Speech treatment for Parkinson's disease

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Abstract. Researchers estimate that 89% of people with Parkinson's disease (PD) have a speech or voice disorder including disorders of laryngeal, respiratory, and articulatory function. Despite the high incidence of speech and voice impairment, studies suggest that only 3–4% of people with PD receive speech treatment. The authors review the literature on the characteristics and features of speech and voice disorders in people with PD, the types of treatment techniques available, including medical, surgical, and behavioral therapies, and provide recommendations for the current efficacy of treatment interventions and directions of future research.

Keywords: Parkinson's disease, speech and voice disorders, speech and voice treatment, hypokinetic dysarthria, hypophonia

1. Introduction

Successful treatment of speech disorders in people with progressive neurological diseases, such as Parkinson disease (PD) can be challenging. Historically, people with PD have been particularly resistant to speech treatment [5,6,62,139,169] resulting in reports that of the 89% of these people with voice and speech disorders only 3–4% receive speech treatment [68,115]. The reduced ability to communicate is considered to be one of the most difficult aspects of PD by many people with the disease and their families. The common perceptual features of reduced loudness (hypo-phonia), reduced pitch variation (monotone), breathy and hoarse voice quality and imprecise articulation [32, 33,99,148], together with lessened facial expression (masked facies), contribute to limitations in communication in the vast majority of people with PD [3,119, 120]. Consequently, it has been reported that people with PD are "less likely to participate in conversations" or have "confidence in communication" as compared to healthy aging adults [51].

Although medical treatments, including neuropharmacological as well as neurosurgical methods, may be effective in improving limb symptoms, their impact on speech production remains unclear [7,58,88,90, 132,166,171]. In addition, previous speech treatment approaches for people with PD, focusing on articulation and rate, have limited efficacy data and limited evidence of long-term success. Recently, however, a speech treatment approach called LSVT (Lee Silverman Voice Treatment) has generated the first high quality Level I efficacy data for successfully treating voice

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and speech disorders in this population. The purpose of this paper is to 1) review speech and voice characteristics associated with PD, 2) discuss medical, surgical, and behavioral speech treatment approaches for PD, 3) summarize components of the LSVT speech treatment approach, and 4) highlight ongoing and future research directions in speech treatment for PD.

2. Speech and voice characteristics in Parkinson disease

Disorders of laryngeal, respiratory, and articulatory function have been documented across a number of perceptual, acoustic, and physiological studies in people with PD [8,13,92,93,109,173,174]. Although the neural mechanisms underlying these voice and speech disorders are unclear [1,2,46,66,78,79], they have traditionally been attributed to the motor signs of the disease (rigidity, bradykinesia, hypokinesia, and tremor). An additional explanation for the speech and voice impairment in PD is a deficit in the sensory processing related to speech [75,76,138]. This section will review characteristics of speech and voice impairment in people with PD, including laryngeal and respiratory disorders, articulatory disorders, and deficits in sensory processing related to speech.

2.1. Laryngeal and respiratory disorders

Darley et al. [34] described perceptual characteristics of speech and voice in people with PD [75,76]. They identified reduced loudness, monopitch, monoloudness, reduced stress, breathy, hoarse voice quality, imprecise articulation and short rushes of speech as the classic features of speech and voice in people with PD. Collectively these speech symptoms are called hypokinetic dysarthria [34]. Logemann and colleagues [99] conducted a study with 200 people with PD to examine vocal-tract control and to quantify and describe features of the disorder. Eighty-nine percent of the people with PD in the study presented with laryngeal disorders, comprising breathiness, hoarseness, roughness, and tremulousness. Ho et al. [73] studied 200 people with PD and found that voice problems were first to occur, with other speech problems (prosody, articulation and fluency) gradually appearing later and accompanying more severe motor signs. Sapir and colleagues [138] studied 42 people with PD who sought treatment for their speech problems. Eighty-six percent of the people with PD had an abnormal voice, and this

problem tended to occur early in the disease course. Later, with symptom progression, prosodic, fluency, and articulation abnormalities occurred. Furthermore, Aronson [6] and Stewart et al. [157] have also observed that voice disorders might occur very early in the disease process.

Acoustic descriptions of voice characteristics of people with PD have also been documented. Vocal sound pressure level (SPL) has been measured. Early studies varied in reporting a reduction in vocal SPL in these people [19–21,101,102,108]. However, Fox and Ramig [51] more recently compared 29 people with PD with an age and gender-matched control group and found that vocal SPL was 2–4 decibels (at 30 cm) lower across a number of speech tasks in people. A 2–4 decibel change is equal to a 40% perceptual change in loudness [51]. Furthermore, Ho and colleagues [77] found voice intensity of people with PD to decay much faster than that observed in a healthy comparison group during various speech tasks.

Results related to fundamental frequency (acoustic correlate of pitch) in the speech of people with PD have consistently reported a reduced frequency [19–21, 55,102,108]. Fundamental frequency variability has been reported to be consistently lower in people with PD as compared to healthy aging people [20,21,102]. These findings support the perceptual characteristics of monopitch or monotonous speech typically observed in this patient population [32,33,98].

Disordered laryngeal function has been documented through a number of imaging studies of the vocal folds (videoendoscopic studies). Hansen et al. [167] reported vocal fold bowing (lack of medial vocal fold closure) in 30 out of 32 people with PD. Smith and colleagues [150] documented that 12 of 21 people with PD in their study demonstrated a form of glottal incompetence (bowing, anterior or posterior chink) on flexible fiberoptic views. Perez et al. [117] studied 29 people with PD and observed 50% of them demonstrated difficulties with phase closure of the vocal folds, 46% demonstrated an asymmetrical vibratory pattern, and 55% had laryngeal tremor (vertical laryngeal tremor being the most common).

