Researchers estimate that 89% of people with Parkinson’s disease (PD) have speech and voice disorders 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. Here, we review the literature on the characteristics and features of speech and voice disorders in people with PD and the types of treatment techniques available (medical, surgical and behavioral), with a focus on behavioral therapies. We provide a summary of the current status of the field of speech treatment in PD and recommendations for implementation of the current efficacy of treatment interventions. Directions for future research, including a speculative viewpoint on how the field will evolve in 5 years time, are discussed.

**KEYWORDS:** dysarthria • hypokinetic • hypophonia • LSVT® • neural plasticity • Parkinson’s disease • speech and voice disorder • speech and voice treatment

Oral communication is vital in education, employment, social functioning and self-expression. The prevalence of disordered communication is particularly high (89%) in the nearly seven million individuals worldwide with Parkinson’s disease (PD); however, only 3–4% receive speech treatment [1–3]. Soft voice, monotone, breathiness, hoarse voice quality and imprecise articulation, together with lessened facial expression (masked faces), contribute to limitations in communication in the vast majority of individuals with PD [4,5]. 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 [6]. Moreover, speech and language changes in PD can have a negative impact upon individuals with PD and their family-life long before frank impairments of intelligibility are apparent [7]. Affected individuals often become disabled or retire early, are forced to give up activities they enjoy, incur substantial medical costs and have increased mortality [8–10]. Based upon 2004 estimates, PD costs the USA US$34 billion annually in direct health-related expenses, disability-related costs, and lost productivity [11,12]. As the number of elderly people greater than 65 years of age increases, these costs are expected to exceed US$50 billion by 2040 [13]. The average age of diagnosis of PD is 60 years, but in many individuals the disease develops at a much younger age. Given the relative slow progression of the disease, improvement in the ability of these individuals to communicate increases the chance that they could maintain an improved, productive quality of life, despite living out the rest of their life with a chronic disease. Thus, the value of an effective treatment for disordered communication in this population is clear.

Although medical treatments, including neuropharmacological as well as neurosurgical methods, may be effective in improving limb symptoms, their impact on speech production remains unclear [14–20]. Moreover, historically, people with PD have been particularly resistant to speech treatment [1,21–26]. Recently, however, a speech treatment approach called Lee Silverman Voice Treatment (LSVT® LOUD) has generated efficacy data for successfully treating voice and speech disorders in this population. The purpose of this review is to:

- Provide a brief summary of speech and voice characteristics associated with PD
- Discuss medical and behavioral speech treatment approaches for PD
- Summarize key components of speech treatment that seem to underlie positive outcomes
- Highlight ongoing and future research directions in speech treatment for PD

Speech & voice characteristics in PD

Disorders of laryngeal, respiratory and articulatory function have been documented across a number of perceptual, acoustic and physiological studies in people with PD [27–33]. The neural mechanisms underlying these voice and speech disorders are unclear [34–39]. Traditionally, they have been attributed to motor signs of the disease such as rigidity, bradykinesia, hypokinesia and tremor. Of particular importance to speech and voice disorders in people with PD are the proposed pathophysiological mechanisms underlying bradykinesia/hypokinesia: inadequate muscle activation [40]. The muscle activation deficits that occur in bradykinesia are believed to result from inadequate merging of kinesthetic feedback, motor output and context feedback within the basal ganglia, that is necessary to select and reinforce an appropriate command in the motor command [40,41]. This is supported by single cell recording studies [42] and recent brain activation imaging studies [43] showing a correlation in activation of neurons or muscle with increasing movement amplitude. In addition to the mechanisms of bradykinesia/hypokinesia, abnormal sensory processing [43–45] and impaired ability to initiate a motor response (problem with internal cueing) [46,47] play an important role in speech and voice disorders.

Perceptually, speech and voice in people with PD are characterized by reduced loudness, monopitch, monoloudness, reduced stress, breathy, hoarse voice quality, imprecise articulation, short rushes of speech, and hesitant and dysfluent speech [48,49]. Collectively, these speech symptoms are called hypokinetic dysarthria [50]. Voice problems are typically the first to occur, with other problems, such as prosody, articulation and fluency, gradually appearing as the speech disorder progresses [2,3,45].

