Aerodynamic Mechanisms Underlying Treatment-Related Changes in Vocal Intensity in Patients With Parkinson Disease

The purpose of this study was to document changes in aerodynamic and glottographic aspects of vocal function in patients with Parkinson disease who received two forms of high effort treatment. Previous reports (Ramig, Countryman, Thompson, & Hori, 1995) have documented increased sound pressure level (SPL) following treatment that trained phonation and respiration (Lee Silverman Voice Treatment: LSVT), but not for treatment that trained respiration only (R). In order to examine the mechanisms underlying these differences, measures of maximum flow declination rate (MFDR) and estimated subglottal pressure (Psub) were made before and after treatment. A measure of relative vocal fold adduction (EGGW) was made from the electroglosstographic signal during sustained vowel phonation. Sound pressure level data from syllable repetition, sustained vowel phonation, reading, and monologue tasks were also analyzed to allow a more detailed understanding of treatment-related change in several contexts. Consistent with increases in SPL, significant increases in MFDR, estimated Psub, and EGGW were measured posttreatment in patients who received the LSVT. Similar changes were not observed following R treatment. These findings suggest that the combination of increased vocal fold adduction and subglottal pressure is a key in generating posttreatment increases in vocal intensity in idiopathic Parkinson disease (IPD).

KEY WORDS: Parkinson disease, treatment efficacy, aerodynamic, phonation, voice treatment

The voices of patients with Parkinson disease are characterized by low vocal intensity, monotonicity, and hoarseness (Aronson, 1990; Logemann, Fisher, Boshes, & Blonsky, 1978), which may contribute to a reduction in speech intelligibility (Ramig, 1992; Maclay, 1992). These characteristics have been associated with reduced vocal fold adduction (Hanson, Gerratt, & Ward, 1984; Perez, Ramig, Smith, & Dromey, in press; Smith, Ramig, Dromey, Perez, & Samandari, 1995) and impaired respiratory function (Critchley, 1981; Murdoch, Chenery, & Bowler, 1989; Solomon & Hixon, 1993). Recently, posttreatment increases in vocal intensity (measured as the sound pressure level of a subject's speech) and fundamental frequency variability have been reported in patients with Parkinson disease following one of two forms of intensive speech treatment (Ramig, Countryman, Thompson, & Hori, 1995). One type of treatment focused on increasing inspiratory and expiratory volumes for speech in order to increase subglottal air pressure, since greater passive recoil forces at high lung volumes can contribute to pressure increases (Hixon, 1973). This approach was termed the respiratory effort treatment (R). The other treatment focused on increasing both respiratory effort and vocal fold adduction and is known as the Lee Silverman Voice Treatment (LSVT). Posttreatment findings documented clinically...
and statistically significant increases in sound pressure level primarily for the subjects who received treatment designed to increase vocal fold adduction as well as respiratory volumes (Ramig et al., 1995). For example, the LSVT subjects increased on average 12 dB for sustained vowels, 6 dB for a reading passage, and 4 dB for a monologue task. On the other hand, subjects who received the respiratory treatment alone decreased on average 2 dB for sustained vowel phonation and increased 2 dB for reading and 1 dB for the monologue task (Ramig et al., 1995).

It was speculated that the combination of increased subglottal air pressure and vocal fold adduction was necessary to optimize the aerodynamic mechanism of intensity control in patients with Parkinson disease. This speculation was based on reports of vocal intensity control described in healthy subjects (Gauffin & Sundberg, 1989; Titze & Sundberg, 1992). For healthy speakers, increases in vocal intensity have been associated with increases in respiratory volumes (Hixon, 1973; Russell & Stathopoulos, 1988), subglottal air pressure (Isshiki, 1964), and vocal fold adduction (Scherer, 1991; Sundberg, Titze, & Scherer, 1993).

The purpose of this study was to evaluate the aerodynamic and adductory changes associated with intensity control in patients with Parkinson disease following one of two forms of intensive speech treatment. It was hypothesized that subjects whose treatment focused on increased vocal fold adduction and respiratory volumes would show changes in aerodynamic measures that would correspond to mechanisms of intensity change in healthy speakers (Dromey, Ramig, & Johnson, 1995). It was hypothesized that those subjects whose treatment emphasized increased respiratory volumes would have limited changes in aerodynamic and glottographic measures accompanying any SPL changes.

