The Science and Practice of LSVT/LOUD: Neural Plasticity–Principled Approach to Treating Individuals with Parkinson Disease and Other Neurological Disorders

Cynthia M. Fox, Ph.D., CCC-SLP,[1] Lorraine O. Ramig, Ph.D., CCC-SLP,[2] Michelle R. Ciucci, Ph.D., CCC-SLP,[3] Shimon Sapir, Ph.D., CCC-SLP,[4] David H. McFarland, Ph.D.,[5] and Becky G. Farley, Ph.D., PT[6]

ABSTRACT

Our 15 years of research have generated the first short- and long-term efficacy data for speech treatment (Lee Silverman Voice Treatment; LSVT/LOUD) in Parkinson's disease. We have learned that training the single motor control parameter amplitude (vocal loudness) and recalibration of self-perception of vocal loudness are fundamental elements underlying treatment success. This training requires intensive, high-effort exercise combined with a single, functionally relevant target (loudness) taught across simple to complex speech tasks. We have documented that training vocal loudness results in distributed effects of improved articulation, facial expression, and swallowing. Furthermore, positive effects of LSVT/LOUD have been documented in disorders other than Parkinson's disease (stroke, cerebral palsy). The purpose of this article is to elucidate the potential of a single target in treatment to encourage cross-system improvements across

[1]Research Associate, National Center for Voice and Speech, Denver, CO Research Lecturer, Department of Neurology, University of Arizona, Tucson, Arizona; [2]Professor, Department of Speech, Language, Hearing Sciences, University of Colorado–Boulder; Senior Scientist, National Center for Voice and Speech, Denver Center for the Performing Arts, Denver, Colorado; Adjunct Professor, Columbia University, New York, New York; University of Colorado–Boulder, Boulder, Colorado; [3]Lecturer, University of Texas-Austin, and University of Colorado–Boulder, Boulder, Colorado; [4]Associate Professor, Department of Communication Sciences and Disorders, Faculty of Social Welfare and Health Studies, University of Haifa, Haifa, Israel, Department of Communication Sciences and Disorders, Faculty of Social Welfare and Health Studies, University of Haifa, Mount Carmel, Haifa, Israel; [5]École d'orthophonie et d'audiologie et centre de recherche en sciences neurologiques, Faculté de médecine, Université de Montréal and School of Communication Sciences and Disorders and Center for Research on Language, Mind and the Brain, McGill University, University de Montréal, Montréal, Québec, Canada; [6]Assistant Research Professor, Department of Physiology, University of Arizona, Tucson, Arizona.

Address for correspondence and reprint requests: Lorraine O. Ramig, Ph.D., CCC-SLP, Department of Speech, Language, Hearing Sciences, University of Colorado, Boulder, Campus Box 409, Boulder, CO 80303. E-mail: ramig@spot.colorado.edu.

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seemingly diverse motor systems and to discuss key elements in mode of delivery of treatment that are consistent with principles of neural plasticity.

KEYWORDS: Parkinson's disease, neural plasticity, vocal loudness

Learning Outcomes: As a result of this activity, the reader will be able to (1) identify common voice and speech disorders associated with Parkinson's disease and the need for speech treatment in Parkinson's disease' (2) describe data supporting positive treatment outcomes following LSVT/LOUD, (3) discuss the effect of a single treatment target (loudness/amplitude) on treatment outcomes, (4) explain the effect of the mode of delivery (intensity/neural plasticity) on treatment outcomes, and (5) discuss implications for clinical practice with people with Parkinson's disease and other neural disorders.

