Intensive voice treatment (LSVT®) for patients with Parkinson's disease: a 2 year follow up

L O Ramig, S Sapir, S Countryman, A A Pawlas, C O'Brien, M Hoehn, L L Thompson

Abstract

***Objectives*—To assess long term (24 months) effects of the Lee Silverman voice treatment (LSVT®), a method designed to improve vocal function in patients with Parkinson's disease.**

***Methods*—Thirty three patients with idiopathic Parkinson's disease were stratified and randomly assigned to two treatment groups. One group received the LSVT®, which emphasises high phonatory-respiratory effort. The other group received respiratory therapy (RET), which emphasises high respiratory effort alone. Patients in both treatment groups sustained vowel phonation, read a passage, and produced a monologue under identical conditions before, immediately after, and 24 months after speech treatment. Change in vocal function was measured by means of acoustic analyses of voice loudness (measured as sound pressure level, or SPL) and inflection in voice fundamental frequency (measured in terms of semitone standard deviation, or STSD).**

***Results*—The LSVT® was significantly more effective than the RET in improving (increasing) SPL and STSD immediately post-treatment and maintaining those improvements at 2 year follow up.**

***Conclusions*—The findings provide evidence for the efficacy of the LSVT® as well as the long term maintenance of these effects in the treatment of voice and speech disorders in patients with idiopathic Parkinson's disease.**

(*J Neurol Neurosurg Psychiatry* 2001;**71**:493–498)

Keywords: voice treatment; efficacy; Parkinson's disease

Department of Speech Language Hearing Sciences, University of Colorado-Boulder, Colorado, USA
L O Ramig

Wilbur James Gould Voice Center, Denver Center for the Performing Arts, 1245 Champa Street, Denver Colorado 80204, USA
L O Ramig
S Sapir
S Countryman
A A Pawlas

Colorado Neurological Institute, Englewood, Colorado, USA
C O'Brien

Department of Psychiatry, University of Colorado Health Science Center, Denver, Colorado, USA
M Hoehn
L L Thompson

Correspondence to:
Dr L O Ramig
ramig@spot.colorado.edu

Received 14 February 2000
and in final form
11 May 2001
Accepted 23 May 2001

About 1.5 million people in the United States have Parkinson's disease. Of these people, at least 75% have voice and speech abnormalities related to their disease.[1 2] Some of these abnormalities—for example, breathy phonation, hoarseness, reduced loudness, imprecise articulation, and reduced prosody—affect speech intelligibility and oral communication. This may adversely affect social, economic, and psychological wellbeing.[3 4]

The physiological and neuropathological mechanisms underlying voice and speech deficits in patients with Parkinson's disease are yet to be determined. Voice abnormalities in such patients have been attributed to inadequate vocal fold adduction, reduced laryngeal muscle activation or synergy, muscle atrophy or fatigue, asymmetric vocal fold tension or movements, stiffness or rigidity of the vocal folds, and/or respiratory muscles.[5–11] Voice and speech abnormalities in people with Parkinson's disease have also been attributed to neurocognitive, neuroaffective, psychomotor, and other higher level cerebral dysfunction.[12 13]

Traditional methods of speech therapy for dysarthric patients with Parkinson's disease, typically administered once or twice a week and emphasising articulation, rate, and prosody intervention, have been largely ineffective.[14–16] By contrast, intensive voice therapy methods, administered almost daily and emphasising simple phonatory effort tasks, have been found to produce favourable results.[17–19]

In 1987 Ramig *et al*[20] developed an intensive treatment programme to improve vocal fold adduction and overall voice and speech production in patients with Parkinson's disease. The programme, known as the Lee Silverman voice treatment (LSVT®), is unique in that it focuses on a simple set of tasks designed to maximise phonatory and respiratory functions. This is done by instructing and constantly stimulating patients to produce a loud voice with maximum effort during sustained phonation and in various speech tasks. These patients are also constantly reminded to monitor the loudness of their voice and the effort it takes to produce it.[13 21]

The loud and effortful phonatory tasks of the LSVT® are aimed at improving respiratory drive, vocal fold adduction, laryngeal muscle activity and synergy, laryngeal and supralaryngeal articulatory movements, and vocal tract configuration. These physiological changes should improve voice quality and loudness, articulatory precision, prosodic inflection, resonance, and speech intelligibility. Such changes accompanying high effort, loud phonation are expected based on similar effects seen in non-disordered speakers.[4 22 23]

