Speech and swallowing disorders in Parkinson disease

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Purpose of review

To review recent research and clinical studies pertaining to the nature, diagnosis, and treatment of speech and swallowing disorders in Parkinson disease.

Recent findings

Although some studies indicate improvement in voice and speech with dopamine therapy and deep brain stimulation of the subthalamic nucleus, others show minimal or adverse effects. Repetitive transcranial magnetic stimulation of the mouth motor cortex and injection of collagen in the vocal folds have preliminary data supporting improvement in phonation in people with Parkinson disease. Treatments focusing on vocal loudness, specifically LSVT LOUD (Lee Silverman Voice Treatment), have been effective for the treatment of speech disorders in Parkinson disease. Changes in brain activity due to LSVT LOUD provide preliminary evidence for neural plasticity. Computer-based technology makes the Lee Silverman Voice Treatment available to a large number of users. A rat model for studying neuropharmacologic effects on vocalization in Parkinson disease has been developed. New diagnostic methods of speech and swallowing are also available as the result of recent studies.

Summary

Speech rehabilitation with the LSVT LOUD is highly efficacious and scientifically tested. There is a need for more studies to improve understanding, diagnosis, prevention, and treatment of speech and swallowing disorders in Parkinson disease.

Keywords

speech disorders and Parkinson disease, swallowing disorders and Parkinson disease, voice disorders and Parkinson disease

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Introduction

Classical signs of Parkinson disease (PD) include bradykinesia, hypokinesia, muscle rigidity, and resting tremor. Other signs of PD, such as speech and swallowing disorders, olfactory disturbances, fatigue, pain, autonomic dysfunction, sleep fragmentation, depression, and dementia, may co-occur, precede, or follow the classical motor signs of the disease [1]. Ahlskog [2] questions some of the traditional notions regarding PD and its pathophysiology. Based on recent research findings, he argues that PD is much more than degeneration of the dopaminergic nigrostriatal system; the first neurons affected in PD are nondopaminergic; the substantia nigra and other dopaminergic nuclei are affected only later in the course of the disease; and most of the disability of advancing PD stems from the involvement of nondopaminergic systems. Ahlskog's [2] commentary regarding the neuropathology and neuropharmacology of many symptoms of PD is especially relevant to speech and swallowing disorders.

Nearly 90% of individuals with PD will develop speech and swallowing disorders during the course of their ill-

ness [3,4]. Given that there are over 600 million individuals in the world age 65 years old or older [5], and that the prevalence of PD in this age range is 1.5% [6], there are probably 8 000 000 or more individuals in the world each year that have or will have speech and swallowing disorders during the course of their PD. These disorders have deleterious effects on communication, health, psychological well-being, and quality of life [7-12]. Unfortunately, pharmacological and surgical interventions for these disorders are minimally effective [3,4,13]. Historically, behavioral therapy methods for speech disorders in PD have yielded disappointing results in terms of treatment gains and/or long-term maintenance of these gains [3]. A unique behavioral treatment program known as LSVT LOUD (Lee Silverman Voice Treatment) has been tested scientifically in numerous studies and has been shown to yield statistically significant and long-term therapeutic effects on speech disorders in individuals with PD; preliminary data also suggest improvements in swallowing [3,14,15°°,16]. The purpose of the present review is to address some of the important findings in recent research studies regarding the

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Dysarthria in Parkinson disease: characteristics

Speech disorders associated with PD have been characterized by reduced voice volume and a tendency for voice volume to decay over time (hypophonia) [17–19], poor voice quality (dysphonia), reduced pitch inflection (hypoprosodia) [17–19], reduced range of articulatory movements (hypokinetic articulation) [15••], a tendency for speech articulation to festinate (rush) [20], and hesitant and/or dysfluent speech [13]. These disorders, collectively termed hypokinetic dysarthria (HKD) have been shown to have a negative impact on speech intelligibility, linguistic and emotive communication, psychological well-being, and overall quality of life [9–12,21].

Dysarthria in Parkinson disease: diagnosis

Perceptual analysis of dysarthric speech is commonly used in clinical practice and research studies of PD. Such analysis can be problematic, however, as it relies on subjective judgment and is influenced by various factors, such as familiarity or lack of familiarity with the patient or his or her speech disorder, overall experience with motor speech disorders, and more. These issues and arguments for and against the use of perceptual analyses are addressed in recent studies [22–24].