TABLE 1. Selected mean (standard deviation in parentheses), minimum and maximum measures on demographic and speech variables, as well as treatment-related changes in SPL, for the subjects in the present study and for the larger group (Ramig et al., 1995) of which they were a subset.

<table>
<thead>
<tr>
<th>Variable</th>
<th>LSVT subset mean (SD)</th>
<th>Range min/max</th>
<th>LSVT main group7</th>
<th>Range min/max</th>
<th>Respiration subset</th>
<th>Range min/max</th>
<th>Respiration main group7</th>
<th>Range min/max</th>
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<td>7</td>
<td>19</td>
<td></td>
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<td></td>
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<td>Age</td>
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<td>49/79</td>
<td>63.5 (11.5)</td>
<td>32/79</td>
<td>63.7 (7.7)</td>
<td>52/71</td>
<td>65.6 (8.9)</td>
<td>51/83</td>
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<tr>
<td>PD Stage</td>
<td>2.4 (5.2)</td>
<td>2/3</td>
<td>2.7 (6.8)</td>
<td>1.5/4</td>
<td>2.4 (6.5)</td>
<td>1/3.5</td>
<td>2.3 (2.6)</td>
<td>1/3.5</td>
</tr>
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<td>Years since Dx</td>
<td>5.3 (5.5)</td>
<td>0/18</td>
<td>7.0 (5.9)</td>
<td>0/20</td>
<td>6.6 (6.2)</td>
<td>1/18</td>
<td>5.6 (4.8)</td>
<td>0/19</td>
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<tr>
<td>UPDRS²</td>
<td>22.9 (11.1)</td>
<td>4/40.5</td>
<td>26.4 (13.4)</td>
<td>1.5/48</td>
<td>28.2 (13.6)</td>
<td>3/43</td>
<td>25.6 (14.4)</td>
<td>1/43</td>
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<tr>
<td>Depression³</td>
<td>6.9 (5.9)</td>
<td>1/20</td>
<td>9.0 (5.6)</td>
<td>0/20</td>
<td>10.6 (6.1)</td>
<td>1/22</td>
<td>9.9 (6.3)</td>
<td>0/25</td>
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<td>Speech severity⁴</td>
<td>2.3 (8.2)</td>
<td>1/3</td>
<td>2.6 (1.0)</td>
<td>1/5</td>
<td>2.7 (1.3)</td>
<td>1/4</td>
<td>2.7 (1.1)</td>
<td>1/4</td>
</tr>
<tr>
<td>/a/ SPL</td>
<td>67.7 (4.2)</td>
<td>61/76.0</td>
<td>68.9 (4.7)</td>
<td>61/76.7</td>
<td>67.6 (4.0)</td>
<td>62/72.1</td>
<td>69.3 (5.1)</td>
<td>61/75.9</td>
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<tr>
<td>Reading SPL⁵</td>
<td>67.5 (3.4)</td>
<td>60.5/72.4</td>
<td>66.3 (4.0)</td>
<td>59.4/76.0</td>
<td>65.0 (1.9)</td>
<td>62/67.6</td>
<td>65.7 (2.7)</td>
<td>61/70.0</td>
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<tr>
<td>Pre/post /a/ SPL difference</td>
<td>14.1 (4.3)</td>
<td>7/21.7</td>
<td>13.2 (5.3)</td>
<td>4.6/22.1</td>
<td>-2.3 (3.6)</td>
<td>-7.6/21.1</td>
<td>-1.3 (5.3)</td>
<td>-11.7/10.3</td>
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<tr>
<td>Pre/post reading SPL difference</td>
<td>6.7 (4.0)</td>
<td>1.3/12.6</td>
<td>8.0 (5.9)</td>
<td>0.4/22.5</td>
<td>1.9 (2.3)</td>
<td>-1.4/4.5</td>
<td>2.5 (3.4)</td>
<td>-1.4/12.5</td>
</tr>
</tbody>
</table>

1Hoehn and Yahr (1967)  
2Motor exam score  
3Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961)  
4Perceptual rating of impairment: 1 = mild, 5 = severe  
5dB SPL at 50 cm; mean of 6 vowels  
6dB SPL at 50 cm; mean over the entire Rainbow Passage  
7Ramig, Countryman, Thompson, and Hori (1995)  