The implementation of high effort, intensive phonatory-respiratory therapy is based on evidence from clinical practices in neurology and physical therapy.[24–26] In line with theories of motor learning,[27–29] Ramig *et al* have argued that intensive high effort treatment of vocal functions, especially when coupled with proprioceptive feedback and auditory-vocal self monitoring, should help those with Parkinson's disease to rescale the magnitude of their speech motor output and habituate this level in conversation.[29] Emphasis on self monitoring is an important part of the treatment as motor deficits in those with Parkinson's disease seem to be related to factors such as impaired sensorimotor processing, inability to appropriately

Table 1 Mean (SD) values of pretreatment group characteristics and mean scores (SD) on the UPDRS and stage of disease for the LSVT® and RET groups before and 24 months after treatment

	LSVT® (n=21)		*RET (n=12)*	
Age (y)	61.3 (11.4)		63.3 (7.1)	
Time since diagnosis (y)	7.2 (5.4)		5.0 (4.6)	
Speech severity rating*	1.2 (1.2)		1.7 (1.9)	
Voice severity rating*	2.5 (1.1)		2.3 (1.1)	
	Before	*24 m FU*	*Before*	*24 m FU*
UPDRS	27.7 (12.0)	29.2 (15.1)	12.9 (12.4)	19.2 (18.3)
Stage	2.6 (0.6)	2.7 (0.7)	2.2 (0.9)	2.4 (1.0)

*Speech and voice severity scale: 1=mild; 2=mild-moderate; 3=moderate, 4=moderate-severe; 5=severe. LSVT®=Lee Silverman voice treatment; RET=respiratory effort treatment.; 24 m FU=24 month follow up.

scale and regulate and internally cue movement parameters, reduced ability to automatically execute learned motor plans, impairment in effort demanding processes, and other abnormalities involving high level executive functions.[26 30–33]

Several acoustic, aerodynamic, stroboscopic, electroglottographic, and perceptual studies have demonstrated significant improvement in glottic closure, vocal fold vibratory movements, sound pressure level (SPL), voice fundamental frequency (Fo) range and modulations, voice quality, and speech intelligibility after LSVT®.[29 34–36]

The LSVT® has been previously compared with an alternative treatment method which emphasises high respiratory effort (RET).[36] The comparison with the RET group was carried out both to evaluate the role of increased respiratory drive alone in the improvement of loudness in those with Parkinson's disease and to rule out extraneous factors such as the Hawthorne or placebo effects in interpreting treatment outcome. The greater improvement in vocal function with the LSVT® compared with the RET previously reported is in line with evidence from physiological studies in animals and normal adult people.[22 37–41] Given these facts, and given the differential effects of LSVT® and RET treatments on acoustic and physiological measures mentioned above, greater improvement would be expected in vocal function after LSVT® than after RET.

The studies documenting the efficacy of the LSVT® programme have been based on data obtained immediately after therapy, or 6 or 12 months after therapy. The long term (2 years post-treatment) efficacy of speech treatment for Parkinson's disease has never previously been studied. The purpose of this study was to assess the impact of LSVT® on vocal functions in these same patients 2 years after treatment. To control for extraneous effects, the LSVT® was again compared with the RET. To compare the two groups, we elected to analyze two objective measures of vocal function: vocal loudness (measured as sound pressure level, or SPL) and inflection in voice fundamental frequency (measured as semitone standard deviation, or STSD). Increases in SPL and STSD typically reflect improvement in vocal function.[6 21–23] These two acoustic variables are among those most often impaired in patients with Parkinson's disease and are important for improving speech intelligibility and naturalness.[37]

Methods

PATIENTS

Thirty three patients from the Denver, Colorado area with idiopathic Parkinson's disease were studied. Patients were recruited through local support groups, newspaper advertisements, and referrals from movement disorder specialists. An otolaryngological history and a videolaryngoscopic examination were obtained before the start of speech treatment, and patients were excluded from the study if on examination there was evidence of laryngeal pathology (for example, severe gastric reflux and benign mucosal lesions) not related to Parkinson's disease that would contraindicate speech and voice therapy. Additional details of the pretreatment otolaryngological studies are reported elsewhere.[42] There was no significant difference between the treatment groups for glottal incompetence at baseline.

