Supraglottal Hyperadduction in an Individual With Parkinson Disease: A Clinical Treatment Note

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Recent treatment for voice problems associated with idiopathic Parkinson disease has primarily focused on increasing reduced vocal loudness and improving true vocal fold hypoadduction, common voice deficits observed in these individuals. This study presents an individual with reduced vocal loudness and supraglottic hyperadduction accompanying Parkinson disease and the outcome following a course of the Lee Silverman Voice Treatment (LSVT). Posttreatment observations included increased vocal loudness, decreased supraglottic hyperadduction, and improved intonation and overall voice quality when compared with pretreatment observations. These results suggest that in this individual, supraglottic hyperadduction was due to a secondary compensatory behavior resulting from mild true vocal fold hypoadduction that responded positively to adduction therapy (LSVT). This study also demonstrates the use of a continuum of observations ranging from functional ratings to physiological measures to evaluate the impact of intensive voice treatment and identify mechanisms underlying treatment-related change in an individual with Parkinson disease.

Common neurological disorders, such as Parkinson disease, are often characterized by laryngeal abnormalities that result in voice disorders (Aronson, 1990; Brin, Fahn, Blitzer, Ramig, & Stewart, 1992; Darley, Aronson, & Brown, 1975; Logemann, Fisher, Boshes, & Blonsky, 1978). These disorders may contribute to reductions in vocal loudness, vocal quality, and overall speech intelligibility (Aronson, 1990; Kent et al., 1990; Ramig, 1992). Recently, Ramig and Scherer (1992) and Smith and Ramig (1995) developed an approach to planning behavioral treatment for voice disorders associated with neurological disease. This approach focuses on the disordered laryngeal function in combination with the neurological etiology and is designed to maximize improvement in overall intelligibility as efficiently as possible.

In this approach, the disordered voice is considered in relation to problems of adduction or phonatory instability (Ramig & Scherer, 1992). Disorders of hypoadduction are distinguished from those of hyperadduction. Disorders of hypoadduction are characterized by inadequate true vocal fold closure, reduced loudness, and breathy, hoarse vocal quality. Hypoadduction may accompany a variety of neurological disorders but is typically associated with lower motor neuron (flaccid) disorders, multiple sclerosis...
Individuals with Parkinson disease (PD) may have mild to severe bowing of the true vocal folds or other forms of vocal fold incompetence (Aronson, 1990; Perez et al., 1996; Smith, Ramig, Dromey, et al., 1995; Ward, Berci, & Calcaterra, 1977) during speech. As a result, many individuals with PD present with reduced loudness and a weak, breathy, or hoarse vocal quality in addition to monotone of pitch and loudness, shortened breath groups, and vocal tremor (Brin et al., 1992; Darley et al., 1975; Ramig & Gould, 1986). Furthermore, individuals with PD may exhibit reduced respiratory volumes for speech resulting from limited thoracic excursion and difficulty coordinating the respiratory and laryngeal systems (Aronson, 1990; Darley, Aronson, & Brown, 1969a, 1969b). Hyperadduction occurs in different forms; it can be constant, as in the strain-strangled phonation of pseudolubular palsy, or variable, as in the alternating adductor laryngospasm in spasmodic dysphonia (Aronson, 1990; Darley et al., 1969b). In some cases, hyperadduction may be so severe as to result in complete termination of phonation or aphony.

A second form of hyperadduction involves adduction of the ventricular (false) vocal folds (Arnold & Pinto, 1960; Aronson, 1990). The exact etiology of supraglottic hyperadduction has been debated (Arnold & Pinto, 1960; Jackson & Jackson, 1935; Roy, 1994; Von Hake, Ganzman, & Mauer, 1989), although it has been associated with laryngeal paralysis, cerebral disease, and cerebellar lesions (Arnold & Pinto, 1960; Von Hake et al., 1989). Supraglottal hyperadduction may also be a means of compensation for true vocal fold hypoadduction (Arnold & Pinto, 1960; Feinstein, Hilger, Szachowicz, & Stimson, 1987; Jackson & Jackson, 1935; Roy, 1994; Von Doersten, Izdebski, Ross, & Cruz, 1992; Woo, Casper, Colton, & Brewer, 1992, 1994). For example, individuals with Parkinson disease may occasionally present with supraglottic hyperadduction (i.e., anterior-posterior foreshortening and ventricular hyperadduction or phonation) (Arnold & Pinto, 1960; Hanson et al., 1984; Von Hake et al., 1989; Smith, Ramig, Dromey, et al., 1995) and true vocal fold hypoadduction. Such observations are consistent with the opinion that ventricular fold adduction may result as a secondary compensatory mechanism in individuals with vocal fold hypoadduction.

