Current Perspectives on the Lee Silverman Voice Treatment (LSVT) for Individuals With Idiopathic Parkinson Disease

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Successful treatment of speech disorders in individuals with progressive neuro logical diseases can be challenging. Hillman, Gress, Haugraf, Walsh, and Bunting (1990) stated that "voice treatment for disorders that are degenerative is controversial since there is no expectation for recovery of function or that any improvement secondary to speech language pathology intervention will be maintained in the long term" (p. 308). Individuals with idiopathic Parkinson disease (IPD) have been particularly resistant to speech treatment, with the conventional wisdom being summarized by the statement that changes observed in the treatment room disappear on the way to the parking lot (Allan, 1970; Aronson, 1985; Greene, 1980; Sarno, 1968; Weiner & Singer, 1989). The consensus that speech treatment has not been effective for individuals with IPD is, perhaps, the basis for the report that of the 75%–89% of these individuals with voice and speech disorders, only 3%–4% receive speech treatment (Hartelius & Svensson, 1994; Oxtoby, 1982).

The reduced ability to communicate is considered to be one of the most difficult aspects of IPD by many patients and their families. Soft voice, monotone, breathy, hoarse voice quality, and imprecise articulation (Darley, Aronson, & Brown, 1969a, 1969b; Logemann, Fisher, Boshes, & Blonsky, 1978), together with lessened facial expression (masked facies), contribute to limitations in communication in the vast majority of individuals with IPD (Pitcairn, Clemie, Gray, & Pentland, 1990a, 1990b). Although medical treatments, including neuro-pharmacological as well as neurosurgical methods, may be effective in improving limb symptoms, their impact on speech production remains unclear (Baker, Ramig, Johnson, & Freed, 1997; Kompoliti, Wang, Goetz, Leurgans, & Raman, 2000; Larson, Ramig, & Scherer, 1994; Rigrodsky & Morrison, 1970; Solomon et al., 2000; Wang, Kompoliti, Jiang, & Goetz, 2000; Wolfe, Garvin, Bacon, & Waldrop, 1975). In addition, previous speech treatment for individuals with IPD, focusing on articulation and rate, has limited efficacy data and limited evidence of long-term success. Recently, there has been great progress in understanding the function of the basal ganglia; this has shed light on the neural bases of IPD (Albin, 1995; Brooks, 1995; Hayes, Davidson, Keele, & Rafal, 1998; Mink, 1996; Wichmann & DeLong, 1993, 1996). Although many studies have used these findings to understand limb function in individu-als with IPD (Rand & Stelmach, 1999; Weiss, Stelmach, Chaiken, & Adler, 1999), their application to voice and speech disorders has been infrequent. At this time, the neural mecha-nisms underlying speech, voice, and swallowing disorders in IPD are not well understood.

Over the past 10 years, our research team has focused on improving speech disorders in individuals with IPD by directing attention to phonation (voice) as a key treatment element. Although disordered voice has been observed in the majority of individuals with IPD (Logemann et al., 1978; Oxtoby, 1982; Streifler & Hofman, 1984), it has until recently been given limited attention in treatment and has been overlooked for its contribution to improv-ing speech intelligibility. Treating voice in individuals with IPD has generated short- and long-term efficacy data for a speech treatment in this population (Ramig, Countryman, O'Brien, Hoehn, & Thompson, 1996; Ramig, Countryman, Thompson, & Horii, 1995; Ramig, Sapir, Countryman, Pawlas, O'Brien, Hoehn, & Thompson, 2001; Ramig, Sapir, Fox, & Countryman, 2001). This treatment— known as the Lee Silverman Voice Treatment (LSVT)—has as its essential concepts (a) exclusive focus on voice (specifically vocal

loudness), (b) stimulation of high-effort productions with multiple repetitions, (c) intensive delivery of treatment (4 individual sessions a week for 4 weeks, 16 sessions in one month), (d) enhancing sensory awareness of increased vocal loudness and effort (calibration), and (e) quantification of behaviors (Ramig, Pawlas, & Countryman, 1995).

The idea of training loudness and the specific techniques of LSVT bring together clinical concepts from literature in the areas of motor speech (Berry & Sanders, 1983; Duffy, 1995; Froeschels, Kastein, & Weiss, 1955; Hardy, 1967; Rosenbek & LaPointe, 1985; Yorkston, Beukelman, & Bell, 1988) and voice (Aronson, 1990; Boone & McFarlane, 1988; Colton & Casper, 1996; Stemple, 1993). LSVT integrates these concepts and techniques in a manner specifically designed for individuals with IPD. In addition, LSVT is administered in a manner consistent with principles of exercise science (Brown, McCartney, & Sale, 1990; Frontera, Merredith, O'Reilly, Knuttgen, & Evans, 1988), skill acquisition (Verdolini, 1997), and motor learning (Schmidt & Lee, 1999)—that is, high effort, multiple repetitions, intensive, simple—together with a focus on sensory awareness. These elements have not previously been systematically combined in a speech treatment program for individuals with IPD (Yorkston, 1996; Yorkston et al., 1988).