To keep groups comparable on variables that may affect measures of voice, patients were stratified on the variables age, time since diagnosis, score on the unified Parkinson's disease rating scale (UPDRS),[43] stage of disease,[44] and clinical speech and voice severity ratings, and then randomly assigned to one of two treatment groups. Twenty one patients (17 men, four women) were in the LSVT® programme and 12 (seven men, five women) were in the RET programme. Patient attrition resulted in unequal group sizes and fewer women. Mean (SD) values of pretreatment group characteristics of age, time since Parkinson's disease was first diagnosed, and clinical speech and voice severity ratings are reported in table 1. Means (SD) for scores on the UPDRS and stage of disease before and 24 months after treatment for each group are also reported in table 1. There were no significant differences between the two groups on any of these variables before treatment or on UPDRS scores and stage of disease over time. The two groups also were not different in changes in medication they received during the 2 year period of the study. During the course of speech treatment, patients did not change medication. All subjects were considered "optimally medicated" by their neurologist, a movement disorders specialist, before and throughout the study.

TREATMENT

Details of treatment have been described previously.[13 36] Both forms of treatment were intensive, with a duration of four 1 hour sessions a week for 4 weeks. Both emphasised high effort levels and encouraged patients to perform at maximum effort level throughout every session. Both types of therapies included repeated exercises for the first half of each session and speech tasks for the second half of each session.

The RET programme targeted increased inspiratory and expiratory respiratory muscle activity to increase respiratory volumes and subglottal air pressure and loudness.[36] Treatment tasks included maximum inspiration and expiration, maximum prolongation of /s/ and /f/, and sustained intraoral air pressure using the Iowa oral performance instrument (IOPI).

Table 2 Pretreatment (pre), post-treatment (post), and follow up (FU) means (SD) of SPL and STSD measures of sustained "AH" phonation ("AH"), reading the "Rainbow Passage" (Rainbow) aloud , and conversational speech (monologue)

		Pre	*Post*	*FU*	*PRE to POST Significance*	*PRE to FU Significance*	*df*
SPL "AH":							
LSVT® (n=21)	Mean	68.26	82.36	76.5	*F*=149.88	*F*=39.32	1,20
	SD	4.45	3.92	4.1	p=0.000	p=0.000	
RET (n=12)	Mean	69.19	68.69	70.12	*F*=0.1160	*F*=0.3618	1,11
	SD	5.31	4.79	7.01	p=>0.20	p > 0.20	
SPL Rainbow:							
LSVT® (n=21)	Mean	66.18	75.31	69.78	*F*=49.68	*F*=14.23	1,20
	SD	3.79	4.22	3.19	p=0.000	p=0.001	
RET (n=11)	Mean	65.79	68.03	66.49	*F*=7.1562	*F*=0.3019	1,10
	SD	2.6	3.36	5.54	p <0.025	p >0.20	
SPL Monologue:							
LSVT® (n=12)	Mean	64.7	69.36	67.02	*F*=31.30	*F*=9.88	1,11
	SD	2.56	3.39	1.87	p=0.000	p=0.009	
RET (n=6)	Mean	64.72	65.76	65.71	*F*=0.2996	*F*=0.3928	1,5
	SD	2.76	2.72	4.32	p >0.20	p >0.20	
STSD Rainbow:							
LSVT® (n=20)	Mean	1.9	2.48	2.29	*F*=35.65	*F*=17.78	1,19
	SD	0.53	0.71	0.65	p=0.000	p=0.000	
RET (n=12)	Mean	1.87	2.17	2.03	*F*=25.44	*F*=3.278	1,11
	SD	0.46	0.36	0.35	p=0.000	p=0.098	
STSD Monologue:							
LSVT® (n=11)	Mean	1.74	2.09	2.39	*F*=7.832	*F*=5.280	1,10
	SD	0.32	0.56	1.03	p=0.019	p=0.044	
RET (n=9)	Mean	2.25	2.14	2.13	*F*=0.285	*F*=0.285	1,8
	SD	0.8	0.73	0.56	p=0.608	p=0.608	

Subjects were encouraged to maximise their respiratory effort and were given frequent encouragement to "breathe" just before each of the sustained productions, and during pauses while reading or performing conversational speaking tasks. Visual feedback of rib cage and abdomen excursions was provided to the patients via NIMS Respigraph system PN SY03.[36] The RET did not address phonation or increasing phonatory effort, vocal fold adduction, or voice pitch modulations.

The LSVT® targeted increasing vocal effort to improve loudness. The main goal of the LSVT® is to maximise phonatory efficiency by improving vocal fold adduction and overall laryngeal muscle activation and control.[13 36] Special care is taken to increase vocal fold adduction without causing vocal hyperadduction and strain. Upper limb pushing and lifting tasks[14] during phonation were implemented to increase vocal fold adduction. Maximum prolongation of "AH" and maximum fundamental frequency range drills were completed. Subjects were encouraged to maximise phonatory effort and were given frequent encouragement to "think loud" during sustained phonation tasks, reading, and conversational speaking tasks.[13 36] Attention was given to the respiratory system in the form of general reminders for subjects to take deep breaths "to be loud". The respiratory system was indirectly stimulated during all "think loud" speech tasks.[13 36]

The treatment intensity, high effort, clinician feedback, daily homework, daily quantification of treatment variables and carryover were all presented and stimulated equally in both treatment groups. Two clinicians delivered the treatment to all the patients; both clinicians gave both forms of treatment and were randomly assigned to individual patients. The clinicians worked together to ensure consistency and equivalent high effort and motivation across both forms of treatment. No other additional treatment was given after the initial 16 sessions.