The combination of supraglottal hyperadduction and glottal hypoadduction in individuals with PD creates a clinical dilemma for the speech-language pathologist, who would typically treat individuals with Parkinson disease for a hypoadducted voice disorder. However, if supraglottic hyperadduction is secondary compensation for inadequate vocal fold closure or functioning, treatment designed to improve true vocal fold adduction or vibratory efficiency should be effective in reducing supraglottic hyperadduction and improving the voices of these individuals. An initial study by Smith, Ramig, Dromey, et al. (1995) noted improved true vocal fold adduction with reduction in mild supraglottic hyperadduction following a course of the Lee Silverman Voice Treatment (LSVT) in individuals with Parkinson disease. This improvement suggests that the mild supraglottic hyperadduction observed in these individuals may have been secondary compensation for an inadequate glottal source.

The LSVT was developed by Ramig and colleagues for remediation of voice and speech disorders associated with Parkinson disease (Countryman & Ramig, 1993; Countryman, Ramig, & Pawlas, 1994; Ramig, Bonitati, Lemke, & Horii, 1994; Ramig, Countryman, O’Brien, Hoehn, & Thompson, 1996; Ramig, Countryman, Thompson, & Horii, 1995) and other neurological disorders (Ramig, Baker, Smith, Luschei, Countryman, & Pawlas, 1997). The LSVT program is based on the underlying vocal fold hypoadduction (Hanson et al., 1984; Smith, Ramig, Dromey, et al., 1995), reduced range of motion in laryngeal musculature, and poor respiratory drive typically observed in individuals with Parkinson disease. Treatment is designed to maximize overall speech intelligibility by focusing on increased or efficient true vocal fold adduction and improved coordination of the respiratory and laryngeal systems (Ramig & Dromey, 1996), as well as improved sensory perception of effort (calibration) (Ramig, Pawlas, & Countryman, 1995). Following conventional wisdom, the speech-language pathologist may determine this method of voice treatment to be inappropriate for an individual with PD who has reduced loudness but moderate to severe supraglottal hyperadduction. There may be a concern that the adduction exercises would exacerbate the hyperfunctioning system. Furthermore, not all individuals with PD with reduced loudness have apparent vocal fold hypoadduction on laryngeal stroboscopic examination (Smith, Ramig, Dromey, et al., 1995). Again, conventional wisdom considers true vocal fold adduction exercises unsuitable for individuals with adequate vocal fold closure. To date, there have been no reports in the literature that specifically examine the effects of the LSVT on an individual with PD who has reduced loudness and a moderate to extreme degree of supraglottic hyperadduction.

The purpose of this paper is to report speech and voice data from one individual with Parkinson disease who had a mild to moderately soft, hoarse voice and severe supraglottic hyperadduction, including ventricular fold vibration during soft, normal, and loud phonation and mild true vocal fold hypoadduction during soft phonation pretreatment during flexible endoscopic exam. It was hypothesized that the LSVT would improve the individual’s primary
deficit (true vocal fold hypoadduction), reduce the need for secondary compensatory behavior (supraglottic hyperadduction), and result in improved loudness, intonation, and overall vocal quality posttreatment.

Method

Participant

The participant was diagnosed with idiopathic Parkinson disease (IPD) and was determined to be stable on his anti-Parkinson medication by a neurologist before entering this study. His medications did not change, and he did not receive other therapies (physical, occupational, etc.) during the study period. Furthermore, the participant had not received previous speech or voice treatment before the start of this program.

The participant was a 60-year-old male who had first been diagnosed with IPD in December 1994. At the time of enrollment in the LSVT program in April 1995, he was classified as stage III (moderate-severe) Parkinson disease (Hoehn & Yahr, 1967). His score on the motor section of the Unified Parkinson Disease Rating Scale (UPDRS; Fahn, Elton, & members of the UPDRS development committee, 1987) was 33, indicating mild impairment (higher scores on the UPDRS indicate greater impairment; scores range from 0 to 108 for the motor section). Anti-Parkinson medication included selegiline hydrochloride (Eldepryl®). An initial speech mechanism exam revealed structure and function of speech musculature within normal limits. Perceptual speech and voice characteristics, as rated by the attending speech-language pathologist, included mild to moderately reduced loudness, monotone pitch, a hoarse vocal quality, and an abnormally low pitch during sustained phonation and conversational speech. The attending speech-language pathologist rated overall speech and voice impairment as mild to moderate. Before treatment, the participant and a family member reported that his speech and voice deficits affected the functionality and effectiveness of his communication. For example, during telephone and in-person conversations, the participant reported that he had to repeat himself numerous times for the listener to hear and understand him. At the time of treatment, the participant was self-employed in a small consulting firm and typically spent 1 to 2 hours speaking per day, usually on the phone. The participant reported no problems with chewing or swallowing.

Treatment

The LSVT was administered to this participant. Treatment techniques focused on increasing vocal loudness and phonatory efficiency by targeting the hypothesized underlying laryngeal pathophysiology and optimizing phonatory and respiratory effort and coordination across the motor speech system using the global variable “loud” (Dromey et al., 1995; Ramig, Countryman, et al., 1995; Schulman, 1989). The LSVT was specifically designed to maximize participant motivation for the speech tasks and facilitate immediate carryover of increased vocal loudness and vocal quality into functional communication. Specific techniques included optimizing vocal fold closure and efficiency through vocal isometric exercises and improving respiratory support by instructing the participant to inspire deeply and frequently before speaking “loud” and to speak on “top of the breath.”