Treatment challenges and successes have guided our understanding of the basic mechanisms of voice and speech disorders in IPD and the development of our approach to treatment. The purpose of this paper is to share current perspectives on LSVT by integrating outcome data within an explanatory motor perspective supporting the role of phonation as an efficacious treatment approach for individuals with IPD and to suggest that sensory processing deficits, as well as neuropsychological changes, may be important considerations for speech treatment approaches with this population.

Development and Outcomes of LSVT From a Motor Perspective

Disordered voice and speech characteristics of individuals with IPD are frequently related to the motor signs of the disease (rigidity, bradykinesia, hypokinesia, tremor). Reduced amplitude of movement (hypokinesia) and slowed movement (bradykinesia), which are observed across motor systems in individuals with IPD, have been associated with reduced excitation of the cortical motor centers from the basal ganglia and subsequent reduced drive to motoneuron pools (Penny & Young, 1983). This may be manifest in reduced movement during walking (reduced arm swing, shuffling gait), writing (micrographia), and talking (soft voice) (Beneke et al., 1987; Hallet and Khoshbin, 1980; Tatton, Eastman, Bed-ingham, Verrier, & Bruce, 1984; Wisendanger & Rüegg, 1978). The initial development of LSVT was based on the hypothesis that reduced drive to respiratory and laryngeal musculature underlies reduced vocal loudness and monotonous speech observed in individuals with IPD (Baker, Ramig, Luschei, & Smith, 1998). Therefore, the primary aim of treatment was to increase drive to the respiratory and laryngeal muscles by stimulating and training increased loudness.

A summary of clinical efficacy studies illustrates outcome data associated with LSVT. Individuals with IPD who received LSVT ($n = 26$) increased vocal sound pressure level (SPL) from 8 to 13 decibels (dB at 30 cm) across a variety of speech tasks in comparison with changes from 1 to 2 dB SPL for an alternative treatment group ($n = 16$) (Ramig, Countryman, et al., 1995). Follow-up studies documented that increases in vocal SPL were maintained above pretreatment levels for the LSVT group up to 1 year (Ramig et al., 1996) and 2 years posttreatment (Ramig et al., 2001). An additional 44 individuals (15 treated IPD, 15 untreated IPD, and 14 untreated healthy age-matched control participants) were studied over 6 months with similar findings (Ramig et al., 2001). Changes that accompanied increased vocal SPL in individuals with IPD included increased duration of sustained vowel phonation, maximum range of fundamental frequency, fundamental frequency variability during speech, and reductions in rate (increased pause time and decreased utterance duration) of speech (see Ramig, Countryman, et al., 1995). In addition, increased subglottal air pressure (2–3 cm H_2O) and improved maximum flow declination rate (200–300 l/s/s) have been reported (Ramig & Dromey, 1996). These findings are supported perceptually by listener ratings of increased loudness and improved voice quality accompanying treatment (Baumgartner, Sapir, & Ramig, 2001; Ramig, Countryman, et al., 1995; Ramig et al., 1996), which were maintained as long as 12 months posttreatment in some individuals with IPD (Sapir, Ramig, Hoyt, & Countryman, 1999; Sapir, Ramig, Hoyt, O'Brien, & Hoehn, in review).

Further examination of phonatory source characteristics pre- to posttreatment have documented improved true vocal fold adduction as measured by videostroboscopy (Smith, Ramig, Dromey, Perez, & Samandari, 1995) as well as electroglottography (Garren, Brosovic, Abaza, & Ramig, 2000; in review) for individuals who received LSVT but not an

alternative treatment (see Smith et al., 1995). It is important to recognize that training increased loudness in individuals with IPD results in a loudness level within normal limits and with a healthy voice quality (i.e., not a pressed or shouted voice); therefore, there was no evidence of increased hyperfunction (ventricular hyperadduction or anterior-posterior foreshortening) posttreatment (Smith et al., 1995). In contrast, mild-to-moderate hyperfunction observed pre-treatment in some individuals with IPD—which was hypothesized to be compensation for hypoadduction of true vocal folds—resolved post-LSVT (Countryman, Hicks, Ramig, & Smith, 1997; Smith et al., 1995). Finally, preliminary data from laryngeal EMG in two individuals with IPD have documented positive increases in thyroarytenoid (TA) muscle activity post-LSVT from a pretreatment reduction in TA activity—as compared to healthy aging individuals (Ramig, Sapir, et al., 2000). Taken together, these findings support the impact of intensive loudness training on the phonatory source for individuals with IPD and are consistent with the hypothesis that increasing drive to the respiratory and laryngeal systems increases amplitude of vocal output, thereby improving vocal loudness and quality.

