an extended 8-week treatment period. The current treatment model was similarly founded upon the principle of training specificity, which states that training should be specific to the movement pattern(s) being targeted in treatment (Cerny & Burton, 2001; Gonyea & Sale, 1982). The principle of specificity was met by using a natural speaking context to foster changes in muscle behavior. It is important to note, however, that once treatment (e.g. the conditioning stimulus) is removed, treatment gains begin to diminish rapidly as the skeletal muscle fibers return to their pre-conditioned state (Bruton, 2002; Cerny & Burton, 2001). For example, muscle mass atrophy has been shown to begin as early as several days after the conditioning stimulus is removed, with the greatest loss occurring in the first three weeks of detraining (Cerny & Burton, 2001). Thus it is important to find treatments that can be carried out by patients long-term, between periods of behavioral therapy. An important aim of the present study was to examine the effect of a muscle conditioning treatment program on vocal intensity adjustments in persons with Parkinson's disease. The effect of increased vocal intensity on the temporal coordination of laryngeal and supralaryngeal gestures (interarticulator timing) and speech intelligibility was then studied. A review of interarticulator timing and the temporal coordination difficulties documented in persons with PD are presented in the sections to follow.

1.1.4 Interarticulator timing

The relative timing of laryngeal and supralaryngeal events is commonly referred to as interarticulator timing. Interarticulator timing plays a robust role in signaling the voiced-voiceless distinction for word-initial English stop consonants (Lisker & Abramson, 1964). A temporal disruption in laryngeal-supralaryngeal coordination is thought to result in less discrete acoustic contrasts, which may have implications for speech intelligibility and the quality and effectiveness of communication (Dromey, Ramig, & Johnson, 1995).

There are two commonly reported acoustic measures of interarticulator timing: voice onset time (VOT) and percent voicing. Voice onset time is defined as the time interval between the release of an oral constriction and the onset of voicing for the following vowel (Kent & Read, 2002; Lisker & Abramson, 1964). In the English language, pre- and inter-vocalic voiced and voiceless stop consonants are signaled, in part, by VOT length. For example, the voiced stop consonants /b d g/ have relatively short VOT values, ranging from approximately -20 to +20 msec, whereas their voiceless cognates /p t k/ have relatively long VOTs, ranging from approximately +40 to +100 msec (Lisker & Abramson, 1964). In production of voiceless aspirated stop consonants, the brief temporal delay (+40 to +100 msec) between the release of the oral constriction and the onset of voicing for the following vowel is mediated, in part, by a maximally abducted vocal fold position (peak glottal opening) at the time of the oral release (Lisker & Abramson, 1964). Maintaining the voiced-voiceless contrast for English stop consonants requires intricate and precise temporal coordination across the speech mechanism (Lofqvist & Yoshioka, 1981).

The presence or absence of voicing during a stop closure interval also plays an important role in signaling the voicing contrast for English stop consonants. For example, the absence of voicing in a stop closure interval signals production of a voiceless stop consonant such as [p] [t] or [k]. In contrast, if voicing is present in the stop closure interval it signals production of a voiced cognate [b] [d] or [g]. Percent voicing is an acoustic measure used to reflect the proportion of a stop closure interval that is voiced (Duffy, 2005; Weismer, 1984).

During production of voiced-voiceless sound sequences, a speaker must coordinate laryngeal and the supralaryngeal events in a timely manner in order to terminate voicing from the preceding sound. At the laryngeal level, the vocal folds abduct through the mechanical action of the intrinsic laryngeal muscles (known as the laryngeal devoicing gesture). At the supralaryngeal level, an oral constriction simultaneously forms to obstruct airflow. The synchronous timing between the formation of the oral constriction (supralaryngeal event) and the onset of vocal fold abduction (laryngeal event) facilitates the equalization of pressure above and below the larynx. The laryngeal devoicing gesture and the equalization of pressure serve to arrest vocal fold vibration. In studies of non-neurologically impaired speakers, voicing has been shown to terminate several milliseconds (one or two glottal pulses) after the oral constriction has formed for a voiceless consonant (Hixon, Weismer, & Hoit, 2008). If voicing continues to be present during a normally-silent voiceless closure interval, however, it may degrade the voicing contrast (Lisker, 1986). While percent voicing has been used to study dialectal variations in stop consonant production (Jessen, 2002; Lyle, 2008), it should be a useful addition to the investigation of voicing termination in neurologically-involved populations such as PD.

1.1.5 Voice onset time in individuals with PD

A small body of VOT research on individuals with PD has yielded mixed results. Weismer (1984) examined the voiceless VOT interval in individuals with PD, healthy older adults, and healthy young adults during a structured speaking task. Results of the study revealed shorter VOTs for neurologically-impaired speakers, as compared to healthy older and younger speakers. Weismer's (1984) data is consistent with the more recent work of Flint and colleagues (1992). In the analysis of word-initial voiceless stops, individuals with PD were found to exhibit significantly shorter VOTs compared to neurologically-healthy controls (Flint, Black, Campbell-Taylor, Gailey, & Levinton, 1992). Weismer (1984) offered a tenable explanation for the observed VOT decrease in speakers with PD. He postulated that increased stiffness of the laryngeal musculature leads to a more adducted vocal fold position during a normally open glottal configuration (Weismer, 1984). As a result, the vocal folds are able to achieve a fully adducted position more quickly at the onset of voicing, which is reflected acoustically by a shorter VOT (Weismer, 1984). Weismer's (1984) findings, however, were in contrast with a follow-up study by Forrest, Weismer, and Turner (1989) who reported longer VOTs in production of word-initial voiced bilabial stops for individuals with PD, compared to age-matched controls. Interestingly, no significant VOT differences were found between individuals with PD and controls for the voiceless cognates (Forrest et al., 1989). Forrest and colleagues (1989) attributed the longer VOTs for the voiced bilabial stops to a deficit in motor initiation and coordination at the level of the larynx. In summary, although the VOT literature suggests that some individuals with PD

exhibit temporal deficits with phonatory onset behavior (voicing initiation), the findings are mixed as to whether the VOT interval is longer or shorter than non-neurologically involved speakers.

Incongruous data on VOT intervals may be partly attributable to inter-speaker variability in speech rate. VOT studies involving neurogenic populations, such as PD, have traditionally not accounted for speech rate differences. The recent work of Fischer and Goberman (2010) examined utterance-initial VOT in individuals with PD and age- and sex-matched controls. A conventional measure of VOT was applied, as well as a measure of VOT ratio which examines changes in VOT with the effect of rate removed. Results of their study indicated that VOT and VOT ratio were in agreement with the following two exceptions. First, removing the effect of speech rate was found to be an important manipulation when comparing open and closed vowels (Fischer & Goberman, 2010). Second, a greater medication effect was found for the conventional measure of VOT, compared to VOT ratio (Fischer & Goberman, 2010). The authors concluded that changes in VOT during the “on” versus “off” medication state were likely reflective of rate-related changes rather than true-VOT changes. Overall, there is sufficient evidence to support the use of examining both VOT and VOT ratio in neurogenic populations, such as PD, where rate of speech has been shown to be highly variable.