A typical session included a high number of successive repetitions of the following activities: maximum duration of sustained vowel phonation (/a/), generation of the highest and lowest fundamental frequencies the participant could obtain, and speech production tasks using the same phonatory and respiratory techniques used in sustained phonation. The high number of repetitions during the treatment as well as the intensiveness of treatment (four times a week for 4 weeks) are consistent with theories of motor learning and skill acquisition (Schmidt, 1975, 1988) and muscle training (Saxon & Schneider, 1995). Treatment techniques also enhance the participant’s overall “knowledge of results,” which is critical in skill acquisition (Salmoni, Schmidt, & Walter, 1984). A tape recorder was used to provide feedback on vocal quality and loudness to the participant during each task. In addition, during the 4-week program, the participant was trained in self-monitoring of vocal effort, loudness, and vocal quality (sensory calibration) and in habituation of the increased loudness and improved vocal quality into conversational speech. At no point during treatment was the participant taught to sustain phonation or speak in a voice that was pressed or strained. The LSVT method has been described in further detail elsewhere (Ramig, Pawlas, & Countryman, 1995).

Measurement Variables and Rationale

Variables that assess a continuum of glottic and supraglottic functioning as well as objective and perceptual characteristics of the participant’s speech and voice were chosen to document pre- to posttreatment changes. The variables presented are sound pressure level, mean fundamental frequency and its variability, maximum duration of sustained vowel phonation, electroglottographic data, perceptual ratings of the participant’s speech and voice by experienced practitioners, and videolaryngostroboscopic data.

Sound Pressure Level. Because of the high frequency of occurrence of reduced loudness in individuals with Parkinson disease (Aronson, 1985; Canter, 1965; Critchley, 1981; Darley et al., 1969a, 1969b; Fox & Ramig, 1997), the impact of reduced loudness on speech intelligibility (Ramig, 1992), and its close relationship to vocal fold adduction and function, sound pressure level (SPL) during sustained phonation, reading, and monologue was measured.

Maximum Duration of Sustained Vowel Phonation. The maximum duration of sustained vowel phonation was chosen for measurement because of its relationship to laryngeal (Yanagihara, Koike, & von Leden, 1966) and respiratory functioning (Boone, 1977). Stimulation of these two mechanisms is important for training respiratory and laryngeal coordination and improving overall vocal loudness and functioning in individuals with PD (Ramig, 1992).

Mean Fundamental Frequency and Fundamental Frequency Variability. Mean fundamental frequency and
its variability (semitone standard deviation [stsd]) were chosen for measurement to evaluate whether changes in phonatory and respiratory effort and vocal intensity as well as the treatment exercises generalized to other aspects of the participant’s speech. Changes in fundamental frequency often accompany changes in vocal intensity (Jacob, 1968; Linville & Korabic, 1987). Positive changes in these two variables may indicate better intonation and overall vocal quality in this participant. Changes in the measures of fundamental frequency variability and intensity have been previously documented in IPD patients following a course of the LSVT (Ramig et al., 1994; Ramig, Countryman, et al., 1995; Ramig et al., 1996).

Electroglottographic Width 50% (EGGW50). The electroglottographic width 50% was chosen for analysis because of its theoretical relationship to true vocal fold adduction (Scherer & Vail, 1988; Scherer, Vail, & Rockwell, 1995; Titze, 1984). EGGW50 provides information about the duration of glottal closure relative to the duration of complete glottal cycle. EGGW50 is a measure of glottal adduction and is defined as the width of the EGG signal at 50% of the wave’s amplitude divided by the period, where the width is defined between the positive-going (glottal closing) and negative-going (glottal opening) portions of the EGG waveform (Scherer et al., 1995). It is highly correlated with other measures of glottal adduction, such as the abduction quotient (Titze, 1984).

Expert Listener Perceptual Ratings. To determine the extent changes in speech and voice characteristics from pre- to immediately posttreatment, expert raters unfamiliar with the participant completed a perceptual listening task based on a tape recording of the participant reading the “Rainbow Passage” (Fairbanks, 1960). The variables rated by the listeners were “strained” voice quality, “hoarse or rough” voice quality, and “strong” voice. Hoarseness, roughness, and weak voice are all symptoms of PD and would be expected to decrease following a course of the LSVT. Because of the participant’s pretreatment supraglottic hyperadduction and the treatment focus on true vocal fold adduction, “strained” voice quality was included in the ratings.

Videolaryngostroboscopic Examination and Ratings. After completion of an otolaryngological history and examination, a laryngostroboscopic examination was done to evaluate laryngeal and vocal fold functioning. To obtain a nonbiased description of the participant’s laryngeal functioning pretreatment to immediately posttreatment, experienced raters unfamiliar with the participant completed a blind perceptual study. Variables assessed by perceptual videostroboscopic ratings included supraglottal functioning (anterior-posterior compression and false vocal fold adduction) and degree of glottal competence (i.e., true vocal fold adduction).