DATA ACQUISITION

Pretreatment experimental data were collected within the week before speech treatment was initiated. Post-treatment data were collected within the week after treatment and were collected at the same time after medication. All experimental data were collected by the primary investigator, who did not administer treatment and was blind to the form of treatment each subject received. Additional post-treatment speech data collection sessions were completed at 6, 12, and 24 months after the initial therapy programme. The results of the 6 and 12 month follow up, including routine neurological and neuropsychological evaluations of the patients before and after treatment have been reported elsewhere[29] and will not be included in this study. Routine neurological assessment at 24 month follow up, including standardised testing (UPDRS and Hoehn and Yahr staging),[43 44] was completed. The results of these tests suggest comparable levels of neurological functioning across the two treatment groups and stable neurological functioning before treatment and throughout the 2 years of follow up. This "stability" is not considered uncommon in patients with idiopathic Parkinson's disease under the care of a movement disorders specialist.

Microphone and SPL data were collected in an IAC sound treated booth while patients performed the following tasks: maximum duration of sustained vowel "AH" phonation, reading of the phonetically balanced "Rainbow Passage",[45] and 25 to 30 seconds of conversational speech (a monologue). Details have been described previously.[36]

SPEECH ASSESSMENT

For clinical purposes, standard speech and voice assessments (for example, motor speech examination) were completed at the time of the first pretreatment speech data collection session. None of the patients exhibited oral motor or speech and voice characteristics uncommon to Parkinson's disease. The severity of speech disorder ratings presented in table 1 was determined by clinical observations.

DATA ANALYSIS

Vocal loudness, fundamental frequency, and its variability were analyzed using standard procedures described previously.[36] Differences between means were analyzed statistically using a two factor time (immediately pretreatment to immediately post-treatment to 24 month follow up) by treatment group (LSVT® *v* RET) repeated measures analysis of variance (ANOVA). Comparisons between groups were done with *t* tests at each point in time.

Results

The means (SD) of the SPL and STSD data are summarised in table 2. The *F* and p values after statistical analysis (ANOVA) are also provided in table 2. Figures 1–5 provide graphic

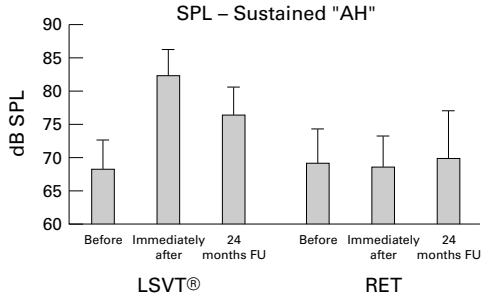

Figure 1 Mean SPL of sustained "AH" immediately pretreatment (before), immediately after, and 24 months after treatment (FU) in the LSVT® and RET groups. Differences from before to immediately after and from before to FU are significant in the LSVT® group (p=0.000) but not in the RET group.

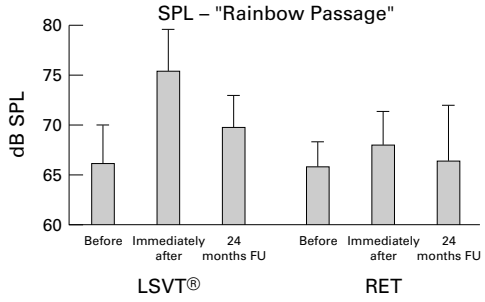

Figure 2 Mean SPL of reading the "Rainbow Passage" immediately pretreatment (before), immediately post-treatment (after), and 24 months after treatment (FU) in the LSVT® and RET groups. Differences from before to immediately after and from before to FU are significant in the LSVT® group (p=0.000 and p=0.001, respectively) and from before to immediately after in the RET group (p<0.025).