1.1.6 Voicing during the voiceless stop closure in individuals with PD

Similar to VOT research, limited data is currently available on phonatory offset behavior (voicing termination) in neurogenic populations such as PD. Weismer (1984) qualitatively examined the termination of voicing during production of voiced-voiceless sound sequences in individuals with PD and their age-matched controls. A dichotomous decision-making task was used to reflect the presence or absence of voicing during the voiceless stop closure interval. Results of the study indicated a higher occurrence of voicing during the voiceless closure interval for speakers with PD, compared to controls (Weismer, 1984). It was further reported that the speakers with PD tended to exhibit continuous phonation throughout the voiceless closure interval (Weismer, 1984). However, the author attributed this result to the speech patterns of two individuals with PD who voiced approximately 45% of the voiceless closure interval.

More recently, Goberman and Blomgren (2008) examined phonatory offset behavior in individuals with PD and age- and sex-matched controls. Participants were instructed to read the first paragraph of the Rainbow Passage in their habitual manner. Fundamental frequency was measured across the last ten cycles of a periodic waveform as the speech mechanism transitioned from production of a voiced nasal [n] to a voiceless fricative [f] in the production of “one finds” (Goberman & Blomgren, 2008). Both speaker groups (PD and control) significantly decreased their mean fundamental frequency at the end of the voiced segment, similar to findings from older adults in Watson (1988). The authors concluded that speakers in the PD group, as well as their matched controls, likely terminated voicing through mechanical action of the vocal folds (vocal fold abduction) without any significant increase in laryngeal muscle tension (Goberman & Blomgren, 2008). The decrease in fundamental frequency observed for the speakers with PD and their matched controls

suggests age-related changes in devoicing rather than disease-related changes. In contrast, Watson (1988) demonstrated that young adults terminate voicing through increased vocal fold tension and vocal fold abduction, as supported by his finding of a relatively stable (or slightly increased) fundamental frequency during the phonatory offset gesture. Despite growing empirical evidence that individuals with PD have difficulty with the termination of voicing, there is a need to further quantify changes in phonatory offset behavior.

1.1.7 The present study

The present study offers a preliminary examination of the effect of increased vocal intensity on interarticulator timing in a group of individuals with PD before and after treatment involving the Lombard effect, as well as four-weeks post-treatment. The Lombard effect, an automatic or unconscious response to auditory stimuli, was used to naturally elicit increases in vocal intensity during daily living activities. The effect of treatment and detraining on vocal intensity was assessed using sound pressure level (dB SPL) data. SPL data were collected without the Lombard effect present. The effect of treatment and detraining on interarticulator timing was indexed using acoustic measures of voice onset time (VOT), VOT ratio, and percent voicing. The following four hypotheses were investigated:

1. Speakers would show increased vocal intensity after 8-weeks of treatment involving speaking-in-noise (Lombard effect);
2. As a result of increased vocal intensity, speakers would show improved interarticulator timing during a structured speaking task. Improvement would be reflected in acoustic measures of VOT, VOT ratio, and percent voicing that approached target values previously reported for healthy English-speaking adults;
3. Speakers would not maintain SPL and interarticulator timing gains 4-weeks after speech-in-noise treatment was removed due to the effects of detraining; and
4. Speakers would show improved speech intelligibility at the end of treatment.

2.1 MATERIALS AND METHODS

2.1.1 Participants

Data collection procedures were approved by committees on the Use of Human Research Participants at Purdue University and at the University at Buffalo. Written informed consent was obtained from all participants. Data was collected from ten adults (8 men, 2 women) diagnosed with idiopathic Parkinson's disease (PD). Participants ranged in age from 66 to 86 years, with a mean age of 74.3 years ($SD \pm 6.4$ years). The speaker codes M01, M02, M04, M06, M07, M13, M16, and M18 refer to male speakers with PD, and the speaker codes F02 and F03 refer to female speakers with PD. Pre-treatment respiratory and laryngeal aerodynamic data are presented for these speakers in previously published work (Stathopoulos et al., 2014).

Criteria for inclusion were: 1) a diagnosis of idiopathic PD by a neurologist; 2) presence of hypophonia as determined by a speech-language pathologist; 3) speaker of Standard American English; 4) no history of other neurological diseases other than PD; and 5) unaided hearing in at least one ear.

Eight of the ten participants were taking medication to alleviate their PD-related symptoms and were not receiving other forms of behavioral, surgical, or prosthetic treatment for speech or voice at the time of participation. All participants who were taking medication were tested during the “on” state of their medication cycle. One participant (F03) was receiving electrical stimulation to the subthalamic nucleus for management of levodopa-induced dyskinesias. To mitigate any potential changes in speech due to stimulator condition, participant F03 was only recorded under the stimulator “on” condition.

Judgments of hypophonia were made during conversational speech by a certified speech-language pathologist who did not participate in any other aspects of the study. Hypophonia was defined as low intensity speech and/or caregiver complaints of difficulty hearing the speech from the individual with PD. Auditory-perceptual judgments of speech and voice characteristics were also noted by the speech-language pathologist during diadochokinetic rate tasks, sustained vowel productions, and connected speech. Finally, to index global speech and voice severity, speech and voice characteristics were assessed during connected speech using a 150mm visual analog scale adapted from Cannito and colleagues (1997). The speech and voice severity ratings, reflected in Table 1, were based upon consideration of consonant precision, intonation, voice quality, and speaking rate, without regard to speech intelligibility. The speech and voice severity ratings range from 0%–100%, indicating normal to severely impaired speech and/or voice characteristics, respectively. An audiologist, not involved in other aspects of the study, performed audiological evaluations to document hearing thresholds.

Table 1 includes a description of participant characteristics including age on date of testing, time since diagnosis, Hoehn and Yahr stage (Hoehn & Yahr, 1967), average pure tone thresholds for the better ear, PD-related medications, hypophonia severity rating, auditory-perceptual speech and voice characteristics, and speech and voice severity ratings.

2.2 EQUIPMENT

2.2.1 Acoustic recordings

The speech signal was transduced using a head-mounted omnidirectional condenser microphone (Countryman E6 Model #E610P5L2 or Shure Beta 53 Model #WBH53B). For each participant, the same microphone was used for all recordings. The speech signal was digitally recorded at a frequency of 44.1 kHz using a digital audio recorder (Marantz PMD-670). The speech signal was then downsampled and filtered using GoldWave (Version 5.25, 2008) at a sampling rate of 22 kHz with a low-pass filter of 9 kHz applied for anti-aliasing. The microphone was calibrated for SPL on the day of testing to a known 1 kHz calibration tone, generated at a known decibel level (either 94 or 110 dB) (Quest Piston Phone, Model CA22). Gain was provided to the microphone signal by the digital audio recorder and gains were factored into the microphone calibration.

2.2.2 Device for eliciting the Lombard effect

Device description—A specially engineered device (SpeechVive™) was used to present multi-talker babble noise to each participant. The 20-talker multi-talker babble noise (Auditec of St. Louis) sounded like unintelligible background talking. The voice-activated

SpeechVive™ delivered the multi-talker babble noise to one ear when the individual wearing the device was speaking.

Device fitting—The SpeechVive™ detected voicing through an accelerometer affixed superficially to the participant's thyroid lamina or suprasternal notch. When the signal from the accelerometer reached a preset level, the SpeechVive™ played the multi-talker babble noise. When the individual stopped speaking, the multi-talker babble noise turned off. The accelerometer detection level and the amplitude of the noise were both adjusted by the examiner (described in *Device setting*). The multi-talker babble noise was presented through a small hearing aid type speaker and was delivered to one ear of the participant through thin tubing which terminated with an open ear fitting (Phonak Fit'n'Go). The SpeechVive™ was fit to the ear with better hearing sensitivity based on the results of pure tone air conduction thresholds (see Table 1).

