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Speech dysfunction, cognition, and Parkinson’s disease

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Abstract

Communication difficulties are a ubiquitous symptom of Parkinson’s disease and include changes to both motor speech and language systems. Communication challenges are a significant driver of lower quality of life. They are associated with decreased communication participation, social withdrawal, and increased risks for social isolation and stigmatization in persons with Parkinson’s disease. Recent theoretical advances and experimental evidence underscore the intersection of cognition and motor processes in speech production and their impact on spoken language. This chapter overviews a growing evidence base demonstrating that cognitive impairments interact with motor changes in Parkinson’s disease to negatively affect communication abilities in myriad ways, at all stages of the disease, both in the absence and presence of dementia. The chapter highlights common PD interventions (pharmacological, surgical, and non-pharmacological) and how cognitive influences on speech production outcomes are considered in each.

Keywords

Parkinson’s disease; Speech; Voice; Communication; Spoken language; Conversation; Speech-language therapy; Communication participation

1 Introduction

Although cognitive impairments develop typically later in Parkinson’s disease (PD), the finding that for some persons, cognitive changes manifest early underscores the importance of considering the effects of cognition on communication at all stages of the disease (Aarsland et al., 2021; Postuma et al., 2012). Detailed elsewhere in this book, PD commonly affects working memory, attention, executive control, and visuospatial domains. Recently the PD cognitive phenotype has expanded to include impairments in action semantics (Bocanegra et al., 2015; Fernandino et al., 2013; Roberts et al., 2017), verb

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retrieval (Bocanegra et al., 2015; Herrera and Cuetos, 2012; Salmazo-Silva et al., 2017), complex syntax processing (Hochstadt, 2009; Johari et al., 2019; Natsopoulos et al., 1993), spontaneous discourse (Ash et al., 2012; Murray, 2000; Roberts and Post, 2018), and social cognition (Alonso-Recio et al., 2021; Palmeri et al., 2017; Roca et al., 2010).

Family members, naive listeners, and individuals with PD appear sensitive to these cognitive changes and their effects on communication abilities (Miller et al., 2006; Whitworth et al., 1999). In addition to motor symptoms, communication partners report that frequent pauses, abandoned thoughts, difficulty starting conversation turns, and misalignment between facial expressions and emotional intent negatively affect communication and quality of life (Miller et al., 2006). When asked to compare feelings about their communication to before their diagnosis, persons with PD reported a “loss of control” during communication events, frustration because of difficulty communicating their intended messages, and feelings of reduced confidence (Miller et al., 2007). Collectively, these symptoms contribute to a perceived loss of independence for some individuals with PD (Miller et al., 2007; Schalling et al., 2017).

Although communication impairments are ubiquitous in PD, referrals for speech-language therapies (SLT) are underutilized in this population, particularly in early and late-stage disease (Roberts et al., 2021). One plausible reason for SLT underutilization, that is supported by survey responses of persons with PD and their care partners, is the limited availability of evidence-informed behavioral interventions at all stages of the disease that generalize to everyday communication challenges broadly, beyond speech and voice issues (Miller et al., 2015).

This chapter overviews a growing evidence base demonstrating that cognitive impairments can interact with motor changes in PD to negatively affect communication abilities in myriad ways, at all stages of the disease, both in the absence and presence of dementia. The chapter highlights common PD interventions (pharmacological, surgical, and non-pharmacological) and how each takes into consideration the interaction between cognition and speech production outcomes.

2 Linkages between cognition and speech production in Parkinson’s disease

Central to contemporary speech production theories is the idea that speakers use auditory, sensory, and kinesthetic feedback to monitor whether an actualized speech/vocal output matches the intended production target (Guenther and Hickok, 2015; Nozari and Novick, 2017; Tourville and Guenther, 2011). When misalignments between planned targets and actual productions occur, or when monitoring and conflict detection processes are impaired, so-called “error signals” can manifest as misarticulated speech sounds, long pauses, verbal fillers (“uh” and “uhm”), disfluent productions (e.g., repeated sounds, syllables, or words), word choice, grammatical errors, and reformulated/revised productions (Levelt, 1980; Postma, 2000). These speech manifestations are commonly observed in speakers with PD during spontaneous, connected speech (Illes et al., 1988; McNamara et al., 1992). Although less well understood, sensorimotor control appears to involve several cognitive processes including decision making, integration of multiple sensory inputs, determining the current state of a complex system, and goal-directed intention, among other processes that rely heavily on attention and working memory (Chivukula et al., 2019; McDougle and Taylor, 2019).

