It is a stunning time to be in rehabilitation today. The basic science evidence for the value of exercise in Parkinson’s disease (PD) has been documented in animal models of the disease and is being increasingly explored in humans with PD [1–3]. In addition, research has identified key principles of exercise that drive activity-dependent neural plasticity (modifications in the CNS in response to physical activity) [4–5]. These collective data have elevated the role of exercise and/or rehabilitation in the overall management of PD beyond a reactive referral for secondary impairments (e.g., aspiration due to swallowing impairments or hip fracture due to falling) to a legitimate therapeutic option prescribed early, upon diagnosis, that may slow or halt symptom progression [2,6]. As a result of this basic science evidence, there has been a steady increase in the number of physical therapy trials in individuals with PD from 1980 through 2010 [7], and the practice variables that have been documented to be effective in the animal models (e.g., intensity and specificity) are being reported in human trials of PD and stroke [1,3].

The purpose of this article is to examine this new paradigm of exercise-based interventions and activity-dependent neural plasticity in PD in relation to literature in speech treatment, with a focus on the Lee Silverman Voice Treatment (LSVT) as an example therapy. This article will briefly review the literature on the characteristics and features of speech and voice disorders in individuals with PD, and will discuss the impact of pharmacological and surgical treatment techniques on these disorders. This will be followed by a focus on behavioral speech treatment, specifically Lee Silverman Voice Treatment, including development of the treatment approach, documenting efficacy, discovery of unexpected outcomes and insights into the mechanism of speech disorders in PD gained from treatment-related changes. This research will be placed in the context of other previous and current speech treatment approaches in development for individuals with PD and will highlight future directions for research.

Keywords: Lee Silverman Voice Treatment • LSVT • neural plasticity • Parkinson’s disease • speech and voice disorders • swallowing disorders
Hypokinetic dysarthria in PD
Parkinson’s disease affects 1–2% of individuals over the age of 60 years, as well as younger individuals in their 30s and 40s [8]. Nearly 90% of these individuals are likely to develop speech disorders during the course of their illness [9]. These disorders, collectively called hypokinetic dysarthria [10], may include any or a combination of the following: reduced vocal loudness and abnormal vocal decay [11]; a breathy, hoarse, or harsh voice quality [12]; imprecise consonants and vowels due to reduced range of articulatory movements, with the tendency of these movements to decay and/or accelerate toward the end of a sentence [13–15]; reduced voice pitch inflections, or monotone; and rushed, dysfluent or hesitant speech [13–16]. Other less frequently occurring speech abnormalities may include palilalia and stuttering-like dysfluencies [17]. These speech problems have been traditionally attributed to muscle rigidity and hypokinesia secondary to dopamine deficiency [18]. However, as we will discuss, additional factors, such as deficits in scaling and maintaining movement amplitude, internal cueing, high-level sensory processing, neuropsychological functions (attention to action and vocal vigilance), along with nondopaminergic or special dopaminergic mechanisms, may provide a more comprehensive or complimentary explanation for the etiology of the dysarthria.

Physiologic abnormalities associated with voice & speech disorders in PD
Studies of vocal function in individuals with PD have documented poor vocal fold closure; reduced amplitude, asymmetrical or slow vibratory patterns of the vocal folds and voice tremor (the latter might be a form of essential tremor) [19–20]. Respiratory studies of individuals with PD have documented a reduction or abnormalities in vital capacity, chest wall movements, respiratory muscle activation patterns, amount of air expended during maximum phonation tasks and intraoral air pressure during consonant/vowel productions [21]. Electromyographic (EMG) studies of individuals with PD revealed a reduction of neural drive to the laryngeal muscles [22], abnormally elevated laryngeal muscle activity [23] or poor reciprocal suppression of laryngeal and respiratory muscles [24]. Kinematic and EMG studies of orofacial movements during various speech tasks in individuals with PD have indicated 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 [25–30]. These studies, which have included healthy control subjects for comparison, suggest that hypokinetic speech movements in PD may be associated with abnormal neural drive to the speech periphery and abnormal sensorimotor gating and may be a major cause of the speech movement abnormalities in PD.

Medical treatment for voice & speech disorders in PD
Pharmacological treatments with dopamine-replacement therapy (levodopa or dopamine agonists) has marked therapeutic effects on rigidity, akinesia, bradykinesia and tremor in the limb motor system [31]; however, its effects on hypokinetic dysarthria in individuals with PD have yielded variable findings [32–33] in terms of magnitude and long-term maintenance of treatment outcomes. While some studies have documented dopamine-induced improvement in speech motor function [23,34–38], other studies have failed to show systematic changes or clinically significant improvement in the speech of individuals with PD following levodopa treatment [33,39–42]. These findings suggest that neurochemical or neurobehavioral factors other than, or in addition to dopamine deficiency may play a significant role in hypokinetic dysarthria associated with PD, as will be discussed later [32,41].

Neurosurgical procedures such as deep brain stimulation (DBS) of the thalamus, pallidum or subthalamic nucleus, ablative surgeries (pallidotomy and thalamotomy) and fetal cell implantation have been demonstrated to result in dramatic improvement in limb motor function, yet these procedures have yielded contradictory results on speech functions in individuals with PD [42–45]. The most common neurosurgical approach used today is DBS of either the subthalamic nucleus (STN) or the internal segment of the globus pallidus (GPi). Although there have been reports of improvements in components of speech production with STN-DBS [46–49], the impact on functional speech intelligibility remains unclear [50–51]. Adverse effects of STN-DBS on speech have received increasing attention in the neurology and speech literature in recent years [52–54]. Explanations for the negative effects of STN-DBS on speech include high-frequency stimulation [55], location of electrode contacts and amplitude of stimulation in the STN [51], the spread of current to nearby fibers such as the cerebellothalamic fibers [56] and/or lesions of a neural network that mediates sensorimotor speech control [57].