Subjects in the present study were numbered 3, 6, 7, 9, 12, 15, 17, 18, 20, 22, 27, 35, 36, 38, 39, 43, 45 in the Ramig et al. (1995) study.
treatment characteristics of the two groups reported in the present study, who were a subset of the randomly assigned participants in the larger study of 45 subjects. Of those who remained after the removal of subjects with unusable aerodynamic data, 10 were from the LSVT group and 7 from the R group. There was one female in each treatment group. A one-way analysis of variance revealed no pretreatment differences between these two treatment groups prior to the study on the variables of age, $F(1,15) = .0476, p = .830$, disease stage, $F(1,15) = .0168, p = .899$, score on the motor section of the UPDRS, $F(1,14) = .7282, p = .408$, time since diagnosis, $F(1,15) = .1982, p = .663$, depression, $F(1,15) = 1.7787, p = .202$, or severity of speech disorder, $F(1,15) = .8827, p = .422$. Additionally, there were no pretreatment differences between these groups in vocal intensity for sustained vowel phonation, $F(1,15) = .0078, p = .931$, reading, $F(1,15) = 2.882, p = .110$, or monologue, $F(1,11) = .3701, p = .555$.

**Instrumentation**

A sound level meter (Bruel and Kjaer Type 2230) was positioned at 50 cm and a head-mounted microphone (AKG C410) at 8 cm from the subject’s lips. A Synchrovoice Research Electroglossograph (EGG) was used to obtain the electroglossographic signal. A Rothenberg circumferentially vented pneumotachograph mask (Glottal Enterprises MS 100-A2 with transducer PTW-1) was used to collect the oral air flow signal. An intraoral pressure tube leading to a pressure transducer (Glottal Enterprises PTW-1) mounted on the pneumotachograph mask rested in the center of the oral cavity to allow the estimation of subglottal air pressure during /p/ closure (Smithner & Nixon, 1981). The orientation of the tube was perpendicular to the flow of air. The output of the pressure transducer was monitored on an oscilloscope to ensure proper task performance through monitoring of the shape of the pressure peak.

The pneumotachograph transducer was calibrated against a flow meter across a range of values that exceeded the range of flows produced by subjects in the study. The pressure transducer was similarly calibrated across a range of pressures using a U-tube water manometer and syringe system. Vital capacity was measured using a Collins wet spirometer (Model 2785).

The analog sound pressure level, acoustic, EGG, air flow, and oral pressure signals were stored on a Sony PC-108M 8-channel digital audiotape (DAT) recorder, which allowed signals up to 5 kHz bandwidth to be reproduced. In addition, speech acoustic and EGG signals were recorded on a Panasonic SV 3700 2-channel DAT recorder, which allowed higher bandwidth storage because of its 44 kHz sampling rate. Signals played back from the recorders were digitized into computer files using a 16-bit Digital Sound Corporation A/D converter to a VAX 4000/200 computer.

**Procedure**

Experimental data were collected during the week preceding treatment and during the week following treatment. The following procedure was carried out for each data collection session. The subject was seated in a medical examining chair in an IAC sound-treated booth. To limit extraneous movement, the subject’s arms and legs were secured to arm and foot rests using three-inch wide Velcro bands. After 2 minutes of tidal breathing, forced vital capacity (FVC) was measured. The subject was asked to take his or her deepest breath and blow out the air into a Collins wet spirometer “as hard and fast and long as you can.” This task was repeated three times at the beginning of the session and twice at the end. The best performance was taken as the FVC. Care was taken to ensure that the subject’s lips were tightly sealed on the mouthpiece; to prevent any nasal air flow, nose clips were used for this procedure.

For the collection of air flow and intraoral air pressure data, the subject repeated a series of seven /pae/ syllables with the Rothenberg mask held firmly in place by the experimenter and the air pressure tube in the middle of the oral cavity. Syllables were produced at normal pitch and loudness and flat intonation at a rate of 1.5 syllables per second, as modeled by the experimenter (LR). The /pae/ syllable was selected because the distance between the fundamental frequency and first formant of the vowel facilitates the inverse filtering procedure (Rothenberg, 1973). When F0 and F1 are closely spaced, it can be difficult to adequately filter out the effects of vocal tract resonances in order to obtain a glottal flow waveform (Rothenberg, 1977).

During the data collection session, subjects also read the Rainbow Passage and produced a 20–30 second monologue, from which SPL and fundamental frequency data were extracted (Ramig et al., 1995).