Additional data to support laryngeal closure problems in people with PD comes from Hirose and Joshita [72] who studied data from the thyroarytenoid (TA) muscles in an individual with Parkinsonism who had limited vocal fold movement. They observed no reduction in the number of motor unit discharges and no pathological discharge patterns (such as polyphasic or high amplitude voltages). They reported loss

of reciprocal suppression of the TA during inspiration and interpreted this as evidence of deterioration in the reciprocal adjustment of the antagonist muscles associated with rigidity. This finding is consistent with deficits in sensory gating characteristics of Parkinson's disease [143]. Luschei et al. [103] studied single motor unit activity in the TA muscle in people with PD and suggested the firing rate of the TA motor units was decreased in males with PD in the study. The investigation reported that this finding as well as those in past studies suggest that PD affects rate and variability in motor unit firing in the laryngeal musculature. Baker et al. [8] found that absolute TA amplitudes during a known loudness level task in people with PD were lowest for the group of people with PD when compared to young normal adults and normal aging adults. Relative TA amplitudes were also decreased in both the aging and PD groups when compared to the young normal adults. The authors concluded that reduced levels of TA muscle activity may contribute to the reduced vocal loudness that is observed in people with PD and aging populations. The reduction in TA activity may also reflect sensory gating anomalies and is contrary to the notion of laryngeal muscle rigidity as the cause of hypophonia in PD.

A number of studies have documented evidence of disordered respiratory function in people with PD. Researchers reported reduced vital capacity [30,37,91], a reduction in the total amount of air expended during maximum phonation tasks [110], reduced intraoral air pressure during consonant/vowel productions [104, 110,151], and abnormal airflow patterns [142,165]. The origin of these respiratory abnormalities may be related to variations in airflow resistance resulting from abnormal movements of the vocal folds and supralaryngeal area [165] or abnormal chest wall movements and respiratory muscle activation patterns [46,111,151].

2.2. Articulatory disorders

Imprecise consonants have been observed in people with PD [30,98,99]. Logemann et al. [98,99] reported articulation problems in 45% of the 200 unmedicated people with PD they studied. Sapir et al. [137] found abnormal articulation in 50% of 42 medically treated people with PD.

Disordered rate of speech has also been reported in some people with PD. While rapid rates, or short rushes of speech, have been described in 6–13% of people with PD [3,20,21,65,67], Canter [20] found slower than normal rates. Pallilalia or stuttering-like speech dysflu-

encies have been observed in some people with PD [32, 137].

Acoustic correlates of disordered articulation have been studied and include problems with timing of vocal onsets and offsets (voicing during normally voiceless closure intervals of voiceless stops), and spirantization (presence of fricative-like, aperiodic noise during stop closures) [1,164,170]. In another study [50], dysarthric speakers with PD showed longer voice onset times (VOTs) than normal. Such abnormal VOTs may reflect a problem with movement initiation [50], which may be related to deficits in internal cueing, timing, and/or sensory gating [1,137].

Disordered articulatory movements have been documented in people with PD through kinematic analysis of jaw movements [15,17,18,26,27,71]. Researchers consistently report that people with PD show a significant reduction in the size and peak velocity of jaw movements during speech when compared to healthy people with normal speech [26,43,50]. The reduction in range of movement has been attributed to rigidity of the articulatory muscles [57,134]; however, this may be related to a problem with sensorimotor perception and/or scaling of speech and non-speech movements [1, 73–76].

Electromyographic (EMG) studies of the lip and jaw muscles in people with and without PD have provided some evidence for increased levels of tonic resting and background activity [81,92,93,109,114] as well as for loss of reciprocity between agonist and antagonistic muscle groups [71,72,81,92,93]. These findings are consistent with evidence for abnormal sensorimotor gating in the orofacial and limb systems, which are presumably related to basal ganglia dysfunction [16, 143,144]. Whether or not these abnormal sensorimotor findings are indicative of excess stiffness or rigidity in the speech musculature is not clear [15,16,26,27].

2.3. Sensory observations

Although the speech problems associated with PD are considered to be related to the motor dysfunctions of the disease, sensory problems in these people have been recognized for years [9,87,144]. Numerous investigators documented sensorimotor deficits in the orofacial system [16,40,143,144] and abnormal auditory, temporal, and perceptual processing of voice and speech [1, 61,74–76,143,144,152], and they have been implicated as important etiologic factors in speech and voice abnormalities secondary to PD [52]. Behavioral evidence from limb and speech motor systems for sensory pro-

cessing disorders in PD include errors on tasks of kinesthesia [39,82,86]; difficulties with orofacial perception, including decreased jaw proprioception, tactile localization on tongue, gums and teeth, and targeted and tracking head movements to perioral stimulation [143]; problems utilizing proprioceptive information for normal movement [82,143]; and abnormal higher order processing of afferent information as demonstrated by abnormal reflex and voluntary motor responses to proprioceptive input [130]. Overall, the basal ganglia may be an area in the brain where sensory information related to movement is filtered [143] in that it "gates out" sensory information when it is not relevant for a motor action, or when it is overly familiar. Thus one aspect of PD might include complex deficits in the utilization of specific sensory inputs to organize and guide movements.

Problems in sensory perception of effort have been identified as an important focus of successful speech and voice treatment [125]. Specifically, it is often observed that soft-speaking people with PD report that their voices are not reduced in loudness, but rather, their spouse, "needs a hearing aid" [51,105]. When these same people 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 range. If persons with PD hear a tape recording of themselves using increased loudness, they can easily recognize that their voice sounds within normal limits, despite the fact they feel they are talking too loud. This suggests that the breakdown may be in online feedback (auditory and proprioceptive) while speaking.