Hypotheses regarding neural mechanisms underlying voice & speech disorders in PD
It has been suggested that some motor disorders in PD, including hypokinetic dysarthria, may be partially related to abnormal non-dopaminergic or special dopaminergic mechanisms, which impair several high-level processes that are important for the regulation and control of speech movements [16,32–33,53–54]. We will briefly present four high-level processes we hypothesize to be impaired and to underlie hypokinetic dysarthria in PD. These include scaling movement amplitude, sensory processing, internal cueing and vocal vigilance.

Scaling movement amplitude
One likely cause of hypokinetic dysarthria in PD is reduced amplitude of output (hypokinesia) and abnormal scaling and maintenance of movement amplitude. Reduced range (hypokinesia) of respiratory, laryngeal and orofacial movements during speech sound production in individuals with PD, with the tendency of the amplitude of the movements to decay (become more hypokinetic) within and across utterances, have been documented in various studies [11,14,25,58–59]. This reduction may manifest as a systematic reduction and decay of vocal loudness, pitch intonation and precision of vowels, consonants and other sounds of speech [14–15,58,60–62], and may be attributed to the inability of individuals with PD to scale and/or maintain movement amplitude, force or related parameters [63–68].
**Sensory processing**

A second factor is abnormality in sensory processing. Behavioral and physiological studies of speech and nonspeech oral and head and neck functions in individuals with PD have documented sensory abnormalities, manifested as errors on tasks of kinesthesia [69–71]; difficulties with orofacial perception, including decreased jaw proprioception, tactile localization on tongue, gums and teeth, and targeted and tracked head movements to perioral stimulation [72]; problems utilizing proprioceptive information for normal movement [70,72]; abnormal higher order processing of afferent information as demonstrated by abnormal reflex and voluntary motor responses to proprioceptive input [27,73–75]; and abnormal sensory processing of voice and speech in individuals with PD [25,76–80]. Laryngeal sensorimotor deficits in PD have also been documented [81,82]. These sensorimotor abnormalities may account, at least partially, for the deficits in scaling and maintaining speech movement amplitude.

One aspect of sensory processing deficits in individuals with PD is misperception of self-produced voice and vocal effort. Individuals with PD are often unaware of the magnitude of their reduced vocal loudness and will report, ‘my voice is fine, but my spouse needs a hearing aid’ [83]. This is similar to the inaccurate perception of body awareness and lack of self corrections of smaller and slower movements in everyday living, even in early PD [2]. Furthermore, when individuals with PD and hypokinetic dysarthria are asked to produce ‘loud’ speech (i.e., increase amplitude of motor output), they typically increase their speech to a normal conversational volume level, yet they complain that this louder voice is ‘way too loud’. This phenomenon has been documented experimentally by Ho and colleagues [79]. These researchers found that even though individuals with PD spoke with a softer voice than healthy controls, they nevertheless perceived their own speech to be louder than that judged by the healthy controls. In addition, individuals with PD overestimated the loudness of their speech during both reading and conversation. Furthermore, sensorimotor abnormalities in auditory–vocal feedback and feed-forward mechanisms have been indirectly demonstrated in individuals with PD by behavioral [84] and neurophysiological [85] studies. Finally, Ramig and colleagues [86–88] have shown that addressing problems of sensory processing deficits is an important therapeutic goal for a successful treatment of hypokinetic dysarthria in individuals with PD [86–88].

**Internal cueing**

A third factor is deficits in internal cueing. One of the most striking characteristics of hypokinetic dysarthria in individuals with PD is the dramatic improvement in voice and speech when these individuals are externally cued or instructed to speak loudly and or clearly. The improvement in voice and speech with external cuing is most likely a compensatory response to deficits in internal cuing [89]. This conclusion is empirically supported by a series of experimental studies conducted by Ho and colleagues [77–79]. It is also consistent with the phenomenon of micrographia (the abnormally small handwriting) in individuals with PD, which tends to improve dramatically (though transiently) when these individuals are verbally instructed to ‘write big’ or when they are provided with dots or lines on the paper, and asked to write so that the letters touch the dots or the lines (external cues) [90]. The use of external cuing has also been demonstrated to positively impact gait in PD, with long-term retention, when cueing strategies were systematically trained over several weeks’ time [91].

**Vocal vigilance**

A fourth factor is impairment in self-monitoring and self-regulation of voice and speech motor output due to deficits in attention to action or vocal vigilance. There is evidence that attention to action is impaired in individuals with PD, and that this impairment may contribute to the abnormal control of movements in PD [92,93]. By inference, impaired vocal vigilance in individuals with PD may have an adverse effect on motor speech control, which may partially account for the hypokinetic dysarthria in these individuals [54]. Importantly, it has been shown that attention to action in individuals with PD can be improved significantly by intensive training [80,94]. As will be discussed later, the LSVT treatment regimen, which has been designed to train individuals with PD to pay attention to their speech output and to monitor the effort to produce this output, has been shown to be effective in the long-term maintenance of treatment outcome [87,95].

**Summary of origins of speech & voice disorders in PD**

To summarize, the neural mechanisms underlying speech disorders in individuals with PD appear to be complex and not well delineated. Additional research is needed to explore these deficits and their interaction and contribution to the speech disorders observed in individuals with PD. Given this complexity, it is not surprising that traditional medical and behavioral treatments have had only limited success in managing these problems and in improving speech in the long term. New insights about the nature of speech disorders in PD have been gained from testing new hypotheses regarding the pathogenic mechanisms underlying these disorders and from studies of treatment effects, such as those induced by the LSVT regimen.