The effects of neurological disease reach beyond communication and include impairment of swallowing, limb function, and activities of daily living. Often, the rehabilitation clinician (e.g., speech, physical, or occupational therapist) approaches the motor system that relates to his or her specialty without the context of general principles of motor control. However, there is increasing evidence that common motor control processes (and perhaps common neural systems) underlie a variety of motor behaviors including those from apparently divergent motor systems such as speech, swallowing, and limb movements. Understanding these principles as well as factors that promote neurorehabilitation by capitalizing on neural plasticity will help to guide the clinician in choosing the most appropriate approach in terms of both treatment target and mode of treatment delivery. The purpose of this article is to elucidate the potential importance of a single, overriding treatment target that may encourage cross-system improvements across seemingly diverse motor systems and to discuss key elements in mode of delivery of treatment that are consistent with principles of neural plasticity. These two themes will be discussed in terms of an effective speech treatment for Parkinson's disease (PD), specifically the Lee Silverman Voice Treatment (LSVT/LOUD). The success of LSVT/LOUD may be related to the uniquely pervasive effect of training vocal loudness across the entire speech production mechanism, the intensive mode of delivery that is consistent with neural plasticity promoting principles, or some combination of the two. We will discuss the need for speech treatment in PD, data supporting positive treatment outcomes following LSVT/LOUD, the effect of a single treatment target (loudness/amplitude) on treatment outcomes, the effect of the mode of delivery (intensity/neural plasticity) on treatment outcomes, and implications for clinical practice with people with PD and other neural disorders.

THE SCIENCE AND PRACTICE OF LSVT/LOUD

The Significant Need

Oral communication is vital in education, employment, social functioning, and self-expression. Nearly 300 million children and adults worldwide have a neurological disorder that may impair their ability to communicate orally. The prevalence of disordered communication is particularly high (89%) in the nearly six million individuals worldwide with idiopathic 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 facies), contribute to limitations in communication in the vast majority of individuals with PD.[4,5] In addition, dysphagia (disordered swallowing), which may be associated with life-threatening pneumonia, has been reported in as many as 95% of individuals with PD.[6] Despite optimal medical management,[7–9]

most individuals with PD have significant speech deficits that negatively affect their quality of life.[10] Affected individuals become disabled or retire early, are forced to give up activities they enjoy, incur substantial medical costs, and have increased mortality.[11] With PD predicted to increase fourfold by 2040,[12] the value of an effective treatment for disordered communication and swallowing in this population is clear.

Progress toward a Solution

Over the last 15 years, our research team has focused on improving speech and voice in individuals with PD. The approach is known as LSVT/LOUD,[13] and our research has generated the first level 1 evidence for efficacious speech treatment in PD (Ramig et al,[14,15] and C. Goetz, personal communication, 2003). LSVT/LOUD trains amplitude (increased vocal loudness) as a single motor control parameter, thereby targeting inadequate muscle activation, the pathophysiologic mechanism underlying bradykinesia (slowness of movement) and hypokinesia (reduced amplitude of movement) in PD.[16–18] LSVT/LOUD incorporates enhancing the voice source, which is consistent with improving the carrier in the classic engineering concept of signal transmission[19]; using vocal loudness as a trigger for distributed system-wide effects across the speech production system; and recalibrating sensorimotor processes so individuals with PD integrate improved speech into functional communication. Unlike other forms of speech treatment, LSVT/LOUD requires intensive, high-effort speech exercise combined with a simple, redundant, and salient treatment target to transfer loudness into functional daily living. The standardized protocol for LSVT/LOUD adheres to 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[20] and human stroke-related hemiparesis.[21] The effectiveness of LSVT/LOUD is beginning to be globally documented for people with PD,[22–25] with speech therapists in 30 countries successfully delivering the treatment. The unique combination of targeting vocal loudness and self-monitoring of vocal loudness in a training mode that is high effort and intensive may be essential elements of why LSVT/LOUD has been successful.

Effect of LSVT/LOUD on Speech and Swallowing in PD

Initial studies on LSVT/LOUD compared it to a respiratory treatment in which both treatments were matched for mode of delivery (e.g., intensive, high effort, homework, positive reinforcement).[13–15,26] Findings documented that in individuals with PD, the combined approach of treating vocal fold adduction and respiratory drive (LSVT/LOUD) generated the greatest and most lasting positive (statistically significant) effect on vocal sound pressure level (SPL) and other laryngeal and respiratory measures.[13,27–31] These findings were consistent with perceptual data demonstrating improved loudness and voice quality.[32] There was no evidence of vocal hyperfunction posttreatment; to the contrary, LSVT/LOUD has been shown to reduce laryngeal hyperfunction[33] and improve voice quality.[32]