Acoustic descriptions of voice characteristics of people with PD have also been documented. Early studies varied in reporting a reduction in vocal sound pressure level (vocSPL) in these people [51–55]. More recently, Fox and Ramig compared 29 people with PD with age- and gender-matched neurologically healthy individuals (control group) and found that vocSPL was 2–4 decibels (at 30 cm) lower across a number of speech tasks [6]. A 2–4 decibel change is equal to a 40% perceptual change in loudness [6]. In addition to an overall low level of vocal loudness, Ho and colleagues found that voice intensity in people with PD tend to decay much faster than that observed in a control group during various speech tasks [56]. Results related to the voice frequency (an acoustic correlate of pitch) in the speech of people with PD have consistently reported a reduced frequency range [51–53,54–57]. These findings support the perceptual characteristics of monopitch or monotonous speech typically observed in this patient population [3,48–49,58]. 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), reduced range of vowel articulation [59] and spirantization (presence of fricative-like, aperiodic noise during stop closures) [34,60,61].

Physiologic abnormalities associated with speech & voice disorders in PD

Physiologic (videolaryngostroboscopic and electroglossographic) studies of voice in people with PD have documented voice tremor, poor vocal fold closure and reduced amplitude, asymmetry or slow vibratory patterns of the vocal folds [62,63]. Respiratory studies in people with PD have documented reduction or abnormalities in vital capacity, amount of air expended during maximum phonation tasks, intraoral air pressure during consonant/vowel productions, chest wall movements and respiratory muscle activation patterns during speech breathing [64]. Electromyographic (EMG) studies of vocal function in people with PD indicate either a reduction of neural drive to the laryngeal muscles [27] or abnormally elevated laryngeal muscle activity [65], and poor reciprocal suppression of laryngeal and respiratory muscles [66]. Kinematic and EMG studies of orofacial movements during speech in PD indicate a reduction in the size and peak velocity of jaw movements, increased levels of tonic resting and background neuromuscular activity, and loss of reciprocity between agonist and antagonistic muscle groups [30,34,35,67–69]. Thus, these studies collectively suggest decreased amplitude of speech movements in people with PD associated with abnormal neural drive to the speech periphery and abnormal sensorimotor gating. Importantly, these studies also indicate that muscle rigidity is not a major cause of the speech movement abnormalities in PD. Moreover, the severity of speech disorders in PD may change markedly as the function of the specific speech tasks being performed and in the absence or presence of external cues, such as the instruction to speak loudly and clearly [70,71]. These, and other findings to be reviewed later, suggest that the neuropathological mechanisms underlying speech disorders in PD probably involve high level motor dysfunctions and sensory processing abnormalities.

Sensory & internal cueing deficits associated with speech & voice disorders in PD

It has been suggested that unlike certain limb motor disorders in PD, which are strongly and directly related to dopamine deficiency and rigidity, some motor disorders in PD, including speech, may also involve abnormal nondopaminergic or special dopaminergic mechanisms, which impair internal cueing, sensorimotor gating and scaling of movement parameters, thus resulting in poor regulation and control of speech movement initiation, amplitude and timing [71–73]. Numerous investigators documented sensorimotor deficits in the orofacial system [67,74–76] and abnormal auditory, temporal, and perceptual processing of voice and speech [34,43,44,46.75–78]. These abnormalities have been implicated as important etiologic factors in the speech and voice impairment associated with PD [79]. Behavioral evidence from limb and speech motor systems for sensory processing disorders in PD include: errors on tasks of kinesthesia [80–82]; difficulties with orofacial perception, including decreased jaw proprioception, tactile localization on tongue, gums and teeth, and targeted and tracking head movements to
perioral stimulation [75]; problems utilizing proprioceptive information for normal movement [75,81]; and abnormal higher order processing of afferent information as demonstrated by abnormal reflex and voluntary motor responses to proprioceptive input [83]. Thus, one aspect of hypokinetic dysarthria in PD might include complex deficits in the utilization of specific sensory inputs to organize and guide speech movements.

Additional insights into the sensory deficits affecting speech and voice in people with PD have been provided by Ho and colleagues [43,46]. 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 an impairment with online or autophonic scaling of loudness in people with PD; an impairment that can be overridden, in the short term, with explicit external cueing. This impairment is analogous to the micrographic handwriting in individuals with PD when asked to write on a blank piece of paper. Once these individuals are provided with dots on the paper, and asked to write so that the letters touch the dots (external cues), or once they are provided with the word “big” spoken to them (another external cue) the handwriting improves dramatically, but only during the short time that they are exposed to the cues and/or immediately after the cue is given [84].