**Behavioral speech treatment for individuals with PD**

Historically, speech and voice disorders in individuals with PD were thought to be resistant to behavioral speech therapy [96–99]. This view of speech therapy mirrored the view of physical therapy and exercise in that it was perceived as not helpful (see [2] for full historical physical therapy review). In the mid 1980s and early 1990s there began to be a shift in the view of both physical and speech therapy for individuals with PD. In speech treatment, studies in the UK and the USA reported on speech treatment protocols that documented positive impact on voice and speech outcomes [100–103] (see [35] for more detailed review). Two consistent features of these studies emerged and were later recommended as key treatment components to focus on in speech therapy in PD: high dosage of the therapy (almost daily therapy for 2–4 weeks) and a focus on improving the voice either through exercise of prosody, loudness or a combination of these [104]. To our knowledge, of these published initial promising studies, only the Ramig et al. protocol (known today as LSVT) was standardized and
Further researched to assess treatment efficacy in the short and long term. Today, there are more than 20 years of research on the development and research process of the LSVT program for people with PD. Thus, the study of LSVT has provided a unique opportunity to examine a behavioral treatment regimen through the various phases of efficacy research [105], as well as to improve our understanding of motor speech disorders in PD and their neurophysiologic underpinnings.

The Lee Silverman Voice Treatment
Initial development of the LSVT began in the late 1980s under the direction of Lorraine Ramig and her former student Carolyn Bonitati at the Lee Silverman Center for Parkinson's disease in Scottsdale (AZ, USA). Initial Phase I and II studies were completed where the protocol of LSVT was established and tested in clinical conditions. These initial data were reported in 1988 by Ramig and colleagues [106] and the standardized protocol was introduced as the LSVT in honor of Mrs Lee Silverman, a woman with PD. These initial data sets were the foundation for the Phase III efficacy studies that were completed in the 1990s. Two randomized controlled trials were completed. The first study compared two treatments, both designed to improve vocal loudness in people with PD. One treatment focused on increasing respiratory drive and vocal fold adduction (known today as LSVT) and the other treatment focused on increasing respiratory support only. Both treatments were matched for intensity of dosage, positive reinforcement from the clinicians, and homework and carryover assignments. Additional details can be found in Ramig et al. [106]. Subject groups were studied immediately before treatment, after treatment and at 6, 12 and 24 months after treatment (with no additional therapy). Multiple acoustic, perceptual, aerodynamic and neuropsychological measures were collected. These data were published in a series of studies [87–88,103,107]. The primary treatment outcome variable from these studies was vocal sound pressure level (vocSPL). In this series of studies, LSVT was superior in improving vocal loudness and vocSPL over the alternative treatment approach. In the second randomized controlled trial, LSVT was compared with an untreated group of individuals with PD and an age- and sex-matched healthy control group at three time points: before treatment, after treatment and at 6 months after treatment. This study was published in 2001 [88]. We have summarized the primary outcome variable of vocSPL across this series of studies and added effect size values in Table 1. As can be seen, in most studies, the effect size was larger than 0.80, indicating that the changes induced by the LSVT are clinically highly significant [108].

Review of secondary studies/outcomes
In the process of documenting treatment efficacy, there were a series of secondary studies and discoveries that occurred. Most notably, the recognition of the distributed effects across the speech production system following LSVT that extended beyond vocal loudness [20] to include improved articulation [15,61,109], facial expression [110] and swallowing efficiency [111]. These distributed effects have been of great interest, given the potential clinical efficacy of a single treatment target (vocal loudness) to improve multiple speech system disorders. In addition, it became clear that it is important to distinguish between acute responses to a single episode of physical activity (i.e., stimulating increased loudness) from chronic effects associated with long-term adaptation accompanying repetitive physical exercise (i.e., training increased loudness) [112]. Across a series of stimulated vocal loudness studies, we have documented short-term improvements in a number of voice, respiratory and articulatory measures in nondisordered and disordered speakers [113–115], as have other authors examining stimulated vocal loudness [116,117]. However, Liotti et al. documented that changes in brain activation in five subjects with idiopathic PD occurred only following training vocal loudness (LSVT) for 1 month [118]. These changes were not observed before treatment with brief stimulated increases in loudness, as will be discussed later. Will et al. also documented a difference between stimulated and trained loudness, reporting that only in the trained condition (not the stimulated condition) did significant acoustic differences in vowel space accompany increased loudness in individuals with PD [119]. Taken together, these findings suggest that while stimulating loudness does impact speech production in the short term [120], lasting changes in speech–motor coordination and neural reorganization appear to require intensive training. This is consistent with basic research studies that suggest there is a need for continued practice of a new motor skill (e.g., intensive training) for long-term structural changes in neural functioning [121]. Acquisition alone is not sufficient for sustained neural plasticity (i.e., resistance to decay) nor may it be sufficient for transfer and carryover outside the therapeutic environment.

Brain imaging studies using O9 PET have also documented marked changes in brain function in conjunction with speech improvement following LSVT [85,118,122,123]. Specifically, while stimulated loud phonation prior to the administration of LSVT activated cortical premotor areas, particularly the supplementary motor area (SMA), after LSVT, SMA activity was normalized, and increased activity in the basal ganglia (right putamen) suggested a shift from abnormal cortical motor activation to normal subcortical organization of speech–motor output. The excessive activity in the auditory cortex before LSVT and its reduction to a normal level after LSVT, as discussed earlier, also suggests improvement in brain function. The post-LSVT changes also indicated an increase in activity in right anterior insula and right dorsolateral prefrontal cortex, suggesting that LSVT may recruit a phylogenetically old, preverbal communication system involved in vocalization and emotional communication (consistent with the multisystem effects of LSVT). Finally, the shift in brain activity from left to right hemisphere and in regions that involve attention to action and self-monitoring of action [122] indicates that the long-term maintenance of treatment outcomes may, at least partially, be related to improvement in vocal vigilance.

Fundamentals of LSVT: target, mode & calibration
Lee Silverman Voice Treatment is a PD-specific, standardized, research-based approach and differs from the way in which traditional speech therapy was delivered at the time LSVT was developed. Table 2 highlights these key differences. The LSVT regimen is unique in a number of key ways. First, the target...
of treatment is the voice. Specifically, LSVT trains amplitude (increased vocal loudness) as a single motor control parameter. Second, the mode of treatment delivery is intensive and requires high effort, consistent with principles that drive activity-dependent neural plasticity [124], theories of motor learning [125] and skill acquisition [126]. Third, calibration addresses the sensory, internal cueing and neuropsychological deficits (e.g., impaired attention to action or vocal vigilance) that can make generalization and lasting treatment effects difficult to obtain for individuals with PD.