While supervisory cognitive processes are integral to psycholinguistic theories of language processing and production (Garrett, 1989; Hickok, 2012; Levelt, 1980), only recently have researchers put forward unifying accounts that integrate higher-level (cognitive and linguistic) and lower level (motor speech and vocal control) processes (Gauvin and Hartsuiker, 2020; Guenther and Hickok, 2015; Nozari and Novick, 2017). This theoretical shift is supported by a growing body of experimental evidence that highlights the importance of attention and working memory in sensorimotor feedback monitoring and conflict detection processes during speech and voice production generally (Guo et al., 2017; Liu et al., 2018; Miller et al., 2021; Segawa et al., 2019) and in PD specifically (Gauvin et al., 2017; Li et al., 2021; McNamara et al., 1992).

PD affects cognitive domains that are integral to supervisory processes in language and speech production (Aarsland et al., 2021). For example, PD appears to affect temporal processing in both motor and perceptual domains (Jones and Jahanshahi, 2014, for review), which is associated with cognitive performances on standardized tests (Singh et al., 2021) and has the potential to affect speech production (Johari and Behroozmand, 2018). Moreover, network level disruptions in PD overlap with brain regions central to rule generalization, speech monitoring, and conflict detection processes (Behroozmand et al., 2015; Golfinopoulos et al., 2010; Zheng et al., 2010). Neuroimaging studies in PD found reduced connectivity and perfusion in fronto-striatal and parietal networks responsible for cognitive control of motor behaviors, and for speech output specifically (Burton et al., 2004; Kendi et al., 2008). PD-related changes are also seen in neural connectivity involving brain regions involved in phonological planning and sequencing, thought to be critical for speech error monitoring and detection (Manes et al., 2018). Taken together, emerging experimental and theoretical work underscores the importance of considering the cognitive-motor interface in relation to PD speech and communication outcomes.

3 Overview of communication difficulties in Parkinson's disease

Herein, is an overview of the communication challenges in PD. Beyond the scope of this chapter, the reader is directed to comprehensive reviews of motor speech and voice (Broadfoot et al., 2019; Tjaden, 2008), language (Altmann and Troche, 2011; Bastiaanse and Leenders, 2009), social cognition (Prenger et al., 2020), and spoken language and communication (Roberts and Orange, 2013; Smith and Caplan, 2018) consequences of PD.

3.1 Motor speech

Motor speech impairments, consistent with a hypokinetic dysarthria profile, occur in 70%–90% of speakers with PD (Ramig et al., 2008). Frequently reported motor speech changes in PD include reduced overall loudness, less variable prosody and loudness, hoarse vocal quality, weakened vocal strength, shortened breath groups, imprecise articulation with blurring of consonant boundaries, and excessive speech rate that is complicated by longer speech pauses occurring at non-grammatical boundaries (Ho et al., 1998, Tjaden, 2008, Broadfoot et al., 2019, for review). Voice quality impairments appear early in the disease with articulation and speech fluency changes appearing later in PD (Ho et al., 1998). Changes with disease progression include reduced lung volume initiation and termination (Huber and Darling-White, 2017), worsening voice quality (Skodda et al., 2013), and impaired articulatory precision and speed in connected speech (Skodda et al., 2013). However, whether disease progression affects prosody is less clear with significant longitudinal changes in pitch variability seen in women, but not men (Skodda et al., 2009).

Interestingly, motor speech performance is affected by language and cognitive task demands. Speech timing differences between persons with PD and healthy controls are reported and are magnified by cognitively demanding speaking tasks (Lowit et al., 2018). Additionally, Huber and Darling-White (2017) found that participants with PD had greater challenges coordinating respiratory support and language formulation efforts in a cognitively demanding spontaneous speaking task compared to a reading task. Furthermore, recent findings from the second and senior authors' lab show that attention and working memory scores significantly predicted syllable repetition rates in 136 participants with early stage PD (unpublished data). Collectively, these studies highlight the varied speech production changes associated with PD and underscore the importance of considering cognitive changes and task demands in the evaluation of motor speech impairments in PD.

3.2 Connected speech

Working memory and attention show modest to robust associations with a broad range of verbal impairments in PD including word retrieval for nouns (Cotelli et al., 2007) and verbs (Péran et al., 2003), processing of complex syntax structures (Lieberman et al., 1992), reduced information content in connected speech (Roberts and Post, 2018), and increased disruptions in speech fluency (Goberman et al., 2010). In connected speech elicited from picture descriptions and other structured tasks, people with PD produce significantly longer pauses, uncorrected lexical and verb tense errors, and sentences with grammatical errors when compared to healthy controls (Ash et al., 2012; Lee et al., 2019; Murray, 2000; Roberts, 2014). Spoken discourse in PD, even in the absence of dementia, is marked by fewer information content units and fewer main event structures (Murray, 2000; Roberts and Post, 2018). Discourse impairments are exaggerated in the face of PD-cognitive impairment and worsen over time (Ash et al., 2017). Higher episodic memory task demands also affect language production in individuals with PD compared to healthy controls with fewer information content units and higher speech disfluency rates found in a story retelling task compared to a picture description task for persons with PD (Roberts, 2014). On whole, this literature confirms (1) that communication impairments in PD extend beyond the motor domain and (2) that cognitive task demands influence performance on both speech and language output measures.