Data Collection. SPL, mean fundamental frequency and its variability, and electroglottographic data were collected three times pretreatment and two times immediately posttreatment to establish a baseline and account for potential variability in speech production associated with Parkinson disease (King, Ramig, Lemke, & Horii, 1994). All data collection sessions were conducted by the same experimenter and were scheduled at approximately the same time of day to minimize effects of medication fluctuation.

During each data collection session, the participant was seated in an IAC-sound-treated booth with a headset microphone (AKG-410) positioned 8 cm in front of his lips (Titze & Winholtz, 1994). After preamplification through an ATI-1000, the microphone signal was recorded onto a Sony Digital PC-108M (DAT) eight-channel recorder. The microphone signal was used to collect speech samples for the perceptual listening task as well as mean fundamental frequency and its variability. To collect sound pressure level data, a Bruel and Kjaer 2230 sound level meter was placed 30 cm from the participant’s mouth. During each speaking and voicing task, the experimenter hand recorded the peak vocal SPL measures that were continuously displayed at 1-second intervals from the digital output of the sound level meter. To collect electroglottographic data, the electrodes of a Synchrovoice Inc. Research Electroglossograph (EGG) were placed on the participant’s neck over the thyroid lamina during each recording session. After amplification (Tektronix Amplifier 502 TM 506), the EGG signal was recorded onto the eight-channel DAT recorder.

Measures of SPL, mean fundamental frequency, and fundamental frequency variability for reading and speaking were collected while the participant read the “Rainbow Passage” and spoke for 30 seconds on a topic of interest (monologue) at a comfortable rate and loudness. SPL, maximum duration, and electroglottographic data of sustained vowel phonation were collected while the participant sustained phonation of the vowel /a/ for as long as possible. A timer with a second hand was placed within the participant’s view to encourage him to monitor his performance and sustain phonations longer with each repetition. Four to six maximally sustained vowels were collected during each recording session.

The speech samples for the perceptual listening task were collected by using the recorded microphone signal from the participant’s second pretreatment and first posttreatment data-collection session reading of the “Rainbow Passage.” These passages were dubbed in random order with loudness normalized onto a master rating tape that contained several samples of individuals with PD reading the same passage. Twenty percent of the samples were repeated to determine intrarater reliability.

For the videolaryngostroboscopic data, an examination was completed one time within the week preceding treatment and one time within the week immediately following treatment. To collect the videolaryngostroboscopic data, the nasal passage was anesthetized with 4% lidocaine spray. Endoscopic examination was conducted with both an Olympus ENP-P3 fiberscope and Nagashima SPT-70 degree rigid laryngoscope using well-described techniques (Bless, Hirano, & Feder, 1987). Images were recorded with a CCD camera, using a 35-mm lens for the fiberscope and a 60-mm lens for the rigid telescope, and a SVHS tape recorder. Examinations were conducted using both a rigid endoscope and a flexible fiberscope because of documented differences between views (Shaik & Bless, Countryman, Hicks, Ramig, Smith, 1995).
Flexible fibrescope views have been reported as more representative of natural speech functioning (Shaik & Bless, 1986; Södersten & Lindestad, 1992; Smith, Ramig, Dromey, et al., 1995). The abnormal posture (i.e., elevation of the larynx) and extension of the tongue, which occurs during a rigid endoscopic examination, may result in incomplete glottal closure during phonation and an abnormally high pitch (Södersten & Lindestad, 1992). In addition, soft phonation may cause differences in closure to become more apparent, making the glottis appear much more open during phonation when viewed under the rigid endoscope as compared with the flexible (Södersten & Lindestad, 1992).

The endoscopic examination protocol is briefly summarized as follows: With the fibrescope, the larynx was visualized under constant light during quiet respiration, sustained phonation of the vowel /i/, and counting for 10 seconds. The strobe light source was then used to visualize the larynx and vocal folds during phonation of sustained vowel /i/ under several conditions, including (a) normal pitch, normal loudness, (b) normal pitch, soft phonation, and (c) normal pitch, loud phonation. Trials were repeated until adequate samples were obtained as judged by the otolaryngologist completing the examination. The fibrescope was withdrawn, and the stroboscopic examination of the larynx was repeated with a rigid telescope for the sustained-vowel tasks only. For the purposes of this paper, only the stroboscopic conditions will be reported.

Two master study videotapes were created that included normal, loud, and soft phonation segments of the participant during stroboscopic examination, before and after treatment. The participant’s samples were included on this tape as part of a larger study. Each phonation segment was shown as viewed through both a flexible fibrescope and a rigid endoscope. The soft phonation condition during rigid scope exam is not reported because of compliance issues with the participant during this task posttreatment. The order of samples was randomized, and 20% of the recording samples were repeated to assess intrarater reliability. The audio signal from each individual on the tape was removed to eliminate auditory perceptual cues. Instead, a prompt was overdubbed onto the audio track that identified the participant number and tasks performed during each sample to allow raters to identify them.