Target: vocal loudness (amplitude)

We hypothesize that training-induced increases in movement amplitude (i.e., vocal loudness) target the proposed

<table>
<thead>
<tr>
<th>Study</th>
<th>Treatment groups</th>
<th>Subjects (n)</th>
<th>SPL ‘AH’ Before</th>
<th>SPL ‘AH’ After</th>
<th>Effect size</th>
<th>SPL Rainbow Before</th>
<th>SPL Rainbow After</th>
<th>Effect size</th>
<th>SPL Monologue Before</th>
<th>SPL Monologue After</th>
<th>Effect size</th>
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<td>Ramig et al.‡</td>
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Note the total subject numbers for Monologue or Rainbow passage are reduced across both treatments in some studies. The data include the number of subjects in each treatment group, the mean (SD) vocal sound pressure level (in dB) before treatment, after treatment and at follow-up, and the effect size measure of the difference between before and after or before and follow-up. According to Cohen [108] effect size >0.80 is considered a highly significant difference (clinically speaking), a value of 0.50 indicates medium difference, and a value 0.20 indicates a small difference. Note the large effect size values for the LSVT and the small effect size values for the other treatment (RESP) or no treatment (UNTX). There are some exceptions to the subject numbers given in the table, and these are as follows: Ramig et al. [175]: Monologue LSVT 13, RESP 8; Ramig et al. [87]: Rainbow passage RESP 11; Monologue LSVT 12, RESP 6. LSVT: Lee Silverman Voice Treatment; mo: Month; RESP: Speech therapy with the same schedule and intensity as LSVT, but with emphasis only on respiratory effort, rather than on respiratory-phonatory effort, which is the basis of the LSVT regimen; SPL: Sound pressure level; UNTX: The group of individuals with Parkinson’s disease who did not receive speech therapy at the time of the study.

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Table 2. Traditional speech therapy versus Lee Silverman Voice Treatment LOUD therapy for individuals with Parkinson’s disease.

<table>
<thead>
<tr>
<th>LSVT LOUD</th>
<th>Traditional speech therapy</th>
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<tr>
<td>Ramig et al. [87–88,103]</td>
<td>e.g., Till et al. [176]</td>
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**Intensity**

<table>
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<th>LSVT LOUD</th>
<th>Traditional speech therapy</th>
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<tbody>
<tr>
<td>Standardized</td>
<td>Variable</td>
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**Dosage**

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<th>Traditional speech therapy</th>
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<tr>
<td>4 days per week for 4 weeks (16 sessions in 1 month)</td>
<td>Twice per week for 4–16 sessions over several months</td>
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**Repetitions**

<table>
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<tr>
<td>Minimum 15 repetitions per task</td>
<td>Variable per task</td>
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**Effort**

<table>
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<th>LSVT LOUD</th>
<th>Traditional speech therapy</th>
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<tr>
<td>Push for maximum patient-perceived effort each day (8 or 9 on scale of 1–10 with 10 being the most)</td>
<td>Moderate to low exertion, reactive to patient response to treatment</td>
</tr>
</tbody>
</table>

**Focus**

<table>
<thead>
<tr>
<th>LSVT LOUD</th>
<th>Traditional speech therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simple focus: LOUD</td>
<td>Complex focus:</td>
</tr>
<tr>
<td>Increased movement amplitude directed predominately to respiratory/laryngeal systems</td>
<td>Voice, respiration, articulation, rate, posture, resonance, orofacial movement, pacing, intonation</td>
</tr>
</tbody>
</table>

**Daily tasks**

<table>
<thead>
<tr>
<th>LSVT LOUD</th>
<th>Traditional speech therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>First half of the treatment session (25 min)</td>
<td>• Variable from therapist to therapist – no standardized protocols</td>
</tr>
<tr>
<td><strong>Task 1</strong>: maximum sustained movements:</td>
<td>• Variable from day to day (e.g., one session focus on voice loudness and intonation, next session focus on pacing, next session orofacial movements and posture)</td>
</tr>
<tr>
<td>• 15 reps: sustain ‘ah’ in good-quality, loud voice as long as possible</td>
<td></td>
</tr>
<tr>
<td><strong>Task 2</strong>: directional movements:</td>
<td></td>
</tr>
<tr>
<td>• 15 reps each: say ‘ah’ in loud good-quality voice going high in pitch</td>
<td></td>
</tr>
<tr>
<td>• 15 reps each: say ‘ah’ in loud good-quality voice going low in pitch</td>
<td></td>
</tr>
<tr>
<td><strong>Task 3</strong>: functional movements:</td>
<td></td>
</tr>
<tr>
<td>• Patient self-identifies ten phrases or sentences he/she says daily in functional living (e.g., ‘Good morning’), five reps of the list of ten phrases. Read phrases using same effort/loudness as you did during the long ‘ah’</td>
<td></td>
</tr>
</tbody>
</table>

**Hierarchy**

<table>
<thead>
<tr>
<th>LSVT LOUD</th>
<th>Traditional speech therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Second half of the treatment session (25 min)</td>
<td>• Speech practice often does not relate back to CORE exercises focused on rescaling effort, strength or some other movement variable</td>
</tr>
<tr>
<td>• Designed to train rescaled amplitude/effort of movement achieved in CORE exercises from daily tasks into context-specific and variable speaking activities</td>
<td>• Progression across treatment sessions of increasing complexity of speech tasks, often starting with speech that is more complex than single words</td>
</tr>
<tr>
<td>• Incorporate multiple repetitions with a focus on high effort (e.g., list of 20 phrases/sentences repeated ten times for 200 repetitions)</td>
<td>• Diffuse focus during practice, for example, think about taking a deep breath, articulate, increase loudness and/or slow down</td>
</tr>
<tr>
<td>• Tasks increase complexity across weeks (words &gt; phrases &gt; sentences &gt; reading &gt; conversation) and can be tailored to each subject’s goals and interests (e.g., golf vs cooking)</td>
<td></td>
</tr>
<tr>
<td>• Tasks progress in difficulty by increasing duration (maintain LOUD for longer periods of time), amplitude (loudness – within normal limits) and complexity of tasks (dual processing, background noise and attentional distracters)</td>
<td></td>
</tr>
</tbody>
</table>