Further studies have documented that the intensive, high-effort, amplitude training applied to the speech motor system in LSVT/LOUD also results in distributed effects across the speech production system. These effects extend beyond phonation as discussed above[27] to include improved articulation (Sapir and colleagues, submitted for publication, 2006; Dromey et al[34]) facial expression,[35] and swallowing.[36] The effect of LSVT/LOUD on articulation has been reflected in measures of transition duration, rate, and extent.[34] A recent study by Sapir and colleagues (personal communication, 2006) examined various measures of the first (F1) and second (F2) formants of the vowels /i/, /u/, and /a/; vowel triangle area; and perceptual vowel ratings among groups of subjects with PD who received either LSVT/LOUD or no treatment and among an age-matched healthy control group. Results revealed that F2 of the vowel /u/ (F2u), and the ratio F2i/F2u, along with perceptual vowel ratings, improved posttreatment in the group who received LSVT/LOUD.[35] These findings support an effect of LSVT/LOUD on vocal tract

movement; they also indicate that the effect of loudness training may go beyond a simple scale up of effort and loudness and may affect motor speech reorganization as well. Furthermore, the effect of LSVT/LOUD on improving facial expression,[35] together with improvements in vocal loudness and intonation, underscores the relationship between face and voice that has been demonstrated in expression studies involving individuals with neurological disorders[37–42] and indicates that these communicative behaviors may have some overlapping neural mechanisms.

The effects of LSVT have been shown to improve the swallowing function in dysphagic patients.[36] An assessment of the effect of LSVT/LOUD on swallowing used standardized radiographic studies to define any changes that may have resulted posttreatment. Results indicated a 51% reduction in the number of overall oropharyngeal swallow disorders after LSVT/LOUD on several liquid and solid bolus types.[36] The nature of the swallow disorders were oral tongue and tongue base disorders resulting in prolonged oral and pharyngeal transit times, difficulty with bolus formation, and lingual and pharyngeal stasis, which resolved with LSVT/LOUD. This indicates that the effect of treatment extended to the musculature of the aerodigestive tract from the focus of the treatment on respiratory/laryngeal function. These findings are important in that they indicate that the swallow mechanism is also responsive to treatment that capitalizes on targeting increased amplitude of motor output in people with PD. It is not surprising that this occurs, as many muscles and brain regions involved in speech production share function for swallowing (see McFarland and Tremblay, this issue). Additional perspectives come from two recent positron emission tomography (PET) studies of voluntary swallowing in healthy volunteers.[43,44] These studies found that in addition to primary sensorimotor cortex (pharynx–larynx representation) and brainstem, the other region most strongly activated during voluntary swallowing was right anterior insular cortex[43,44]—one of the sites that significantly changed with LSVT/LOUD.[45] It is therefore likely that improved right anterior insular function (phylogenetically the old communication system) may also contribute to the mechanism of improved voluntary swallowing following LSVT/LOUD.

Effect of Stimulated versus Trained Vocal Loudness

These distributed effects across articulation, facial expression, and swallowing have been of great interest to us, and we have conducted several studies to evaluate the effect of both stimulated and trained (i.e., treated during a course of speech therapy) vocal loudness. The term stimulation refers to situations in which the patient is asked or instructed to perform a task, such as speaking in a loud voice (e.g., "say that twice as loud") in a single session. That is, stimulation induces a transient behavior in response to an external cue. The term training refers to a systematic and intensive program (e.g., 16 sessions of individual LSVT/LOUD therapy lasting 60 minutes each in 1 month) that is designed to change a behavior such that a person will internally cue him or herself for the behavior—the behavior will not depend on external cueing and will be sustained over a protracted time (i.e., over months or years). Thus, training involves learning, memory, and reliance on internal sources (self-cueing, self-regulation) to maintain the acquired behavior. Across a series of stimulated vocal loudness studies, we have observed improved or maintained motor stability[46] and enhanced respiratory, articulatory, and phonatory behaviors.[47] A recent study by Will and colleagues[48] documented significant acoustic differences in vowel space (acoustic working space constructed by the first and second formants of vowels /i/, /ae/, /u/, and /a/) accompanying increased loudness only in the trained condition (i.e., following LSVT/LOUD) in individuals with PD and not the stimulated condition. Liotti and colleagues[45] documented changes in brain activation in five participants with PD following training LSVT/LOUD for 1 month. These neural changes were not observed pretreatment, with brief experimenter-cued stimulated vocal loudness indicating that although stimulating loudness does affect speech production,[49] lasting changes in speech-motor coordination and