**Treatment**

The subjects participated in one of two types of treatment. Both forms of treatment were designed to be intensive (16 1-hour sessions within 4 weeks) and high effort. Subjects were strongly encouraged to employ maximum effort in all treatment tasks. The first half of each session consisted of high effort drills, whereas the second half was used to carry over the increased effort to speech tasks. The implementation of high effort, intensive treatment is based on neurology and physical therapy practices (England & Schwab, 1959; Hallet & Khosbin, 1980; McDowell, Lee, & Sweet, 1986) that suggest when pushed to higher effort levels, patients with Parkinson disease can compensate for bradykinesia and improve task performance. The daily training of high effort to increase magnitude of speech motor output is based upon the potentially similar problems in scaling the magnitude of output (e.g., stride length in walking, letter stroke in writing) observed in patients with Parkinson disease (Brooks, 1986; Muller & Stelmach, 1991; Stelmach, 1991). We speculate that intensive high effort speech treatment teaches patients to rescale the magnitude of speech motor output (Ramig, 1995; Ramig, Bonitati, Lemke, & Horii, 1994) and that daily treatment with intensive practice (Schmidt, 1975, 1982) and feedback (knowledge of results) (Adams, 1971, 1986) facilitates this training.

The respiratory (R) treatment was designed to increase the activity of the respiratory musculature in order to generate

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increased volumes and subglottal air pressure for speech (Netsell & Daniel, 1979; Yorkston, Beukelman, & Bell, 1988). Treatment tasks included those designed to increase ventilation as well as train expiratory muscles (Leith & Bradley, 1976; McKenzie & Gandevia, 1986): maximum inhalation and exhalation (Hardy, 1983; Netsell & Rosenbek, 1986), maximum duration of voicelessness fricatives (/s/ and /f/), maximum duration of counting on one breath and sustaining oral air pressure for as long as possible using the IOP (Iowa Oral Performance Instrument; Robin, Goel, Somodi, & Loschei, 1992) both with and without the leak tube. This device, which was originally designed to measure tongue strength, had been modified to allow subjects to view the level of air pressure they were producing during treatment tasks. During the second half of each treatment session, subjects were encouraged to take frequent, deep breaths during speech (e.g., “think breathe”) and to use increased respiratory volumes throughout a hierarchy of speech tasks. Subjects were given visual feedback (NIMS Respiragraph PN SY03) concerning chest wall movements during the maximum sustained fricative productions, reading and speaking tasks. The visual feedback allowed the subjects to view the relative magnitude of their respiratory maneuvers as they implemented the treatment techniques. The impact of the respiratory effort treatment on maximum duration vowel phonation and utterance and pause duration during reading and speaking have been reported in a study by Ramig, Countryman, Thompson, and Horii (1995). The Lee Silverman Voice Treatment (LSVT) targeted increased vocal intensity through improved vocal fold adduction and increased respiratory effort (Ramig, Pawlas, & Countryman, 1995). Treatment drills included tasks to increase respiratory/pharyngeal effort and coordination: maximum duration sustained vowel phonation and maximum fundamental frequency range. The tasks that were used to increase vocal fold adduction included stimulation to generate “loud” phonation or pushing the hands together, or pulling or pushing on the arms of a chair while phonating a sustained vowel (Aronson, 1990; Froeschels, Kastein, & Weiss, 1955). Feedback regarding intensity was provided with a voice light (a device that illuminates more LEDs as intensity increases) and a tape recorder. Subjects were encouraged to “think loud” and to use increased vocal intensity in a hierarchy of speech tasks during the second half of each treatment session. The impact of the LSVT on vocal intensity and fundamental frequency, as well as several neuropsychological variables, has been reported in a study by Ramig et al. (1995). Changes in pre- to posttreatment videolaryngostroboscopic ratings have been reported previously by Smith et al. (1995).

It is important to point out that the treatment goal of increasing vocal fold adduction in Parkinson disease is designed to maximize the efficiency of the phonatory source. It is never the goal of treatment to increase vocal fold adduction so that the voice becomes pressed or hyperadducted. The goal is a voice with sufficient loudness, generated with maximum phonatory efficiency (Ramig, 1995).

The treatment intensiveness, daily homework, daily quantification of treatment variables and carryover were all stimulated equally in both treatment groups. No direct attention was given to improving intonation, articulation, or rate in either treatment group. Details of the daily treatment sessions, patient progress, and clinician training have been summarized in Ramig et al. (1995), and Ramig, Pawlas, and Countryman (1995).