Device setting—For the initial SpeechVive™ fitting, the experimenter determined each participant's average comfortable SPL across several minutes of speech-in-quiet (without the SpeechVive™ in place). SPL values were obtained from a digital SPL meter placed 12 inches from the participant (Quest sound level meter Model 155 set at C-weighting and slow response). The SpeechVive™ was then placed on the participant. The experimenter adjusted the detection level of the SpeechVive™ until it activated and deactivated at the onset and offset of the participant's speech. The amplitude of the multi-talker babble noise was then increased during conversational speech until the participant spoke approximately 3 dB above his/her speech-in-quiet SPL. In this way, the gain level of the SpeechVive™ was adjusted to each person's own hearing level. The amplitude of the multi-talker babble noise was set on the first day of treatment and then further increased biweekly by the examiner using the same procedures. The biweekly noise adjustment was performed in order to elicit higher levels of vocal intensity over the course of treatment as the participant's speech-in-quiet SPL increased.

2.3 SPEECH TASKS AND PROCEDURES

2.3.1 Treatment paradigm

Each participant served as his or her own control in the within-subject, baseline-treatment-baseline design employed (Kirk, 1968). To obtain a stable baseline measure for each participant, speech data from four of the eight baseline sessions were collapsed by stimulus word (Pre_{Avg}). Immediately following the baseline period, treatment was introduced. Over the 8-week course of treatment, participants were instructed to wear the SpeechVive™ two to eight hours per day during conversation and during 30 minutes of oral reading five days per week. The SpeechVive™ digitally recorded the date and time the multi-talker babble noise was played, thereby allowing for an estimate of device use for each participant. To control for the potential carry-over of Lombard-elicited changes in SPL, participants were instructed not to wear the SpeechVive™ on days of testing until after their appointment with the research team. On the last day of treatment (PT_{WK1}), the SpeechVive™ was removed from the participants' custody. Speech data were collected 4-weeks after treatment termination to examine the effects of detraining (PT_{WK4}).

2.3.2 Speech tasks

Speech data were collected in two environments; in a quiet room in the participant's home, or in an institutional setting (University at Buffalo, Purdue University, or a satellite location at the Rehabilitation Hospital of Indiana in Indianapolis, IN). The same models of recording equipment were used across all collection sites. All stimuli were displayed on a computer monitor situated in front of the participant. The stimuli consisted of stimulus words differing in word-initial voiceless stop consonant. The consonants varied in place of articulation (/p, t, k/) and were presented in fixed vowel height contexts /_i, _u/. The vowel context was constrained to include only high vowels to limit the confounding influence of vowel height on VOT values (Klatt, 1975). The stimulus words were randomly presented within the carrier phrase "It's a ___ again." Three repetitions of each sentence were produced, for a total of 18 sentence productions per session. In total, 1080 sentences were recorded across participants [10 participants $\times$ 18 sentences $\times$ 6 recording sessions]. The six recording sessions included four baseline sessions, one immediate post-treatment session, and one detraining session. During speech recordings, the head-mounted microphone was positioned 6 cm from the lips at a 45-degree azimuth. Participants were instructed to read each sentence at a comfortable loudness and pitch. No feedback was provided by the examiner. Data was collected while the participants were not wearing the SpeechViveTM.

2.3.3 Experimental conditions

Treatment—To examine treatment-related changes for SPL, VOT, VOT ratio, percent voicing, and speech intelligibility, baseline speech data (Pre_{Avg}) was compared to the end of treatment speech data (PT_{WK1}).

Detraining—To examine detraining-related changes for SPL, VOT, VOT ratio, and percent voicing, end of treatment speech data (PT_{WK1}) was compared to speech data collected 4-weeks post-treatment (PT_{WK4}).

2.4 MEASUREMENTS

2.4.1 Acoustic data

The speech waveform was displayed and measured spectrographically in TF32 (Milenkovic, 2005) using a wide-band filter of 300 Hz and an upper frequency limit of 11 kHz. Measurements were made directly from the wide-band spectrogram coupled with the acoustic waveform.

1. for SPL (dB), root-mean-square voltage was measured across target utterances or speech runs and converted to dB SPL. A speech run was operationally defined as a segment of speech bound by a silent pause of 200 milliseconds or greater (Tjaden & Wilding, 2004; Turner & Weismer, 1993). In total, 17% of the sentences produced (187/1080) were divided into multiple speech runs. A correction constant was applied relative to the calibration and the gain level used at the time of the recording.
2. VOT (msec) was measured as the time interval between the release of the voiceless consonant constriction and the onset of voicing for the following vowel (Dromey &

Ramig, 1998a; Lisker & Abramson, 1964). The release of the oral constriction was acoustically marked by the presence of a stop burst release (brief period of high intensity transient noise). If multiple stop burst releases were present, the initial burst was used for VOT measurement (Wang, Kent, Duffy, Thomas, & Weismer, 2004). The onset of voicing for the following vowel was acoustically marked by the first vertical striation or glottal pulse of voicing (Forrest & Weismer, 1997; Wang et al., 2004). VOT could not be measured for 1.4% of the trials (15/1080) due to the absence of a clear stop burst release and/or a weak vocalic nucleus (Wang et al., 2004).

3. VOT ratio was calculated using the formula described by Ravizza (2003):

$$\left(\frac{\text{Voice onset time (msec)}}{\text{Word duration (msec)}} \right) \quad [\text{Eq. 1}]$$

Word duration (msec) was measured as the time interval between the release of the voiceless consonant constriction and the offset of voicing for the following vowel (Ravizza, 2003; Fischer & Goberman, 2010). The release of the oral constriction was acoustically marked by the presence of a stop burst release and the offset of voicing at the end of the vowel was marked by the last full glottal pulse and a decrease in first formant energy.

4. Percent voicing was calculated using the formula described by Frisch and Wright (2002):

$$\left(\frac{\text{Voicing during the closure (msec)}}{\text{Closure duration (msec)}} \right) \times 100 \quad [\text{Eq. 2}]$$

Closure duration (msec) was measured as the time interval between the onset of the voiceless consonant constriction and its subsequent release (Dromey & Ramig, 1998b). The onset of the oral constriction was acoustically marked by a decrease in the root-mean-square amplitude trace and a decrease in first formant energy for the preceding vowel. The release of the oral constriction was marked by the presence of a stop burst release. For voicing during the closure, the time between the onset of the voiceless consonant constriction and the termination of postvocalic voicing was measured (Smith, 1979). The termination of postvocalic voicing was acoustically marked by the presence of the last periodic pulse in the voiceless closure interval.

2.4.2 Speech intelligibility data

To obtain perceptual judgments of speech intelligibility, the sentence portion of the Speech Intelligibility Test (SIT) (Yorkston, Beukelman, Hakel, & Dorsey, 1996) was administered pre- and post-treatment. The SIT sentences were transcribed by two certified speech-language pathologists who were not involved in data collection. Each judge had more than 10 years of clinical experience working with the adult neurogenic population. Each judge passed a bilateral, pure tone hearing screening for the octave frequencies 250 to 8000 Hz presented at 20 dB HL. The recorded speech samples were presented to each judge

binaurally through over-the-ear headphones (Sony MDR-V300). The SIT sentences from the pre-treatment and post-treatment sessions were randomly presented to each judge in a single block. The judges were blind to the participants' identity and the treatment session presented. The speech intelligibility test was administered and scored according to the standard SIT protocol. An average percent-intelligible score was calculated for each participant, pre- and post-treatment, by averaging the percent of words correctly identified across the two judges.