Summary of PD-related speech & voice dysfunction
In summary, perceptual, acoustic and physiological 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 vocSPL, reduced vocSPL range, reduced frequency range and abnormal articulatory acoustics. 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. In addition, studies of sensory aspects of PD have revealed sensorimotor deficits that include errors on tasks of kinesthesia, difficulties with oro-facial perception, including decreased jaw proprioception, tactile localization on tongue, gums and teeth, and targeted and tracking head movements to perioral stimulation. More research is needed to clarify the impact of sensory processing and internal cuing deficits in hypokinetic dysarthria associated with PD.

Treatment for speech & voice disorders in PD
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 neuropharmacological therapies, neurosurgical procedures, behavioral speech therapy or a combination thereof [85,86]. At this time, a combination of medical therapy (e.g., optimal medication) with behavioral speech therapy appears to offer the greatest improvement for speech dysfunction [85]. There are a number of recent papers that have reviewed the literature related to speech treatment in PD including medical and behavioral interventions for this population [73,86–90].

Medical treatment for speech and voice disorders in PD
In contrast to the marked therapeutic effects of dopamine therapy (levodopa or dopamine agonists) on rigidity, akinesia, bradykinesia and tremor in the limb motor system [91], the magnitude, consistency, and long-term effects of levodopa or dopamine agonist therapy on speech in PD are far from satisfactory [72,73]. Specifically, while several studies have documented dopamine-induced improvement in speech motor function, such as a reduction in excessive laryngeal neuromuscular activity, better laryngeal control of voice onset and vocal fold closure during speech [65], better speech intelligibility [92], greater prosodic voice fundamental frequency (Fo) inflection, greater vocSPL, better voice quality [93,94], enhanced lip function during speech and nonspeech tasks [95], and less voice tremor [96], many other studies have failed to show systematic changes or clinically significant improvement in speech with levodopa [16,73]. These findings have led researchers to suggest that voice and speech disorders in PD might be related to non-dopaminergic or special dopaminergic mechanisms [45,72]. In line with this suggestion, it has been shown that clonazepam, a non-dopaminergic agent, when given at 0.25–0.5 mg/day dosages, can significantly improve some aspects of speech, such as imprecise consonants, short rushes of speech, and inappropriate silences in an individual with PD [97].

Neurosurgical procedures such as deep brain stimulation (DBS) of the thalamus, pallidum or subthalamic nuclei (STN), ablative surgeries (pallidotomy and thalamotomy) and fetal cell implantation, have also been shown to result in dramatic improvement in limb motor function, but produce inconsistent effects on speech, with some showing dramatic improvement, others showing improvement, others no changes and still others reporting dysarthria as a significant side effect of DBS in some individuals [14,15,98–102]. The adverse effects on speech might be related to voltage spread and/or lesions of a neural network that mediates sensorimotor speech control [87]. In addition, stimulator placement and adjustments may play a key role in speech outcomes [102]. The effects of different electrical parameter settings on the intelligibility of speech in patients with PD treated with subthalamic DBS need further research [102].

Given that neuropharmacological and neurosurgical approaches alone do not improve speech and voice consistently and significantly [85,87], behavioral speech therapy
should be used to improve speech and voice even for optimally medicated people with PD and for those who have undergone neurosurgical procedures.

Behavioral speech & voice therapy for PD

Reviews of evidence-based practice (EBP) for behavioral speech therapy for people with PD have been recently summarized by Trail and colleagues [73]. Summary statements from that review as well as additional reviews not included in that summary will be discussed here including: Movement Disorders review [88], Cochrane reviews [103,104], NICE PD treatment guidelines [105] and the Academy of Neurology review [106]. These reviews used the criteria of only including randomized controlled studies and most analyzed the quality of the studies based on Consolidated Standards of Reporting Trials (CONSORT) guidelines.

Summary findings from these reviews concluded that there was insufficient evidence to conclude on the efficacy of speech therapy in the following areas:

- Prevention of disease progression in PD
- As a sole treatment in any indication of PD
- As an adjunct treatment to medication and/or surgery
- In preventing motor complications in PD
- On motor and nonmotor complications of PD [88]

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 (Cochrane). Furthermore, there was insufficient evidence to determine if any specific speech therapy modality is superior to another [104,106]. 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 [88].