3.3 Conversation

Conversation is defined as talk in a social interaction between two or more speakers.

Conversation difficulties can be a sensitive marker of motor, cognitive, and language impairments in day-to-day interactions. Individuals with PD report significant challenges carrying out conversations (Miller, 2017; Wolff and Benge, 2019). Additionally, increased fatigue and the presence of cognitive and emotional issues are associated with lower communication participation in everyday activities in PD (Barnish et al., 2017; McAuliffe et al., 2017). The suggestion that motor and cognitive impairments in conversations are intertwined is supported by Wolff and Benge's (2019) finding that everyday language difficulties in persons with PD are associated with increased motor dysfunction and increased severity of cognitive impairment.

Using robust Conversation Analysis methods, multiple researchers report that conversations including a person with PD are marked by increased episodes of overlapping talk (both partners talking simultaneously) compared to controls, often caused by longer pauses and quieter voice volume (Griffiths et al., 2012; Rinne and Roberts, 2019). Overlapping talk is a frequent source of breakdowns in conversation exchanges in PD (Griffiths et al., 2012, Rinne and Roberts, 2019). However, sources of conversation breakdowns are not limited to motor symptoms. Semantic and word-retrieval issues (e.g., use of incorrect words, non-specific vocabulary, and reformulated utterances) also caused conversation breakdowns in PD (Saldert et al., 2014; Saldert and Bauer, 2017). In a recent study of conversations between persons with PD without dementia and their family members, two-thirds of conversation breakdowns occurred for reasons unrelated to articulation difficulties or quiet voice volume, such as difficulties with topic initiation and maintenance, word retrieval challenges, syntax errors, and pausing at non-linguistic boundaries (Rinne and Roberts, 2019).

These behaviors, which reflect the joint motor and cognitive challenges resulting from PD, lead to maladaptive communication behaviors within family conversations and can result in the person with PD withdrawing from conversation (Griffiths, 2013; Griffiths et al., 2012, 2015; Saldert and Bauer, 2017; Saldert et al., 2014). Conversation difficulties in PD are complicated by social cognition impairments including difficulty perceiving and interpreting emotional and prosodic social cues (Dara et al., 2008; Pell et al., 2014; Schwartz and Pell, 2017).

Flattened facial expressions (i.e., hypomimia) and reduced spontaneous limb movements, common motor symptoms of PD, can also negatively affect the ability to communicate non-verbally through gestures (Gomez-Gomez et al., 2020; Prenger et al., 2020).

These literatures converge on the point that motor impairments do not account fully for difficulties in everyday conversations for persons with PD, even in the absence of measurable cognitive impairment. This underscores a limitation of current therapies for communication impairments, which focus almost exclusively on motor symptom affects and only rarely consider the intersection of motor speech and cognition changes in PD.

4 Parkinson's disease treatments and speech, language, and communication

The effect of mainstream PD treatments such as carbidopa-levodopa (L-dopa) and deep brain stimulation (DBS) on limb motor and gait symptoms are well established, but the effects on speech outcomes are less understood. Behavioral interventions, typically as a component of SLTs, are the mainstay intervention for communication impairments in PD. However, while studies of high intensity therapies that incorporate motor learning principles demonstrate efficacy for improving voice loudness, articulation, and respiratory support in persons with PD, there are limited clinical trials of SLT for those with advanced disease and those with cognitive impairments (Broadfoot et al., 2019; Xu et al., 2020, for review). In the sections that follow, we provide a brief overview of pharmacologic, surgical, and behavioral interventions that affect speech and communication impairments in PD.

4.1 Pharmacological interventions

While there is strong evidence supporting the benefits of L-dopa, on limb motor and gait symptoms, evidence regarding the effects of dopaminergic medications on speech is limited (Pinho et al., 2018; Rusz et al., 2016). Individual studies report positive effects of L-dopa ON-state for speech symptoms and acoustic features including increased voice intensity (De Letter et al., 2006; Ho et al., 2008); longer maximum phonation time in females (Cavallieri et al., 2021); faster speech rates (Ho et al., 2008) increased articulatory movement and precision (Thies et al., 2021); and improved fundamental frequency, harmonic to noise ratio and jitter (Fabbri et al., 2017; Sanabria et al., 2001). However, recent meta-analyses and reviews emphasize the highly variable effects of dopamine replacement therapy on acoustic and perceptual speech measures (Pinho et al., 2018). For example, Plowman-Prine et al. (2009) showed no significant effect of medication state on 35 perceptually-rated aspects of speech and voice quality (e.g., resonance, harshness). Additionally, changes in speech rate in L-dopa ON state are not a consistent finding in the literature (De Letter et al., 2006; Skodda et al., 2011). Researchers investigating L-dopa effects on speech prosody (e.g., pitch variation, loudness variation) also report high within-subject variability with no significant effect of L-dopa status (De Letter et al., 2007; Plowman-Prine et al., 2009; Skodda et al., 2011).