Data analysis

From the /pae/ syllable train task, simultaneous recordings of air flow, intraoral air pressure, and SPL were lowpass filtered at 10 kHz and digitized at a sampling rate of 20 kHz to allow detailed waveform preservation for the analysis of maximum flow declination rate. The signals were subsequently analyzed with custom software that interpolated between the pressure peaks during /p/ closure to allow the estimation of mid-vowel subglottal pressure (Smitherton & Hixon, 1981). Flow and SPL values for their respective time-aligned channels were also obtained in this way, so that values for these measures represented the temporal midpoint of the vowel in each syllable. These analyses were performed on a VAX 4000/200 computer, using the middle three syllables from each of three syllable trains from each recording session. Thus, nine tokens contributed to the mean for each subject on each occasion.

Maximum flow declination rate (MFDR), which reflects the interaction of subglottal air pressure and vocal fold adduction and is an index of the speed of glottal flow “shut-off,” correlates highly with vocal intensity (Holmberg, Hillman, & Perkell, 1988; Titze & Sundberg, 1992). To measure maximum flow declination rate, the air flow signal was transferred to a 486 PC and was inverse filtered with CSpeech 4.0. The maximum flow declination rate was measured as the magnitude of the downgoing peak from the derivative of the glottal flow signal (see Figure 1a) for 10 successive cycles at the vowel midpoint. Again, nine tokens were examined to derive a mean value for each subject on each recording date.

Open quotient (OQ), defined as the time during which the vocal folds are open, divided by the period of the vibratory cycle, decreases as intensity increases (Dromey, Stathopoulos, & Sapienza, 1992; Timcke, von Leden, & Moore, 1958) in healthy speakers. Open quotient was measured using a custom software program (Stathopoulos & Sapienza, 1993a, 1993b) at a 20% AC flow criterion level. Thus, the time of glottal opening was operationally defined as the point at which the inverse-filtered flow reached 20% of the height of the waveform between the minimum flow offset (DC offset) and the peak flow. The beginning of the closed period was defined as the time at which the flow again decreased to the 20% level (see Figure 1b). Between 30 and 50 consecutive cycles were measured from the glottal flow waveform at the vowel midpoint during the /pae/ task. The mean values from three vowels from each of three trials for each subject for each session were calculated.

EGGW-25, which is calculated as the relative width of the electrolaryngographic (EGG) waveform at 25% of its height (Scherer & Vail, 1988; Scherer, Vail, & Rockwell, 1995) has been found to increase with vocal fold adduction (Brosovic, 1994). Ten consecutive cycles of the electrolaryngographic (EGG) signal from the temporal midpoint from each of six
maximum sustained vowel productions for each subject on each date were analyzed. This analysis was performed with an in-house software program on a VAX 4000/200 computer following digitization of the two-channel DAT recording at 20 kHz. Figure 1c shows how this measure was derived.

SPL analysis was performed for six vowels and one reading passage for each subject on each recording date. The analog output of the sound level meter, which had been stored in digital format on the eight-channel DAT recorder, was redigitized at 1 kHz into computer files. The mean SPL was calculated using a custom software program that allowed an SPL baseline to be set by the operator. By setting the baseline at an intensity corresponding to the lowest level during a phrase, intensity points falling below this level during inhalation were excluded from analysis, in order to measure the mean SPL during speech, rather than during speech and pauses combined.

Measurement Reliability

Twenty percent of the data on each measure were reanalyzed after several months by the same investigator to assess measurement reliability. A paired t-test revealed no significant differences between original and repeated analyses. Pearson correlation coefficients ranged from .995 to 1.0 between the original and the reanalyzed data. On the measures of estimated subglottal pressure and mid-vowel SPL, there was no measurement error, since these variables were automatically extracted from the waveforms. Mean measurement errors on the other variables were in the order of 0.5%.

To assess test-retest intrasubject reliability, 9 (53%) of the subjects had a second pretreatment recording session and 5 (29%) of the subjects had a second posttreatment recording. Paired t-tests revealed that there were no significant differences between pretreatment recording sessions for any variables except the measure of forced vital capacity, which was smaller (0.12 L) for the second pretreatment recording, t(9) = 2.45, p = .037, than for the first. There were no differences on any measures between the first and second posttreatment recordings.

Results

Means and standard deviations for each measure are presented in Table 2. A two-factor time (pre-to-post) by treatment group (LSVT vs. R) repeated measures analysis of variance (ANOVA) was performed on each of the dependent measures to examine changes that occurred following treatment, as well as differences in these pre/post changes according to the type of treatment that was given. The F-ratios and p-values for these ANOVAs are reported in Table 3.