Although there was inconclusive evidence for one speech treatment over another, all reviews acknowledged the potential for speech treatment to improve function in people with PD. More specifically, all reviews proposed that behavioral speech therapies should be intensive and focus on loudness (e.g., LSVT LOUD) or prosody based on the evidence reviewed [106–109].

Since the publication of the Movement Disorders review, other studies for speech therapy in PD have been published. One study by Ramig and colleagues [110] was independently reviewed by the primary author of the section responsible for speech therapy and it was concluded to be of high-quality Level I evidence [Gortz C. Pers. Comm., 2003]. This paper was not included in any of the other reviews listed above. Currently, an update of information from the Cochrane review for speech therapy and PD 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.

The UK NICE recently updated their national clinical guidelines for diagnosis and management of PD. As part of the guidelines, they conducted a review of speech treatment literature including the Cochrane review discussed above as well as several studies published after that review [110,111]. Their recommendations for clinical care were that speech and language therapy should be available to people with PD. One of the specific recommendations from the NICE guidelines, consistent with recommendations from other reviews, is that speech therapy should focus on improving vocal loudness and pitch range, as in programs such as the LSVT LOUD.

Caveats to behavioral treatment evidence-based reviews

There are a few important considerations to keep in mind regarding reviews of behavioral treatment literature, both those presented here or encountered in the future. Montgomery and Turkstra discuss the limitations of evidence-based medicine (EBM), which is the standard for judging medical treatment efficacy, and its counterpart, EBP, which is the growing standard for judging rehabilitative treatment efficacy [112]. While these guidelines are helpful to understand and judge the quality of clinical research, there are limitations to the information they can provide. Three points for caution in interpreting data from EBP guidelines discussed include [112]:

- Evidence that is “statistically significant” is not necessarily “clinically meaningful”
- Group data will not always apply to the individual patient, clinical judgment is also necessary
- Randomized controlled trials (RCTs), the current gold standard in EBM and EBP, may not be feasible or achievable for answering many clinical questions

The authors summarized, “evaluations and recommendations for clinical practice should not be based only on the amount of RCT or other evidence but also on reasoned assessments of the problems inherent in attributing treatment cause to experimental effect; the degree of generalizability; and the scientific, social, and ethical implications of a decision in favor of or against assessing a cause to an effect. Also, such evaluations and recommendations should not discount the role of reasoned judgments made by experienced clinicians” [112].

In line with the above comments, the Academy of Neurologic Communication Disorders and Sciences (ANCDS) has offered two additional reviews [89,113]. These authors did not limit the review to RCTs; rather they included case, single subject and group designs. The first review examined evidence for behavioral management of respiratory and phonatory dysfunction from dysarthria including studies of speech therapy for people with PD [89]. The strength of evidence was based upon the following factors: type of study (e.g., case, single subject or group); primary focus of treatment (e.g., biofeedback or LSVT LOUD);
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: three studies of biofeedback devices totaling 39 people; five studies with devices (e.g., delayed auditory feedback) totaling 16 people; 14 studies of LSVT LOUD totaling approximately 90 people; and three miscellaneous studies of group treatment. For a table outlining details of these studies, see [89].

Conclusions from the review reported that LSVT LOUD 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 [89]. 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). Recommendations for future research in LSVT LOUD included additional documentation of long-term maintenance effects, large multisite 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.

The second, more recent review examined evidence for effectiveness of global treatment parameters including loudness, rate, prosody or general instructions such as “clear” speech for individuals with dysarthria [113]. The evidence was characterized based upon Phases of research as defined by Robey and Schultz [114]. Phase I studies included experimental manipulations, typically a single dosage/session of loudness, rate or prosody manipulations. Phase II studies included treatment protocols carried out in a speaker with dysarthria in case reports and small groups with no control or treatment comparison. Phase III studies included treatment protocols tested with single subject or group designs with control groups or treatment comparisons. This review included PD and other etiologies; however, the majority of data were from individuals with PD. A full comprehensive summary of findings can be found in Table 2 of the Yorkston et al. review with brief summary statements below [113].