An alternative, or complementary, method to perceptual rating of dysarthria is acoustic analysis. Acoustic analysis can potentially provide an objective, quantitative, noninvasive, reliable, valid, and precise means to index the sensitivity and characteristics of dysarthria in general and HKD in particular [15**]. Tjaden and Sussman [25] found that certain acoustic cues are critical for the perception and perceptual assessment of dysarthric speech. Rosen et al. [26] sought to identify acoustic signatures of HKD that are robust to phonetic variation. Of the different acoustic measures they tested, they found that percentage pause time and spectral range were two acoustic indices that were highly specific (95%) and accurate (95%) in the differentiation of HKD and normal conversational speech. Sapir *et al.* [15**] used acoustic analysis of vowel formants to assess differences between HKD and normal speech, and to assess treatment (LSVT LOUD) effects. They found that the ratio of the second formant (F2) of the vowels /i/ and /u/, denoted as F2i/F2u, was the only acoustic index of vowel articulation, among many tested, that significantly differentiated between HKD and normal speech, and was highly sensitive, in terms of statistical significance and large effect size, to treatment effects. The studies by Rosen et al. and Sapir et al. encourage further investigation to delineate additional acoustic indices of dysarthric speech.

Treatment of hypokinetic dysarthria in Parkinson disease

Treatment of hypokinetic dysarthria will be discussed in terms of the role of dopamine, neurosurgical, transcortical stimulation, laryngosurgery, and behavioral (speech) treatments.

The role of dopamine, rigidity, and high level deficits

Traditionally, speech disorders in PD have been attributed to dopamine deficiency and muscle rigidity. Recent studies of dysarthria in PD provide some support for this attribution, as there is evidence for improved speech functioning with levodopa treatment [17–19]. The improvement is in respiratory function, prosodic pitch and loudness variation, and speech intelligibility. There is also electromyographic evidence for hypertonicity of laryngeal muscles at rest in individuals with PD [27]. However, recent reviews indicate that the majority of other studies have failed to find a causal relationship between dopamine and speech, or rigidity and speech, or a positive impact of dopamine therapy on functional speech intelligibility in individuals with PD; moreover, there is evidence to suggest other etiologies for speech problems in PD, such as deficits in internal cueing, scaling movement force and amplitude, sensorimotor gating, self-perception of voice, and self-regulation of vocal output. These findings are detailed elsewhere [3,14,13].

An animal model may help delineate the neuropharmacology of vocalization in PD. Ciucci and colleagues [28**] introduced a rodent model of vocalizations to systematically examine any relationship between dopamine depletion and ultrasonic vocalization (USV). One underlying tenet for using the rat model is that the impairment in voice and speech in individuals with PD is at least partially related to phylogenically old neural mechanisms subserving phonation. Initial data suggest that mild transient dopamine depletion with haloperidol or even unilateral destruction of dopamine neurons via 6-hydroxydopamine are associated with changes in USV acoustic signal, specifically, decreased frequency bandwidth.

Effects of deep brain stimulation of the subthalamic nucleus

Deep brain stimulation of the subthalamic nucleus (DBS-STN) has been shown to yield dramatic improvement in global motor functions of the limbs and to reduce tremor [29,30], but its effects on speech are varied and inconclusive. Several studies have reported dysarthria and word or phonemic fluency problems as side effects of

the DBS-STN [29,31–34], other studies have reported no significant deleterious effects of DBS-STN on speech [35]) and still other studies have reported improvement in some aspects of speech with DBS-STN [30]. The reasons for these diverse outcomes are not clear, but they are likely related to factors such as lesions associated with the stimulating electrode, stimulus parameters, neuroanatomical and neurophysiological differences across patients, patient's medical status, and the surgeon's skills. Research is needed to examine these and other potential factors to optimize the effects of DBS-STN and reduce side effects.

Effects of repetitive transcranial magnetic stimulation

Dias et al. [36] studied the effects of repetitive transcranial magnetic stimulation (rTMS) on voice and speech in 30 individuals with PD. rTMS of the M1-mouth, but not of the dorsolateral prefrontal cortex, resulted in a significant improvement of the voice fundamental frequency and voice intensity. These findings indicate that rTMS of the M1-mouth might improve vocal function in PD. However, longitudinal studies are needed to assess long-term therapeutic or side effects of the rTMS.