Additional outcome data from LSVT suggest that vocal loudness training may stimulate increased amplitude and coordination of motor output (beyond the phonatory system) to the orofacial system as well. Improvements in articulation have been documented following LSVT as reflected in measures of formant transition duration, rate and extent of movement (Dromey, Ramig, & Johnson, 1995), and increases in vowel space (decreased centralization) during speech of individuals with IPD (Spielman, Ramig, Story, & Fox, 2000). The increase in vowel space reflected differential changes in formant 1 and formant 2, which rose or fell depending on the vowel, despite an overall increase in vocal SPL. This suggests that the formant frequency changes may represent improvements in articulatory range and coordination rather than simply the increase in vocal SPL. Additionally, increased neural drive to orofacial muscles has been associated with increased vocal effort (McHenry, 1997; Ramig, Sapir, et al., 2000; Wohlert & Hammen, 2000) and may have contributed to improvements in articulatory function as well. These changes in articulatory measures post-LSVT are functionally relevant as it is well documented that individuals with IPD have imprecise articulation and reduced amplitude and speed of articulatory movements (Ackermann, Hertrich, Daum, Scharf, & Spieder, 1997; Caligiuri,

1989; Connor, Abbs, Cole, & Gracco, 1989; Forrest, Weismer, Turner, 1989; Leanderson, Meyerson, & Persson, 1971; Netsell, Daniel, & Celesia, 1975).

Recent data using the spatiotemporal index (STI), a measure of spatial and temporal variability (Smith, Goffman, Zelaznik, Ying, & McGillem, 1995), provided additional evidence for changes in the orofacial system associated with vocal loudness. This study compared the effects of loudness and rate manipulations on lower lip movements of young adults, non-neurologically impaired adults, and adults with IPD (Kleinow, Smith, & Ramig, 2001). Data revealed that loudness manipulation produced spatiotemporal indexes that were closest to habitual speech patterns for all groups. In addition, increased loudness resulted in more stable motor output than changes in rate—in particular, slowing rate—which produced the greatest amount of motor variability. Similarly, studies of the gait of individuals with IPD have documented that manipulations of amplitude (e.g., large steps, large arm swing) resulted in more normal (habitual) gait patterns than manipulations in velocity (rate) (Behrman, Teitelbaum, & Cauraugh, 1998). Finally, Dromey (2000) used STI to examine individuals with IPD who were instructed to speak loudly or to speak with exaggerated articulation. Speaking loudly was associated with lower STI values (representing more stable motor output) than exaggerated speech.

Post-voice-treatment data have also documented changes in facial expression (Spielman, Ramig, & Borod, 2001). Observations of increased facial expression accompanying improved loudness and improved intonation following voice treatment—but not following an alternative treatment—suggest these facial changes may reflect more than just posttreatment feelings of happiness or the results of positive reinforcement from one month of treatment (Spielman et al., 2001). Rather, these findings suggest that training loud phonation may also stimulate neural centers considered to be important in the regulation and conscious experience of affect and emotion (Borod, 2000; Eccles, 1980) and the vocal expression of emotion (Cummings, Benson, Houlihan, & Gosenfeld, 1983; Jurgens & von Cramon, 1982; Meyers, 1976; Porges, 1995).

In addition, positive improvements in the nonspeech motor function of swallowing in eight individuals with IPD having a mild swallowing disorder (El Sharkawi et al., 1998, 2002) have been reported. El Sharkawi et al. (2002) documented a 51% reduction in swallowing motility disorders for these individuals on several bolus types. The swallow

disorders that resolved with voice treatment were oral-tongue and tongue-base disorders. These swallowing observations may be considered in light of a recent report (Ward, Theodoros, & Murdoch, 2000) documenting statistically significant increased tongue strength in individuals with IPD following LSVT. Future studies are needed to clarify the simultaneous effects of LSVT on voice and swallowing of individuals with IPD.