The strongest evidence regarding treatment effectiveness was for modification of loudness, specifically the LSVT LOUD protocol, in people with PD. This work demonstrated progression through all three phases of research, had a well-defined, replicable protocol, measured multiple aspects of speech and included a relatively large subject pool. Continued research should focus on areas such as measures of outcomes in natural social situations and examining factors to optimize learning (e.g., dosage). Studies modifying rate included Phase I and II research and revealed it may be a powerful technique for improving intelligibility in some speakers with dysarthria. There are a variety of techniques used across studies. Areas for future research include examining generalization of techniques, treatment studies comparing various techniques, parameters of treatment scheduling (e.g., intensity or dosage) and descriptions of how optimal rate is selected and trained. Prosody studies also included Phase I and II research. The current status indicates that manipulating prosody may enhance linguistic information, thus intelligibility. Future research needs to include areas such as comparison of approaches, generalization of training, techniques for perceptual ratings of prosody and documenting social validity. Finally, there were studies of general instructions, such as instructions to speak “clearly.” The current status documents that this type of approach may take advantage of compensatory abilities and be applied to many types of dysarthria. Future research should focus on larger groups of speakers, candidacy issues for the approach and studying actual rather than simulated communication breakdowns [113].

**Intensive voice treatment (LSVT LOUD) for PD**

Given the large research base supporting training-increased vocal loudness via LSVT LOUD, it is beneficial to examine this specific treatment protocol in more detail as it pertains to current and future speech treatment research [73,87,89]. The fundamentals of LSVT LOUD are based upon the hypothesized features underlying voice disorders in people with PD [79]. These features include:

- 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” [115–117]
- Problems in sensory perception of effort that prevents a person with PD from accurately monitoring his/her vocal output and results [116,118]
- The individual’s difficulty in independently generating (internal cueing/scaling) the right amount of effort to produce adequate loudness [119,120]

The result is hypophonia, hypoprosodia and hypokinetic articulation characteristics of the dysarthria associated with PD. It is critical to recognize that the loudness target in LSVT LOUD is a healthy increase in vocal loudness. Patients are not trained to yell or scream or to use pressed voice, rather the speech clinician trains a voice that is louder with appropriate voice quality.

The mode of delivery of LSVT LOUD differs from traditional forms of speech treatment. It requires intensive, high effort speech exercise combined with a simple, redundant and salient treatment target to facilitate transfer of loudness into functional daily living. The standardized protocol for LSVT LOUD embodies many of the fundamental principles of exercise and motor training that have been shown to promote neural plasticity and brain reorganization in animal models of PD [121] and human stroke-related hemiparesis [122]. Such intensive training, especially when practiced continuously and at the onset of the disease or following brain injury, has been shown...
to impact molecular changes associated with cell survival, cell growth and functional recovery in animal models of PD. These changes reflect neural plasticity and brain reorganization [121,123–126], and challenge the assumption that there is no potential for recovery of function in PD or other neurodegenerative disorders. Thus, translation of the principles of neural plasticity to therapeutic approaches will require a significant paradigm shift in rehabilitation sciences [127]. Our ability to embrace these principles and integrate them into the mode of delivery of treatment will be essential for advancing rehabilitation science in parallel with neuroscience. While the standardized protocol for LSVT LOUD was developed before these recent neuroscience investigations, it adheres to key principles of neural plasticity, such as intensity, complexity, saliency, use it or lose it, and use it and improve it, which may explain, in part, why it has been successful (for details, see [125]).

The target of training vocal loudness (increased amplitude of motor output) may stimulate generalized neural motor activation across the speech production system and potentially other motor systems. The improvements in articulation, facial expression, swallowing and limb movements are consistent with the concept of global parameters, whereby a single treatment target impacts common control mechanisms that, in turn, influence motor behaviors beyond the specific targeted function [128–130]. The neurological bases of such global motor effects are not known. However, McLean and Tasko reported evidence for neural coupling of orofacial muscles to neural systems of laryngeal and respiratory control in human studies [131]. These authors suggest that a potential source of this observed neural coupling might be from efferent drive from a common brain region to motor neurons innervating orofacial, laryngeal and respiratory muscles. Such common neural structures and coupling may explain, in part, the potential spread of effects from stimulation of increased vocal effort and loudness (respiratory and laryngeal systems) to orofacial muscles and swallowing function. Another explanation for the distributed and lasting impact of LSVT LOUD is that it involves and stimulates phylogenetically old neural systems, especially the emotive brain, which is an important part of the survival mechanism. Speech production is a learned, highly practiced motor behavior, with many of its movements regulated in a quasiautomatic fashion [132,133]; loudness scaling is a task that both animals and humans engage in all their lives [43,44,134–137]. Thus, the regulation of vocal loudness for speech may involve a phylogenetically old system that has been adapted, through learning, for speech production and comprehension purposes.