Findings relative to L-dopa effects on speech disfluency are mixed. While previous investigators reported a reduction in speech disfluencies in ON state, the effect was limited to those participants with the most severe disfluencies in OFF state (Im et al., 2019). In other studies, increases in speech disfluencies in ON state are reported (Louis et al., 2001) with the suggestion that longer-term exposure to L-dopa in higher doses may contribute to increased disfluencies and stuttering-like behavior in persons with PD (Tykalová et al., 2015).

These mixed findings highlight the need for further research into the interactions between PD medication states and speech production in PD. Unresolved issues include whether the combined effect of dopaminergic replacement and DBS therapies affect speech symptoms differently than either intervention alone (Tripoliti et al., 2014). Additionally, more research is needed to understand how (and whether) speech symptoms manifest differently as a function of medication scheduling and L-dopa wearing-off across dosing cycles (De Letter et al., 2006; De Letter et al., 2010). Studying this phenomenon, in relation to cognitive performance, is particularly intriguing given the potential role of cognition on speech output, and the complex and often paradoxical, relationship between cognitive performance and dopaminergic replacement therapies (Cools et al., 2010; Hanna-Pladdy et al., 2015; MacDonald and Monchi, 2011; McGuigan et al., 2019).

While L-dopa is the primary pharmacologic agent used in PD, additional research into the relationship between speech outcomes and other medications used to manage cognitive symptoms in persons with PD (e.g., cholinesterase inhibitors) is also warranted. Improvements in word retrieval, following commands, and comprehension of spoken language have been reported in persons with PD-related dementia in response to rivastigmine compared to placebo (Schmitt et al., 2010). However, investigation of medication effects on speech, beyond L-dopa, are scant.

4.2 Deep brain stimulation (DBS)

Deep brain stimulation (DBS) of the subthalamic nucleus (STN), globus pallidus interna (GPi) and ventral intermediate nucleus of the thalamus (VIM) are established treatments for PD motor symptoms. However, the benefits of DBS on speech symptoms are less clear, and highly variable with both improvements and declines noted across patients and behaviors (Aldridge et al., 2016; Skodda, 2012). Described by some as “stimulation-induced dysarthria,” a systematic review by Skodda (2012) reported prevalence rates of dysarthria following STN-DBS as high as 70% at three-years post-surgery with an average prevalence of 9.3%. Documented declines following STN-DBS include reduced articulatory precision and also symptoms of spastic dysarthria (Aldridge et al., 2016; Tsuboi et al., 2017; Wertheimer et al., 2014). However, researchers also reported improvements in speech rate, phonation duration for maximum effort tasks, reduced glottic tremor, and improved intelligibility (Klostermann et al., 2008; Tripoliti et al., 2011). Still yet, other investigators reported equivocal findings between STN-DBS ON versus OFF stimulation in vocalic transitions measured by F2 slopes (second formant frequency, significantly correlated with intelligibility), although at an individual level, high heterogeneity was observed (Martel-Sauvageau and Tjaden, 2017).

Stimulation parameters appear to impact speech outcomes post STN-DBS. In a double-blind study, Tripoliti et al. (2008) found that when compared to low voltage stimulation (2V), high voltage stimulation (4V) resulted in deterioration of speech intelligibility but not voice loudness. This finding is consistent with other recent studies reporting that lower frequency stimulation is associated with better speech outcomes (Knowles et al., 2018). Contact site also appears to play a role in speech outcomes with better intelligibility outcomes for posterior STN electrode placement (Tripoliti et al., 2008) compared to medial and anterior placements (Mossner et al., 2020; Tripoliti et al., 2008).

While beyond the scope of this chapter, cognitive changes can also occur post-DBS, and have the potential to mediate speech, language and communication outcomes (Arten and Hamdan, 2020; Foley et al., 2017; Mikos et al., 2010). A recent longitudinal study of

25 patients following STN-DBS found that patients whose speech intelligibility and/or naturalness deteriorated over the 2-year follow-up period had lower baseline scores on cognitive measures of working memory and inhibition (Tanaka et al., 2020). By contrast, the PD group whose speech remained stable over 2 years, and who realized greater ON-stimulation benefit for speech symptoms (e.g., increased voice loudness, increased loudness variability, and lower perceptual ratings of asthenia), had higher baseline cognitive scores (Tanaka et al., 2020).