Data Analysis

Sound Pressure Level. SPL during sustained vowel phonation, the “Rainbow Passage,” and conversational monologue were calculated using the continuously hand-recorded peak vocal SPL that was displayed at 1-second intervals from the digital output of the sound level meter. Because peak vocal SPL can only be recorded from the sound level meter during speech output, pauses and hesitations were not included in the analysis. Using this method, mean vocal SPL measures have been reported to be reliable (Fox & Ramig, 1997) and valid when compared to a custom-built software program for measuring SPL from the sound level meter signal (Countryman & Ramig, 1993; Ramig, Countryman, et al., 1995). The mean and standard deviation of each set of output data for each task (i.e., sustained phonation, reading, and monologue) were calculated.

Maximum Duration of Sustained Vowel Phonation. To calculate duration measures of maximally sustained vowel phonation, each phonation (four to six tokens for each recording session) was input into a Hewlett Packard Model 54503A MHz digitizing oscilloscope at a sampling rate of 10 samples per second. Cursors were hand-positioned to mark the monitor-displayed zero crossing preceding the first negative-going peak at the onset and the zero crossing following the final positive-going peak at the offset of each vowel. The four to six tokens were then measured, averaged, and reported for each session in seconds.

Mean Fundamental Frequency and its Variability. To determine measures of mean fundamental frequency and fundamental frequency variability (std) during reading and conversational monologue, the microphone signal was digitized at 5000 samples per second and analyzed on a 486 computer using C-Speech software (Milenkovic, 1987). The mean fundamental frequency and hertz standard deviation were then calculated and displayed by the program. The hertz standard deviation was then converted using a standard formula to express frequency variability in semitones (std).

Electroglottographic Data (EGGW50). The electroglottic signal was low-pass filtered at 10 kHz and digitized at a sampling rate of 20 kHz onto a VAX computer system. To calculate EGGW50, in-house software averaged 13 to 20 consecutive EGG cycles from the temporal midpoint of each vowel obtained during each voice recording session. The mean of four vowels from each pretreatment and posttreatment session were then averaged.

Perceptual Data of Expert Listeners. Three speech-language pathologists with ASHA certification and 3 or more years of experience who were unfamiliar with the participant served as raters for the listening task. The listeners were individually seated in an IAC sound-treated booth approximately 3 feet from the loudspeaker. The tape was played at approximately 75 dB at 30 cm for all samples. The listeners were given a visual analog (VA) scale and asked to rate the perceptual variables of “strained” voice quality, “hoarse or rough” voice quality, and “strong” voice quality during participant taped readings of the “Rainbow Passage.” On one side of the VA scale was the word always and the other side never (e.g., always a “strong” voice, never a “strong” voice). The listeners were asked to make a mark along the line that would best represent their impression of the participant’s voice during reading. Due to high intralistener reliability (Pearson product correlation coefficients > 0.90), data from all three listeners were used in this study. Standard procedures for analysis of visual analog scales (Beckstyns & Backer, 1989) were used to analyze and obtain perceptual data. The data from each expert listener are reported individually.

Videostroboscopic Data. The variables assessed for the study from the laryngostroboscopic examinations included glottal incompetence and two supraglottal hyperadduction variables—anterior/posterior compression and false fold movement—seen under five observation conditions. These
TABLE 1. Means and standard deviations (in parentheses) for the speech variables sound pressure level (SPL), maximum duration of sustained vowel phonation, mean fundamental frequency, fundamental frequency variability (STSD), and EGGW50. Means are listed for the participant for the three pretreatment sessions, for the three pretreatment sessions combined (mean pre), for the two posttreatment sessions, and the two posttreatment sessions combined (mean post).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre 1 (SD)</th>
<th>Pre 2 (SD)</th>
<th>Pre 3 (SD)</th>
<th>Mean Pre (SD)</th>
<th>Post 1 (SD)</th>
<th>Post 2 (SD)</th>
<th>Mean Post (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SPL (dB) 30 cm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sustained vowel</td>
<td>78.80 (0.95)</td>
<td>76.50 (1.70)</td>
<td>80.90 (0.58)</td>
<td>78.46 (3.13)</td>
<td>86.30 (0.82)</td>
<td>87.80 (0.38)</td>
<td>87.08 (2.62)</td>
</tr>
<tr>
<td>Rainbow</td>
<td>73.50 (2.68)</td>
<td>71.15 (2.54)</td>
<td>74.11 (2.78)</td>
<td>72.94 (2.84)</td>
<td>87.30 (2.83)</td>
<td>84.68 (2.37)</td>
<td>84.62 (2.43)</td>
</tr>
<tr>
<td>Monologue</td>
<td>72.50 (2.42)</td>
<td>68.56 (3.35)</td>
<td>71.94 (2.76)</td>
<td>70.99 (3.44)</td>
<td>86.00 (2.34)</td>
<td>81.50 (3.14)</td>
<td>83.30 (3.51)</td>
</tr>
<tr>
<td>Max duration (seconds)</td>
<td>34.55 (3.06)</td>
<td>35.75 (3.21)</td>
<td>31.15 (7.01)</td>
<td>34.15 (4.46)</td>
<td>32.97 (3.26)</td>
<td>34.11 (3.05)</td>
<td>33.50 (3.1)</td>
</tr>
<tr>
<td>Mean F0 (hertz)</td>
<td>92.09</td>
<td>86.68</td>
<td>86.75</td>
<td>88.51 (3.10)</td>
<td>123.45</td>
<td>118.54</td>
<td>122.47 (5.55)</td>
</tr>
<tr>
<td>STSD</td>
<td>2.17</td>
<td>2.29</td>
<td>2.59</td>
<td>2.35 (0.22)</td>
<td>3.59</td>
<td>3.56</td>
<td>3.58 (0.02)</td>
</tr>
<tr>
<td>EGGW50</td>
<td>0.671 (0.020)</td>
<td>0.629 (0.010)</td>
<td>0.684 (0.020)</td>
<td>0.650 (0.18)</td>
<td>0.671 (0.010)</td>
<td>0.688 (0.020)</td>
<td>0.673 (0.014)</td>
</tr>
</tbody>
</table>