In summary, these findings suggest the effects of loud phonation may be uniquely pervasive across the speech production system by stimulating common neural mechanisms for speech and other motor systems, and/or by stimulating neural systems that mediate emotive vocalization via an integrated, phylogenetically old neural mechanism.

Brain changes induced by LSVT LOUD as measured with PET imaging [124,138] reflect improvements in the basal ganglia, limbic system, prefrontal cortex and right hemisphere functions. As indicated previously, these neural systems are involved in vocalization, loudness regulation and vocal learning, which collectively may account for the significant and long-term effects of LSVT LOUD on speech in individuals with PD. This may also help to explain how a cross-system, such as swallowing, may also be affected by training vocal loudness. Perspectives such as these elucidate why LSVT LOUD improves voice and speech production in PD and other neural conditions, as compared with previous treatments that have focused on rate or articulation, which involve primarily cortical, phylogenetically newer neural centers. Thus both mode of treatment delivery and target of the treatment approach are areas of study in future speech treatment research.

Expert commentary & five-year view

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. Suggested areas for research related to manipulations of loudness, rate, prosody and general instructions were summarized previously. Some areas of ongoing and future research in our collaborative laboratories include:

- Comparative studies evaluating the impact of training vocal loudness (LSVT LOUD) to matched treatment targeting high effort articulatory exercise (ENUNCIATE), with neural imaging of treatment-related changes [139]
- Systematically documenting the impact of DBS surgery on speech in people with PD and their response to speech therapy post-surgery
- Applying principles of successful speech therapy (LSVT LOUD) to limb motor systems and creating a combined amplitude-based speech and physical therapy program
- Evaluating the potential neuroprotective impact of exercise-based speech therapies in humans with PD
- Developing animal models of PD-like vocalization
- Increasing accessibility to speech treatment through technology

Published pilot data from training loudness (LSVT LOUD) have documented that effects generalize beyond vocal loudness to improve swallowing, speech articulation, communicative gestures, facial expression and neural functioning [59,124,140–143]. Ongoing randomized controlled studies are further examining this spread of effects by evaluating and comparing the system-wide generalized impact of the therapies (voice [LSVT LOUD] and articulation [ENUNCIATE]) on speech articulation, facial expression and swallowing in idiopathic PD, and the system-wide generalized impact of these therapies on limb gesture and limb motor functioning in PD. Functional imaging investigations (PET) of these two treatments will identify changes in
neural connectivity and functioning and identify any differences associated with different treatment targets. 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 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 [87]. People and families consistently rate problems in speech and swallowing following DBS-STN as significant and persistent. 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. The 1st International Symposium on Basal Ganglia Speech Disorders and Deep Brain Stimulation was held in 2007 at the Institute of Neurology, Queen Square, London. This meeting brought together researchers (neurosurgeons, neurologists, speech scientists and speech-language pathologists) from around the world to address the issues surrounding DBS surgery and changes in speech. Advances in surgical techniques, stimulator settings and use of behavioral speech treatments will likely emerge in the next 5 years given the concentrated focus on this area of research.

Recently, the principles of LSVT LOUD were applied to limb movement in people with PD. 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 [144,145]. 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 (big and loud) has been proposed. Results from pilot work have been completed in 11 people with PD: nine stage I (three de novo) and two stage II. Data revealed all subjects increased vocSPL (loudness) during sustained vowels and reading (average 10 db SPL increase across both tasks), and increased stride length/velocity during gait (average of 9 cm) [146,147]. The gains in vocal SPL and gait stride length were comparable to previously published data from independently training LSVT LOUD (speech; range 8–13 dB SPL) [110–148] or LSVT BIG (limbs; range 9–30 cm) [145]. These changes in speech and gait function had a positive impact of 28% on disease severity (Unified Parkinson’s Disease Rating Scale [UPDRS]-motor section) and 27% on quality of life (Parkinson’s Disease Questionnaire [PDQ]-summary score). These data document that individuals with early PD are able to improve beyond baseline levels. There is a great need to simplify rehabilitation approaches for people with PD owing to the progressive nature of the disorder, cognitive challenges that make motor learning difficult, and logistical and financial burdens that intensify speech and physical therapies present. A whole body, amplitude-based treatment program may be one possible solution. This was a Phase I study and further research is required.