**Shaping techniques**

<table>
<thead>
<tr>
<th>LSVT LOUD</th>
<th>Traditional speech therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Train vocal loudness that is healthy (i.e., no unwanted strain or excessive vocal fold closure).</td>
<td>Typically use extensive verbal explanations, modeling behavior, biofeedback</td>
</tr>
<tr>
<td>• Shape the quality and voice loudness through use of modeling (‘do what I do’) or tactile/visual cues.</td>
<td>• Complex for patient</td>
</tr>
<tr>
<td>• Minimal cognitive loading: behavior is not achieved through extensive instructions or explanations (that are often too complex for patient to generalize outside of treatment room) but through modeling</td>
<td></td>
</tr>
</tbody>
</table>

LSVT: Lee Silverman Voice Treatment.
pathophysiologic mechanisms underlying bradykinesia/hypokinesia – that is, inadequate muscle activation [127]. 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 gain in the motor command [66,127]. In addition to increasing movement amplitude, the distributed effects of vocal loudness training on articulation, facial expression and swallowing are consistent with the concept of global parameters [128–130]. The neurological bases of such global motor effects are not known; however, vocal loudness might be regulated, or at least influenced, by a biomechanical and neurophysiologic linkage between the articulatory and phonatory systems [131–135]. Specifically, articulatory positions or movements have been shown to influence laryngeal muscle activity, vocal fold closure, laryngeal tension, transglottal air flow, maximum air flow, declination rate, and air pressure, with some of these adjustments also correlating strongly with vocSPL or vocal loudness level [131–135]. Whether these articulatory influences on laryngeal function are intentional is not clear [132]. In singers, at least, it appears that articulatory adjustments are intentionally used to influence laryngeal function; for example, in the control of vibrato, vocal loudness and pitch, as argued elsewhere [135,136]. Additional evidence of the orofacial articulatory–laryngeal interaction is provided by a study showing that treatment of hypertensive voice disorders in otherwise healthy individuals improves both phonation and vowel articulation [137].

Another explanation for the distributed and lasting impact of LSVT is that the target involves and stimulates phylogenetically old neural systems, especially the emotive brain, which play an important role in vocalization and the intensity of vocalization, both important parts of the survival mechanism [138]. As noted earlier, speech production is a learned, highly practiced motor behavior, with many of its movements regulated in a quasi-automatic fashion; loudness scaling is a task that both animals and humans engage in all their lives [77,79,139–141]. Thus, the regulation of vocal loudness for speech may involve a system that has been adapted, through learning, for speech audibility and intelligibility purposes [142–145]. Physiologic evidence suggests that the three subsystems of speech – respiration, phonation and articulation – are involved in vocal loudness regulation in a highly orchestrated, integrated, quasi-automated manner [134,144,145]. The central mechanism underlying this regulatory system is not clear. We have hypothesized that this system involves a phylogenetically old neural network that regulates emotive vocalization and that is subjugated to higher neural networks involving linguistically driven motor control [146]. Indeed, brain lesions and/or brain stimulation in monkeys and humans indicate that the emotive limbic system and its connections to the medial prefrontal cortex, thalamic, parathalamic, basal ganglia, periaqueductal gray and reticular formation networks are involved in the regulation or mediation of vocal intensity control [138,143,147–150].

It is important to point out that LSVT aims to produce healthy vocal loudness and speech clinicians are trained to increase vocal loudness without any strain or hyperfunction. de Swart et al. have claimed that the LSVT results in vocal hyperfunction [151]. However, their claim is not substantiated empirically as they did not study the effects of LSVT on the voice; rather, they tested a single session of loud voice, which is far from the 16 carefully planned sessions of training of healthy phonation in the LSVT regimen [152]. In fact, post-LSVT videostroboscopic data [50] and perceptual ratings of voice [12] indicate improved laryngeal function and voice quality rather than vocal hyperfunction or deterioration following treatment. These multilevel findings (physiological, acoustic and perceptual) support healthy vocal loudness in patients who receive LSVT. This is achieved by teaching LSVT clinicians how to increase loudness in patients with PD without causing any vocal strain or hyperfunction. See Table 2 for a description of the shaping techniques that are inherent in LSVT exercises and prevent any hyperfunctional behaviors during treatment.

Table 2. Traditional speech therapy versus Lee Silverman Voice Treatment LOUD therapy for individuals with Parkinson’s disease.

<table>
<thead>
<tr>
<th>LSVT LOUD</th>
<th>Traditional speech therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ramig et al. [87-88,103]</td>
<td>e.g., Till et al. [176]</td>
</tr>
</tbody>
</table>

**Sensory calibration**

**Treatment**: focus attention on how it feels and sounds to talk LOUD

**Carryover activities**: start day 1; daily assignments (treatment and nontreatment days); use loud voice in real-life situations; difficulty of the assignment matches the level of the hierarchy where the person is working; make patient accountable and look for comments from patient that people in their daily living have said, such as, ‘I can hear you better’

- No systematic approach to sensory problems associated with speech disorders in Parkinson’s disease
- Carryover activities are typically used as a part of generalization. They do not typically start the very first day of treatment; rather after some period of acquisition of the trained skill

**Homework**

**Start day 1**: daily assignments to practice at home (daily tasks and hierarchy exercises); treatment days (one other time for 5–10 min); nontreatment days (two times for 10–15 min); homework book provided and patient made accountable

**Homework exercises are typically provided. Frequency and accountability for doing homework varies**
Mode: intensive, high-effort therapy
The mode of delivery of LSVT is intensive and high effort. It is consistent with theories of motor learning, skill acquisition and principles that drive activity-dependent neural plasticity. The LSVT dosage involves 60-min individual treatment sessions, 4 days a week for 4 consecutive weeks. In addition, there are daily homework practice and carryover assignments on all 30 days of the month.