An initial examination of neural correlates of the speech and voice disorder in IPD and neural changes associated with behavioral improvements post-LSVT was conducted using Positron Emission Tomography (PET) in five individuals with mild IPD. This pilot study revealed a reduction of abnormally increased activation in cortical premotor areas pre-LSVT and a shift to greater activation in the basal ganglia and anterior insula region post-LSVT (Liotti et al., 1999, in review). These observations suggest a change from an abnormally effortful volitional control (cortex) to compensate for disordered voice and speech, to more effortless and automatic implementation of speech motor actions (basal ganglia, anterior insula). It is important to note that these effects required training of vocal loudness (LSVT), because stimulated increases in loudness pre-LSVT had no effect on the pretreatment abnormalities. These are initial data documenting potential neural changes accompanying LSVT and will be followed up with studies of a similar nature to confirm these findings.

In integrating these outcome data, we suggest that LSVT may affect speech production at two levels. (a) Increased loudness can improve vocal fold closure and enhance the phonatory source, consistent with improving the carrier in the classic engineering concept of signal transmission (Titze, 1993). (b) Increased loudness may stimulate increased effort and coordination across the respiratory, laryngeal, and orofacial systems. Similar findings have been reported in studies of gait of individuals with IPD, where increasing amplitude of one gait variable—for example "take large steps"—automatically activated increased amplitude in other gait variables, such as arm swing (Behrmen et al., 1998). Taken together, these findings support the possibility that a single treatment goal may activate system-wide increases in amplitude of movement, thus enhancing efficiency and simplicity of treatment.

Additionally, we suggest that by targeting vocal loudness in treatment, well-established, centrally stored motor patterns for speech may be triggered. Speech production is a learned, highly practiced motor behavior that becomes relatively automatic; loudness scaling is a task we engage in all our lives. For example, it is common to increase loudness to improve speech intelligibility when speaking against noise or when the listener is far away. Therefore, intensive loudness training may provide the stimulation needed for individuals with IPD to activate and appropriately modulate speech motor programs that are still intact. Accordingly, increasing loudness does not involve deautomatization of speech production by requiring individuals to focus on specific speech parameters such as rate, pauses, or articulatory precision; rather the speaker simply speaks louder (Dromey, 2000; Klienow et al., 2001; Ramig, Pawlas, et al., 1995). Furthermore, training loud phonation may modify vocal behavior by targeting an emotive, phylogenetically old neural system, which involves the limbic system, basal ganglia, thalamus, and periaquaductal gray and the circuits that interconnect these subsystems (Cummings et al., 1983) This observation is supported by recent PET work that demonstrated increased activity in paralimbic regions (left anterior insula and, to a lesser extent, anterior cingulate cortex) post-LSVT, suggesting a greater mobilization of corticolimbic circuits involved in emotional communication (Liotti et al., 1999, in review). The specificity of treatment effects to training vocal loudness versus alternative treatment goals, such as over articulate, remains to be determined

Although studies of LSVT for individuals with IPD are promising, there are clearly limitations to our existing knowledge. First, current published clinical efficacy studies represent approximately Phase I–Phase III studies (Robey & Schultz, 1998) that have examined treatment effects in ideal experimental conditions. Although reports of real world clinical application have been positive, large scale, multisite clinical trials have yet to be conducted. Second, prognostic variables for predicting treatment success remain to be clearly defined. Although the Ramig, Countryman, et al. (1995) study examined the magnitude of treatment-related change and subject characteristics and reported no significant correlations among age, stage of disease, rating on motor UPDRS, time since diagnosis, severity of pretreatment speech disorder, glottal incompetence, cognitive ability, and depression, future studies examining these factors with a larger number of participants may indicate otherwise. Third, studies examining modifications of LSVT at different levels of intensity, with variable versus blocked practice of treatment tasks, treatment in groups, and shorter or longer periods of treatment, will help to elucidate the best mode of administration for optimal treatment results. Fourth, there is a need for studies comparing individuals with IPD who receive LSVT (with its focus on

phonation) with individuals who receive alternative treatment approaches administered in a parallel mode and intensity, but with a focus on articulation or rate. These comparison studies will help delineate key elements for treatment success. For example, is it the focus on phonation, intensity of treatment, or sensory awareness training that contributes most to successful outcomes. Finally, despite positive long-term (2-year) treatment outcomes for group data, successful maintenance of treatment effects for individual patients continues to offer challenges. Efforts to develop feedback and at-home data collection devices to document generalization and maintenance of treatment outcomes are ongoing.

Although data support LSVT from a motor perspective, generalization of improvements in motor function outside the treatment room and maintenance over time have historically been challenging. Our clinical observations suggest that for optimum treatment outcomes, sensory processing and neuropsychological deficits may be important considerations in planning and delivering treatment to individuals with IPD.