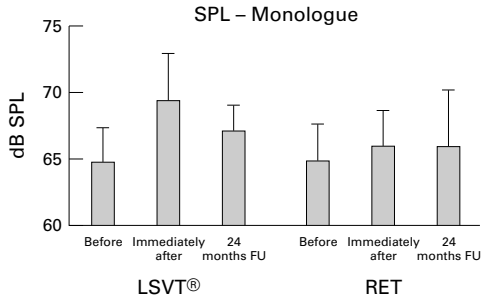

Figure 3 Mean SPL of the monologue immediately pretreatment (before), immediately post-treatment (immediately after), and 24 months after treatment (FU) in the LSVT® and RET groups. Differences from before to immediately after and from before to FU are significant in the LSVT ® group (p=0.000 and p=0.009, respectively) but not in the RET group.

displays of the means of SPL and STSD as a function of treatment group, speech tasks, and time of speech recordings (pretreatment *v* immediately post-treatment *v* follow up 24 months post-treatment).

Twenty per cent of the data were reanalyzed to determine measurement reliability. Repeated measures of SPL and STSD data yielded correlation coefficients greater than 0.97. Test-retest reliability for vocal loudness measures have been assessed in previous studies and have been shown to yield correlation coefficients between 0.75 and 0.95, with most correlation coefficients in the upper range.[29 36]

As seen in table 2 and figures 1–5, the LSVT® resulted in a significant improvement

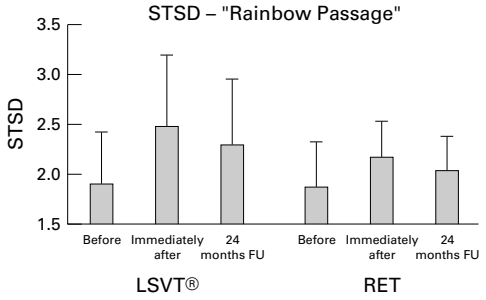

Figure 4 Mean STSD of reading the "rainbow passage" immediately pretreatment (before), immediately post-treatment (immediately after), and 24 months after treatment (FU) in the LSVT® and RET groups. Differences from before to immediately after and from before to FU are significant in the LSVT® group (p=0.000). The difference from before to immediately after is also statistically significant in the RET group (p=0.000). The difference from before to FU in the RET group is not significant.

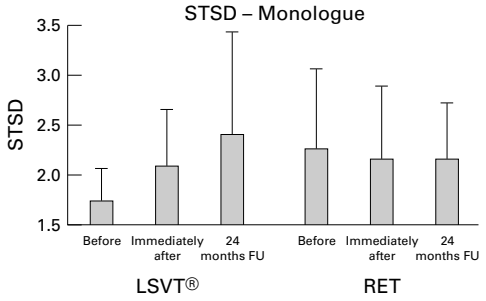

Figure 5 Mean STSD of the monologue immediately pretreatment (before), immediately post-treatment (immediately after), and 24 months after treatment (FU) in the LSVT® and RET groups. Differences from before to immediately after and from before to FU are significant in the LSVT ® group (p=0.019 and p=0.044, respectively) but not in the RET group.

in mean SPL and STSD for the three speech tasks from pretreatment to immediately post-treatment and from pretreatment to 24 months follow up. Specifically, compared with pretreatment, mean SPL values for post-treatment were significantly higher for sustained "AH" (by 14.1 dB, p=0.000), "Rainbow Passage" (by 9.13 dB, p=0.000), and monologue (by 4.66 dB, p=0.000). Compared with pretreatment, mean SPL values for the 24 month follow up were statistically higher for sustained "AH" (by 8.24 dB, p=0.000), "Rainbow Passage" (by 3.6 dB, p=0.001), and monologue (by 2.3 dB, p=0.009). Compared with pretreatment, mean STSD values for post-treatment were significantly higher for the "Rainbow Passage" (by 0.58 STSD, p=0.000), and monologue (by 0.35 STSD, p=0.019). Compared with pretreatment, mean STSD values for the 24 month follow up were statistically higher for the "Rainbow Passage" (by 0.39 STSD, p=0.000), and monologue (by 0.65 STSD, p=0.044).

The RET failed to show significant improvement in SPL or STSD for any but a single speech task from pretreatment to post-treatment and no significant differences from pre-treatment to 24 month follow up. The exceptions were a significant improvement in SPL (by 2.24 dB, p<0.025) and STSD (by 0.30 STSD, p=0.000) from pretreatment to

immediately post-treatment for the "Rainbow Passage".

Comparisons between groups (LSVT® *v* RET) at each point in time showed the following differences: mean SPL for "AH" was significantly higher for LSVT® than RET at post-treatment (p=0.000) and follow up (p=0.006), mean SPL for the "Rainbow Passage" was significantly higher for LSVT® than RET at post-treatment (p=0.000) and follow up (p=0.046) and mean SPL for monologue was significantly higher for LSVT® than RET at post-treatment (p=0.016); mean STSD for the "Rainbow Passage" was significantly greater for LSVT® than RET at post-treatment (p=0.05).