reorganization of neural control processes appear to require intensive training and a recalibration of the patient's internal sensorimotor system.

Recently, neural imaging pre- to post-LSVT/LOUD using PET (O^{15}) in both activation and connectivity studies revealed short-term changes in the speech motor network of people with PD post-LSVT/LOUD.[45,50] These data indicate that LSVT/LOUD facilitates a strategy shift during speech tasks by recruiting right hemisphere speech motor areas as well as recruiting right hemispheric multimodal sensory integration and auditory areas.[45,50] A significant finding from this work was the appearance of activity in right Brodmann area 21/22 and the superior temporal sulcus both in activation and correlation maps post-LSVT/LOUD. It was suggested that this might reflect improved self-monitoring of paralinguistic features of speech (clinically referred to as sensory/auditory recalibration) following LSVT/LOUD. These are the first imaging data of any kind to document neural changes (i.e., neural plasticity) following a speech treatment for people with PD.

Effect of LSVT/LOUD on Limb System in PD

We have discussed above that LSVT/LOUD affects several processes within and across the speech production mechanism, including facial expression and swallowing. It seems reasonable to assume that similar principles may be operating on other aspects of motor function associated with PD including those involved in limb movements (such as walking and reaching). Recently, principles of LSVT/LOUD were applied to limb movement in people with PD (Training BIG) and have been documented to be effective in the short term.[51] Similar to the LSVT/LOUD, Training BIG involves a single treatment parameter—scaling of increased movement amplitude—and training involves intense, task-specific exercises designed to capitalize on neural plasticity underlying neurorehabilitation. Specifically, training-increased amplitude of limb and body movement has resulted in improvements in amplitude of trunk rotation and gait as well as generalized improvements in the speed of upper and lower limb movements, balance, and quality of life.[51] In addition, participants were able to maintain these improvements when challenged with a dual task. The extension of this work to a novel integrated treatment program that simultaneously targets speech and limb motor disorders in people with PD (Training BIG and LOUD) has been developed. Results from pilot work in 11 people with PD revealed that all subjects increased vocal SPL (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).[52–54] The gains in vocal SPL and gait stride length were comparable to previously published data from independently training LSVT/LOUD (range 8–13 dB SPL)[13–15] or Training BIG (range 9–30 cm).[51] These data indicate that it is possible that training amplitude, as a single control parameter, may facilitate organization across divergent motor systems (speech and limb), thereby increasing the cross-system effects in people with PD (for additional information on cross-system interactions, see McFarland and Tremblay, this issue). Furthermore, the additional complexity of training dual tasks (e.g., walking Big while talking Loud) during therapy may have the potential to increase neural plasticity associated with treatment outcomes.

Effect of LSVT/LOUD beyond PD

Although LSVT/LOUD was developed to target the proposed pathophysiologic mechanisms underlying speech disorders in PD, it is important to note that this approach has been successfully applied to a range of other neurological disorders. Outcome data from application of LSVT/LOUD to a series of individuals with severe speech problems accompanying Parkinson's plus syndromes (Shy Drager, multisystem atrophy), as well as post–deep brain surgery, revealed positive outcomes in these very challenging patients.[55,56] In addition, LSVT/LOUD has been applied successfully to select individuals with the following conditions: cerebellar ataxia,[57] multiple sclerosis,[58] stroke,[59] and aging voice.[60] Of particular interest was the evidence of improved vocal loudness and quality, as well as the distributed effects of

loudness training as measured in some of these individuals.[57] The effect of LSVT/LOUD has also been examined in pediatric populations, documenting improved vocal loudness, voice quality, and articulatory precision in speech of children with cerebral palsy[61] and Down syndrome[62] posttreatment. These findings provide preliminary support that training vocal loudness can make improvements in speech production in neurological conditions beyond PD (where amplitude scaling is the primary deficit) and that the distributed system-wide improvements following LSVT/LOUD (e.g., improved articulation) are observed in other neurological conditions as well.