Some insights into the sensory deficits affecting speech and voice in people with PD have been provided by Ho and colleagues [74,75]. One study examined the regulation of speech loudness to increased levels of background noise and instantaneous auditory feedback in soft speaking people with PD and age and gender matched controls. The people in the control group automatically adjusted the loudness of their voice while reading aloud and during conversation by decreasing their loudness when presented with increasing levels of instantaneous auditory feedback and increasing their loudness with more background noise. The people with PD demonstrated an abnormal pattern of speech loudness modulation and failed to increase or decrease loudness in response to the auditory feedback and background noise in the same manner as people in the control group. When given explicit auditory cues to increase loudness, the people with PD were able to increase their speech loudness. These findings further suggest a problem with online or autophonic scaling of loudness in people with PD that can be overridden, in the short term, with explicit external cueing.

2.4. Summary of Parkinson related speech dysfunction

In summary, perceptual, acoustic, physiological, and sensory processing data have documented varying degrees of dysfunction in different aspects of speech in people with PD. The most common perceptual speech characteristics are reduced loudness, monopitch, hoarse voice and imprecise articulation. Acoustic studies of speech of people with PD appear to parallel perceptual studies and have shown evidence of reduced vocal SPL, reduced vocal SPL range, reduced fundamental frequency range, and abnormal articulatory acoustics, such as spirantization. Physiological studies of articulatory muscles have revealed reduced amplitude and speed of movements from a kinematic analysis, EMG activity, and abnormal vocal fold closure patterns. Finally, sensory studies have revealed sensorimotor deficits that include errors on tasks of kinesthesia, difficulties with oralfacial perception, including decreased jaw proprioception, tactile localization on tongue, gums and teeth, and targeted and tracking head movements to perioral stimulation. The neurophysiological mechanisms underlying speech and voice disorders in PD are still poorly understood at this time, particularly in regard to deficits in sensory processing.

3. Treatment for speech and voice disorders

Management of speech and voice disorders in people with PD has been challenging for both medical and rehabilitation practitioners. This has been due, in part, to the lack of precise understanding of the neuropathology of speech and voice disorders in PD. Current treatments for speech and voice disorders in people with PD consist of medical therapies, surgical procedures, behavioral speech therapy, or a combination thereof [146, 147]. Medical therapies alone are not as effective for treating speech symptoms as they are for limb motor symptoms. Thus, speech symptoms are often grouped with other axial symptoms (e.g., balance, gait, posture) that are also considered less-responsive to traditional medical therapies. At this time, a combination of medical therapy (e.g., optimal medication) with behavioral speech therapy appears to offer the greatest improve-

ment for speech dysfunction [146]. There are a number of recent papers that have reviewed the literature related to speech treatment in PD including medical, surgical and behavioral interventions for this population [118, 147,153,175]. This paper will highlight key findings from those recent reviews and report ongoing research in the area of speech and PD. This review of treatment options will help guide clinician choices for recommendations for speech treatment and set the stage for future work in the area of speech treatment and PD.

3.1. Medical treatments

Neuropharmacological approaches for the treatment of PD have had positive outcomes on motor function. However, the impact of these treatments on speech, voice, and swallowing production is highly variable across published reports. While some studies have reported general positive effects of levodopa on limb function [31,106,107,112,132,171,172], the magnitude and consistency of improvement in speech tends to be less impressive [132,171]. More recent studies reported little variation in speech, voice, and respiratory characteristics at different points in the drug treatment cycle [90,151]. In a review of speech and levodopa treatment, Goberman and Coelho reported improvements in overall intelligibility and in phonation, articulation, and speech rate [59]. In another review, Pinto et al. [118] reported that pharmacological methods of treatment alone do not appear to significantly improve voice and speech function in PD people.

A study by Kompoliti et al. [88] used apomorphine to look at the impact of dopaminergic agents on voice and articulation in a randomized, double blind, placebo controlled trial. Results revealed no significant improvement on either function due to apomorphine administration. They proposed that laryngeal and articulatory speech components, like gait, postural instability and cognitive impairment that are also not responsive to dopaminergic therapy, are in fact caused by non-dopaminergic lesions. A follow-up study by Wang et al. [166] reported similar conclusions as the previous report. These studies are important in that they emphasize the need for speech impairments to be treated primarily via non-pharmacologic methods.

Interestingly, Biary et al. [14] performed a double blind placebo controlled study of 12 men with PD to look at the benefits of clonazepam on hypokinetic dysarthria. Clonazepam is a benzodiazepine nd anticonvulsant that has shown some benefit in decreasing parkinsonian tremor. It is not a dopaminergic agent.

This study found that doses of 0.25–0.5 milligram of clonazepam improved speech, specifically short speech rushes, imprecise consonants, and inappropriate silences. Investigators reported little improvement in breathy voice quality or low pitch. When they administered larger doses of clonazepam, no benefit was appreciated.

The actions of pharmacological therapies on speech and voice functioning in people with PD are not well understood. Reports in the literature cite variable responses to medication, with no report documenting a consistent and significant impact of functional communication abilities in people with PD. At this time, pharmacological treatment alone is not sufficient for managing the symptoms of hypokinetic dysarthria in people with PD.