For false fold movement, 1 = no false fold overclosure, laryngeal ventricles easily seen, 2 = mild, one or both false folds obscure laryngeal ventricles, 3 = moderate, one or both false folds obscure ventricles and a portion of the true folds, 4 = severe, true folds barely visible, and 5 = extreme, false folds touching and covering entire glottis and may interfere with glottal vibration.

Reliability
For the variables SPL, maximum duration of sustained vowel phonation, and fundamental frequency and its variability, intraexaminer measurement reliability using Pearson product correlation coefficients ranged between 0.99 and 1.0. Interexaminer reliability for the SPL measurement method has been shown reliable (0.93–0.99 [Fox & Ramig, 1997]). For the expert listener, perceptual rating task intralistener reliability ranged between 0.92 and 0.97. For the two videolaryngostroboscopy raters, intrarater reliability was 0.79 and 0.90. Interlistener and interrater reliability are not reported for the perceptual listening and videostroboscopy rating tasks because the results from each rater are reported and discussed individually.

Statistical Design
To evaluate pre- to postspeech treatment changes in the measures of SPL, fundamental frequency and its variability, and maximum duration, a comparison of the means of the participant’s results were made (Barlow & Hersen, 1984; Kratochwill & Levin, 1992; McReynolds & Kearns, 1983). Differences in the pre- to posttreatment means were considered noteworthy if the mean of the two posttreatment sessions exceeded the mean of the three pretreatment sessions, where applicable, by ±1 standard deviation. This criterion is considered meaningful and acceptable and is analogous to a statistical measurement of a large size effect (Cohen, 1988). The expert listener and videolaryngostroboscopic perceptual ratings are reported separately for each rater.

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Results

Sound Pressure Level

As shown in Table 1, the participant improved on measures of SPL during sustained vowel phonation, reading, and the 30-second monologue pre- to posttreatment. The participant increased mean SPL pre- to posttreatment 8.32 dB for sustained phonation, 13.09 dB for reading, and 12.75 dB for conversational monologue. The posttreatment increases in SPL exceeded the pretreatment means by 3 SD for sustained phonation, 4 SD for reading, and 3.5 SD for monologue.

Maximum Duration of Sustained Vowel Phonation (seconds)

Maximum duration of sustained vowel phonation remained relatively stable pre- to posttreatment (Table 1). The posttreatment means were within .15 SD of the pretreatment means.

Mean Fundamental Frequency

The participant increased mean fundamental frequency pre- to posttreatment by 30.95 Hz during reading and 27.50 Hz during monologue (Table 1). These posttreatment means exceeded pretreatment means by 7 and 8 SD for reading and monologue, respectively.

Fundamental Frequency Variability (std)

The participant increased semitone standard deviation (std) pre- to posttreatment by 1.23 std during reading and 0.57 std during monologue (Table 1). These posttreatment means exceeded pretreatment means by 5.5 and 2 SD for reading and monologue, respectively.

EGGW50

As shown in Table 1, the EGGW50 data remained stable pre- to posttreatment. These posttreatment means were within .1 SD of pretreatment means. This stability suggests no noticeable decrease or increase of true vocal fold adduction pre- to posttreatment during sustained vowel phonation. Pre- and posttreatment, these values are within the high range of normal phonation (i.e., not characteristic of a breathy or pressed voice) for this measure (Scherer et al., 1995).

Perceptual Measurements

For the variable never “hoarse or rough,” Raters 1 and 2 noted a positive change of 6 (from 85% to 91%) and 25 (from 68% to 93%) percentage points, respectively, pre- to posttreatment. Rater 3 indicated relatively no change (-1 percentage point [from 97% to 96%]) pre- to posttreatment for this variable. For the variable always “strong,” Raters 1, 2, and 3 noted a positive change of 15 (from 73% to 78%), 36 (from 64% to 100%), and 2 (from 95% to 97%) percentage points, respectively, pre- to posttreatment. The participant was rated never “strained” 99% and 100% of the time both pre- and posttreatment by Raters 1 and 2, respectively. Rater 3 entered a change of -6 percentage points (from 98% to 92%) for the participant pre- to posttreatment for this variable.