Within each treatment session, the first half of the session is spent on three daily tasks, which are core exercises designed to rescale the amplitude of motor output. These daily tasks are completed with multiple repetitions (e.g., minimum of 15 repetitions per task per day), and continually increased requirements for effort, consistency and accuracy of vocal loudness [86]. The second half of the session is spent on the speech hierarchy. The rescaled vocal loudness achieved in the daily tasks is now systematically trained into speech. Across the weeks of the hierarchy the duration of time that a person needs to keep their voice loud and the complexity of the speech task increase from words/phrases, to sentences, continuous reading and finally, conversational speech.

Within the structured setting of the LSVT treatment sessions, many elements of exercise that promote activity-dependent neural plasticity are embedded [4–5]. Tables 2 & 3 highlight these elements. Key elements of exercise that promote neural plasticity, neural protection and neural restoration in animal models included intensive training of motor tasks, increased practice of motor tasks, active engagement or salience of tasks, complexity of tasks, saliency, and the sensory experience of the motor task [53,54]. In LSVT, for example, intensive treatment is achieved both across and within treatment sessions. The dosage across treatment sessions is four individual, 1-h treatment sessions 4 consecutive days a week for 4 weeks. Within treatment sessions, patients are pushed to high vocally nonabusive intensity levels through increased repetitions of treatment tasks (e.g., minimum of 15 repetitions per task), driving high effort and continuous motor speech exercise (e.g., patients report self-perceived high effort during tasks), and increasing accuracy requirements as treatment progresses for achieving target goals. While the LSVT protocol is standardized, saliency is achieved by tailoring speech materials and homework and carryover assignments to each individual’s interests, hobbies and communication goals. Saliency is important because the more meaningful or rewarding a task, the greater potential impact on neural plasticity [55,56]. Complexity of tasks is built into the speech hierarchy exercises, moving from simple reading of words and phrases from paper, to carrying on conversational speech outside of the treatment room in a noisy environment (e.g., cafeteria). Finally, the sensory experience of increasing vocal loudness is a major focus of calibration, as discussed later.

Calibration: sensory, internal cueing & neuropsychological barriers to generalization
As discussed earlier, the etiology of the speech and voice disorder in individuals with PD is complex. It appears that addressing the motor deficit in isolation is not sufficient for lasting treatment outcomes that generalize beyond the treatment room [83]. Thus, LSVT specifically trains individuals with PD to ‘recalibrate’ their motor and perceptual systems so that they are less inclined to downscale (reduce amplitude) speech movement parameters. In addition, it is geared toward overcoming or compensating for deficits in internal cueing, vocal vigilance and self-regulation of vocal effort during speech production. We hypothesize that LSVT re-trains amplitude scaling via intensive sensorimotor training that teaches patients to recognize the effort for louder speech, and use it during everyday living. By directly addressing this sensory mismatch, LSVT teaches subjects with PD to recalibrate their perception of normal loudness and vocal effort so that by the end of 1 month of therapy, they spontaneously speak with greater vocal loudness. A few examples of specific treatment tasks related to calibration include recording the patient’s voice while reading with a louder voice that they self-perceive as ‘too loud’ and then playing it back to them. They recognize and comment when they hear the audiotapes that what felt and sounded too loud to them while reading actually sounds within normal limits (or in some cases still too soft) when played on the tape. In addition, carryover activities are assigned daily that require patients to use their louder voice in a specific communication setting outside of the treatment room, such as, order dinner at the restaurant, or greet coworkers. Convincing patients to speak in a voice that they previously perceived is too loud and having positive reactions and communication in their daily living, help convince them that the louder voice is actually within normal limits. This improvement may be due to some restoration of the internal cueing mechanism, and/or compensation through vocal vigilance. Although the treatment delivery is complex for the clinician who is administering the therapy, the focus for the individual with PD is kept purposefully simple and redundant (think loud, speak louder), which may maximize the ability of individuals with PD to learn this one target. If one treatment target can make an impact across the speech production system, this may allow us to improve the efficiency and effectiveness of treatment.

To date, LSVT is the behavioral therapy with the most data to support positive outcomes addressing the type of speech impairments experienced by individuals with PD, as judged by the members of the USA Academy of Neurologic Communications Disorders and Sciences (ANCDS) [57] and others [58]. Specifically, the LSVT has been shown to significantly improve (statistically, and in terms of effect size measures) laryngeal function, vocal loudness, voice quality, prosodic voice fundamental frequency (and its perceptual correlate pitch), inflection, vowel articulation, speech quality and overall speech intelligibility (e.g., [12,15,20,61,87–88,95,159]; see also reviews in [53,54]). Furthermore, the LSVT has been shown to improve facial expression, swallowing and tongue function [110–111,160]. In addition, these LSVT-induced changes in speech and nonspeech orofacial functions have been shown to be associated with improved brain function [159,161,162]. Moreover, improvement in vocal function owing to LSVT has been shown to be maintained over months and up to 2 years of follow-up [87,95]. The effectiveness of the LSVT program may be variably compromised by several factors, including atypical Parkinsonism, coexisting dystonia, depression, dementia, abulia and adverse
neurosurgical effects. Although data have been reported documenting improvements in individuals after thalamotomy [165], fetal cell transplant and DBS [164–166], as well as in individuals with multisystem atrophy, progressive supranuclear palsy and Parkinson’s plus syndromes [167], these outcomes may not be of the same magnitude as in idiopathic PD and may require follow-up treatment sessions to maintain improvements over time. Nevertheless, the functional impact for individual patients can be quite significant in these more advanced or complicated cases of PD. There are a number of ongoing studies examining the impact of alternative cues for speech in PD (e.g., clear speech) and the use of external auditory devices to either cue vocal loudness or improve speech fluency [158]. It may be that for more complicated speech disorders associated with PD, a combination of approaches will be the most effective.