Effects of transoral collagen injection

Sewall *et al.* [37] used acoustic and perceptual methods, as well as the Voice Handicap Index (VHI) questionnaire, to prospectively assess the effects of transoral vocal fold collagen injection on dysphonia in six individuals with PD. Five of the six patients benefited markedly from the procedure. Sewall et al. [37] concluded that this medical procedure is safe, well tolerated, and an effective temporary method of subjectively improving voice and speech in selected PD patients.

Effects of behavioral speech treatment

A recent review by Yorkston et al. [16] examined evidence for effectiveness of global treatment parameters including loudness, rate, prosody, or general instructions such as 'clear' speech for individuals with dysarthria. The evidence was characterized on the basis of the phase of research, as defined by Robey and Schultz [38]. The strongest evidence regarding treatment effectiveness was for modification of loudness, specifically the LSVT LOUD protocol, in PD patients. This work demonstrated progression through Phase III research had a well defined, replicable protocol, measured multiple aspects of speech, and included a relatively large subject pool. It was recommended that continued research should focus on areas such as measures of outcomes in natural social situations and examining factors to optimize learning (e.g., dosage). Studies modifying rate included Phase I and II research and revealed it may be a powerful technique for improving intelligibility in some speakers with dysarthria. There were a variety of techniques used across studies. Areas for future research include examining generalization of techniques, treatment studies comparing various techniques, parameters of treatment scheduling (e.g., intensity, dosage), and descriptions of how optimal rate is selected and trained. Prosody studies also included Phase I and II research. The current status indicates that manipulating prosody may enhance linguistic information, thus intelligibility. Future research needs include areas such as comparison of approaches, generalization of training, techniques for perceptual ratings of prosody, and documenting social validity. Finally, there were studies of general instructions, such as instructions to speak 'clear'.

To summarize, then, various speech manipulations may take advantage of compensatory abilities and be applied to many types of dysarthria. Future research should focus on larger groups of speakers, candidacy issues for the approach, and studying actual rather than simulated communication breakdowns.

Effects of LSVT LOUD speech treatment in Parkinson disease

LSVT LOUD has been shown, clinically and scientifically, to be a powerful method of improving speech and related functions such as swallowing and facial expression. Improvement has been documented in vocal loudness [15**,39,40], voice quality [41], prosody [42,43], and speech articulation [15**]. Most of these effects have been shown to be sustained at 1-year and 2-year follow ups [3,42,44]. Published pilot data from training loudness (LSVT LOUD) indicate that treatment effects generalize beyond vocal loudness to improve swallowing, articulation, communicative gestures, facial expression, and neural functioning [3,4,13,14,15°°].

LSVT LOUD targets increased amplitude of motor output across the speech mechanism by training increased vocal effort and loudness while training individuals to monitor vocal output. LSVT LOUD is delivered in accordance with key principles of motor learning and neural plasticity, such as intensity, complexity, saliency, use it or lose it, and use it and improve it [3,14].

Ongoing randomized controlled studies by these authors and their colleagues are further examining this transfer of effects by evaluating and comparing the systemwide generalized impact of two therapies [voice (LSVT LOUD) and articulation (LSVT ENUNCIATE)] on speech articulation, facial expression, swallowing in idiopathic PD, and the systemwide generalized impact of these two therapies on limb gesture and limb motor functioning in PD. Positron Emission Tomography (PET) studies of these two treatments are being used to identify changes in functional connectivity and neural functioning and identify any differences associated with different treatment targets. Results from these studies

will further clarify the neural bases for voice and speech disorders in PD patients, as well as guide development and modifications for optimal speech treatment approaches for this population.

For some patients, the intensive schedule of four therapy sessions a week for 4 consecutive weeks, as the LSVT LOUD requires, is difficult to implement. Spielman et al. [40] examined a revision of the traditional LSVT LOUD regimen, whereby treatment is delivered over an extended period of time, that is, 2 days a week in the treatment room and 2 days a week the patient practices on his/her own for 8 consecutive weeks. This extended treatment (LSVT-X) successfully increased vocal SPL (which was consistent with improvements following traditional LSVT LOUD), decreased perceived voice handicap, and improved functional speech in individuals with PD. Furthermore, delivery of the LSVT LOUD via Telehealth has documented positive outcomes in people with PD. The method is likely to enhance accessibility of treatment to a large number of patients who cannot come to the clinic [45].