Discussion

The main finding of this study is that, as a group, patients with idiopathic Parkinson's disease who are treated with LSVT® are likely to maintain treatment related improvement in vocal function up to 2 years after treatment. The fact that patients treated with RET did not show such long term effects, despite intensive therapy, suggests that the LSVT® results are treatment specific and cannot be attributed to extraneous factors such as placebo or Hawthorn effects, or to the mere process of being followed up and recorded.

We offer three possible explanations for why LSVT® but not RET produced these long term effects. The first explanation is that the patients learned to increase vocal fold adduction and improve laryngeal muscle activation and synergy, thus rendering the phonatory system more efficient. This interpretation is in line with previous physiological studies of patients treated with LSVT®, demonstrating improved glottic closure and greater vibratory motions of the vocal folds after treatment.[10 37] It is not clear whether the increase in STSD with the LSVT® reflects simply an increase in vocal fold tension and subglottal pressure associated with increasing loudness or whether it also reflects intentional activation of laryngeal muscles to improve intonation. We suspect that both explanations are correct as, perceptually, patients treated with LSVT® often improve both loudness and prosody.[29]

The second explanation is that the LSVT®, by emphasising loud phonation, high vocal effort, and self monitoring of both loudness and effort, helped the patients overcome some of the higher level deficits associated with Parkinson's disease, especially deficits in proprioceptive processing, scaling motor output variables, motor learning, programming and memory, and servoregulation of movement.[26 30 46 47] Physical therapy treatment techniques used to rehabilitate patients with Parkinson's disease often emphasise intensive motor relearning, maximising motor output and effort, increasing drive and goal directed activity, and enhancing sensory awareness to promote internal cueing, self monitoring, and upscaling of motor output.[48–50] These techniques help patients to maximise motor performance and maintain that performance over a long period of time. Because the RET involved similar intensive treatment, why did it did not produce favourable results? One reason is that the target of treatment was respiration rather than phonation and that the lack of emphasis on the phonatory system did not allow patients treated with this method to maximise phonatory output.

The third explanation for the long term effect with the LSVT® is that the emphasis on loud phonation and high effort levels stimulated centres in the brain that are associated with drive and goal directed activities. These neuropsychological activities are highly related to the limbic system, which is also involved in the regulation of emotive vocalisation and intensity of vocalisation.[51 52] For the second, Jürgens and von Cramon[51] have argued that the limbic system, and the neocortical and subcortical systems associated with it, do not participate in motor coordination, nor in the execution of phonatory gestures; rather, they seem to function as a drive controlling mechanism that determines, by its activity, the readiness to phonate as well as the intensity of phonation. Thus, the LSVT®, by emphasising loud and effortful phonation, may have constantly stimulated these systems in the brain that may be impaired in patients with Parkinson's disease and that may have become more functional with LSVT®. Recent findings from a PET study[53] provide preliminary support for this explanation. However, it is the combination of these explanations that most likely accounts for the significant improvement and long term effects of the LSVT®.

Recent studies suggest that the effects of LSVT® extend beyond loud phonation and include improved voice quality, prosody, articulation, speech intelligibility, and swallowing.[29 34 37 54] We suspect that these effects are related to increased motor drive as well as improvement in self monitoring skills. These explanations are tentative and are obviously in need of further research.

Improved oral communication can make a significant positive impact on quality of life. An improvement in vocal loudness of the magnitude reported in this paper has a significant impact on functional communication.[34] Both patients and their spouses and family have reported this phenomenon anecdotally. The results of this and previous efficacy studies of the LSVT® should encourage physicians and other clinicians to refer patients with Parkinson's disease for speech treatment similar to the LSVT®. This type of intensive voice therapy is different from the more traditional methods and has been documented as successful in producing long term effects on voice and speech.

The research presented here was supported by NIH grant No R01DC01150. Our deepest gratitude is extended to the patients who participated in this research study. We thank Drs C Dromey, K Baker, and S Hensley for their assistance during various parts of this research.

1 Logemann J, Fisher H, Boshes B, et al. Frequency and concurrence of vocal tract dysfunctions in the speech of a large sample of Parkinson patients. *J Speech Hear Disord* 1978;**42**:47–57.
2 Streifler M, Hofman, S. Disorders of verbal expression in Parkinsonism. In: Hassler R, Christ J, eds. *Advances in neurology*. New York: Raven Press, 1984.