3.2. Surgical treatments

Recently, much attention has been paid to the effects of neurosurgery, in particular deep brain stimulation procedures, on speech and voice of people with PD. The results of studies looking at speech outcomes post-surgery are variable [118]. Ablative surgeries, including pallidotomy and thalamotomy, had significant negative effects on speech, voice, and swallowing following bilateral surgery and variable results following unilateral surgery [28,58]. Pinto et al. [118] summarized published studies looking at the effects of deep brain stimulation (DBS) on speech and reported that thalamic stimulation, although it improved some motor components of speech, had a worsening effect on perceptual assessment and electrophysiological measurements of speech post-surgery. Pallidal stimulation was observed as having both beneficial and worsening effects for perceptual assessment of speech post-surgery. Similarly, studies examining speech following deep brain stimulation of the subthalamic nucleus (DBS-STN) reported variable outcomes for perceptual assessment of speech and electrophysiological measurements [118]. Pinto et al. [118] concluded that, of the surgical therapies, DBS-STN had some efficacy for improving subcomponents of speech (e.g., lip movements). However, there was an overall worsening of speech intelligibility when it was clinically assessed. In a five-year follow-up study of bilateral DBS-STN in advanced PD, significant post-operative improvements occurred in all parkinsonian motor signs except speech when the people were off dopaminergic medication [89].

At this time, speech and voice disorders appear to be less responsive to deep brain stimulation surgeries

than global limb motor functioning. This outcome may be predictable given that DBS-STN should improve levodopa responsive symptoms. Since speech disorders in PD do not respond well to levodopa, it can be predicted that they will not respond well to DBS-STN. This does not account for the appearance of speech symptoms post-surgery when there were no pre-surgery speech problems or the worsening of speech symptoms after surgery. One hypothesis for these unpredictable outcomes is that there is a spread of voltage to surrounding structures that negatively impact speech [118]. Based upon stimulation site within the STN, speech outcomes may vary in a predictable manner. If the voltage spread is in proximity to the internal capsule, the result in speech may be characterized by hesitations and face muscle tightness, whereas, proximity to cerebello-thalamic fibers may result in speech characterized by slurred articulation and stuttering. It has also been suggested that speech functioning may be susceptible to micro lesioning as a result of electrode placement resulting in worsening of speech post-surgery, particularly when the placement is in the dominant hemisphere [167]. Further research into effects of DBS-STN is needed to fully understand its impact on speech in people with PD.

Another surgical intervention that has been reported in the literature is fetal cell transplantation in people with PD. Baker and colleagues [7] observed limited effect of fetal dopaminergic cell transplant on speech functioning of people with PD. Similar to DBS-STN, the people with PD who had fetal cell transplation surgery improved limb motor functioning, but not speech. Other surgical procedures include augmentation of vocal folds with collagen. Hill et al. [70] augmented the vocal folds of 12 people with collagen injections and achieved temporary improvement in hypophonia, with an average benefit lasting 7.8 to 8.5 weeks. While augmentation of vocal folds will help with the laryngeal aspect of speech disorders in people with PD, it does not address the sensory aspect of the speech disorder. It may be that, for people with PD who have moderate to severe degrees of incomplete vocal fold closure, a combination of vocal fold augmentation and behavioral speech therapy will offer the greatest improvements.

Current data reveal that neuropharmacological and neurosurgical approaches alone do not improve speech and voice consistently and significantly [118,146]. Behavioral speech therapy should be considered as an adjunct for improving speech and voice even for optimally medicated people with PD and for those who have undergone neurosurgical procedures.

3.3. Behavioral speech and voice therapy for PD

For many years, speech and voice disorders in people with PD were considered resistant to traditional behavioral speech therapy [5,6,62,139,168]. Although changes in speech may be achieved in the treatment room, the challenge of carryover and long-term treatment outcomes has been encountered consistently over a wide range of speech therapies that have been applied to this population [3]. These approaches have included training in control of speech rate, prosody, loudness, articulation and respiration [172]. Speech therapy with assistive instruments, such as delayed auditory feedback (DAF), voice amplification devices, and pacing boards have also shown limited long-term success [3, 41,69]. Reviews of evidence-based practice for behavioral speech therapy for people with PD have recently been reported in the literature [153] and will be summarized here including: Movement Disorders review [153], Cochrane review [35,36], and Academy of Neurologic Communication Disorders and Sciences (ANCDS) review [175]. Table 1 augments the summary statements below with specifics of the studies reviewed.

The Evidence Based Medical Review for the Treatments of Parkinson's Disease sponsored by the Movement Disorder Society published a review of speech therapy for PD in 2002. This review reported there were a varied number of speech therapies reported in the literature, but very few clinical trials. This report critiqued four Level-I randomized controlled studies with the following inclusion criteria: randomized controlled studies, treatments with a duration of at least 2 weeks, a minimum of 10 people with idiopathic PD, and objective assessments of speech functioning before and after the speech therapy protocol. One of the four critiqued studies was a combination of two published articles on the same group of people with PD [125,126]. The Johnson and Pring [83] and Robertson and Thompson [133] studies compared a speech therapy protocol to no therapy in people with PD. The Ramig et al. [125, 126] and Scott and Cairn [149] studies compared two forms of speech therapy in people with PD. The quality of these Level 1 studies was measured according to CONSORT guidelines. The qualities ratings are listed in Table 1.