2.5 STATISTICAL ANALYSES

2.5.1 Outlier analysis

An outlier analysis was conducted for each participant and dependent measure using the protocol described by Tukey (1970). The 75th and the 25th quartiles and the H-spread (the difference between the 75th and 25th quartiles) were calculated using JMP 8.0.2 (2009). The upper and lower quartile "fences" were computed to determine the acceptable range of data points. The upper fence was calculated as [75th percentile +1.5(H-spread)] and the lower fence was calculated as [25th percentile -1.5(H-spread)]. Data points located above the upper fence and below the lower fence were identified as outliers and excluded from analysis. As a result of the outlier analysis, 3.2% of the data points (35/1080) were excluded from study. A trial average was computed for each stimulus word produced by a speaker in a given session, using data points that fell within the acceptable fence range.

2.5.2 Reliability

Acoustic data—Acoustic measures of SPL, VOT, VOT ratio, and percent voicing were remeasured by the original examiner (intra-measurer reliability) and an independent examiner (inter-measurer reliability). For intra-measurer and inter-measurer reliability, 10% of the sentences were randomly selected for re-measurement. The independent examiner was a graduate student who did not participate in other aspects of the study and who was trained in spectrographic analysis.

Intra-class correlation (ICC) coefficients were computed using PASW Statistics 18.0 (2009) to index intra-measurer and inter-measurer reliability. For intra-measurer reliability, a mean intra-class correlation coefficient of 0.991 was reported across dependent measures (ICC range= 0.984 to 0.998), indicating strong agreement between the first and the second set of measurements for the original examiner. For inter-measurer reliability, a mean intra-class correlation coefficient of 0.989 was reported across dependent measures (ICC range= 0.950 to 0.997), also indicating strong agreement between the original and the independent examiner.

Speech intelligibility data—A Pearson product-moment correlation coefficient was used to measure the degree of similarity between the judges' assigned speech intelligibility scores. The expert listeners' speech intelligibility scores were found to be strongly correlated (Pearson product-moment correlation coefficient $r=0.92$, $p<0.05$).

2.5.3 Analysis of variance

Acoustic data—For each experimental condition, mean data were analyzed separately for each of the four dependent measures (SPL, VOT, VOT ratio, and percent voicing). All statistical tests were two-tailed with statistical significance set at an alpha level of 0.05. Differences between pre-treatment and immediately post-treatment were analyzed separately from differences between immediately post-treatment and 4-weeks post-treatment. Participant was included as a random effect in all statistical models due to expected inter-subject differences in responses to treatment and detraining.

SPL data were assessed using a one-way repeated measures analysis of variance (ANOVA) (SAS 9.2). The within-group factor was Session (2 levels: pre-treatment vs. immediately post-treatment or immediately post-treatment vs. 4-weeks post-treatment).

Percent voicing, VOT, and VOT ratio data were assessed using a two-way repeated measures ANOVA (SAS 9.2). The two within-group factors were Session (2 levels: pre-treatment vs. immediately post-treatment or immediately post-treatment vs. 4-weeks post-treatment) and Place of articulation (3 levels: bilabial, alveolar, and velar).

Post hoc testing was completed as appropriate for significant ANOVA findings. In consideration of the multiple comparisons being made, Tukey-Kramer's Honestly Significant Difference (HSD) adjusted p-values were used to ascertain statistical significant in all post-hoc comparisons. Cohen's test of effect size (*d*) was completed for all significant findings and is reported in-text in Section 3.1 *Results*. Individual participant data was also qualitatively inspected and are reported in Section 4.1 *Discussion*.

Speech intelligibility data—All percent correct speech intelligibility scores were arcsine transformed to normalize the distribution of the data set (Winer, 1962). A two-tailed, paired samples t-test was then used to assess for a pre to post difference in speech intelligibility scores. A significance level of 0.05 was used for statistical testing.

3.1. Results

3.1.1 Preliminary data analyses

Inspection of conventional VOT data and VOT ratio data indicate identical patterns of statistical significance for each experimental condition: VOT and VOT ratio significantly increased by the end of treatment and significantly decreased after the detraining period (see Table 2). Thus, it can be reasonably inferred that any observed changes in VOT are true VOT-related changes, and not rate-related VOT changes. The conventional VOT data will therefore be presented and discussed since it is a more commonly reported measure of phonatory onset timing (Lisker & Abramson, 1964).

In addition, a Pearson's product-moment correlation was performed (SPSS, 2012) to examine the relationship between device training time and adjustments in vocal intensity. The results of the correlation analysis indicated no significant relation between device training time and post-treatment increases in SPL ($r = -0.006$, $p = 0.987$). As a result,

individual differences in device training time were not a variable of interest in the statistical analyses.

3.1.2 Group data analysis

Group data will be presented by condition (Treatment and Detraining) for each dependent measure: SPL, VOT, and percent voicing. Session effects were examined for SPL to determine the participants' response to a Lombard-elicited voice treatment. Session effects were examined for VOT and percent voicing to explore treatment-related responses in interarticulator timing patterns.

3.1.3 Treatment effects (pre-treatment vs. immediately post-treatment)

Sound pressure level (SPL)—A significant Session effect was observed for SPL. Speakers were found to significantly increase their comfortable vocal intensity at the end of treatment [$F_{(1,9)}=31.27$; $p=0.0003$, $d=0.50$]. A mean SPL increase of +2.9 dB SPL was observed for the group, with a range of +1.6 to +3.33 dB SPL.

Voice onset time (VOT)—A significant Session effect was observed for VOT [$F_{(1,9)}=5.53$, $p=0.0433$, $d=0.20$]. Significantly longer VOTs, exemplifying the voiceless characteristic of the target consonants, were observed across speakers at the end of treatment ($M=78.68$ msec, $SD=30.84$ msec), compared to the onset of treatment ($M=71.16$ msec, $SD=24.90$ msec). A main effect of Place of articulation [$F_{(2,18)}=133.09$, $p<0.0001$, $d=0.50$] was also observed. The speakers showed VOTs appropriate to place of articulation (Lisker and Abramson, 1964). Post hoc testing for Place of articulation revealed significantly shorter VOTs for word-initial [p] ($M=60.68$ msec, $SD=22.33$ msec), compared to both [t] ($M=77.88$ msec, $SD=26.13$ msec) [$t_{(18)}=15.10$; Adj $p<0.0001$, $d=0.91$] and [k] ($M=84.01$ msec, $SD=31.22$ msec) [$t_{(18)}=24.17$; Adj $p=0.0000$, $d=1.12$]. In addition, VOT was found to be significantly shorter for word-initial [t] compared to [k] ($t_{(18)}=0.31$, Adj $p=0.0430$, $d=0.28$). There was no significant Session by Place of articulation interaction effect [$F_{(2,18)}=0.51$, $p=0.6103$].

Percent voicing—A significant Session effect was observed for percent voicing [$F_{(1,9)}=6.11$, $p=0.0355$, $d=0.26$]. Percent voicing was found to be significantly higher at the end of treatment ($M=22.70\%$, $SD=11.97\%$), compared to the onset of treatment ($M=18.70\%$, $SD=6.63\%$). There was no significant main effect of Place of articulation [$F_{(2,18)}=2.71$, $p=0.0646$] and no significant Session by Place of articulation interaction effect [$F_{(2,18)}=0.66$, $p=0.5297$].