Findings from studies of spontaneous discourse post-DBS further hint at a relationship between DBS cognitive changes and speech/language outcomes. Recent studies reported that STN-DBS ON-stimulation state was associated with higher word and clause production rates, a measure of language formulation speed (Ehlen et al., 2020); increased proportion of open class words, a measure of word retrieval (Ehlen et al., 2020; Tiedt et al., 2021); and shorter pause durations at non-linguistic boundaries indicating more efficient language planning (Ehlen et al., 2020; Klostermann et al., 2008). However, these improvements may be offset by an increased proportion of language production errors in STN-DBS ON-stimulation (Ehlen et al., 2020) and by reductions in lexical complexity reported in VIM-DBS ON-stimulation state (Tiedt et al., 2021).

Collectively, this literature underscores the complexity of DBS stimulation effects on speech and spoken language production. While more work is needed, these data provide preliminary evidence of possible associations between cognition and communication outcomes broadly post-DBS, which may be an important consideration for pre- and post-surgical counseling and assessment.

4.3 Transcranial magnetic stimulation

Transcranial Magnetic Stimulation (TMS) is a non-invasive stimulation intervention that may improve motor and non-motor symptoms of PD (Wu et al., 2008). Multiple mechanisms are implicated in these improvements including improved cortical excitability, normalization of neural network activity, and increased dopamine release (Wu et al., 2008). Using repetitive TMS (rTMS), Dias et al. (2006) randomized people with PD to undergo 10 sessions of either 15Hz rTMS of the left dorsolateral prefrontal cortex or sham stimulation of the same area during a 2-week period. Results indicated that people with PD had no significant improvements in loudness or fundamental frequency. However, both groups showed improvement in voice-related qualify of life, irrespective of stimulation group, suggestive of a placebo effect. Brabenec and colleagues found improvements in articulatory accuracy and rhythmicity of speech when the right posterior superior temporal gyrus (STG) was targeted at low frequency (1Hz) stimulation levels (Brabenec et al., 2019). A follow up study by the same group compared a randomized sham stimulation group to a group with PD undergoing 1Hz rTMS of the right STG for 10 total sessions across 2 weeks (Brabenec et al., 2021). They found improved intrinsic connectivity within the targeted stimulation area, caudate nucleus, and orofacial sensorimotor cortex when the right STG was targeted and noted corresponding improvements in intelligibility and prosody post-treatment for both groups (Brabenec et al., 2021). However, only the treatment group maintained these changes post-stimulation.

Interventions targeting neural connectivity hold promise for treating PD-related speech deficits. Previous studies showed that altered connectivity between the right caudate nucleus and left sensorimotor cortex differentiated healthy controls from persons with PD, while decreased connectivity between the left STG and left putamen distinguished persons with PD and speech impairments from those without speech impairments (Manes et al., 2018). Further research around stimulation targets and dosing, as well as the impact on functional communication and participation, need to be done before non-invasive brain stimulation techniques can be implemented as mainstream interventions for PD-related speech impairments. However, preliminary studies suggest the potential of such treatments either in isolation or in combination with behavioral interventions (Li et al., 2021).

4.4 Behavioral interventions

Voice loudness and speech clarity are often the primary targets of interventions for communication difficulties in Parkinson's disease. The overwhelming majority of interventions in PD are high-intensity interventions based in motor learning principles. While several systematic reviews report positive benefits of speech-language interventions for persons with PD, their efficacy is largely limited to voice loudness and "weakened" voice quality (Atkinson-Clement et al., 2015; Xu et al., 2020) leaving individuals with potentially unmet functional communication needs and communication participation limitations.

Lee Silverman Voice Treatment (LSVT LOUD®).—Consistent with principles of neural plasticity, therapeutic activities in LSVT LOUD include high effort exercises intended to increase vocal intensity by targeting respiratory and laryngeal systems. LSVT LOUD is implemented through clinician modeling, immediate direct and self-evaluative feedback, and recalibration of muscular and respiratory effort in a variety of speaking contexts. Cognitive aspects of speech production are not directly targeted. Nonetheless, LSVT LOUD proposes to re-calibrate the internal model of adequate speech loudness and muscular effort through repeated practice and internalization of the concept of "thinking loud" to minimize the cognitive effort required to maintain vocal effort during contextualized speaking tasks. Although LSVT LOUD claims to be low cognitive effort, there are no systematic, well-controlled studies of LSVT LOUD in persons with moderate to advanced disease or in those with PD-related cognitive impairment. As a result, it remains unclear whether treatment effects are consistent across a broad range of PD severity levels and symptom profiles.

Of the published speech interventions, LSVT LOUD has the more robust body of evidence including randomized control studies with no treatment, respiratory treatment, and alternative LSVT treatment format conditions. Results from efficacy studies and systematic reviews of LSVT LOUD show increased loudness and improved Voice Handicap Index scores for more structured speaking tasks following treatment, that is sustained for up to 24 months post-treatment in those with mild to moderate PD (Ramig et al., 2001, 2018; Scobie et al., 2021). Additionally, Bryans et al. (2021) using a within subject design found post-treatment increases in communication participation and effectiveness following LSVT LOUD that were maintained for upwards of 6-months in a cohort with a wide distribution of motor severity scores. These results suggest communication impairments in PD may be helped through treatments like LSVT LOUD that target loudness and intelligibility.