Summary findings from this review concluded that there was insufficient evidence to conclude on the efficacy of speech therapy in the following areas: a) prevention of disease progression in PD, b) as a sole treatment in any indication of PD, c) as an adjunct treatment

Table 1
Behavioral studies for speech and voice rehabilitation of Parkinson's disease

Study	Study purpose	Subjects	Statistical design	Findings
S. Scott, F.I. Caird (1983)	Assess benefits of speech therapy with prosodic exercise plus the value of a visual reinforcement device, The Vocolite for 2 to 3 weeks.	*Group A* N = 13 MA = 66 DD = 13 *Group B* N = 13 MA = 66 DD = 10	average of 2 visual analogue scores	2-weeks of therapy substantially improved speech – *Prosadic Abnormality Score* Group A ($p \leqslant 0.001$) Group B ($p \leqslant 0.005$) *Intelligibility rating* Group A ($p \leqslant 0.025$) Group B ($p \leqslant 0.05$)
S. Robertson, F. Thomson (1984)	Efficacy and long term affects of intensive voice TX for PD patients. Group TX for techniques to improve respiration, voice production, speech rate, intelligibility, articulationm 40 hrs. of therapy over 2 weeks.	*2 TX Groups* N = 12 – MA = 58.4 *Controls* N = 10 MA = 78.1	planned comparison, Page's L Trend Test	Breathing and prosodic exercises led to measurable improvement in almost every aspect of speech. Within TX group significant improvement between 1st assessment and 2 post-therapy assessments combined ($p \leqslant 0.01$)
J.A. Johnson, T.R. Pring (1990)	Effects of smaller amounts of TX (10 therapy sessions over 4 wks.). Emphasis on prosodic features of pitch and volume.	3 groups (12 pts. with PD) *Treatment* N = 6 MA = 63.5 *Control* N = 6 MA = 64.8 *Normals* N = 4 MA = 65	Wilcoxon test	Frenchay Dysarthria Scores: *TX'd group* showed significant improvement ($p \leqslant 0.05$) *Control Group* showed slight deterioration ($p \leqslant 0.05$)
[124] Ramig et al. (1995)	Assess effects of 2 types of PD speech TX: 1) LSVT Group-TX to increase vocal fold adduction & respiratory support 2) R Group-TX to increase respiratory support for speech (LSVT)	*LSVT Group* N = 26 MA = 63.5 DD = 8.3 *R Group* N = 19 MA = 65.6 DD = 5.9	T-tests, analysis of variance, AVOVA	Significant post-TX changes only with LSVT group. SPL reading ($p \leqslant 0.037$) Fundamental frequency variability for monologue ($p \leqslant 0.007$) Sickness Impact Profile for impact of PD on communication ($p \leqslant 0.09$) Family ratings for overall intelligibility ($p = 0.014$)
[126] Ramig et al. (1996)	Assess long term (12 months) effects of 2 forms of speech TX for PD: !) LSVT Group 2) R Group – respiratory TX only	*LSVT Group* N = 22 MA = 63.23 DD = 6.55 *R Group* N = 13 MA = 65.31 DD = 4.77	MANOVA, nonorthogonal contrasts	*LSVT Group* Sustained phonation ($p \leqslant 0.0001$) Rainbow Passage ($p \leqslant 0.009$) *R Group* No significant difference after 12 months
[128] Ramig et al. (2001)	To assess long term (24 months) effects of LSVT. One group received LSVT, 2nd group (R) received respiratory TX alone, R-Group. (see Ramig et al. [124])	*LSVT Group* N = 21 MA = 61.3 DD = 7.2 *R Group* N = 12 MA = 63.3 DD = 5.0	two factor time, ANOVA	*LSVT Group* showed significant improvement post-TX and at 24 months. SPL (sound pressure level) phonation ($p = 0.000$), SPL Rainbow Passage ($p \leqslant 0.001$), SPL monologue ($p \leqslant 0.009$) *R-Group* SPL phonation ($p \geqslant 0.20$) SPL Rainbow Passage ($p \geqslant 0.20$) SPL monologue ($p \geqslant 0.20$)
[129] Ramig et al. (2001)	Compare changes in speech in patients TX'd with LSVT, in untreated patients with PD and in neurologically normal age-matched controls.	*PD-TX group* N = 14 Mean Age 67.9 years *PT-NoT group* N = 15	Mean & SD, analysis of variance with repeated measures	*PD-Treated group* showed significant improvement on tests (< 0.0001 and < 0.005) *PD – Nontreated group* showed no significant difference (NSD) and *normal controls* showed NSD.

Table 1, continued

Study	Study purpose	Subjects	Statistical design	Findings
		Mean Age 71.2 *Norman Controls* N = 14 Mean age 69.8 years		

Key: MA = mean age, DD = disease duration, LSVT = Lee Silverman Voice Treatment Technique.

to medication and/or surgery, d) in preventing motor complications in PD, and e) on motor and non-motor complications of PD. The authors recommended future clinical research should include larger, randomized, prospective and controlled studies. In addition, the use of functional neural imaging studies to examine people with PD pre- and post-speech therapy to determine the functional and anatomic changes related to speech treatment was suggested [153]. Furthermore, the authors proposed that behavioral speech therapies should be intensive and focus on loudness or prosody based on the evidence reviewed [83,126,149].

Since the publication of the Movement Disorders review, other Level 1 studies for speech therapy in PD have been published. One study by Ramig and colleagues [126] was independently reviewed by the primary author of the section responsible for speech therapy and it was concluded to be of high quality Level 1 evidence [60].

Deane and colleagues in the Cochrane Review [35, 36] also examined behavioral speech therapy studies. These authors included only randomized controlled studies and analyzed quality of the studies based on CONSORT guidelines. In two publications, the results of studies comparing speech therapy to a placebo or no intervention and studies comparing two forms of speech therapy were analyzed. In the first publication three randomized controlled trials totaling 63 people comparing speech and language therapy with placebo or no intervention for speech disorders in PD were examined. These studies included Johnson and Pring [83], Roberston and Thompson [133], and Ramig et al. (unpublished data). The authors concluded there was insufficient evidence to prove or disprove the benefit of speech and language therapy for speech disorders in people with PD due to the methodological flaws, the small number of people examined, and the possibility of publication bias. In the second publication two randomized controlled trials totaling 71 people with PD comparing two different forms of speech therapy were analyzed. These studies included Scott and Cairn [149] and Ramig et al. [124]. Again, the authors concluded

there was insufficient evidence to support or refute the efficacy of one form of speech therapy over another.