3.1.4 Detraining effects (immediately post-treatment vs. 4-weeks post-treatment)

Sound pressure level (SPL)—A significant Session effect was observed for SPL. Vocal intensity was significantly lower 4-weeks after treatment was removed as compared to immediately post-treatment [$F_{(1,9)}=7.75$; $p=0.0213$, $d=0.40$]. A mean SPL decrease of -2.53 dB SPL was observed for the group, with a range of -1.2 to -3.13 dB SPL.

Voice onset time (VOT)—A significant Session effect was observed for VOT [$F_{(1,9)}=7.15$, $p=0.0255$, $d=0.20$]. Significantly shorter VOTs were observed 4-weeks after

treatment was removed ($M=72.52$ msec, $SD=28.22$ msec), as compared to immediately after treatment ($M=78.68$ msec, $SD=30.84$ msec). A main effect of Place of articulation [$F_{(2,18)}=72.2$, $p<0.0001$, $d=0.50$] was found that parallels the results previously reported for the Treatment condition. A non-significant Session by Place of articulation interaction effect was observed [$F_{(2,18)}=0.84$, $p=0.4463$].

Percent voicing—A significant Session effect was found for percent voicing [$F_{(1,9)}=4.16$, $p=0.0417$, $d=0.26$]. Percent voicing was significantly lower 4-weeks after treatment was removed ($M=21.5\%$, $SD=5.93\%$), as compared to immediately after treatment ($M=18.70\%$, $SD=6.63\%$). The Place of articulation ($F_{(2,18)}=1.62$, $p=0.3244$) and the Session by Place of articulation interaction ($F_{(2,18)}=0.37$, $p=0.6942$) effects were not significant.

3.1.5 Speech intelligibility data

Results of the paired samples t-test indicated a significant difference in mean speech intelligibility scores pre- to post-treatment [$t = -1.96$, $p=0.04$]. Mean speech intelligibility scores were found to be significantly higher across speakers at the end of treatment (98% speech intelligibility score), compared to the onset of treatment (93% speech intelligibility score).

4.1 DISCUSSION

The purpose of the current study was twofold: to examine the effect of Lombard-elicited changes in vocal intensity and to examine the effect of increased vocal intensity on interarticulator timing patterns in individuals with hypophonia secondary to PD. Increased vocal intensity and improved interarticulator timing were successfully elicited for the majority of speakers with PD. A detraining effect was observed for vocal intensity and interarticulator timing, further substantiating the presence of a treatment effect.

4.1.1 Lombard-elicited changes in vocal intensity

The significant finding of increased SPL (Figure 1) supports the first hypothesis and shows that use of the Lombard effect to increase vocal intensity is an effective treatment for hypophonia for patients with PD. The present Lombard-elicited increases in SPL are consistent with vocal intensity increases reported for other voice treatment programs (e.g. LSVT®) (Ramig & Sapir, 2001; Ramig, Pawlas, & Countryman, 1995; Sapir et al., 2007).

The effect of successful training on SPL was further supported by a loss of treatment gains (detraining) after the conditioning stimulus of multi-talker babble noise was removed. As illustrated in Figure 1, all participants were shown move back toward pre-treatment SPL levels four weeks after treatment ended. The observed loss in SPL treatment gains is consistent with physiologic reports of muscle deconditioning once a conditioning stimulus is removed (Bruton, 2002; Cerny & Burton, 2001). Although the loss of SPL gains may appear inconsistent with maintenance data reported for other voice treatment programs (e.g. LSVT®) (Ramig & Sapir, 2001), this discrepancy can be attributed to differences in methodology. For example, following completion of LSVT's® intensive voice treatment program, patients are encouraged to continue applying the treatment strategy “Think Loud” in everyday communicative contexts and to continue to complete vocal training exercises

daily. In the present study, however, speakers did not continue to participate in any form of speech and/or voice remediation once treatment was removed.

The present post-treatment increase in SPL suggests underlying improvement in motor function and/or coordination across one or more of the speech subsystems. It is well accepted that adjustments in vocal intensity are mediated through the synergistic actions of the respiratory, laryngeal, and supralaryngeal subsystems (Issiki, 1964). The clinical importance of the interaction of the three speech subsystems in supporting increased vocal intensity has been realized in previous studies of individuals with PD (Dromey et al., 1995; Huber, Stathopoulos, Curione, Ash, & Johnson, 1999; Smith et al., 1995; Tom, Titze, Hoffman, & Story, 2000).

4.1.2 Interarticulator timing patterns in response to voice treatment for speakers with PD

Given that all speakers with PD significantly increased vocal intensity in response to treatment, the researchers sought to examine treatment-related improvements in interarticulator timing. At first glance, the group data appears to reflect a trading relationship between VOT and voicing into the silent closure gap to enhance the voicing distinction as both group averages increased in duration. Inspection of the individual participant data, however, suggests that this is not the case. Given the heterogeneous nature of PD and the inter-speaker variability observed for the present VOT and percent voicing data, a detailed discussion of post-treatment changes in laryngeal and supralaryngeal timing will be presented with reference to individual participant results (below).

The second hypothesis was partially supported by the present VOT and percent voicing data. Inspection of individual VOT and percent voicing data revealed two distinct treatment responses. Six of the ten speakers with PD (M01, M04, M06, M18, F02, and F03) demonstrated improved temporal control for voicing initiation and termination when speaking at higher SPLs post-treatment (referred to as the *improved voicing group*). The six speakers in the improved voicing group demonstrated larger post-treatment SPL increases, on average 3.2 decibels. In contrast, four of the ten speakers with PD (M02, M07, M13, and M16) demonstrated a lack of post-treatment improvement in interarticulator timing (referred to as the *unimproved voicing group*) and demonstrated smaller post-treatment SPL increases, on average 2.3 decibels. Thus, it may be reasonable to suggest that the magnitude of the SPL response contributed, in part, to the differential treatment effect on interarticulator timing patterns. Interarticulator treatment responses will first be presented for the improved voicing group, followed by a discussion for the unimproved voicing group.

4.1.3 Improved voicing group

Voice onset time (voicing initiation)—The six participants in the improved voicing group demonstrated improved temporal control of voicing initiation when speaking at higher SPLs post-treatment. As illustrated in Figure 2, the mean VOTs for the six improved voicing speakers (M01, M04, M06, M18, F02, and F03) approached typical VOT values previously reported for neurologically-healthy older adults (Fischer & Goberman, 2010) across most places of articulation. Consistent with the third hypothesis, a VOT detraining effect was observed for the six improved voicing speakers. As shown in Figure 2, the six individual

speakers were found to move back toward baseline VOT levels 4-weeks after treatment was removed. The interarticulator timing adjustments observed for the six speakers in the improved voicing group may reflect changes to motor performance and/or improved laryngeal muscle activation (Ludlow, Yeh, Cohen, Van Pelt, Rhew, & Hallett, 1994).

Percent voicing (voicing termination)—Treatment-related improvements were similarly observed for the temporal control of voicing termination. Post-treatment improvement in voicing termination was reflected by a post-treatment decrease in voicing during the voiceless closure segment. As shown in Figure 4, the six improved voicing speakers (M01, M04, M06, M18, F02, and F03) demonstrated an average 7.5% decrease in voicing during the voiceless closure interval when speaking at higher SPLs immediately post-treatment, with individual speakers showing a 4% to 11% decrease in voicing. Consistent with the third hypothesis, each of the six improved voicing speakers shown in Figure 4 moved back toward baseline voicing levels 4-weeks after treatment was removed.