Sensory Processing Deficits in IPD and Potential Effect on Speech Treatment

Through the years, two consistent and frustrating challenges to treating individuals with IPD using LSVT have been a failure to recognize that their loudness is reduced pretreatment and a persistent resistance to using increased loudness during treatment. Specifically, it is often observed that soft-speaking individuals with IPD report that they are not speaking less loud, but that their spouse "needs a hearing aid" (Fox & Ramig, 1997; Marsden, 1982). When these same individuals are asked to speak in a louder voice, they often comment, "I feel like I am shouting," despite the fact that listeners judge the louder voice to be within normal limits. If individuals with IPD hear a tape recording of themselves using increased loudness, they can easily recognize that their voice sounds within normal limits, despite their feeling that they are talking too loudly. This suggests that receptive listening is not impaired; rather, the breakdown may be in online feedback (auditory and proprioceptive) while speaking.

In an attempt to understand these challenges, an explanation outside the realm of commonly described motor signs in IPD may be required. Examination of the literature reveals increasing evidence of sensory deficits associated with the neural degenerative process in Parkinson disease. This evidence includes primary sensory symptoms in individuals with IPD, such as complaints of numbness, tingling, pain and achiness, and coldness or burning (Koller, 1984). These may be the result of a "release" of sensory centers from inhibitory control normally mediated by the basal ganglia (Koller, 1984; Snider, Fahn, Isgreen, & Cote, 1976). Neural evidence includes a documented loss in selectivity of firing of globus pallidus neurons to passive sensory stimulation in monkeys made parkinsonian (Filion, Tremblay, & Bedard, 1988); decreased threshold of sensory-triggered jaw reflexes in cats with bilateral globus pallidus lesions (Schneider, 1987); decreased sensory-evoked brain activation, as measured by PET scan data, in cortical (parietal and frontal) and subcortical areas (basal ganglia) in individuals with IPD in comparison with healthy individuals (Boecker et al., 1999); and increased latency in the M2/M3 component of reflexes (mediated by supraspinal levels) in rigid individuals with IPD (Tatton & Lee, 1975; Tatton et al., 1984). Behavioral evidence includes errors on tasks of kinesthesia (Demirci, Grill, McShane, & Hallett, 1997; Jobst, Melnick, Byl, Dowling, & Aminoff, 1997; Klockgether, Borutta, Rapp, Spieder, & Dichgans, 1995); difficulties with orofacial perception including decreased jaw proprioception, tactile localization on tongue/gums/teeth, and targeted and tracking head movements to perioral stimulation (Schneider, Diamond, & Markham, 1987); problems utilizing proprioceptive information for normal movement (Jobst et al., 1997; Schneider et al., 1987); and abnormal higher order processing of afferent information as demonstrated by abnormal reflex and voluntary motor responses to proprioceptive input (Rickards & Cody, 1997).

Overall, the basal ganglia may be a place in the brain where sensory information related to movement is filtered (Schneider, 1987) and, as a result of Parkinson disease, it may no longer be able to effectively filter nonrelevant sensory information (Markham, 1987). Consequences of this deficit (e.g., reduced weighting of sensory information or saturation of sensory signals in the basal ganglia) may include difficulty in recognizing inaccurately scaled amplitude of movement (impaired error detection) or difficulty executing automatic motor behaviors (Demirci et al., 1997; Rickards & Cody, 1997). Thus, it has been suggested that reduced amplitude of movement in individuals with IPD may be perpetuated by abnormally processed sensory feedback (Klockgether et al., 1995; Rickards & Cody, 1997). On the basis of sensory deficit findings, Jobst et al. (1997) suggested that increasing emphasis on kinesthesia as part of physical therapy might be important for individuals with IPD. Similarly, Connor, and Abbs (1990) suggested the following: "In humans, a

breakdown of sensory systems may contribute to the motor abnormalities observed in patients with PD.…These findings have profound implications clinically and offer direct encouragement for sensory enhancement as a form of treatment" (p. 868).

There are limited experimental data (Schneider et al., 1987) documenting the effects of sensory deficits in the speech disorder and associated treatment of individuals with IPD; however, consistent anecdotal observations and evidence from neurology literature support consideration of its role. In LSVT, sensory awareness training has been incorporated through the essential treatment concept of calibration, which is defined as follows: "The patient knows and accepts the amount of effort needed to consistently increase vocal loudness to a level that is within normal limits" (Ramig, Pawlas, et al., 1995, p. 15). As part of generalizing treatment effects, calibration has always been a component of LSVT. Upon observing the persistent complaint, "I can't talk like this, I feel like I am shouting," from individuals with IPD and recognizing evidence of sensory deficits, LSVT has been modified to place a greater emphasis on this element of the treatment program.