Recent advances in neuroscience reveal that exercise and motor training impacts molecular changes associated with cell survival, cell growth and functional recovery in animal models of PD. This challenges the assumption that there is no potential for recovery in PD or other neurodegenerative disorders. Altogether, these background tenants emphasize the need for human studies of exercise-based programs that are continuous, immediately available at the time of diagnosis, and that promote neural plasticity and brain reorganization [79,124,126,144]. The translation of the principles of neural plasticity to therapeutic approaches will require a significant paradigm shift in rehabilitation sciences [127]. Current rehabilitation approaches are typically not developed specifically for the deficits in PD (or other neural disorders), nor do they implement the principles that promote plasticity in a standardized manner. Our ability to embrace these principles and integrate them into the mode of delivery of treatment will be essential for evolving rehabilitation science in parallel with neuroscience. We need future studies to specifically evaluate the impact of intensive behavioral speech therapy on neuroplasticity and the potential for neuroprotection as measured by dopamine-related changes in imaging studies over time. These data would have the potential to facilitate early referral to speech and physical therapies.

As discussed above, speech and voice deficits associated with PD have been largely resistant to pharmacological and surgical treatment, but have responded to intensive speech treatments. The mechanism underlying this phenomenon is not well understood and is difficult to systematically test in human models. Thus, we turn to the rodent as a model, which may be helpful, especially if human and rodent vocalizations share similar underlying neural events. Our underlying tenet for using the rat model is that the impairment in voice and speech in individuals with PD is at least partially related to phylogenically old neural mechanisms subserving phonation [45]. Initial data suggest that mild transient dopamine depletion with haloperidol or even unilateral degeneration of dopamine neurons are associated with changes in the ultrasonic vocalization (USV) acoustic signal, such as decreased frequency bandwidth [149]. USV in the rodent model of PD may be a useful model to study parkinsonian-like vocalization deficits. This work sets the stage for future research aimed at acquiring neurobiological and behavioral evidence on the effects of vocal exercise in an animal model as a translational approach from basic science to human clinical science in the area of PD speech treatment. In addition, this model offers the
ability to evaluate issues of timing and medication confounds on these neurobiological and behavioral outcomes, studies that cannot be easily done in humans with PD. Thus, animal data offer an important opportunity to advance our understanding of speech motor control in neural diseases, such as PD.

It is recognized that there are practical challenges of delivering speech treatment intensively (four individual sessions per week for 4 weeks). In fact, any treatment regime (speech, physical or occupational therapy) that is consistent with plasticity promoting principles and incorporates elements such as intensity and multiple repetitions will require going beyond the one-to-one (patient to clinician) classic paradigm of treatment delivery. There are not enough therapists to deliver this efficacious dosage of treatment to all the people with PD in need; a need which will only increase dramatically in the coming years with the aging of the baby boomer population. Furthermore, continued exercise following the conclusion of speech treatment and tune-up sessions may be needed for maintenance of vocal loudness as the disease progresses. Moreover, intensive treatment can be costly, especially for the elderly, whose source of income might be limited. Thus, there is a need for expanding service delivery while containing costs and human resources.

Key issues

- Speech and voice disorders negatively impact people with Parkinson’s Disease (PD)
  - Between 85 and 90% of individuals with PD develop voice and speech disorders during the course of their illness. Many patients develop speech and voice problems early in the course of their disease. These disorders, along with reduced facial expression, adversely affect communication and quality of life.
  - There are limited effects of medication, neurosurgery, and traditional speech on voice and speech disorders in PD
  - Neurosurgical and levodopa treatments for PD have yielded minimal, inconsistent or adverse effects on voice and speech functions.
- Current reviews of speech treatment evidence suggest more data are needed to clarify the most effective speech treatment approach in PD.
- Criteria for reviewing behavioral treatment evidence may differ from pharmacological studies owing to difficulties with concealed placebo treatment. The use of comparison treatments and statistical analysis with smaller sample sizes may be warranted.
- Speech treatment that has a focus on vocal loudness and pitch or increasing vocal effort and loudness is recommended for people with PD.
- Effects of the Lee Silverman Voice Therapy have greatest amount of data supporting impact on voice, speech and other orofacial functions, as well as on brain reorganization.
- Future areas of research in treatment studies, neural imaging, deep brain stimulation, neural plasticity and animal models of vocalizations will help clarify neural basis of speech disorders in PD and guide development and refinement of speech treatment approaches, and support early referral to speech treatment.