Both of the Cochrane Review publications were based upon studies published before February of 2001. Currently, an update of information from the Cochrane Review for speech therapy and Parkinson disease is taking place. The updated Cochrane Review will include and analyze randomized controlled studies that have been published or are in progress from 2001 to the present.

Members of the Academy of Neurologic Communications Disorders and Sciences (ANCDS) reviewed the evidence for behavioral management of respiratory and phonatory dysfunction from dysarthria including studies of speech therapy for people with PD [175]. These authors did not limit the review to randomized controlled trials; rather included case, single subject, and group designs. The strength of evidence was based upon the following factors: type of study (e.g., case, single subject, group), primary focus of treatment (e.g., biofeedback, LSVT), number of people, medical diagnosis, replicability, psychometric adequacy (e.g., reliability), evidence for control, measures of impairment, measures of activity or participation, and study conclusions. For speech therapy related to PD this review included 3 studies of biofeedback devices totaling 39 people; 5 studies with devices (e.g., delayed auditory feedback) totaling 16 people; 14 studies of LSVT totaling $\sim$ 90 people, and 3 miscellaneous studies of group treatment. For a table outlining details of these studies see Yorkston et al. [175].

Conclusions from the review reported that LSVT has the greatest number of outcome measures associated with any speech treatment examined. Furthermore the authors summarized that for the most part outcomes were positive and can be interpreted with confidence [175]. Recommendations for future research for biofeedback, devices, and group treatment approaches included having a larger number of people in studies, well-controlled replicable and reliable studies of well-defined populations, and control or comparison group studies (randomized controlled studies). Recommen-

dations for future research in LSVT included additional documentation of long-term maintenance effects, large multi-site effectiveness studies (clinical trials), alternative modes of administration (e.g., different dosages of intensity), and further study of treated people with PD to better define predictors of success or failure with the treatment.

4. Intensive voice treatment for PD

The newest and generally perceived state-of-the-art treatment for PD is the LSVT (Lee Silverman Voice Treatment) [118,175]. The fundamentals of LSVT are based upon the hypothesized features underlying the voice disorder in people with PD [52]. These features include i) an overall amplitude scale down of the speech mechanism (reduced amplitude of neural drive to the muscles of the speech mechanism) that may result in a "soft voice that is monotone" [4,9,116], ii) problem in sensory perception of effort that prevents a person with PD from accurately monitoring his/her vocal output [9, 12] and iii) the individual's difficulty in independently generating (internal cueing/scaling) the right amount of effort to produce adequate loudness [38,156]. Key elements of LSVT and details on outcome measures that range across perceptual, acoustic, and physiological levels are summarized.

LSVT is based upon elements derived from neurology, physiology, motor learning, muscle training, and neuropsychology. The five essential concepts of the LSVT include: i) focus on voice (increase amplitude of movement/increase vocal loudness), ii) improve sensory perception of effort, i.e., "calibration," iii) administer treatment in a high effort style, iv) intensity (4 times a week for 16 sessions in one month), and v) quantify treatment related changes. The LSVT approach centers on a specific therapeutic target: increasing vocal loudness (increasing amplitude of movement). This key target acts as a "trigger" to increase effort and coordination across the speech production system. By incorporating sensory awareness training with motor exercises, LSVT facilitates acceptance and comfort with increased loudness, and the ability to self-monitor vocal loudness. Addressing this apparent sensory challenge in people with PD may facilitate generalization and maintenance of treatment effects. Furthermore, a simple, redundant and intensive treatment may help accommodate the processing speed, memory, and executive function deficits observed in some individuals with PD, and promote overlearning and internalization

of the vocal effort required for normal loudness [52]. Incorporation of systematic education, homework exercises and carryover tasks (e.g., assignments to use new LOUD voice outside of the treatment room) further assist in generalization of therapeutic gains to daily living situations.

Findings from initial treatment studies on 45 people with PD documented post-voice treatment SPL increases ranging from 8–13 dB SPL (at 30 cm, across a variety of speech tasks) for those treated with LSVT compared to an alternative treatment group (respiratory treatment; changes from 1–2 dB) [124]. Follow-up studies documented that these SPL increases were maintained for the LSVT group out to one year [126] and two years post-treatment [129]. An additional 44 people (15 treated PD, 15 untreated PD and 14 healthy age-matched control group) were studied over 6 months and findings were similar [128]. The data from these combined studies [124,126,128,129] offer strong support for the short- and long-term efficacy of voice treatment for PD. People who had intensive voice treatment (LSVT) had significant improvements in vocal fold closure, as measured by videostroboscopy as well as electroglottography [56,150], subglottal air pressure (2–3 cm H^{2}0) and maximum flow declination rate (MFDR) (200–300 l/l/sec) [127]. An alternative treatment group (respiratory) did not improve on these measures. Increased vocal effort in the LSVT treated group improved vocal fold valving to contribute to increased vocal SPL and improved speech production. There was no evidence of increased vocal hyperfunction (unwanted strain or excessive vocal fold closure) post-treatment in any person with PD [56,124,150]. These findings are supported further by perceptual reports [11,138] documenting increased loudness and improved voice quality accompanying LSVT. Taken together, these findings support the positive impact of voice treatment for people with PD.