### Ongoing research with the LSVT & speech treatment in PD

The aforementioned findings and recent pilot data indicate that training to increase vocal loudness with the LSVT regimen generalizes beyond laryngeal function to improve speech articulation [55,61], tongue function [160], swallowing [111], communicative gestures [168], facial expression [110] and neural functioning [61,85,118,123]. Randomized controlled studies are ongoing to

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### Table 3. Translation of some of the proposed principles underlying neural plasticity to proposed deficits in Parkinson’s disease and the corresponding rationale and task in Lee Silverman Voice Treatment/LOUD.

<table>
<thead>
<tr>
<th>Principle</th>
<th>Deficit specific to PD</th>
<th>LSVT/LOUD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intensity</strong></td>
<td>Intensive, high-effort training can be difficult in PD due to sensory deficits, force control, fatigue, depression and progressive loss of cardiac sympathetic innervation</td>
<td>Train intensively 1 h per day, 4 days per week, for 4 weeks; multiple repetitions (15 or more); increase resistance, amplitude (within healthy range) effort, accuracy, and daily homework exercises. Train maximum perceived effort</td>
</tr>
<tr>
<td><strong>Complexity</strong></td>
<td>As basal ganglia pathology progresses, there is a loss in automaticity, requiring greater conscious attention to task. When required to perform dual tasks, insufficient attentional resources results in the decrement in one or both of the concurrent tasks</td>
<td>Train complexity of movement with single patient focus (LOUD) to multiple, motor tasks. Retrain automaticity of amplitude (LOUD) in familiar movements. Progress complexity over 4 weeks by varying contexts, adding dual cognitive/motor loads and increasing duration and difficulty of speech tasks (progress from words to conversation)</td>
</tr>
<tr>
<td><strong>Saliency</strong></td>
<td>People with early PD may experience lack of awareness of subtle motor deficits, depression, loss of motivation and a feeling of ‘helplessness’. Thus, they do not feel they need, or would not benefit from speech therapy</td>
<td>We train salient familiar movements (core patterns) of speech promoting success. We provide homework tasks that reinforce success of LOUD in emotionally salient social interactions. We provide extensive positive feedback</td>
</tr>
<tr>
<td><strong>Use it or lose it/use it &amp; improve it</strong></td>
<td>Deficits are subtle – not ‘red flag’ to seek speech therapy. Getting early PD patients to recognize need for exercise and then convincing them to continually exercise is challenging. Decreased physical activity may be a catalyst in degenerative process</td>
<td>Educate people with PD on subtle deficits and improve motor function that directly impacts real life. Retrain a new way of speaking in everyday life (LOUD or ENUNCIATE); thus, normal activity offers continuous exercise</td>
</tr>
<tr>
<td><strong>Timing matters</strong></td>
<td>People with early PD have subtle physical underactivity (small movements/soft voice). This may be coupled with a lack of awareness or self-correction, leading to further inactivity</td>
<td>Train people with early PD when they may not have deficits in all systems (laryngeal and orofacial). Train strategies to raise awareness/avoid neglect and increase muscle activation for normal effort/amplitude required for within-normal-limits vocal loudness</td>
</tr>
</tbody>
</table>

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Author Proof

[83]

Data taken from [83].

*Author Proof*
examine this spread of effects and its treatment specificity. Studies are also planned to assess the reasons for the heterogeneous speech outcomes following DBS-STN. Such studies may involve simultaneous quantitative measures of pre- and post-surgical speech functioning and details of surgical and stimulator optimization. Knowledge gained from these studies is likely to facilitate the development of rehabilitative speech treatment approaches for speech problems in people with DBS-STN either before surgery (as preventative) or after surgery (as rehabilitation).

We are presently assessing the effectiveness of technology-supported delivery of LSVT to increase accessibility of treatment, promote home practice, augment the effects of LSVT, reduce clinician time, cut costs of treatment and support long-term practice. Telepractice delivery of LSVT allows a patient to receive LSVT online (e.g., at home or in their office). Published data have documented that LSVT delivered online has comparable outcome data as LSVT delivered in the clinic [161,169]. In addition, a study by Tindall et al. completed a cost analysis comparing live, traditional delivery of LSVT versus a telehealth delivery of LSVT [162]. The computed mean amount of patient time and money across these two modes of delivery were strikingly different. The live delivery mode required 51 h for 16 visits (travel and therapy time), US$953.00 on fuel/mileage expenses and US$269.00 for other expenses (e.g., food). By contrast, the telepractice delivery option required 16 h of time (therapy and no travel) and no additional costs for fuel/mileage or other expenses. Recent advances in software allow patients to carry out LSVT sessions with software support. The LSVT COMPANION™ is programmed to collect acoustic data and provide feedback as it guides the patient through the LSVT exercises. Outcome data document that treatment effects are comparable when half of the sessions were delivered by software [159].

Another area of research is the role of sensory and perceptual functions in speech and voice disorders and rehabilitation. As discussed earlier, it appears that self-perception of voice in individuals with PD is defective but the nature of this defect is not well understood. Therefore, by experimentally altering the patient’s sidetone (his own voice/speech fed back to his auditory system as he speaks) and simultaneously recording the response of the patient to this manipulation we can gain insight into the nature of the auditory-vocal mechanism underlying Parkinsonian speech in general [84], and in response to treatment in particular. Using this method, we have collected pilot data on one patient with PD and found that auditory-vocal responses to experimental manipulation were absent prior to LSVT but present, as is normally observed [170], after LSVT. However, these findings must be replicated in order to ascertain their generalization to other patients with PD before and after LSVT.