HOW DO WE EXPLAIN THE DISTRIBUTED EFFECT OF TREATING LOUDNESS ACROSS MOTOR SYSTEMS?

Our findings have led us to predict that training vocal loudness (amplitude) may stimulate generalized neural motor activation across the speech production system, and potentially across 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 affects common control mechanisms that, in turn, influence motor behaviors beyond the specific targeted function.[47,63,64] The neurological bases of such global motor effects are not known; however, McClean and Tasko[65] reported evidence for neural coupling of orofacial muscles to neural systems of laryngeal and respiratory control in human studies. These authors suggest that a potential source of this observed neural coupling might be from efferent drive from a common brain region to motoneurons 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. Smith and colleagues[66] suggested that the respiratory drive underlying vocal loudness may be a powerful force for distributed effects in that it entrains other aspects of motor functioning. Similar findings have been reported in the limb motor system, where a focus on training-increased amplitude (large, axial body movements) resulted in improvements in balance, as well as more distal functioning (e.g., reaching).[51] Brain imaging indicates that treatment effects extend beyond the periphery and include central nervous system elements.[45,50]

Another explanation for the distributed and lasting effect 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[67,68]; loudness scaling is a task in which both animals and humans engage all their lives.[69–74] 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 humans, lesions to different parts of the central nervous system, especially the limbic system, the anterior cingulate cortex, the thalamus, and the basal ganglia produce hypophonia, hypoprosodia, and hypokinetic articulatory movements.[75–79] Studies in animals indicate that the limbic system, anterior cingulate cortex, thalamus, and basal ganglia are involved in emotive and other survival-related vocalizations[80]; they also indicate that these neural structures are involved, directly or indirectly, in the readiness to vocalize and the intensity of vocalization.[71,81–87] In addition, brain stimulation and cell recordings in primates indicate that emotive vocalization typically involves coactivation of the respiratory, phonatory, and orofacial muscles.[87,88]

In summary, these findings indicate that 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 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[45,50] reflect improvements in the basal ganglia, limbic system, prefrontal cortex, and right hemisphere functions. As indicated

above, 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.

Effect of Mode of Delivery of LSVT/LOUD

Unlike other forms of speech treatment, LSVT/LOUD has been delivered in high-effort (perceived effort expended by patients) and intensive-mode (frequency and duration of treatment sessions: 16 individual 60-minute treatment sessions in 1 month) with a focus on sensory retraining. This intensive mode of delivery has not been a part of previous speech treatment approaches focused on articulation or rate. Although principles such as intensity of motor training have long been accepted in terms of behavioral recovery and improved function, only recently have the neurobiological phenomena underlying such principles been stringently validated for the positive effects on central nervous system functioning.[89–91] For example, it was previously thought that the adult brain had limited capacity to "heal itself"; however, there is now increasing evidence to the contrary,[89,90] and the adaptive capacity of the nervous system, known as neural plasticity, has begun to be quantified. Data from experimental animal models have documented that exercise or motor training (i.e., behavioral training) may interfere with multiple mechanisms involved in cell death and promote plasticity in the healthy and injured central nervous system.[90–93] Many of these neurobiological processes are triggered by the direct effect of exercise on increasing the expression of neurotrophic factors that promote cell survival in the brain.[89,94]

Although LSVT/LOUD was developed long before these more recent investigations, today we recognize that the mode of delivery of LSVT/LOUD is consistent with principles that promote neural plasticity,[90,95] such as intensive practice, complexity of practice, saliency of treatment tasks, and timing of interventions. Although all the factors that influence neural plasticity are yet to be defined or fully understood, certain key principles have emerged from the literature.[95] An example of five such principles, their application to PD, and their integration into LSVT/LOUD are detailed in Table 1 and briefly summarized in the following sections.