It is important to acknowledge that the voice and speech disorders associated with IPD can be noticeably improved by simply cueing individuals to speak loudly and clearly (Aronson, 1990; Ho, Bradshaw, Iansek, & Alfredson, 1999; Ho, Iansek, & Bradshaw, 1999; Ramig, Pawlas, et al., 1995). This is similar to visual cueing that has been documented to improve limb motor performance in IPD (Oliveira, Gurd, Nixon, Marshall, & Passingham, 1997). Markham (1987) described similar situations in which individuals with IPD could perform strikingly well on motor tasks (take slow, big steps; stand up straight; speak loud and clear) under the careful direction of a spouse or clinician and then immediately revert to their previous behavior. He commented, "The failure to relearn correct posture, etc., in Parkinson's disease suggests a defect in processing sensory input or some defect in the sensory information itself. The former is more likely" (p. 166). A concurrent explanation may be that it simply takes too much effort on the part of the individual with IPD to maintain a level of increased amplitude of movement during such activities as walking, talking, and writing.

The implication of these observations for speech treatment is to be aware that individuals with IPD may easily use increased loudness (loudness that is within normal limits) in the treatment room. However, because this loudness feels too loud or requires excessive effort, individuals with IPD may find it difficult to use increased loudness in daily living. Therefore, the sensory mismatch between perceived vocal effort and vocal output may present a significant barrier to generalization and maintenance of treatment effects over time. LSVT assists individuals with IPD in overcoming this barrier by helping them recognize that their pretreated voice is too soft, and convincing them that the louder voice is within normal limits. In addition, LSVT provides continuous, direct, and intense simultaneous motor with sensory awareness training (e.g., consistently asking individuals "Do you feel that effort, do you hear that loudness? That is the amount of effort and loudness you need to use when you speak if you want people to hear and understand you"). This helps individuals with IPD learn the appropriate amount of vocal effort required to self-generate (internally cue) a louder voice. Finally, increased practice through treatment activities, carry-over exercises, and homework helps these individuals become comfortable with using increased loudness.

Recognizing the potential negative impact that sensory processing deficits may have on speech treatment outcomes may be a key variable to consider in any speech-treatment approach for this population. Future studies are needed to clarify the role of sensory deficits in the speech disorder of individuals with IPD and to evaluate the impact of sensory training in speech treatment success.

Neuropsychological Deficits in IPD and Potential Impact on Speech Treatment

Although neuropsychological impairment does not necessarily play a role in the voice and speech disorder observed in individuals with IPD, cognitive functioning may affect an individual's ability to benefit from speech treatment. It has been estimated that 40%–60% of individuals with IPD experience decreased cognitive functioning (Mahler & Cummings, 1990). In some cases, these deficits are part of a global process of dementia (Agid, Ruberg, Dubois, & Pillon, 1987), but many other individuals experience a pattern of cognitive deficits specific to IPD that are not part of a dementia (e.g., Raskin, Borod, & Tweedy, 1990; Taylor & Saint-Cyr, 1995). It is important to recognize that these cognitive deficits in non-demented individuals with IPD and no dementia, though apparent during formal neuropsychological testing, may be subtle or undetectable when interacting with them. In addition, the relationship between changes in

cognitive functioning and degree of motor impairment (stage of disease) of individuals with IPD is not clear (Flowers & Robertson, 1985; Growdon & Corkin, 1986), as cognitive deficits can be seen at all stages of Parkinson disease severity. It may be important for planning speech treatment to recognize that even individuals mildly affected with IPD can have subtle neuropsychological deficits.

The range of neuropsychological changes associated with IPD have been well described in the literature and include slow thinking (Lees, 1994), slow learning (Taylor, Saint-Cyr, & Lang, 1990; Tweeedy, Langer, & McDowell, 1982), problems shifting cognitive sets (Cools, Van Den Bercken, Horstink, Van Spaendonck, & Berger, 1984; Fimm, Bartl, Zimmermann, & Wallesch, 1994; Owen et al., 1992), problems internally cueing (Brown & Marsden 1988), and problems in procedural memory (Harrington, Haaland, Yeo, & Marder, 1990). The design of LSVT, as it was developed from a motor perspective, includes treatment components (simple, redundant, and intensive) that may be favorable for treating individuals with this range of neuropsychological deficits.