**Expert commentary**

Speech disorders are highly common in individuals with PD, producing adverse effects on communication, health and quality of life. Traditionally, these disorders have been attributed to dopamine deficiency and rigidity and have been considered highly resistant to medical, surgical and behavioral forms of treatment. We hypothesize that neither dopamine deficiency nor rigidity are sufficient to account for the speech abnormalities in PD, and that additional factors involving nondopaminergic or special dopaminergic mechanisms, as well as deficits in scaling movement amplitude, sensory processing, internal cueing and self-regulatory mechanisms, add to the speech disorders in PD. We present the LSVT as a scientifically proven effective treatment for speech disorders in PD, most likely because it addresses the aforementioned deficits and because its mode of delivery is consistent with principles that drive activity-dependent neural plasticity. Neural imaging has provided preliminary evidence for neural plasticity induced by the LSVT. We argue that intensive treatment such as the LSVT should be administered at early stages of the disease. Early intervention may potentially slow speech symptom progression and can be administered before adverse conditions such as depression, fatigue or dementia occur, thus allowing optimal treatment outcomes. The feasibility of intensive treatment can be enhanced by technology (telepractice and software) to improve accessibility and to support continued practice. Research studies are needed to improve our understanding of the neural mechanisms underlying the speech abnormalities in PD, as well as the neural changes that are induced by LSVT and other modes of treatment.

**Five-year view**

In the next 5 years, it is expected that the following developments will take place:

- The significant therapeutic impact of LSVT on voice, speech and other orofacial functions, as well as on brain reorganization in individuals with a progressive disease, provides a means to explore the brain mechanisms underlying the unique symptoms of PD and the mechanisms underlying the improvement of these symptoms, including neural plasticity and, possibly, neural protection. Future studies with the LSVT or other behavioral treatments should therefore be designed to explore the role of non-dopaminergic and special dopaminergic mechanisms in voice and speech disorders, and in other motor disorders that are only partially or completely unresponsive to dopamine treatment;

- Explore animal models that may help us understand vocal motor deficits related to dopamine depletion in disorders such as PD. Two emerging models of vocal motor deficits following dopamine depletion in rodents [171] and songbirds [172] offer promise for the feasibility and value of these models;

- Explore the specific role of internal cueing, sensorimotor gating, temporal processing, motor-to-sensory (vocal-to-auditory) gating, perceptual mechanisms, vocal vigilance and self-regulation on voice and speech functions in general, and in individuals with PD in particular;

- Explore the impact of combined treatments and tasks, such as speech and gait, on functional recovery and treatment efficacy. Preliminary evidence suggests that individuals with PD trained
to increase gait amplitude (LSVT BIG) and speech loudness (LSVT) can perform both tasks simultaneously without interference from the other task and while maintaining therapeutic gains [173];

- Explore the interactions between behavioral treatment and neurosurgical or neuropharmacologic treatments;
- Explore the impact of computer-based technology (e.g., internet, virtual clinician, web camera and videophones) on treatment efficacy and availability to patients. Preliminary studies suggest that such technology is highly effective in the delivery of LSVT in individuals with PD [161–162,174].

**Key issues**

- Between 85 and 90% of individuals with Parkinson’s disease (PD) develop voice and speech disorders during the course of their illness. These disorders, along with reduced facial expression and hand gestures, adversely affect communication and quality of life.
- Neurosurgical and levodopa treatments for PD have yielded minimal, inconsistent or adverse effects on voice and speech functions. Traditional speech therapy has also yielded modest outcomes, in terms of both magnitude and long-term effects.
- To date, the most efficacious behavioral method to improve voice and speech and related orofacial functions such as swallowing and facial expression is the Lee Silverman Voice Therapy (LSVT): an intensive, 1-month treatment regimen that emphasizes upscaling speech movement amplitude and self-perception and regulation of vocal effort and loudness.
- Follow-up studies indicate that therapeutic effects of the LSVT are maintained up to 2 years after treatment.
- We suggest that voice and speech disorders in PD are related, at least partially, to complex, high-level mechanisms, reflected by deficits in scaling amplitude of speech movement patterns, sensorimotor gating, internal cueing, self-perception and regulation of vocal output and vocal vigilance. These high-level deficits are probably mediated through non-dopaminergic or special dopaminergic mechanisms. We also argue that the LSVT is effective, in part because it specifically addresses these high-level deficits, and in part because it adheres to principles of neural plasticity and motor learning.
- Brain imaging studies provide preliminary evidence for LSVT-induced changes toward normality in cortical and subcortical activation patterns and in basal ganglia dopamine activity, as well as top-down improvement in vocal vigilance, as right hemisphere activation. These findings, along with the intensive, repetitive and cognitively nondemanding features of the LSVT, suggest that this behavioral treatment can potentially play an important role in neural plasticity. However, the small number of subjects included in the aforementioned brain imaging studies of LSVT, and the lack of follow-up imaging studies of the effects of the LSVT, deems the brain imaging findings and their interpretation tentative; thus, there is an urgent need for a more extensive research to delineate the neural mechanisms underlying the short- and long-term effects of the LSVT.
- Present studies are being conducted to assess the impact of LSVT on non-speech functions, and the impact of a combined speech therapy and physiotherapy (LSVT HYBRID) on speech and limb functions by emphasizing loud phonation and big limb movements, respectively. Preliminary findings indicate that the combined approach, which is a dual task, yields results similar to those obtained with each of the treatments administered alone.
- Computer-based technology (e.g., virtual clinician treatment delivery via portable devices) and telephone and internet communication systems (e.g., remote clinician treatment delivery via Skype™) are being developed to avail treatment, enhance the effects of LSVT and to allow home practice. Preliminary findings support the use of such technology, both in terms of its therapeutic effects and its cost-effectiveness and potential availability to a large number of individuals suffering from PD.
- It has been our clinical experience that individuals with early stages of PD respond better than individuals at a later stage of PD to LSVT. In part, the advantage of early versus late intervention seems to be related to intervening factors such as severity of the disease, motor impairment, depression, cognition, motivation and fatigue, which are likely to increase as the disease progresses. Therefore, an important goal would be to study early intervention with LSVT as a means to improve communication and quality of life, optimize neural plasticity and perhaps help decelerate the disease process with regard to neural protection.

**Financial & competing interests disclosure**

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

**Perspective**

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Perspective


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