Intensity

Intensive training is paramount to eliciting long-term functional changes in behavior.[20,96,97] Intensity can be achieved via frequency of treatment (e.g., days per week), repetitions within sessions, or in requiring greater force, effort, or accuracy during motor tasks. There is a need for continued practice of a new motor skill (e.g., intensive training) for long-term structural changes in neural functioning; acquisition alone is not sufficient for neural plasticity.[98] LSVT/LOUD delivers treatment in an intensive dosage (60-minute individuals sessions 4 days/week for 4 weeks), with multiple repetitions of each task (e.g., minimum of 15 repetitions per task per day), and continually increases requirements for effort, consistency, and accuracy of vocal loudness in speech tasks.[99]

Complexity

Complex movements promote greater neural plasticity.[100–102] With PD there is a loss in automaticity of movements; thus, dual tasks or complex movements become increasingly difficult for people with PD.[103,104] Retraining automaticity of a single treatment target (e.g., vocal loudness) may allow generalization of this target to multiple motor tasks (e.g., speaking while walking or driving).[105] LSVT/LOUD trains vocal loudness across simple (words/phrases while seated) to more complex (conversational speech while

Table 1 Translation of Some of the Proposed Principles Underlying Neural Plasticity to Proposed Deficits in Parkinson's Disease, and the Corresponding Rationale and Task in LSVT/LOUD

Principle	Deficit Specific to PD	LSVT/LOUD
Intensity[20,96,97]		
Intensive practice is important for maximal plasticity. Intensity can be increased via frequency, repetitions, force/resistance, effort, and accuracy. Intensity increases activation of corticostriatal terminals inducing synaptic plasticity in striatum.	Intensive, high-effort training can be difficult in Parkinson's disease as a result of sensory deficits, force control, fatigue, depression, and progressive loss of cardiac sympathetic innervation.	Train intensively 1 hour/day, 4 days/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.
Complexity[99–102,127]		
Complex movements or environmental enrichment have been shown to promote greater structural plasticity (spine density, protein expression/ synapse number) in adjacent and remote interconnected regions than in simple movements.	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 result in the decrement in one or both of the concurrent tasks.	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 difficultly of speech tasks (progress from words to conversation).
Saliency[45,105–107]		
Practicing rewarding tasks (success/ emotionally salient) activates basal ganglia circuitry. Rewards are associated with phasic modulation of dopamine levels critical to induction of striatal plasticity and learning/relearning in PD.	People with early PD may experience lack of awareness of subtle motor deficits, depression, loss of motivation, and a feeling of "helplessness." Thus, they feel they do not need or would not benefit from speech therapy	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.
Use it or lose it/use it and improve it[90,108,109]		
Spared but compromised DA neurons are highly vulnerable to bouts of inactivity/activity. Inactivity may accelerate deficits. Post–exercise intervention, there may be a minimum-use requirement to maintain positive effects.	Deficits are subtle—not "red flag" to seek speech therapy. Getting early Parkinson's disease patients to recognize need for exercise and then convince them to continually exercise is challenging. Decreased physical activity may be a catalyst in degenerative process.	Educate people with Parkinson's disease on subtle deficits and improve motor function that directly affects real life. Retrain a new way of speaking everyday life (LOUD or ENUNCIATE); thus, normal activity offers continuous exercise.

Timing matters[108,115–118]		
Early exercise has the potential to rescue DA neurons, prevent chronic disuse, promote system-wide plasticity, and halt disease progression—particularly to the asymptomatic side.	People with early Parkinson's disease have subtle physical underactivity (small movements/soft voice). This may be coupled with a lack of awareness or self-correction, leading to further inactivity.	Train people with early Parkinson's disease 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.

walking to cafeteria and ordering coffee) tasks.[99]

Saliency

The more meaningful or rewarding a task, the greater the effect on neural plasticity.[106,107] In LSVT/LOUD, there is a reward associated with using the trained target of increased loudness in daily living (comments such as, "I can hear you better," "You sound great"). This reward makes the target behavior LOUD more salient (i.e., "if I use LOUD, I get a rewarding response").[105] This saliency may mediate greater experience-dependent plasticity.