SPL

For sustained vowel phonation, there was a significant pre/post by treatment group interaction (p < .001). The LSVT subjects increased on average by 14 dB (SD 4.3). The R group subjects decreased 2.3 dB (SD 3.5). For the mid-vowel intensity in the syllable repetition task there was a significant pre/post by treatment group interaction of (p = .042). The LSVT subjects increased on average 6.5 dB (SD 7.4) and the R subjects decreased 0.1 dB (SD 2.8) during the syllable repetition task (see Figure 2a) from pre- to post-treatment. For the reading passage, there was a significant pre/post by treatment group interaction (p = .013). The mean increase for the LSVT subjects was 6.7 dB (SD 4.2) and for R group it was 1.9 dB (SD 2.3). In the monologue task, the pre/post main effect was significant (p = .005), but there was not a significant interaction with treatment group at the p < .05 level. Nevertheless, there was a tendency for

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FIGURE 1. a = Measurement of maximum flow declination rate (MFDR) from the derivative of the glottal flow signal. MFDR represents the most rapid decrease in flow during vocal fold closure; b = Measurement of open quotient (OQ) at 20% of the height of the glottal flow pulse. OQ = open duration/open + closed durations; c = Measurement of EGGW-25—the relative width of the electroglottographic waveform at 25% of its height. EGGW-25 = a/a + b.
TABLE 2. Means (and standard deviations) for each measure before and after treatment as a function of treatment type.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre-Tx</th>
<th>SD</th>
<th>Post-Tx</th>
<th>SD</th>
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<tbody>
<tr>
<td>SPL for /a/</td>
<td>67.7</td>
<td>(4.2)</td>
<td>81.7</td>
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<tr>
<td>SPL for /pae/</td>
<td>68.5</td>
<td>(4.2)</td>
<td>75.0</td>
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<tr>
<td>SPL for reading</td>
<td>67.5</td>
<td>(3.4)</td>
<td>74.2</td>
<td>(3.5)</td>
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<td>SPL for monologue</td>
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<td>(2.9)</td>
<td>70.0</td>
<td>(3.5)</td>
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<td>6.6</td>
<td>(2.5)</td>
<td>8.6</td>
<td>(2.9)</td>
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<td>MFDR</td>
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<td>FVC</td>
<td>3.57</td>
<td>(.760)</td>
<td>3.53</td>
<td>(.824)</td>
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SPL measures are dB SPL at 50 cm; mean for 6 vowels, 9 /pae/ syllables or entire passage or monologue.
Paub = estimated subglottal pressure (cmH2O); mean for 9 /pae/ syllables.
MFDR = maximum flow declination rate (L/s/s); mean for 9 /pae/ syllables.
OQ = open quotient using 20% AC flow criterion; mean for 9 /pae/ syllables.
EGGW-25 = EGG pulse width adduction measure using 25% height criterion; mean for 6 vowels.
FVC = forced vital capacity (liters); maximum value of three attempts.

The LSVT subjects to show larger SPL increases (5.5 dB, SD 3.1) than the R group subjects (2.2 dB, SD 3.8).

The treatment-related SPL changes reported for the 17 subjects here are similar to those for the larger group of 45 subjects of which they were a subset (Ramig, Countryman, Thompson, & Horii, 1995—see Table 1).

For estimated subglottal pressure, there was a significant pre/post by treatment group interaction (p = .003). LSVT subjects increased on average 2 cmH2O (SD 1.4), and the R group decreased 0.3 cmH2O (SD 1.3) (see Figure 2b).

There was a significant pre/post by treatment group interaction (p = .001) for maximum flow declination rate. The mean increase for the LSVT subjects was 215 L/s/s (SD 119) and the mean decrease for the R group was 2 L/s/s (SD 103) (see Figure 2c).

The changes in open quotient following treatment were not statistically significant for main or interaction effects. The LSVT group decreased by .042 (SD .056), whereas the R group increased by .014 (SD .086). A lower value on this measure is associated with greater vocal fold adduction.

There was a significant pre/post by treatment group interaction (p = .003) for EGGW-25. The LSVT group increased on average by .073 (SD .050) following treatment, whereas the R group decreased by .035 (SD .063) (see Figure 2d). A higher value on this measure is associated with greater vocal fold adduction (Scherer et al., 1995).

The changes following treatment were not statistically significant for main or interaction effects for forced vital capacity.