These studies have also provided important information about mechanisms underlying speech and voice disorders in PD and have identified fundamental elements of treatment-related change. Data have documented that successful speech treatment generates other important effects across the vocal tract, encompassing positive changes in articulation, swallowing, and facial expression [42,45,155]. Preliminary imaging results with Positron Emission Tomography (PET) have identified post-speech treatment changes consistent with improved neural functioning in two studies [94,95,113]. Specifically, pre-LSVT, loud phonation in people with PD activated cortical premotor ar-

eas, particularly supplementary motor area (SMA). In the same people with PD post-LSVT, cortical premotor activity during spontaneously loud voicing (LSVT-induced) normalized hyperactivity in the SMA, and increased activity in the basal ganglia (right putamen) suggesting shift from abnormal cortical motor activation to more normal subcortical organization of speech-motor output. Furthermore, post-LSVT changes in people with PD demonstrated an increase in activity in right anterior insula and right dorsolateral prefrontal cortex. Right insula activation has been associated with non-linguistic vocalization (singing, emotional expressive prosody) and emotional expression. This suggests, along with right hemisphere lateralization of post-LSVT effects in people with PD, that LSVT may recruit a phylogenetically old, preverbal communication system involved in vocalization and emotional communication.

Challenges that diminish treatment outcomes with LSVT include people with PD who have severe depression, moderate to severe dementia, atypical parkinsonism (e.g., multiple-system atrophy, progressive supranuclear palsy), or people who have had neurosurgery for their PD (e.g., deep brain stimulation). These people are more challenging to treat during therapy due to factors such as difficulty putting forth maximum effort, more difficulty staying on task, easily confused, or on/off drug effects. Many times the ultimate treatment outcomes are adjusted for these people with advanced PD or those who have had surgical intervention. Instead of striving for self-generated improved loudness in daily conversation, the end treatment goal may be self-generated loudness in 10 functional phrases and cued loudness during conversational speech. Although treatment outcomes are adjusted in these individuals, they can, and do, make significant gains in communication abilities that are important to both the person with PD and his or her family members.

The documentation of Level I evidence for the efficacy of behavioral speech therapy for speech and voice disorders associated with PD is ongoing. To date, LSVT appears to be the most promising form of behavior therapy to address the type of speech impairments experienced by people with PD (Table 1).

5. Summary and future directions

Positive gains have been made over the years towards recognizing key variables for successful speech treatment outcomes in people with PD. Ongoing and future investigations have the potential to further clarify underlying mechanisms of speech disorders in PD while addressing key variables for improving speech treatment outcomes. Some areas of *ongoing research* include: 1) evaluating the impact of training loudness (LSVT) on other systems, such as articulation, swallowing, and neural functioning, 2) systematically documenting the impact of deep brain stimulation surgery on speech in people with PD and their response to speech therapy post-surgery, and 3) increasing accessibility to intensive speech therapy (e.g., LSVT) through use of technology. Areas of *future research* will focus on: 1) understanding the role of sensory deficits in speech disorders in PD, 2) applying principles of successful speech therapy (LSVT) to limb motor systems and creating a combined amplitude-based speech and physical therapy program (Big and Loud), and 3) evaluating the potential neuroprotective impact of exercise-based speech therapies in humans with PD.

5.1. Ongoing research

Published pilot data from training loudness (LSVT) have documented that effects generalize beyond vocal loudness to improve swallowing, articulation, communicative gestures, facial expression, and neural functioning [44,45,95,122,123]. Ongoing randomized controlled studies are further examining this spread of effects by (1) evaluating and comparing the system-wide generalized impact of two therapies [voice (LSVT) and articulation (LSVTA)] on: (a) speech articulation, (b) facial expression, and (c) swallowing in idiopathic PD; (2) evaluating and comparing the system-wide generalized impact of these two therapies on limb gesture and limb motor functioning in PD; (3) investigating external cueing of vocal loudness in people with PD and the impact of these two therapies on this deficit; and (4) investigating the effects of LSVT on brain function activity using Positron Emission Tomography (PET). Results from these studies will further clarify the neural bases for voice and speech disorders in people with PD as well as guide development and modifications for optimal speech treatment approaches for this population.

While deep brain stimulation of the subthalamic nucleus (DBS-STN) has been a valuable treatment for many symptoms of PD, speech outcomes have been variable. Reports range from improvements in selective aspects of speech to severe problems in speech and swallowing following DBS-STN [118]. People and families consistently rate problems in speech and swallowing following DBS-STN as significant and persis-

tent. We need systematic studies of these heterogeneous speech outcomes following DBS-STN that include simultaneous quantitative measures of pre- and post-surgical speech functioning and details of surgical and stimulator optimization. This careful definition of speech outcomes following DBS-STN will provide guidance to surgical stimulation targets for speech. Furthermore, this knowledge will facilitate development of rehabilitative speech treatment approaches for speech problems in people with DBS-STN either pre-surgery (as preventative) or post-surgery (as rehabilitation).

Currently, several research groups are undertaking these tasks. Preliminary data from Tripoliti et al. [163] from Institute of Neurology in London have reported outcomes with LSVT in 5 people with PD post DBS-STN as compared to 5 people with PD and no surgical intervention. Immediate improvements in vocal loudness across these two groups were comparable, but the DBS-STN group did not maintain treatment effects at 2 months post-LSVT as compared to the non-surgical group. This work is ongoing and addressing possible impact of STN stimulation on learning and maintaining new motor behaviours.

An additional area of continued research is addressing the practical challenges of delivering speech treatment intensively (four individual sessions a week for four weeks). Halpern et al. [63,64] reported on the use of a personal digital assistant (PDA), as an assistive device for delivering LSVT to people with PD. This PDA, named the LSVT Companion (LSVTC), was designed to meet the challenges of treatment accessibility and frequency that people with PD often encounter. The LSVTC is specially programmed to collect data and provide feedback as it guides people through the LSVT exercises, enabling them to participate in therapy sessions at home. Fifteen people with PD participated in this study during which nine voice treatment sessions were completed with a speech therapist and seven sessions were completed independently at home utilizing the LSVTC. Acoustic data collected in a sound-treated booth before and after the 16 treatment sessions demonstrated that following treatment the people with PD made significant gains in vocal loudness across a variety of voice and speech tasks. These results were similar to previously published data on 16 face-to-face sessions both immediately post-treatment and at six-month follow-up [63,64]. These pilot findings support feasibility of the LSVTC and support further development of technology-based approaches to enhance treatment accessibility.