Use It or Lose It

Inactivity may accelerate the degenerative process in PD and exacerbate speech or other motor disorders.[90,108,109] Because of sensory deficits associated with the speech disorder in PD,[105] many people live with a speech disorder without seeking treatment, which results in an insidious decrease in their amount of speaking and in gradual social withdrawal and isolation.[110] Thus, it is important to engage people with PD early in their disease state and to improve their communication with treatments. LSVT/LOUD targets the voice problem in PD, which is the first problem to occur,[2,3,111] and directly addresses the sensory mismatch that makes people with PD feel they are speaking loud enough, when in fact they are speaking too softly.

Use It and Improve It

Speech can improve with focused practice on increasing vocal loudness and active attention to the sensory mismatch that accompanies increased vocal loudness.[13–15,26] Active attention to sensory feedback may be essential for facilitating the neural plastic changes of cortical sensorimotor maps during behavioral treatment.[112,113] Repetitive passive practice without a sensory focus has been shown to generate minimal enduring neural plastic changes.[112–114] Although speech behavior can improve with speech exercises focused on vocal loudness, animal and human studies indicate that the benefits of exercise quickly degrade[109]; thus, the need for continued use to promote maintenance is necessary. The novelty of LSVT/LOUD is that it trains everyday loudness for speech, so that everyday life is continuous exercise. For example, a person uses a Loud voice when making phone calls, speaking at work, or socializing. Thereby, specific strategies to override hypokinesia/bradykinesia are learned and used in daily living to retrain normal or optimal use.

Timing Matters

When exercise is introduced early in disease process in animal models of PD, there is the potential to slow or halt the progression of the disease.[115–118] If exercise for speech is engaged early, there may be a slowing of the speech degeneration. Early referral to behavioral therapies (e.g., speech, physical therapy) in people newly diagnosed with PD is uncommon. Instead, referrals are typically made after functional impairments have progressed to the point of disability. It may be important to train people with PD in speech therapy before the symptoms are apparent or severe enough to interfere with functional communication.

The use of exercise as a physiological tool to promote neural plasticity, slow progression of motor symptoms (speech or limb), and promote quality of life in PD is a virtually untapped resource. To date, the majority of the research efforts in human PD has been focused on pharmacological agents or exogenous neurotrophic factors (e.g., introducing neurotrophins such as glial-derived neurotrophic factors into the brain) to provide hope for slowing disease progression. Recent literature from animal models of PD, however, have offered data that are so compelling that they have lead basic science researchers to suggest that physical training interventions might halt the progression of the disease.[89–92,109,117,118] These animal studies are beneficial in that they offer insight into the effect of physical activity on PD by allowing direct observation of morphologic, neurochemical, and neurophysiologic changes associated with exercise. It is unknown how these data will translate to human PD because of the difference in acute animal models of PD

versus the chronic dopamine neuronal death in human PD.[119,120] Nonetheless, we need studies designed to translate findings from animal models of exercise/motor training to the effect of intensive behavioral speech therapy on neural plasticity and the potential for neural protection as measured by dopamine-related changes in imaging studies over time. Preliminary studies of PET-related changes pre-/post-LSVT/LOUD have already documented treatment-dependent functional reorganization in people with PD. These data indicate that speech therapy may go beyond treating the symptoms of PD and may have the potential to affect progression of speech disorders associated with the disease over time.

WHAT ARE THE IMPLICATIONS FOR CLINICAL PRACTICE?

Single Treatment Target/LOUD Is Special

As discussed in earlier sections, a single, relevant cue may be the most effective means to elicit a behavioral change in PD.