There are several cognitive deficits common in IPD that can interfere with the initial stages of information processing and, potentially, speech treatment success. First, bradyphrenia (slowed cognitive processing speed) may reduce the ability of individuals with IPD to process information as quickly as it is being presented. Slowness in the rate of thinking is akin to the slowing of motor movements that occurs in Parkinson disease (bradykinesia). Second, reduced working memory capacity, also described as short-term memory (Baddeley, 1995), has often been found in individuals with IPD (Dalrymple, Kalders, Jones, & Watson, 1994; Gabrieli, Singh, Stebbins, & Goetz, 1996; Owen, Iddon, Hodges, Summers, & Robbins, 1997). This may interfere with the ability of individuals with IPD to process all of the information presented in treatment.

LSVT appears to be well suited to individuals with IPD who have these types of cognitive impairments because the goals and procedures of treatment are simple. The concept of talking louder is easy to comprehend and concrete. It does not require lengthy explanations, which bradyphrenic individuals may find difficult to follow, and these instructions are not likely to exceed the working memory capacity of even mildly demented individuals with IPD. In addition, techniques used to shape a good-quality, louder voice rely heavily upon modeling—for example, "Do what I do," as opposed to verbal explanations. Finally, the considerable repetition of treatment tasks and the single goal

of increased loudness during treatment sessions provides enough redundancy that information missed by individuals during the first presentation may be captured when it is re-presented.

Difficulties with learning new information have been reported in non-demented individuals with IPD (Taylor et al., 1990; Tweedy et al., 1982) and are evidenced by the slower rate of learning that occurs in these individuals relative to those without neurological impairment. To facilitate learning, the goals and tasks of treatment should be kept simple, and treatment should be intensive, involving multiple repetitions within each treatment session while keeping intersession intervals short. LSVT, with its simple focus (e.g., increased loudness) and intensive program (four sessions per week/ 4 weeks), incorporates both of these memory-enhancement strategies. In addition, an impaired ability to shift cognitive sets (mental flexibility) may interfere with learning (Cools et al., 1984; Fimm et al., 1994; Owen et al., 1992), making it difficult for some individuals with IPD to learn and to carry over a series of directions such as "Take a deep breath, be loud, over-articulate, and slow down." Set-shifting requirements are minimized in treatment by focusing on a single goal (e.g., loudness).

Impaired generalization in individuals with IPD may be related, in part, to deficits in memory and executive-type functions. The primary memory deficit in individuals with IPD is generally considered to be memory retrieval (e.g., Flowers, Pearce, & Pearce, 1984) and is characterized by the intact ability to encode new information into long-term memory but an impaired ability to access the previously stored information into consciousness. Therefore, memory retrieval deficits may reduce the ability of individuals with IPD to spontaneously recall previously learned behaviors in new situations. Because over-learned material is easier to recall than newly learned information, the redundancy of the target goal of increased loudness promotes over-learning and may help to overcome this barrier to generalization.

LSVT has been administered to individuals with IPD with a range of cognitive impairments and to individuals with mild to moderate depression. In the Ramig, Countryman, et al. (1995) study, no significant correlations between cognitive ability and magnitude of treatment-related change were found; however, a limited number ($n = 45$) of participants were included. Our clinical experience suggests that individuals with mild to moderate dementia can achieve positive treatment outcomes. However, the end goal of treatment may be modified from using spontaneously increased loudness in conversational speech to using increased

loudness in 10 trained functional phrases, or cued loudness. In addition, some individuals with advanced-stage Parkinson disease or Parkinson-Plus syndromes may require an additional (fifth) week of treatment (Countryman, Ramig, & Pawlas, 1994).

We also recognize that such factors as motivation and compliance are issues in any treatment success. One goal of LSVT is to immediately demonstrate a positive functional impact of increased loudness in the daily lives of individuals with IPD through carry-over activities. Often when individuals with IPD receive positive reinforcement for increased loudness outside the treatment room, it encourages compliance and frequently increases motivation. This is particularly important for individuals who are not initially motivated to improve speech (e.g., individuals who were brought to treatment by a frustrated spouse) or who may be experiencing some degree of depression. Examining LSVT in large numbers of individuals with IPD who have varying degrees of cognitive impairment, dementia, and depression is needed to fully understand the effect of each on speech treatment