Interestingly, the post-treatment strategies used to enhance the production of a voiceless obstruent varied across the six improved voicing speakers. For example, as illustrated in Figure 4a, participants F02 and F03 showed evidence of a “supralaryngeal adjustment” (i.e., longer closure duration), when speaking at higher SPLs post-treatment, with little laryngeal contribution. On average, participants F02 and F03 showed a 20% increase in the duration of the oral constriction, with no corresponding decrease in voicing during the voiceless segment. In contrast, participants M06 and M18, shown in Figure 4b, showed evidence of a “laryngeal adjustment” (i.e., a reduction in voicing during the closure), with little supralaryngeal contribution. Participants M06 and M18 showed an average 9.5% decrease in voicing during the voiceless closure, with little change in the duration of the voiceless segment. Finally, participants M01 and M04, shown in Figure 4c, appeared to use a combination of laryngeal and supralaryngeal adjustments to signal the termination of voicing in the voiceless closure. Participants M01 and M04 showed an average 11% increase in closure duration, coupled with an 8.5% decrease in voicing during the voiceless interval. The phonetic trading relationships observed for the improved voicing speakers are consistent with acoustic cue trading strategies reported for healthy adult speakers (Repp, 1981, Repp, 1983).

In summary, the observed improvements in interarticulator timing (voicing initiation and termination) are consistent with system-wide improvements documented following programs training SPL (Dromey et al., 1995; Dromey & Ramig, 1998a, 1998b; Ramig et al., 1995). It is further speculated that improvements in temporal coordination may reflect a reallocation of central processing resources. According to Kahneman’s (1973) fixed capacity model, when total effort is high one activity receives most of the attention processing capacity, leaving fewer resources for the other processes. Since the present vocal intensity changes were elicited using a less cognitively demanding task (the Lombard effect), resource reallocation may have occurred due to the lower overall effort required to regulate vocal intensity, thus leaving more resources available for laryngeal-supralaryngeal subsystem interaction. Not all individuals, however, improved their laryngeal-supralaryngeal timing in response to treatment, which may reflect underlying differences in their perceived

level of effort. The interarticulator treatment-responses for the unimproved voicing speakers will now be presented and discussed.

4.1.4 Unimproved voicing group

Voice onset time (voicing initiation)—Although the majority of participants with PD demonstrated post-treatment improvement in voicing initiation patterns, four participants (M02, M07, M13, and M16) lacked VOT improvement at the end of treatment (see Figure 3). The mean VOTs for the four unimproved voicing speakers were 43, 56, and 67 milliseconds for word-initial [p] [t] and [k], respectively, at the onset of treatment and 45, 58, and 67 milliseconds at the end of treatment. The four individual speakers demonstrated shorter VOTs across most places of articulation, compared to the VOT data reported for neurologically-healthy older adults (Fischer & Goberman, 2010), though still within the VOT range expected for voiceless stops. The shorter VOTs observed for the four individual speakers suggests poor temporal coordination between the release of the oral closure and the initiation of vocal fold vibration. The lack of VOT improvement may reflect reduced motor control during the laryngeal adduction gesture or difficulty regulating aerodynamic events under a condition of increased subglottal pressure.

Percent voicing (voicing termination)—The group-level finding of a significant increase in voicing during the voiceless closure interval was primarily attributed to the post-treatment performance of the four unimproved voicing speakers. As shown in Figure 5, participants M02, M07, M13, and M16 demonstrated an average 12% increase in voicing during the voiceless closure (range of 7% to 22%) immediately following completion of voice treatment. Four weeks after treatment was removed, three of the four speakers (M07, M13, and M16) shown in Figure 5, moved back toward baseline levels showing a 12.3% decrease in voicing during the voiceless interval. However, as illustrated in Figure 5, one participant (M02) showed a further reduction in laryngeal-supralaryngeal timing 4-weeks after treatment was removed. Interestingly, participant M02 was not receiving pharmacological or surgical management for PD-related symptoms at the time of participation and the declines in timing could be related to deteriorating overall function as a result of PD. While the VOT data for the four unimproved speakers suggested maintenance of a VOT interval associated with [p] [t] and [k], the four speakers also increased the duration of voicing during the normally-silent closure gap. The combination of unimproved VOTs and increased voicing would signal production of unintended voiced obstruents. In contrast, the improved voicing group demonstrated improved VOT and a decreased presence of voicing in the voiceless stop closure gap for more accurate production of voiceless stops.

Two plausible explanations are offered to account for the observed increase in voicing during the voiceless segment for the unimproved voicing group. First, it is hypothesized that the four unimproved voicing speakers had difficulty maintaining adequate vocal tract closure under a condition of increased subglottal pressure (higher SPLs) due to weakness of the upper vocal tract musculature. Examination of the stop closure duration data provided in Figure 5, suggests that the four speakers in the unimproved voicing group exhibited shorter oral constrictions both pre-treatment and post-treatment. On average, the four unimproved voicing speakers demonstrated average closure duration values of 88, 58, and 52 msec, for

[p] [t] and [k], respectively. In comparison, the six improved voicing speakers demonstrated average closure duration values of 107, 100 and 96 msec, respectively, for [p] [t] and [k], which is consistent with closure duration data previously reported by Stathopoulos and Weismer (1983) for neurologically-healthy adults. If the oral cavity is not adequately sealed during production of a voiceless stop consonant, glottal vibration will likely continue during the voiceless segment since the subglottal pressure-oral air pressure gradient will be maintained.

A second possible explanation for the continued presence of voicing during the voiceless segment is that the vocal folds did not maximally abduct during the laryngeal devoicing gesture. The importance of the abductory gesture to achieve laryngeal devoicing has been highlighted by Watson (1988) in studies of neurologically-healthy older adults and in individuals with PD. If the vocal folds remain in close proximity during production of a voiceless segment, glottal vibration is likely to continue as airflow velocity increases through the narrowed glottal opening. As previously suggested by Weismer (1984), the increased stiffness of the laryngeal musculature associated with PD may lead to a more adducted vocal fold posture during a normally open glottal configuration.

4.1.5 General interarticulator timing patterns observed for speakers with PD

The individual VOT data found before treatment supports previous acoustic reports of aberrant phonatory onset behavior in individuals with PD (Flint et al., 1992; Forrest, Weismer, & Turner, 1989; Weismer, 1984). As illustrated in Figure 2, three speakers (F03, M01, and M04) demonstrated an atypical lag in the onset of voicing following the release of the oral constriction (longer VOTs than healthy older adults) for at least two places of articulation. The longer VOTs observed for speakers F03, M01, and M04 is consistent with VOT data previously reported by Forrest et al. (1989) for voiced bilabial plosives produced by individuals with PD. In contrast, seven speakers (F02, M02, M06, M07, M13, M16, and M18) exhibited a rapid onset of voicing following the release of the oral constriction (shorter VOTs than healthy older adults; Figures 2–3) for most places of articulation. The shorter VOTs observed for these 7 individual speakers is consistent with VOT data previously reported for similar word-initial voiceless stop consonants in studies of individuals with PD (Flint et al., 1992; Weismer, 1984). It is also important to note that the mean VOTs for word-initial [p], shown in Figures 2 and 3, fell closest to the voicing boundary previously described by Lisker and Abramson (1964), which may affect a listener's perception of the voiceless bilabial plosive. Regardless of the nature of the laryngeal timing deficit observed prior to treatment (e.g. delayed onset of voicing vs. rapid onset of voicing), six of the ten participants with PD exhibited more typical VOTs as a result of treatment.