3 Oxtoby M. *Parkinson's disease patients and their social needs*. London: Parkinson's Disease Society, 1982.
4 Ramig L. The role of phonation in speech intelligibility: a review and preliminary data from patients with Parkinson's disease. In: Kent R, ed. *Intelligibility in speech disorders: theory, measurement, and management*. Amsterdam: John Benjamin, 1992:119–55.
5 Baker K, Ramig L, Luschei E, *et al*. Thyroarytenoid muscle activity associated with hypophonia in Parkinson disease and aging. *Neurology* 1998;**51**:1592–8.
6 Darley F, Aronson A, Brown J. Differential diagnostic patterns of dysarthria. *J Speech Hear Disord* 1969;**12**:246–69.
7 Darley F, Aronson A, Brown J. Clusters of deviant speech dimensions in the dysarthrias. *J Speech Hear Disord* 1969;**12**:462–9.
8 Hanson D, Gerratt B, Ward P. Cinegraphic observations of laryngeal function in Parkinson's disease. *Laryngoscope* 1984;**94**:348–53.
9 Omori K, Slavit D, Matos C, *et al*. Vocal fold atrophy: quantitative glottic measurement and vocal function. *Ann Otol Rhinol Laryngol* 1997;**106**:544–51.
10 Smith M, Ramig L, Dromey C, *et al*. Intensive voice treatment in Parkinson's disease: laryngostroboscopic findings. *J Voice* 1995;**9**:453–9.
11 Solomon N, Hixon T. Speech breathing in Parkinson's disease. *J Speech Hear Disord* 1993;**36**:294–10.
12 Sapir S, Pawlas A, Ramig L, *et al*. Speech abnormalities in Parkinson disease: relation to severity of motor impairment, duration of disease, medication, depression, gender, and age. *Journal of Medical Speech-Language Pathology* (in press).
13 Ramig L, Pawlas A, Countryman S. *The Lee Silverman voice treatment (LSVT®): a practical guide to treating the voice and speech disorders in Parkinson disease*. Iowa City, IA: National Center for Voice and Speech, 1995.
14 Aronson A. *Clinical voice disorders*. New York: Thieme-Stratton, 1990.
15 LeDorze G, Doinne L, Ryalls J, *et al*. The effects of speech and language therapy for a case of dysarthria associated with Parkinson's disease. *Eur J Disord Commun* 1992;**27**:213–24.
16 Weiner W, Singer C. Parkinson's disease and nonpharmacological treatment programs. *J Am Geriatr Soc* 1989;**37**:359–63.
17 Johnson J, Pring T. Speech therapy and Parkinson's disease: a review and further data. *Br J Disord Commun* 1990;**25**:183–94.
18 Robertson S, Thompson F. Speech therapy in Parkinson's disease: A study of the efficacy and long-term effect of intensive treatment. *Br J Disord Commun* 1984;**19**:213–24.
19 Scott S, Caird F. Speech therapy for Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1983;**46**:140–4.
20 Ramig L, Mead C, Scherer R, *et al*. Voice therapy and Parkinson's disease: a longitudinal study of efficacy. Paper presented at the Clinical Dysarthria Conference; 1988. San Diego, CA: 1988.
21 Ramig L, Bonitati C, Lemke J, *et al*. Voice treatment for patients with Parkinson disease: development of an approach and preliminary efficacy data. *Journal of Medical Speech-Language Pathology* 1994;**2**:191–209.
22 Dromey C, Ramig L. Intentional changes in sound pressure level and rate: their impact on measures of respiration, phonation, and articulation. *Journal of Speech and Hearing Research* 1998;**41**:1003–18
23 Schulman R. Articulatory dynamics of loud and normal speech. *J Acoust Soc Am* 1989;**5**:295–312.
24 England A, Schwab R. The management of Parkinson's disease. *AMA Arch Intern Med* 1959;**104**:439–68.
25 Brooks V. *The neural basis of motor control*. New York: Oxford University Press, 1986.
26 Muller F, Stelmach G. Scaling problems in Parkinson's disease. In: Requin J, Stelmach G, eds. *Tutorials in motor neuroscience*. Netherlands: Kluwer Academic, 1991:161–74.
27 Adams J. Use of the model's knowledge of the results to increase observer's performance. *Journal of Human Movement Studies* 1986;**12**:89–98.
28 Schmidt R. *Motor control and learning*. Champaign, IL: Human Kinetic, 1988.
29 Ramig L, Countryman S, O'Brien C, *et al*. Intensive speech treatment for patients with Parkinson's disease: short- and long-term comparison of two techniques. *American Academy of Neurology* 1996;**47**:1496–504.