An evolution of the LSVTC has been the development of an LSVT virtual speech therapist (LSVTVT).

This is a perceptive animated character, modeled after expert LSVT speech therapists, that delivers LSVT in a computer-based program. This work builds upon the well-established foundation of experimental efficacy data [124,126,128,129] and state-of-the-art learning tools, incorporating intelligent animated agents [10, 22–25,97,158]. A prototype of the LSVTVT has been developed and clinical testing has begun. In addition, research into effectiveness of delivering intensive speech therapy via Telehealth systems or other web-enabled speech therapy systems will continue to enhance accessibility to the intensive sensorimotor training important for successful speech outcomes.

5.2. Future research directions

Further research on the role of sensory problems in speech and voice will likely enhance our understanding of the relationship of this characteristic to our ability to effectively treat speech and voice in people with PD. Pilot research has begun exploring evidence of a sensory component to the speech disorder in PD speech-motor activity, suggesting impaired audio-vocal gating. One area of pilot work examined sensory (auditory) feedback control on speech of a person with PD using behavioral perturbations of both amplitude (loudness) and pitch (frequency) during voicing tasks pre/post LSVT [80]. Pre-LSVT, the person with PD demonstrated a lack of vocal response to perturbations in amplitude and pitch feedback while sustaining the vowel "ah", consistent with impaired audio-vocal gating. Post-LSVT, behavioral responses (as measured by audio recordings) to perturbations in speech feedback revealed that this individual developed a faster, more *automatic* response to amplitude perturbations as a result of LSVT training. Thus, preliminary data suggest that people with PD may have altered cortical responses to pitch and amplitude perturbations, which are modified immediately post-LSVT training. Future research into the nature of this apparent impaired audio-vocal gating and its role in speech disorders is needed.

Recently, principles of LSVT/Loud were applied to limb movement in people with PD (Training Big) and have been documented to be effective in the short term [47]. Specifically, training increased amplitude of limb and body movement (Bigness) in people with PD has documented improvements in amplitude (trunk rotation/gait), that generalized to improved speed (upper/lower limbs), balance, and quality of life [47,48]. In addition, people were able to maintain these improvements when challenged with a dual task. The extension

of this work to a novel integrated treatment program that *simultaneously* targets speech and limb motor disorders in people with PD (Training Big and Loud) has been proposed. Results from pilot work in three people with PD who received Big and Loud treatment revealed all people improved amplitude of speech (SPL/loudness) and limb movements (reaching or gait) post-treatment. The gains in speech and limb movement were comparable to previously published data from independently training Loud or Big, respectively [53]. Furthermore, these gains were maintained for most measures out to 6 months post treatment [54]. There is a great need to simplify rehabilitation approaches for people with PD due to the progressive nature of the disorder, cognitive challenges that make motor learning difficult, and logistical and financial burdens that intensive speech and physical therapies present. A whole body, amplitude-based treatment program may be one possible solution.

Recent advances in neuroscience have brought exercise to the forefront of therapeutic options for people with PD. The potential neuroprotective effects of exercise in animal models of PD, and key aspects of exercise that contribute to neuroplasticity, compel the need for well-defined exercise-based behavioral speech treatments in humans with PD [85,160,161]. Increased physical activity has been shown in animal models of PD to be neuroprotective (reversal of symptoms, attenuation of dopamine loss) if initiated at the time of exposure to a toxin [161,162]. A more recent study has shown that in animal models where dopamine cells are allowed to degenerate to levels equivalent to humans at the time of diagnosis of PD, progressive exercise promoted functional recovery (neurorestorative) and increased levels of dopamine in the striatum [49].

Key elements of exercise in animal models that promoted neuroprotection or neurorestoration included intensive training of motor tasks, increased practice of motor tasks, active engagement in tasks, and the sensory experience of the motor task [49,164]. A behavioral speech therapy (LSVT/Loud) that targets sensory-motor deficits in people with PD and incorporates elements, such as single focus (increased loudness/amplitude), intensive training (4 days/week for 4 weeks), multiple repetitions, and sensory retraining [52] has been documented [128,129]. These principles are consistent with literature citing key elements of exercise that contribute to neuroplasticity and brain reorganization in animal models of PD [49] and human stroke-related hemiparesis [96].

We need future studies to specifically evaluate the impact of intensive behavioral speech therapy on neuro-plasticity and the potential for neuroprotection as measured by dopamine related changes in imaging studies over time. Preliminary studies of PET related changes pre/post LSVT have already documented treatment-dependent functional reorganization in people with PD. These findings include recruitment of the right hemisphere, and activation of motor regions in the left hemisphere, such as the thalamus and pre-supplementary motor area [95,113]. These data suggest that speech therapy may go beyond treating the symptoms of PD and may have the potential to impact progression of speech disorders associated with the disease over time.

5.3. Conclusion

The majority of people with PD experience speech and voice disorders at some point during the disease course and these deficits impair their quality of life. Medical and surgical treatments alone have not sufficiently alleviated speech disorders for people with PD. Thus a combination of behavioral speech therapy, specifically the LSVT approach, in medically managed people with PD appears at present to be the most effective type of speech intervention, though more Level I studies are needed. There are many exciting avenues of ongoing and future speech research that will clarify our understanding of the underlying mechanism of speech disorders in PD and impact development of rehabilitation strategies over the next decade.

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