Discussion

The present study was undertaken to examine selected aerodynamic and glottographic measures of vocal function in patients with Parkinson disease. A primary objective was to document changes in these measures accompanying two different forms of speech treatment.

Since reduced vocal intensity is one of the primary speech disorders of this patient population and contributes to reductions in speech intelligibility, a treatment program that improves vocal intensity is of prime interest. The data for four speech tasks—syllable repetition, sustained vowel phonation, reading, and monologue—all show that patients receiving the LSVT were able to increase their SPL following treatment. The fact that patients whose treatment focused solely on increased respiratory volumes and subglottal air pressure did not make similar SPL gains suggests that it is necessary to compensate for the documented deficiencies in vocal fold adduction in this population in order to achieve the goal of increased SPL (Ramig, Countryman, Thompson, & Horii, 1995). Although increasing subglottal pressure through respiratory volume improvements leads to increased intensity in the healthy larynx (Stathopoulos &

TABLE 3. F-ratios and p-values for the repeated measures ANOVAs on all variables.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre/post effect</th>
<th>Interaction with treatment type</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>F-ratio</td>
<td>p-value</td>
</tr>
<tr>
<td>SPL for /a/</td>
<td>34.68</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>SPL for /pae/</td>
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<td>.048</td>
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<tr>
<td>SPL for reading</td>
<td>25.97</td>
<td>&lt;.001</td>
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<tr>
<td>SPL for monologue</td>
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<td>.005</td>
</tr>
<tr>
<td>Paub</td>
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<td>.002</td>
</tr>
<tr>
<td>MFDR</td>
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<td>.001</td>
</tr>
<tr>
<td>Open quotient</td>
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<td>.249</td>
</tr>
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<td>EGGW-25</td>
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<td>.095</td>
</tr>
<tr>
<td>FVC</td>
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<td>.215</td>
</tr>
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FIGURE 2. a = Mean and standard deviation sound pressure level (dB SPL at 50 cm) for /pae/ syllable repetition before and after speech treatment; b = Mean and standard deviation estimated subglottal pressure (cmH2O) before and after speech treatment; c = Mean and standard deviation maximum flow declination rate (L/s/s) before and after speech treatment; d = Mean and standard deviation EGGW (25% of AC waveform criterion) before and after speech treatment.

Sapienza, 1993a), greater pulmonary effort alone did not lead to improvements in SPL in the glottally incompetent patients in the present study. The treatment tasks of the LSVT included exercises to increase vocal fold adduction and, as the EGGW data and the videostroboscopic findings (Smith et al., 1995) suggest, posttreatment increases in vocal fold adduction were realized. This adductory improvement allowed the LSVT subjects to increase their vocal intensity. These findings are consistent with data reported by Berke, Hanson, Gerratt, Trapp, Macagba, and Natividad (1990), who found that higher SPL was achieved more efficiently by means of medial adductory compression than by higher air flow levels in an in vivo canine model. The posttreatment SPL for /pae/ syllables in the LSVT group is comparable to values reported by Stathopoulos and Sapienza (1993a) for healthy males at a comfortable intensity level.

It is of particular interest that all subjects receiving respiratory effort treatment did not experience substantial improvements in estimated subglottal pressure; only 3 of the 7 subjects increased subglottal air pressure pre- to post-treatment. Observations of statistically significant post-treatment increases in pause durations during connected speech and increases in maximum duration of sustained vowel phonation (Ramig et al., 1995) both suggest that the R group did implement the target treatment techniques to increase inspiratory volumes prior to speech. The lack of corresponding increases in estimated subglottal pressure and SPL allow the speculation that for individuals with an incompetent glottal valving mechanism, merely increasing respiratory force may not be sufficient to lead to improvements in subglottal pressure for speech. These findings are consistent with Verneuil, Kreiman, Kevorkian, Ye, Gerratt, and Berke (in press), who found that during phonation, subglottal air pressure was primarily dependent upon recurrent laryngeal nerve stimulation and laryngeal muscular contraction rather than lung driving pressure in in vivo canine models. An alternative explanation is that the activities used in treatment to increase respiratory function did not adequately train subglottal pressure or generalize the use of expiratory effort to the syllable repetition task examined in the present study.