30 Elias J, Treland J. Executive function in Parkinson's disease and subcortical disorders. *Semin Clin Neuropsychiatry* 1999;**4**:34–40.
31 Marsden C. Function of the basal ganglia as revealed by cognitive and motor disorders in Parkinson's disease. *Can J Neurol Sci* 1984;**11**:129–35.
32 Pillon B, Dubois B, Cusimano G, *et al*. Does cognitive impairment in Parkinson's disease result from non-dopaminergic lesions? *J Neurol Neurosurg Psychiatry* 1989;**52**:201–6.
33 Weingartner H, Burns S, Diebel R, *et al*. Cognitive impairments in Parkinson's disease: distinguishing between effort-demanding and automatic cognitive processes. *Psychiatry Res* 1984;**11**:223–35.
34 Baumgartner C, Sapir S, Ramig L. Perceptual voice quality changes following phonatory-respiratory effort treatment (LSVT) vs respiratory effort treatment for individuals with Parkinson disease. *Journal of Voice* 2001;**15**:105–14.
35 de Angelis E, Mourao L, Ferraz H, *et al*. Effect of voice rehabilitation on oral communication of Parkinson's disease patients. *Acta Neurol Scand* 1997;**96**:199–205.
36 Ramig L, Countryman S, Thompson L, *et al*. A comparison of two forms of intensive speech treatment for Parkinson disease. *J Speech Hear Disord* 1995;**38**:1232–51.
37 Dromey C, Ramig L, Johnson A. Phonatory and articulatory changes associated with increased vocal intensity in Parkinson disease: a case study. *J Speech Hear Res* 1995;**38**:751–63.
38 Gelfer M. Fundamental frequency, intensity, and vowel selection: effects on measures of phonatory stability. *J Speech Hear Res* 1995;**38**:1189–98.
39 Orlikoff R, Kahane J. Influence of mean sound pressure level on jitter and shimmer measures. *J Voice* 1991;**5**:113–9.
40 Berke G, Hanson D, Gerratt B, *et al*. The effect of air flow and medial adductory compression on vocal efficiency and glottal vibration. *Otol Head Neck Surg* 1990;**102**:212–8.
41 Tang J, Stathopoulos E. Vocal efficiency as a function of vocal intensity: a study of children, women, and men. *J Acoust Soc Am* 1995;**97**:1885–92.
42 Perez K, Ramig L, Smith M, *et al*. The Parkinson larynx: tremor, and videostroboscopic findings. *J Voice* 1996;**10**:354–61.
43 Fahn S, Elton R, Committee MOTUD. *Recent developments in Parkinson's disease*. Vol 2. New York: Macmillan Press, 1987.
44 Hoehn M, Yahr M. Parkinsonism: onset, progression and mortality. *Neurology* 1967;**17**:427–42.
45 Fairbanks G. *Voice and articulation drillbook*. New York: Harper and Brothers, 1960.
46 Homberg V. Motor training in the therapy of Parkinson's disease. *Neurology* 1993;**43**:S45–6.
47 Morris M, Iansek R, Matyas T, *et al*. Ability to modulate walking cadence remains intact in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1994;**57**:1532–4.
48 Muller V, Mohr B, Rosin R, *et al*. Short term effects of behavioral treatment on movement initiation and postural control in Parkinson's disease: a controlled clinical study. *Mov Disord* 1997;**12**:306–14.
49 Soliveri P, Brown R, Jahanshahi M, *et al*. Effect of practice on performance of a skilled motor task in patients with Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1992;**55**:454–60.
50 Viliani T, Pasquetti P, Magnolfi S, *et al*. Effects of physical training on straightening up processes in patients with Parkinson's disease.*Disabil Rehabil* 1999;**21**:68–73.
51 Jürgens U, von Cramon D. On the role of the anterior cingulate cortex in phonation: a case report. *Brain Lang* 1982;**15**:234–48.
52 Sapir S, Aronson A. Aphonia after closed head injury: aetiologic considerations. *Br J Disord Commun* 1985;**20**:289–96.
53 Liotti M, Vogel D, New P, *et al*. *A PET study of functional reorganization of premotor regions in Parkinson's disease following intensive speech and voice therapy (LSVT). Paper presented at the American Academy of Neurology (AAN), 1999, Toronto*. Toronto: AAN, 1999.
54 El-Sharkawi A, Ramig L, Logemann JA, *et al*. *Swallowing and voice effects of the Lee Silverman voice treatment (LSVT): a pilot study. Presented at the annual American Speech-Language Hearing Association annual conference*. San Antonio, TX: ASLHA, 1998.