Advances in computer and web-based technology offer potentially powerful solutions to the problems of delivering an intensive efficacious dosage of treatment, treatment accessibility and long-term maintenance in rehabilitation. For example, a computer training application for upper limb motor deficits following stroke has been developed for delivery of constraint induced therapy, a program which requires intensive motor training (e.g., 6 h/day for 2 weeks). This computerized system, called AutoCITE, was documented to result in comparable outcomes to live delivery of the therapy [150]. Computer technology has also been developed for delivery of an intensive speech treatment (LSVT LOUD) and is discussed below.

Halpem and colleagues [151, 152] reported on the use of a personal digital assistant as an assistive device for delivering LSVT LOUD to people with PD. The LSVT LOUD companion (LSVT LOUD-C) is specially programmed to collect data and provide feedback as it guides people through the treatment exercises, enabling them to participate in therapy sessions at home. A total of 15 people with PD participated in a study during which nine voice treatment sessions were completed with a speech therapist and seven sessions were completed independently at home utilizing the LSVT LOUD-C. Data revealed findings similar to previously published data on 16 face-to-face sessions both immediately post-treatment and at 6-month follow-up [151–152]. An evolution of the LSVT LOUD-C has been the development of a virtual speech therapist (LSVT LOUD-VT). This is a perceptive animated character, modeled after expert LSVT speech therapists, that delivers LSVT LOUD in a computer-based program [153]. A prototype of the LSVT LOUD-VT has been developed and clinical testing has begun. In addition, delivery of intensive speech therapy, such as LSVT, via Telehealth systems or other web-enabled speech therapy systems have documented positive outcomes in people with PD and will enhance accessibility to the intensive sensorimotor training important for successful speech outcomes.

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, and in some cases have exacerbated or resulted in voice and speech impairment. Thus a combination of behavioral speech therapy, specifically the LSVT LOUD 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 5 years.
Financial & competing interests disclosure

Lorraine Ramig is a Professor at the University of Colorado-Boulder, Senior Scientist at the National Center for Voice and Speech (Denver) and Adjunct Professor, Columbia University, New York City. Cynthia Fox is a Research Associate at the National Center for Voice and Speech in Denver and Research Lecturer in the Department of Neurology at the University of Arizona. Shimon Sapir is an Associate Professor at the University of Haifa. This research has been funded in part by NIH grants R01 DC1150 from National Institutes of Deafness and other Communication Disorders (Ramig, Fox and Sapir).

Lorraine Ramig receives a lecturer and travel honorarium from the LSVT Foundation (nonprofit organization), a consulting honorarium from the Kinetics Foundation, receives a lecture honorarium and has intellectual property rights and ownership interest in LSVT Global LLC (for-profit organization that runs training courses and sells products related to LSVT Treatment).

Cynthia Fox receives a lecturer and travel honorarium from the LSVT Foundation (nonprofit organization), receives lecture honorarium and has intellectual property rights and ownership interest in LSVT Global LLC.

The authors have no other relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript apart from those disclosed.

No writing assistance was utilized in the production of this manuscript.

References

Papers of special note have been highlighted as:
• of interest
** of considerable interest

• Documents decreased laryngeal muscle activity (thyroarytenoid muscle) in people with Parkinson disease (PD) as compared with healthy age-matched peers. This reduced muscle activation may be one factor underlying soft, monotone voice in people with PD.


- Compared videostroboscopic ratings of vocal folds in individuals with Parkinson's disease (PD) following two forms of high effort, intensive treatment: Lee Silverman Voice Treatment (LSVT®) and respiratory treatment. Only after LSVT did patients have improvements in vocal fold closure.
Speech treatment for Parkinson’s disease


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**The randomized control trial (RCT) is considered as level 1 evidence for effective speech treatment for PD. The short and long-term (2 year) impact of two forms of speech treatment (LSVT and respiratory) on speech and voice in PD were compared. Only patients who received LSVT had statistically significant increases in SPL post-treatment (vs pretreatment) that were maintained 2 years after 1 month of treatment. Patients who received respiratory treatment had no significant increases in SPL and 2 years after treatment were statistically significantly softer than the patients who received LSVT.**


**This RCT evaluates the short- and long-term (6 months) impact of LSVT on speech and voice in PD when compared with untreated PD and healthy control groups. Only patients who received LSVT had a statistically significant on post treatment sound pressure level which was maintained 6 months after treatment.**


**This paper documents changes in neural functioning through PET imaging in patients with PD following LSVT.**


Speech treatment for Parkinson’s disease

Review


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