SPEAK OUT!®.—SPEAK OUT! and its group-based companion program, The LOUD Crowd®, target "speaking with intent" within the context of structured exercises and communication activities. The program includes a variety of structured and semi-structured treatment activities, with layered cognitive demands, that attempt to simulate "real-world" communication. The program claims to optimize "goal-directed basal ganglia-cortical circuits" to make up for impairments in automatic control of speech and voice (Behrman et al., 2020).

SPEAK OUT! builds on previous studies of clear speech techniques (Lam and Tjaden, 2016; Lam et al., 2012; Tjaden et al., 2013) and voice facilitation approaches described by Boone et al. (2005). In contrast to LSVT LOUD, rather than focusing on vocal intensity in isolation, SPEAK OUT! proposes to target increased loudness, improved articulatory precision, and deliberate speech rate through increasing the person with PD's focus on attending to a goal-directed speech target while speaking with intent (Behrman et al., 2020). Therapeutic techniques include direct instruction and metacognitive strategies (Behrman et al., 2020).

Emerging evidence of SPEAKOUT!, from early stage low to medium quality studies, suggest that post-treatment gains include increased speech volume, improved prosody (variation in intensity and frequency), improved cepstral peak prominence, and reductions in voice handicap (Behrman et al., 2020; Boutsen et al., 2018; Levitt et al., 2015; Levitt and Walker-Batson, 2018). However, not all studies have demonstrated superiority of the treatment over a control condition (Parveen, 2020). Further research on the wider effects of SPEAK OUT! and LOUD Crowd beyond mild to moderate PD and dysarthria is warranted. Currently, there are no published comparative studies of LSVT LOUD versus SPEAK OUT! to guide clinical decision-making regarding the selection of treatment for an individual with PD. Given the overlaps in approach, and claims of distinctive mechanisms, this is an important next step to optimize care for persons with PD.

SpeechVive™.—For some patients with Parkinson's, cognitive and sensory deficits may be a barrier to participating in high intensity exercise programs or intentionally monitoring their own speech volume and output. While not specifically designed for persons with PD who have cognitive impairments, the SpeechVive device has the benefit of increasing speech intensity without volitional effort and thus has potential to lessen the cognitive load associated with treatment participation. The SpeechVive is a wearable device that elicits increased speech volume through the Lombard effect. The Lombard effect refers to the non-volitional increase in speech effort and consequently increased loudness, in ambient noise conditions (Zollinger and Brumm, 2011). The Lombard effect in PD was first tested in PD by Adams and Lang (1992), who found that individuals consistently increased speech volume when attempting to speak over 90dB-SPL ambient white noise.

The SpeechVive device, initially developed by Dr. Jessica Huber, is housed in a casing similar to a hearing aid. It detects when the patient speaks and plays "multi-talker babble noise" designed to trigger the Lombard effect and increase the patient's speech volume (Richardson et al., 2014; Stathopoulos et al., 2014). This results in increased speech volume without interfering with the patient's ability to hear other speakers, as the noise plays only when the patient is speaking. On its own, studies showed that leveraging the Lombard effect in PD improved subglottic pressure and voice intensity through more efficient respiratory and laryngeal system activity (Stathopoulos et al., 2014). When embedded within a structured treatment program SpeechVive demonstrated reliable increases in speech volume in persons with PD (Huber et al., 2019; Richardson et al., 2014). Additionally, longer voice onset time (VOTs) and higher percent voicing after treatment with the SpeechVive device were observed (Huber et al., 2019, Richardson et al., 2014). Those with greater improvements in percent voicing also experience improved intelligibility (Huber et al., 2019, Richardson et al., 2014). While this is a low-cognitive effort treatment approach that requires considerably fewer in-person SLT sessions compared to other behavioral interventions in PD, its efficacy in a cognitively impaired cohort requires further study.

Other behavioral interventions.—Other emerging interventions hold promise for persons with PD. Preliminary data for targeted strengthening of respiratory muscles through resistive expiratory muscle training suggest that post-training, participants with PD demonstrate improved maximum expiratory pressure, subglottic pressure, speech loudness and increased maximum phonation time (Reyes et al., 2020; Rodríguez et al., 2020). Embedded within a therapeutic arts model, recent studies of structured group-based and individual chorale/singing therapy programs suggest that these programs may be effective in improving acoustic, perceptual, quality of life voice, and intelligibility measures in PD (Barnish and Barran, 2020; Han et al., 2018; Higgins and Richardson, 2019; Tamplin et al., 2019).