Training a single focus (increased amplitude) allows for the complexity and intensity shown in animal models of PD and human stroke to promote behavioral recovery, neurochemical sparing, and brain reorganization. The observation of distributed effects following LSVT/LOUD across motor systems for the speech/communication and swallowing mechanisms in individuals with PD, as well as adults and children with other neurological disorders, are of significance at both clinical and scientific levels. Even when people with PD have multiple speech difficulties (e.g., soft, hoarse voice with mumbled speech), training only vocal loudness (amplitude) improves other aspects of speech (e.g., hoarseness, intelligibility), keeping the treatment focus simple and redundant. Most individuals with neurological disorders have multiple communication problems; if one treatment target can have an effect across the speech production system, this may allow us to improve the effectiveness of treatment. A single therapeutic target delivered in a manner that promotes neural plasticity has great potential to improve clinical efficiency.

Mode of Delivery/Intensive Training is Special

Recent advances in neuroscience reveal that exercise and motor training affects 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 promote neural plasticity and brain reorganization.[16,45,105,121] The translation of the principles of neural plasticity to therapeutic approaches will require a significant paradigm shift in rehabilitation sciences.[122] Current rehabilitation approaches are typically not developed specifically for the deficits in PD (or other neural disorders), nor do they implement the principles described in Table 1 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. The standardized protocol for LSVT/LOUD differs from traditional modes of speech treatment delivery and adheres to key principles of neural plasticity as summarized in Table 1. The mode of delivery of this treatment protocol, albeit before widespread dissemination of neural plasticity literature, may account in part for its success.

It is recognized that there are practical challenges to delivering speech treatment intensively (four individual sessions a week for 4 weeks). In fact, any treatment regime (speech, physical, 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 an efficacious dosage of treatment to all the people with PD in need—a need that will only increase dramatically in the coming years with the aging of the baby boomer population. The use of group therapy is not an option, as it does not enable the efficacious requirement of each person working to his or her maximum effort levels

for the entire 1-hour treatment session. Furthermore, continued exercise following the conclusion of speech treatment and tune-up sessions are needed for maintenance of vocal loudness as the disease progresses.

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 that requires intensive motor training (e.g., 6 hours/day for 2 weeks). This computerized system, called AutoCITE, was documented to result in comparable outcomes to live delivery of the therapy.[123] Computer technology has also been developed for the delivery of an intensive speech treatment (LSVT/LOUD) and is discussed below.

Halpern and colleagues[124,125] reported on the use of a personal digital assistant as an assistive device for delivering LSVT/LOUD to people with PD. The LSVT companion is specially programmed to collect data and provide feedback as it guides people through the LSVT/LOUD exercises, enabling them to participate in therapy sessions at home. Fifteen 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, using the LSVT-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.[124,125] An evolution of the LSVT-C has been the development of a virtual speech therapist (LOUD-VT). This is a perceptive animated character, modeled after expert LSVT/LOUD speech therapists, that delivers LSVT/LOUD in a computer-based program.[126] A prototype of the LSVT-VT has been developed, and clinical testing has begun. In addition, delivery of intensive speech therapy, such as LSVT/LOUD, 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.[128]

SUMMARY

Improving speech in individuals with neurological disorders, such as PD, is challenging. Many of these individuals have degenerative conditions, a range of medical problems, multiple speech mechanism disorders, and cognitive deficits, all of which may limit the long-term success of treatment. We have documented, however, that intensive speech treatment focusing on vocal loudness is an effective rehabilitative tool, the results of which can give us insight about the neural mechanisms of disordered oral communication and vocal tract functioning in individuals with PD. The effect of training a single treatment target, such as vocal loudness, is powerful in its ability to have a maximum functional effect while increasing clinical efficiency. Continued research will further evaluate the distributed effects of LSVT/LOUD across speech articulation, facial expression, and swallowing to more clearly discern whether training loudness has a unique effect on speech treatment outcomes (as compared with intensive, high-effort treatment focused on articulation). Furthermore, recent advances in neuroscience reveal that exercise affects molecular changes associated with cell survival, cell growth, and functional recovery in animal models of PD. There is a great need for translation of these data to human PD in speech interventions that are founded on principles of neural plasticity. Improvement in voice, speech, and swallowing functions in the context of a degenerative disease may prove, in future research, to be indicative of neural plasticity, as preliminary brain studies post-LSVT/LOUD already suggest.

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