The observed timing deficits in voicing initiation may be related to disease-state, rather than to the effects of normal aging. Weismer (1984) and Sweeting and Baken (1982) previously reported no age-related differences in VOT for healthy older adults and healthy young adults. Weismer (1984) postulated that the increased rigidity of the laryngeal musculature, associated with PD, leads to a more adducted vocal fold posture during peak glottal opening. As a result, the vocal folds are able to achieve a fully adducted position more quickly at the

onset of voicing, which is acoustically reflected by a shorter VOT. Forrest et al. (1989) further suggested that the observed lag in the onset of glottal vibration for the voiced bilabial plosive may reflect a deficit in motor initiation and coordination at the level of the larynx. In summary, the present VOT data verifies the presence of anomalous voicing initiation timing patterns in some individuals with PD. It is hypothesized that the temporal changes in voicing initiation, observed for the present subset of speakers with PD, likely results from a combination of impaired muscle function and/or motor coordination difficulties.

The present voicing termination data is also in agreement with previous acoustic reports of aberrant phonatory offset behavior in individuals with PD. Prior to treatment, participants with PD voiced, on average, approximately 20% of the voiceless closure interval. As shown in Figures 4 and 5, all participants were observed to follow this voicing trend, with individual speakers voicing approximately 10%–30% of the voiceless segment prior to treatment. Previous research has indicated a tendency toward continuous voicing throughout voiceless segments in individuals with PD (Kent & Rosenbeck, 2004; Weismer, 1984), thus suggesting difficulty with the laryngeal devoicing gesture and/or difficulty coordinating the timing of the laryngeal and supralaryngeal mechanisms. While qualitative studies have provided insight into the prevalence of phonatory timing deficits in PD, the present study offers preliminary quantitative data to support the observed changes in phonatory offset behavior.

4.1.6 Speech intelligibility

In support of the fourth hypothesis, the group data for the speakers with PD showed significantly higher speech intelligibility scores immediately post-treatment. Closer inspection of the individual speech intelligibility data, however, revealed an important finding. The six participants (M01, M04, M06, M18, F02, and F03) who showed post-treatment improvement in interarticulator timing (voicing initiation and termination) followed the group intelligibility effect (Figure 6). The four participants (M02, M07, M13, and M16) whose laryngeal-supralaryngeal timing did not respond favorably to treatment, however, showed no post-treatment improvement in speech intelligibility (Figure 6). Given that all speakers increased their SPLs post-treatment, vocal intensity adjustments alone cannot account for the reported intelligibility improvements. It is hypothesized that other supralaryngeal and/or laryngeal adjustments contributed to the treatment-related improvements in speech intelligibility observed for participants in the improved voicing group. It remains unclear, however, how the enhanced voicing contrasts contributed to the judges' perception of increased intelligibility as other aspects of the participants' voices likely also improved.

The influence of interarticulator timing patterns on the perception of voicing contrasts has largely been studied in the hearing-impaired population (Mangan, 1961; Markides, 1970; Nober, 1967; Millin, 1971; Smith, 1975; Monsen, 1976). One of the most comprehensive perceptual studies of voicing contrasts was conducted by Monsen (1976), who examined the perception of voiced and voiceless cognates produced by hearing-impaired children. The hearing-impaired children who maintained the voiced-voiceless distinction were judged to be highly intelligible, whereas the children who did not maintain the voicing contrast (i.e.

VOTs overlapped for voiced-voiceless cognates) were judged to be less intelligible. Thus, the perceptual consequence of laryngeal-supralaryngeal timing errors is clear: a reduction in the voiced-voiceless distinction may degrade speech communication.

4.1.7 Clinical implications

The individual participant data suggests a highly individualized response to treatment. While the majority of participants with PD showed post-treatment improvement in the temporal coordination of the laryngeal and supralaryngeal mechanisms when speaking at higher SPLs, four speakers showed evidence of reduced temporal coordination or a lack of improvement in voicing in response to treatment. It is interesting to note that improvement in interarticulator timing patterns was observed for the majority of speakers with PD in this lower treatment load condition (e.g. +3 dB SPL). These data demonstrate that significant improvements can be elicited in communication without using high treatment loads and using training paradigms that occur in everyday communication environments. These results support the use of an alternative to traditional behavioral therapy paradigms.

The factors underlying the differential response to treatment remain unclear. One possible contributing factor are the differences in disease characteristics. As shown in Table 1, the participants with PD differed in disease severity level as defined by motor symptoms (Hoehn and Yahr stages II–III), as well as their hypophonia severity level and their global speech and voice severity ratings. These diverse patient characteristics and the observed differences in response to treatment are consistent with previous reports of heterogeneity in PD (Darley et al., 1969, 1975; Duffy, 2005; Logemann & Fisher, 1981). The inclusion of a diverse group of patients with PD supports the ecological validity of this treatment study by exploring a potential treatment for hypophonia in a heterogeneous sample of patients. The use of a within-subject design in this treatment study allowed each patient to be compared to him or herself.

4.1.8 Methodological considerations and conclusion

The present acoustic results provide preliminary evidence of improved laryngeal-supralaryngeal timing, in some individuals with PD, as a result of treatment. Given the relatively small number of participants in the current study, however, a larger scale study would be beneficial to determine which interarticulator timing patterns are more common and to explore the factors underlying an individual's response to treatment. It is also important to note that the interpretation of the present acoustic data is confined to the analysis of word-initial voiceless stop consonants. Further investigation may be warranted to determine if the interarticulator timing patterns observed can be generalized across voicing type (i.e. voiced cognates) and word position. In addition, the current investigation used a sentence production task to limit confounding influences such as stress patterns, fundamental frequency, and vowel context. However, structured speaking tasks may not reflect the interarticulator timing patterns of conversational speech.

In summary, the current analyses were intended to provide evidence concerning the effect of increased vocal intensity on interarticulator timing in individuals with PD. For the majority of speakers with PD after an 8-week training program using the SpeechVive™, measures of

VOT and percent voicing reflected improved voiced-voiceless distinctions during sentence production. In addition, improved speech intelligibility scores were shown post-treatment for the six speakers whose interarticulator timing improved. The individual participant results provide evidence to suggest that voice therapy may improve the temporal coordination of laryngeal and supralaryngeal events in some individuals with PD. Further research is warranted, however, to better understand the factors that determine an individual's response to therapy. Identification of such prognostic indicators would assist in the clinical management of speech and voice disorders in individuals with PD.

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Highlights

- The use of a natural cueing strategy (the Lombard effect) is effective for eliciting increased vocal intensity in individuals with Parkinson's disease
- Speakers with Parkinson's disease show differential changes in voicing initiation and voicing termination patterns when speaking at higher SPLs
- The differential impact of voice treatment on interarticulator timing warrants further investigation

Figure 1.

Mean SPL with standard error bars shown for ten speakers with Parkinson's disease. The asterisk (*) indicates significant comparisons at an alpha of 0.05. SPL data is indicated for pre-treatment (Pre_{Avg}), the onset of post-treatment (PT_{WK1}), and 4-weeks post-treatment (PT_{WK4}). The treatment effect = PT_{WK1}-Pre_{Avg}. The detraining effect= PT_{WK4}-PT_{WK1}.