Laryngostroboscopic Measurements

Flexible and Rigid Scope. All laryngostroboscopic ratings for the participant are summarized in Table 2. Although some differences can be seen across raters, changes from pre- to posttreatment were always in one direction, becoming less severe following treatment. The most significant improvements were observed for anterior/posterior compression and false fold movement. These changes were noted virtually exclusively using the flexible scope.

Discussion

The purpose of this study was to evaluate the effect of the LSVT on an individual with idiopathic Parkinson

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rater 1 Pre</th>
<th>Rater 1 Post</th>
<th>Rater 2 Pre</th>
<th>Rater 2 Post</th>
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<tr>
<td>Glottal Incompetence&lt;sup&gt;a&lt;/sup&gt;</td>
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<tr>
<td>Flexible Scope</td>
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<tr>
<td>Loud phonation</td>
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<td>Soft phonation</td>
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<td>1</td>
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<td>1</td>
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</tr>
<tr>
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<td>n/a</td>
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<td>Anterior/Posterior Compression</td>
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<td>2</td>
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</tr>
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<td>n/a</td>
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<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Loud phonation</td>
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<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Soft phonation</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
</tr>
</tbody>
</table>

<sup>a</sup>Ratings for flexible and rigid scope: 1 = none, 2 = mild, 3 = moderate, 4 = severe, 5 = extreme

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disease (IPD) who had soft, hoarse, monotone voice, moderate to extreme supraglottic hyperadduction, and mild true vocal fold hypoadduction. It was hypothesized that the Lee Silverman Voice Treatment (LSVT) program, which focuses on improving true vocal fold adduction and maximizing phonatory efficiency, would improve the participant’s true vocal fold functioning, decrease his supraglottic hyperadduction, and have positive effects on his soft, hoarse, monotone voice. The findings reported here suggest that the participant responded positively to the LSVT in all of these domains.

After treatment, the participant exhibited higher SPL levels during sustained phonation as well as greater SPL and fundamental frequency and fundamental frequency variability (std) during reading and monologue. These findings are consistent with previous reports of LSVT-treatment related changes in IPD (Dromey et al., 1995; Ramig & Dromey, 1996; Ramig et al., 1995, 1996). In addition, the expert listeners and raters indicated improved voice quality and true vocal fold adduction without straining as well as reduction in supraglottal hyperadduction posttreatment. Although SPL in sustained phonation increased posttreatment and the raters noted no true vocal fold hypoadduction posttreatment, little change was observed in measures of maximum duration of sustained vowel phonation and EGGW50 (measure of adduction) posttreatment. These last two findings are inconsistent with previous post-LSVT reports (Brosovic, 1994; Ramig et al., 1995, 1996). It may be that the supraglottal hyperadduction occurring pretreatment in this participant enabled him to achieve a level of vocal fold adduction that was similar to posttreatment levels. However, it can be postulated that posttreatment adduction levels were achieved through a more efficient manner (greater respiratory support and vocal fold vibratory efficiency) than pretreatment.

Normally, the mechanism responsible for posttreatment increases in SPL and fundamental frequency and its variability in an individual with IPD is attributed to improved true vocal fold adduction and respiratory support (Dromey et al., 1995; Ramig & Dromey, 1996; Smith, Ramig, Dromey, et al., 1995). We suggest that through training optimal vocal fold adduction (i.e., adduction with adequate respiratory support, loudness, and quality), the participant increased his SPL and eliminated the need for secondary compensatory supraglottal hyperadduction posttreatment. Reducing supraglottal hyperadduction or constriction in the vocal tract can increase SPL by alleviating a damping effect on the acoustic signal (Sundberg & Gauffin, 1979). Enlarging the vocal tract will increase SPL as well as improve overall vocal quality. This mechanism together with probable increases in subglottal air pressure are likely responsible for increased SPL posttreatment in this participant. Although subglottal air pressure was not measured here, it has been reported to increase following a course of the LSVT in a group of individuals with IPD (Ramig & Dromey, 1996). The change in SPL suggests the participant improved vibratory efficiency and true vocal fold adduction, reduced constriction in the supraglottal area, and learned to manage his respiratory support at the level of the larynx more effectively posttreatment.