Other Neurological Disorders

The extent to which the effects of LSVT are specific to hypokinetic dysarthria associated with IPD is not clear. Current data from treating select individuals with neurological disorders other than IPD (e.g., ataxia, multiple sclerosis, stroke, traumatic brain injury) using LSVT have documented increased vocal SPL following treatment with corroborating perceptual ratings of improved loudness, voice quality, and functional communication (Fox, Ramig, Countryman, Spielman, & Sapir, 2000; Ramig, Fox, Countryman, & Spielman, 2000; Sapir et al., 2001; Solomon, McKee, & Garcia-Berry, 2001). In addition, articulatory acoustic data of improved formant transitions and increased vowel space along with perceptions of improved articulatory precision in a woman with ataxic dysarthria have been reported (Countryman et al., 2000; Sapir, Spielman, et al, in review). Although positive outcomes in perceptual and acoustic measures have been documented, the physiological mechanism of change associated with improved speech production in these individuals has not been established. At this time, we can speculate that positive outcomes may be related to improved motor stability and/or enhanced coordination of respiratory, laryngeal, and orofacial systems that accompany intensive loudness training.

The nature and extent of sensory deficits associated with neurological disorders other than IPD is varied. Clinical experience at this time indicates that these individuals appear to more readily adapt to increased loudness, more easily recognize that increased loudness is within normal limits, and more quickly carry over increased loudness to daily communication, as self-reported during treatment (Fox et al., 2000; Ramig, Fox, et al., 2000; Sapir et al., 2001). It may be that a focus on sensory awareness, although important for individuals with any neurological disorder, is particularly important for individuals with IPD who demonstrate a persistent feeling of talking too loudly. In addition, individuals with a variety of neurological disorders may have a range of neuropsychological deficits that can hinder speech treatment success. For example, Solomon et al. (2001) discussed the effect of memory deficits on post-LSVT outcomes for a man with hypokinetic-spastic dysarthria associated with traumatic brain injury. Consideration of simple, redundant, and intense treatment may be important for individuals with a variety of motor speech disorders, who can have multiple speech mechanism problems as well as cognitive limitations.

Outcome measures from treating individuals with neurological disorders other than IPD are restricted to case study and single-subject designs at this time. As such, the effect of these findings are limited and not considered comparable to the controlled, randomized efficacy studies that have been conducted on individuals with IPD. Speech clinicians should be judicious when considering this approach for individuals with other neurological disorders. Understanding the rationale for treatment techniques and the hypothesized mechanism of change associated with LSVT will assist speech clinicians in making an informed decision about the appropriateness of this treatment on a case-by-case basis. The role of LSVT in treating individuals with speech disorders of various etiologies and the mechanism of posttreatment changes requires further study.

Summary and Future Directions

In summary, we hypothesize that there are at least three features underlying the voice disorder in individuals with IPD: (1) an overall amplitude scale down to the speech mechanism (reduced amplitude of neural drive to the muscles of the speech mechanism), and (2) a problem in sensory perception of effort which prevents individuals with IPD from accurately monitoring vocal output, which results in (3) difficulty in self-generating (internal cueing/scaling) the right amount of effort to produce

adequate loudness. Post-LSVT improvements in the phonatory source in individuals with IPD are likely related to increased neural drive, which may override hypokinetic and bradykinetic movements of respiratory, laryngeal, and orofacial musculature. Concurrently, changes observed with increased vocal effort and loudness may involve a common central mechanism, such as the fronto-limbic system and its link to the basal ganglia, periaqueductal gray, and reticular formation (Davis, Zhang, Winkworth, & Brown, 1996; Devinsky, Morrell, & Vogt, 1995; Jurgens & von Cramon, 1982; Larson, 1985). By incorporating sensory awareness training with motor exercises, we suggest that LSVT encourages acceptance of and comfort with increased loudness and the ability to self-monitor vocal loudness. Addressing this apparent sensory mismatch between vocal effort and vocal output may contribute to generalization and maintenance of treatment effects. Finally, treatment that is simple, redundant, and intensive may help accommodate the processing speed, memory, and executive function deficits observed in some individuals with IPD. It may also promote overlearning and internalization of the vocal effort required for normal loudness. LSVT does not purport to change either sensory processing or neuropsychological function as it has been documented to change motor function; rather, the manner in which treatment is administered addresses these issues and may contribute to positive outcomes.

In conclusion, attaining improvement in communication that is sustained over time in individuals with neural degenerative conditions such as Parkinson disease continues to be challenging. Although positive gains have been made over the years toward understanding both the speech and voice disorder in individuals with IPD and recognizing key variables for positive speech treatment outcomes, there are limitations to existing data, and many questions remain to be answered. Future investigations will continue to clarify the neural bases for voice and speech disorders in IPD as well as guide development and modifications for optimal speech treatment approaches for this population. In addition, as gains are made in our understanding of the mechanism of change associated with LSVT in individuals with IPD, its applicability to individuals with other types of motor speech disorders can be better assessed.

Acknowledgment

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