While interventions in PD are still largely motor-symptom focused, there is an emerging movement toward developing conversation-based interventions in PD that address everyday communication through the training of targeted conversation strategies (Forsgren et al., 2013). Although the development of these therapies lags far behind those that target motor symptoms.

5 Summary

Theoretical frameworks and experimental evidence are converging on the idea that cognitive changes in PD likely play a role in the manifestation of communication difficulties in PD. Moreover, the literature makes clear that early and sub-dementia cognitive changes are associated with myriad communication changes that span motor and language domains. The fact that this chapter exists is evidence of a shift in our understanding of PD and its effects on communication beyond the motor-speech domain. In keeping with this shift, the last decade has seen an increased interest in the effects of pharmacologic and surgical interventions on speech outcomes and substantial expansions of behavioral treatments for speech and communication impairments in PD. Optimizing clinical care for persons with PD depends on continuing this trajectory and advancing research into the cognitive mechanisms that support speech production, the impact of PD treatments on speech/communication outcomes, and systematic, well-controlled trials of interventions for a broad range of communication challenges.

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References

Aarsland D, Batzu L, Halliday GM, Geurtsen GJ, Ballard C, Chaudhuri KR, Weintraub D, 2021. Parkinson disease-associated cognitive impairment. Nat. Rev. Dis. Primers 7 (1), 1–21. [PubMed: 33414454]

Adams SG, Lang AE, 1992. Can the Lombard effect be used to improve low voice intensity in Parkinson's disease? Eur. J. Disord. Commun 27 (2), 121–127. [PubMed: 1446099]

Aldridge D, Theodoros D, Angwin A, Vogel AP, 2016. Speech outcomes in Parkinson's disease after subthalamic nucleus deep brain stimulation: a systematic review. Parkinsonism Relat. Disord 33, 3–11. [PubMed: 27693195]

Alonso-Recio L, Carvajal F, Merino C, Serrano JM, 2021. Social cognition and cognitive decline in patients with Parkinson's disease. J. Int. Neuropsychol. Soc 27 (7), 744–755. [PubMed: 33243315]

Altmann LJ, Troche MS, 2011. High-level language production in Parkinson's disease: a review. Parkinsons Dis. 2011, 238956. [PubMed: 21860777]

Arten T.L.d.S., Hamdan AC, 2020. Executive functions in Parkinson's disease with and without deep brain stimulation (DBS): a systematic review. Dement. Neuropsychol 14, 178–185. [PubMed: 32595888]

Ash S, Xie SX, Gross RG, Dreyfuss M, Boller A, Camp E, Grossman M, 2012. The organization and anatomy of narrative comprehension and expression in Lewy body spectrum disorders. Neuropsychology 26 (3), 368–384. [PubMed: 22309984]

Ash S, Jester C, York C, Kofman OL, Langey R, Halpin A, Spindler M, 2017. Longitudinal decline in speech production in Parkinson's disease spectrum disorders. Brain Lang. 171, 42–51. [PubMed: 28527315]

Atkinson-Clement C, Sadat J, Pinto S, 2015. Behavioral treatments for speech in Parkinson's disease: meta-analyses and review of the literature. Neurodegener. Dis. Manag 5 (3), 233–248. [PubMed: 26107322]

Barnish MS, Barran SM, 2020. A systematic review of active group-based dance, singing, music therapy and theatrical interventions for quality of life, functional communication, speech, motor function and cognitive status in people with Parkinson's disease. BMC Neurol. 20 (1), 371. [PubMed: 33038925]

Barnish MS, Horton SM, Butterfint ZR, Clark AB, Atkinson RA, Deane KH, 2017. Speech and communication in Parkinson's disease: a cross-sectional exploratory study in the UK. BMJ Open 7 (5), e014642.

Bastiaanse R, Leenders KL, 2009. Language and Parkinson's disease. Cortex 45 (8), 912–914. [PubMed: 19447385]

Behrman A, Cody J, Elandary S, Flom P, Chitnis S, 2020. The effect of SPEAK OUT! And the LOUD crowd on dysarthria due to Parkinson's disease. Am. J. Speech Lang. Pathol 29 (3), 1448–1465. [PubMed: 32421347]

Behroozmand R, Shebek R, Hansen DR, Oya H, Robin DA, Howard III MA, Greenlee JD, 2015. Sensory–motor networks involved in speech production and motor control: an fMRI study. Neuroimage 109, 418–428. [PubMed: 25623499]

Bocanegra Y, García AM, Pineda D, Buriticá O, Villegas A, Lopera F, Trujillo N, 2015. Syntax, action verbs, action semantics, and object semantics in Parkinson's disease: dissociability, progression, and executive influences. Cortex 69, 237–254. [PubMed: 26103601]

Boone DR, McFarlane SC, Von Berg SL, Zraick RI, 2005. The Voice and Voice Therapy, ninth ed. Pearson/Allyn & Bacon, Boston, MA.