It is possible that treatment approaches that focus solely on respiration may even be counter-productive in this population. The decreases in vocal fold adduction reflected in EGGW-25 data, together with previous findings by Smith et al. (1995), suggest that glottal incompetence actually became greater in certain subjects who increased respiratory effort without simultaneously improving vocal fold adduction. Although not demonstrating such a clear trend, the open quotient data also suggest a slight increase in glottal incompetence for the R group, whereas the LSVT group appeared to increase adduction based upon this measure. Berke et al. (1990) found that higher flows in the absence of adductory changes led to increased open quotient in their canine preparation—a similar pattern to that seen in the R treatment group in the present study.

Glottal incompetence may not preclude generation of subglottal air pressure in individuals with a non-neurologically disordered respiratory system. However, for individuals with Parkinson disease, it may be necessary to stimulate...
respiration and phonation simultaneously in order to maximize treatment effectiveness to generate SPL increases.

It might be speculated that even though patients with Parkinson disease in the R group were trained to inhale to higher lung volumes before speaking, they counteracted the unusually high respiratory recoil forces by using inspiratory muscular effort (Hixon, 1973). It is conceivable that the R group subjects might have checked the increases in subglottal pressure to allow speech production to continue in an intensity range to which they were accustomed. This may be similar to the technique employed by singers who have been reported to inhale to high lung volume levels and then use inspiratory muscles to counteract excess pulmonary recoil forces when singing softer passages (Watson & Hixon, 1985).

The pretreatment values for estimated subglottal pressure (Psub) fell within the range reported by Gracco, Gracco, Lofqvist, and Marek (1994) for untreated patients with Parkinson disease. Prior to treatment, Psub values were lower than those reported by Higgins and Saxman (1991) for healthy elderly males. For the LSVT group only, post-treatment estimated Psub values were closer to the normative data. This suggests that the treatment might have brought the subglottal pressure of these subjects into a more normal range for their age, whereas the R treatment did not have this effect. However, the lack of SPL data in the reports of these previous authors precludes a more detailed comparison of the healthy and pathological subject groups.

The increase in SPL for the LSVT group was accompanied by a similar increase in MFDR. This can be taken as evidence that the subjects in this group increased their sound pressure level by the same means documented in the healthy larynx (Dromey et al., 1995), in that glottal flow shut-off occurred more rapidly, allowing a greater excitation of the vocal tract (Gauffin & Sundberg, 1989). These changes could be interpreted as an indication that the LSVT treatment was successful in helping subjects achieve a more normal mode of phonation. The R group, on the other hand, did not increase in SPL, and therefore continued with the weak phonation that is characteristic of patients with Parkinson disease.

The open quotient (OQ) data from the inverse filtered airflow signal, which reflect changes in vocal fold adduction, did not reach statistical significance in any pre/posttreatment analysis. However, EGGW-25 values, which also change with vocal fold adduction, showed a significant time by treatment group interaction. A recent study of patients with Parkinson disease that compared EGGW measures with stroboscopic examination reported that the two measures correlated (Brosovic, 1994), in that visible improvements in vocal fold closure coincided with higher EGGW values. The differences in the strength of the trends for the two types of measure reported here could be due to the nature of the respective speech tasks. The EGGW measure was derived from the midpoint of a sustained vowel, whereas the open quotient measure was obtained from a syllable repetition task. It is possible that the rapid changes in laryngeal configuration in the dynamic speech task contributed to greater variability in the aerodynamic measure, thus precluding statistically significant results. It is also possible that the EGGW measure offers greater validity than airflow open quotient as a measure of laryngeal adduction.

The fact that over 60% of the aerodynamic data collected were eliminated from further analysis in this study has important implications for researchers investigating disordered speech production. Many of the subjects were unable to perform the /pa/-syllable repetition task in a way that allowed valid data to be obtained (Hertegard, Gauffin, & Lindestad, 1995). Some were unable to produce syllables with constant effort in a single breath group or even relatively stable intensity within a single syllable. Others did not achieve a sufficient lip seal around the oral pressure tube, which precluded valid pressure estimates. These difficulties limit the applicability to disordered speakers of protocols or techniques that have been developed using healthy individuals as subjects (Finnegan, Luschei, Barkmeier, & Hoffman, 1996). This is unfortunate, given the value of physiologic data in assessing disordered speech production in order to plan treatment or monitor progress.

In summary, this study was undertaken to document the aerodynamic mechanisms of change in two groups of patients with Parkinson disease, one of which was successful in increasing vocal intensity. Subjects who received the LSVT were able to achieve increases in SPL through improved vocal fold adduction and increases in subglottal pressure. Sound pressure level did not consistently increase pre- to posttreatment for subjects who received only respiratory training.

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