The conflicting pretreatment observations of supraglottal hyperadduction between the rigid and flexible scope views must be addressed. A moderate to extreme level of supraglottal hyperadduction was observed pretreatment only during the flexible scope view by both raters. Only Rater 2 noted a mild level of false fold movement during loud phonation pretreatment during the rigid scope view. We believe this can be attributed to the physical positioning that occurs during the rigid scope exam. It has been well documented that differences exist between flexible and rigid scope views. Smith, Ramig, Dromey, et al. (1995) and Södersten and Lindestad (1992) report respectively more glottal incompetence in individuals with IPD and normals during the rigid scope exam. These differences have been associated with the neck extension, laryngeal elevation, tongue protrusion, and elevated pitch that often occur during the rigid exam (Shaik & Bless, 1986; Södersten & Lindestad, 1992; Smith, Ramig, Dromey, et al., 1995). If the supraglottal hyperadduction observed in this participant was compensatory in nature, the positioning during the rigid scope exam may have prevented the participant from achieving the same degree of supraglottic compensatory behavior that was observed during the flexible scope exam. It has been suggested (Shaik & Bless, 1986; Södersten & Lindestad, 1992; Smith, Ramig, Dromey, et al., 1995) that the flexible scope view is closer to natural speech positioning and more representative of speaking behavior than the rigid view. Thus we feel confident that the level of supraglottal hyperadduction occurring during the flexible exam pretreatment was representative of this participant’s normal speaking behavior.

Another curious observation in this participant is the inconsistent report of glottal incompetence pretreatment. Only Rater 1 during soft phonation rated glottal incompetence. In addition, although it has been reported that glottal incompetence appears more often during rigid scope exam, none was reported for this participant during this exam pretreatment. It is disappointing that soft phonation was not available for comparison pre- to posttreatment for the rigid scope exam. One explanation for lack of incompetence ratings pretreatment may be that it was difficult for the raters to assess it during the flexible exam given the high level of supraglottal hyperadduction. However, glottal incompetence was not observed during normal and loud phonation in the rigid scope exam, when supraglottal hyperadduction was not present. Regardless, given the participant’s increase in SPL, fundamental frequency and its variability, and vocal quality posttreatment, and the reported reduction in supraglottal hyperadduction during flexible scope views, we suggest that the participant did have some deficiency at the level of the glottis pretreatment that improved after treatment.

The changes observed posttreatment in the participant’s supraglottal hyperadduction are not surprising when considering the theory for treating voice disorders accompanying neurological disease suggested by Ramig and Scherer (1992) and Smith and Ramig (1995). The underlying physiological mechanism contributing to the participant’s voice disorder
and supraglottal hyperadduction appears to have been inadequate true vocal fold closure or efficiency. By focusing on this primary deficit rather than the resulting secondary compensatory behavior, overall speech and voice improvement was achieved in a simple and efficient manner. The simple focus of the adduction treatment program eliminated true vocal fold hypoadduction and supraglottal hyperadduction and increased loudness, intonation, and improved vocal quality in this participant. These changes were supported by the objective and perceptual measures as well as the participant’s report that the treatment increased his ability to communicate effectively with coworkers, clients, family, and friends.

A more traditional approach to treating this individual may have been to eliminate the supraglottal hyperadduction through relaxation techniques and then later focus on the decreased loudness and intonation and hoarse voice. We believe this more traditional approach to treatment would not have been as effective or as efficient given the probable origin of the supraglottal hyperadduction and reported difficulty individuals with Parkinson disease have with complex instructions and material. These findings are consistent with past reports that treatment designed to restore true vocal fold adduction or repair true vocal fold pathology may reduce supraglottal hyperadduction (Feinstein et al., 1987; Smith, Ramig, Dromey, et al., 1995; Von Doersten et al., 1992; Von Hake et al., 1989).

It should be noted, however, that the changes observed in this individual with Parkinson disease may not be indicative of all individuals with PD presenting with supraglottal hyperadduction. We advocate treating these individuals on a case-by-case basis. Stimulability testing is recommended during pretreatment screening to determine if the individual is capable of producing a louder voice easily and without straining. If straining is noted during the screening or extreme cases of supraglottal hyperadduction are observed, a trial period of the LSVT is recommended to determine appropriateness of the individual for the treatment.

This study demonstrates the use of a continuum of measures ranging from functional ratings to physiological measures to evaluate the impact of intensive voice treatment and identify mechanisms underlying treatment-related change in an individual with Parkinson disease. Some of the measures used here (SPL, fundamental frequency, maximum duration, expert listener ratings, and videolaryngostroboscopy) are available to the practicing clinician and are important for documentation of treatment-related change and reimbursement. Physiological measures such as the EGGW50 provide a better understanding of the underlying mechanism contributing to treatment-related changes. A wide spectrum of measures may allow a comprehensive and accurate picture of treatment-related change spanning functional impact to physiological bases. Research is needed to determine whether certain simple and cost-effective measures are valid indicators of broad-based changes in voice and underlying physiology due to treatment.

In conclusion, the case presented here is an initial step toward increasing professionals’ knowledge of the origin and treatment of supraglottal hyperadduction in individuals with idiopathic Parkinson disease. The study demonstrates that treatment designed to improve vocal fold adduction and vibratory efficiency can enhance the speech and voice in an individual with PD with mild vocal fold hypoadduction and moderate to extreme supraglottal hyperadduction pretreatment. Clearly, the findings are limited to one case of supraglottal hyperadduction in Parkinson disease. Further research on this topic is needed to generalize treatment effects on supraglottal hyperadduction in these individuals, which would result in effective speech treatment for a wider variety of individuals with IPD.

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