Swallowing disorders in Parkinson disease

Nearly 90% of individuals with PD suffer from dysphagia during the course of the disease [3]. The dysphagia may cause malnutrition and pneumonia, thus posing serious threats to the patient's health and longevity [46]. Dysphagia also impacts negatively on the patient's psychological well-being and quality of life [9]. Early detection and treatment of dysphagia are likely to prevent many of the complications associated with dysphagia.

Manor et al. [47] reported on the development and validation of a new swallowing disturbance questionnaire (SDQ). This SDQ was administered to 57 individuals with PD, along with fiberoptic endoscopic evaluation of swallowing (FEES). They found that on the basis of the SDQ assessment alone, 12 of the 24 (50%) noncomplaining patients had signs suggestive of dysphagia. These findings suggest that the SDQ might be a sensitive tool to detect early dysphagia in PD patients. Alfonsi et al. [48] studied electrophysiologic patterns of oral-pharyngeal swallowing in three Parkinsonian syndromes - PD, a Parkinson variant of multiple system atrophy (MSA-P), and progressive supranuclear palsy (PSP). They analyzed several oral-pharyngeal-laryngeal measures, such as the duration of electromyography (EMG) activity of suprahyoid/submental muscles; duration of laryngealpharyngeal mechanogram; duration of the inhibition of the cricopharyngeal muscle activity (CPEMG-ID); interval between the onset of EMG activity of suprahyoid/ submental muscles and the onset of laryngeal-pharyngeal mechanogram (I-SHEMG-LPM); and swallowing reaction time. They found that prolongation of the

I-SHEMG-LPM was more typical in PD, whereas the most distinctive abnormality in both PSP and MSA-P groups was the reduction or the absence of CPEMG-ID early in the course of the disease. Alfonsi et al. suggested that the method they used might help identify swallowing abnormalities in patients without symptoms of dysphagia, as well as evaluate the severity of dysphagia in all patients. Gross et al. [49] used measures of nasal airflow and respiratory inductance plethysmography to track breathing patterns during swallow in individuals with PD and healthy controls. Surface EMG was used to record the timing of each swallow within the respiratory cycle. These swallows were studied as the participants in the study spontaneously swallowed calibrated pudding and cookie portions. Compared to the healthy controls, the PD patients swallowed significantly more often during inhalation and at low tidal volumes. They also exhibited significantly more postswallow inhalation for both consistencies. Only the healthy controls showed significantly longer deglutitive apnoea when swallows occurred during inhalation than when they occurred during exhalation. Gross et al. concluded that the high incidence of oropharyngeal dysphagia and risk of aspiration pneumonia in PD patients may be partially attributable to impaired coordination of breathing and swallowing. Ciucci et al. [50] studied the effects of DBS-STN on dysphagia in individuals with PD. Significant improvement occurred for the pharyngeal composite score and pharyngeal transit time when the DBS-STN stimulator was on compared to when it was off. However, the degree of hyoid bone excursion and oral stage measures did not improve. Ciucci et al. [50] interpreted these findings to suggest that the pharyngeal motor system and the hyoid-oral motor system may be controlled by different sensorimotor pathways within the basal ganglia. The findings from these studies are important for understanding the neurophysiologic mechanisms underlying dysphagia in PD, as well as for the diagnosis and early detection of dysphagia in PD.

Logemann [51,52] addresses the importance of using research protocols to assess the impact of swallowing therapy in various medical conditions, including PD. She provides helpful guidelines for the conduction of randomized clinical trials (RCTs) and discusses methods and rationale for assessing short and long-term treatment effects.

Conclusion

The studies reviewed here shed light on the nature of speech and swallowing disorders in PD, and on methods of diagnosis and treatment of these disorders. Whereas neuropharmacologic and brain stimulation have marked therapeutic effects on limb motor functions, their effects on speech and swallowing in PD are less impressive, and

in some cases, adverse. The LSVT LOUD program appears to be the most effective behavioral treatment of speech disorders in people with PD as the current research data suggest. Research on the impact of different treatment targets for PD patients (e.g., rate, prosody, and clear speech) is in early stages. New physiologic studies shed light on the nature of dysphagia in PD, and provide important information to improve early detection and diagnosis of dysphagia in PD.

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

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