Figure 2.

Figures 2a–c. Mean voice onset time (VOT) data shown by place of articulation for the six individual speakers who demonstrated improvement in VOT with treatment. Data is shown for pre-treatment average (Pre_{Avg}), immediate post-treatment (PT_{WK1}) and 4-weeks post-treatment (PT_{WK4}). The treatment effect = PT_{WK1} - Pre_{Avg}. The detraining effect = PT_{WK4} - PT_{WK1}. Horizontal line = mean VOT data reported for nine healthy older adult speakers by Fischer and Goberman (2010) for word-initial [p] [t] [k]. Voicing boundary at 25 msec for production of word-initial English stop consonants (Lisker & Abramson, 1964).

Figure 3a. Mean VOT for Voiceless Bilabial Stop

Figure 3b. Mean VOT for Voiceless Alveolar Stop

Figure 3c. Mean VOT for Voiceless Velar Stop

Figure 3.

Figures 3a–c. Mean voice onset time (VOT) data shown for the four individual speakers who did not demonstrate improvement in VOT with treatment. Data is shown for pre-treatment average (Pre_{Avg}), immediate post-treatment (PT_{WK1}) and 4-weeks post-treatment (PT_{WK4}). The treatment effect = $PT_{WK1} - Pre_{Avg}$. The detraining effect = $PT_{WK4} - PT_{WK1}$. Horizontal line = mean VOT data reported for nine healthy older adult speakers by Fischer and Goberman (2010) for word-initial [p] [t] [k]. Voicing boundary at 25 msec for production of word-initial English stop consonants (Lisker & Abramson, 1964).

Figure 4a. Increased Closure Duration

Figure 4b. Decreased Voicing Duration

Figure 4c. Decreased Voicing Duration; Increased Closure Duration

Figure 4.

Figures 4a–c. Illustration of laryngeal and supralaryngeal adjustments associated with post-treatment increases in vocal intensity during production of vocalic to voiceless stop sequences. Data is shown for the six individual speakers from Figure 2 and are pooled across place of articulation. Percent voicing is indicated for each session: pre-treatment average (PreAvg), immediate post-treatment (PTWK1), and 4-weeks post-treatment (PTWK4). The treatment effect = $PTWK1 - PreAvg$. The detraining effect = $PTWK4 - PTWK1$.

Figure 5.

Illustration of laryngeal and supralaryngeal adjustments associated with post-treatment increases in vocal intensity during production of vocalic to voiceless stop sequences. Data are shown for the four individual speakers from Figure 3 and are pooled across place of articulation. The percent voicing is indicated for each session: pre-treatment average (PreAvg), immediate post-treatment (PT_{WK1}), and 4-weeks post-treatment (PT_{WK4}). The treatment effect = PT_{WK1}-PreAvg. The detraining effect= PT_{WK4}-PT_{WK1}.

Figure 6.

Mean speech intelligibility scores shown by speaker group: "Improved", "Unimproved", and all speakers combined (Improved + Unimproved). "Improved" speakers demonstrated a favorable change in interarticulator timing in response to voice treatment. "Unimproved" speakers demonstrated an unfavorable or unremarkable change in interarticulator timing in response to voice treatment. Data is shown for pre-treatment (Pre) and immediate post-treatment (PT_{WK1}). The treatment effect = $PT_{WK1} - Pre_{Avg}$. The detraining effect = $PT_{WK4} - PT_{WK1}$.

Table 1

Participant Characteristics

Speaker (n=10)	Age at Date of Testing (years)	Time Since Diagnosis (years)	Hoehn & Yahr Stage	Pure Tone Average ^a in dB	PD-Related Medications	Hypophonia Severity ^b	Auditory-Perceptual Speech and Voice Characteristics ^c	Speech and Voice Severity Rating (%) ^d	Interarticulator Treatment Response ^e
M01	68	3	2	22	Carbidopa-Levodopa; M Mirapex; Azilect	Mild	Monopitch, monoloud, reduced stress, breathy voice, hoarse voice	10	Improved
M02	79	6	3	23	--	Mild	Monopitch, monoloud, reduced stress, breathy voice, hoarse voice, mildly slow rate, loudness decay	20	Unimproved
M04	68	11	2	17	Carbidopa-Levodopa; Mirapex ER	Mild	Breath voice, hoarse voice	41	Improved
M06	66	6	2	12	Azilect; Amantadine	Mild	None observed	4	Improved
M07	81	5	2	33	Carbidopa-Levodopa; Azilect	Mild	Breath voice, hoarse voice	9	Unimproved
M13	71	6	2	53	Mirapex, Amantadine	Mild	Monopitch, imprecise consonants, breathy voice, hoarse voice, mildly increased rate, short rushes of speech, loudness decay	38	Unimproved
M16	86	2	3	28	Carbidopa-Levodopa; Lodosyn	Mild	Breathy voice, hoarse voice, variable rate, variable loudness	25	Unimproved
M18	74	6	4.5	12	Carbidopa-Levodopa	Severe	Monopitch, breathy voice	21	Improved
F02	74	13	2	33	--	Moderate	Monopitch, imprecise consonants, breath voice, hoarse voice, mildly fast rate, short rushes of speech, loudness decay	30	Improved
F03	76	5	3	35	Requip, Stalevo	Normal	Monopitch, imprecise consonants breathy and hoarse voice, moderately slow rate, loudness decay	29	Improved

^a Average pure tone thresholds reported for the better ear for the octave frequencies 500, 1000, and 2000 Hz.

^b Assigned by a certified speech-language pathologist during connected speech.

^c Assigned by a certified speech-language pathologist during a diadochokinetic rate task, sustained vowel productions, and connected speech.

^d Assigned by a certified speech-language pathologist during connected speech using a 150mm visual analogue scale (Cannito et al., 1997). Results indicated normal (0%) to severely impaired speech and/or voice characteristics (100%).

^e “Improved” denotes a favorable change in interarticulator timing patterns (voicing initiation and termination) at higher vocal intensities; “Unimproved” denotes an unfavorable or unremarkable change in interarticulator timing patterns (voicing initiation and termination) at higher vocal intensities.

-- Not receiving pharmacological treatment at the time of participation.

Table 2

Group means (and standard deviations) for voice onset time and voice onset time ratio by session and place of articulation

	Pre-Treatment	Immediate Post-Treatment	4-weeks Post-Treatment
Voice onset time (msec)			
Bilabial [p]	56.38 (20.87)	66.66 (28.58)	58.69 (22.68)
Alveolar [t]	76.17 (24.08)	82.53 (29.25)	77.86 (25.74)
Velar [k]	80.93 (29.50)	86.85 (32.47)	81.02 (32.82)
<i>Means (SD)</i>	<i>71.16 (24.90)</i>	<i>78.68 (30.84)</i>	<i>72.52 (28.22)</i>
Voice onset time ratio			
Bilabial [p]	0.276 (0.08)	0.307 (0.09)	0.263 (0.07)
Alveolar [t]	0.278 (0.05)	0.318 (0.06)	0.294 (0.07)
Velar [k]	0.291 (0.06)	0.322 (0.07)	0.308 (0.07)
<i>Means (SD)</i>	<i>0.282 (0.06)</i>	<i>0.316 (0.07)</i>	<i>0.288 (0.07)</i>