Boutsen F, Park E, Dvorak J, Cid C, 2018. Prosodic improvement in persons with Parkinson disease receiving SPEAK OUT!(R) voice therapy. Folia Phoniatr. Logop 70 (2), 51–58. [PubMed: 29996139]

Brabenec L, Klobusiakova P, Barton M, Mekyska J, Galaz Z, Zvoncak V, Kostalova M, 2019. Non-invasive stimulation of the auditory feedback area for improved articulation in Parkinson's disease. Parkinsonism Relat. Disord 61, 187–192. [PubMed: 30337204]

Brabenec L, Klobusiakova P, Simko P, Kostalova M, Mekyska J, Rektorova I, 2021. Non-invasive brain stimulation for speech in Parkinson's disease: a randomized controlled trial. Brain Stimul. 14 (3), 571–578. [PubMed: 33781956]

Broadfoot CK, Abur D, Hoffmeister JD, Stepp CE, Ciucci MR, 2019. Research-based updates in swallowing and communication dysfunction in Parkinson disease: implications for evaluation and management. Perspect. ASHA Spec. Interest Groups 4 (5), 825–841. [PubMed: 32104723]

Bryans LA, Palmer AD, Anderson S, Schindler J, Graville DJ, 2021. The impact of Lee Silverman Voice Treatment (LSVT LOUD(R)) on voice, communication, and participation: findings from a prospective, longitudinal study. J. Commun. Disord 89, 106031. [PubMed: 33259945]

Burton EJ, McKeith IG, Burn DJ, Williams ED, O'Brien JT, 2004. Cerebral atrophy in Parkinson's disease with and without dementia: a comparison with Alzheimer's disease, dementia with Lewy bodies and controls. Brain 127 (4), 791–800. [PubMed: 14749292]

Cavallieri F, Budriesi C, Gessani A, Contardi S, Fioravanti V, Menozzi E, Antonelli F, 2021. Dopaminergic treatment effects on dysarthric speech: acoustic analysis in a cohort of patients with advanced Parkinson's disease. Front. Neurol 11, 616062. [PubMed: 33613419]

Chivukula S, Jafari M, Aflalo T, Yong NA, Pouratian N, 2019. Cognition in sensorimotor control: interfacing with the posterior parietal cortex. Front. Neurosci 13, 140. [PubMed: 30872993]

Cools R, Miyakawa A, Sheridan M, D'Esposito M, 2010. Enhanced frontal function in Parkinson's disease. Brain 133, 225–233. [PubMed: 19995871]

Cotelli M, Borroni B, Manenti R, Zanetti M, Ar evalo A, Cappa S, Padovani A, 2007. Action and object naming in Parkinson's disease without dementia. Eur. J. Neurol 14 (6), 632–637. [PubMed: 17539940]

Dara C, Monetta L, Pell MD, 2008. Vocal emotion processing in Parkinson's disease: reduced sensitivity to negative emotions. Brain Res. 1188, 100–111. [PubMed: 18022608]

De Letter M, Santens P, De Bodt M, Boon P, Van Borsel J, 2006. Levodopa-induced alterations in speech rate in advanced Parkinson's disease. Acta Neurol. Belg 106 (1), 19–22. [PubMed: 16776432]

De Letter M, Santens P, Estercam I, Van Maele G, De Bodt M, Boon P, Van Borsel J, 2007. Levodopa-induced modifications of prosody and comprehensibility in advanced Parkinson's disease as perceived by professional listeners. Clin. Linguist. Phon 21 (10), 783–791. [PubMed: 17882694]

De Letter M, Van Borsel J, Boon P, De Bodt M, Dhooge I, Santens P, 2010. Sequential changes in motor speech across a levodopa cycle in advanced Parkinson's disease. Int. J. Speech Lang. Pathol 12 (5), 405–413. [PubMed: 20602581]

Dias A, Barbosa E, Coracini K, Maia F, Marcolin M, Fregni F, 2006. Effects of repetitive transcranial magnetic stimulation on voice and speech in Parkinson's disease. Acta Neurol. Scand 113 (2), 92–99. [PubMed: 16411969]

Ehlen F, Al-Fatly B, Kuhn AA, Klostermann F, 2020. Impact of deep brain stimulation of the subthalamic nucleus on natural language in patients with Parkinson's disease. PLoS One 15 (12), e0244148. [PubMed: 33373418]

Fabbri M, Guimarães I, Cardoso R, Coelho M, Guedes LC, Rosa MM, Antonini A, 2017. Speech and voice response to a levodopa challenge in late-stage Parkinson's disease. Front. Neurol 8, 432. [PubMed: 28878734]

Fernandino L, Conant LL, Binder JR, Blindauer K, Hiner B, Spangler K, Desai RH, 2013. Parkinson's disease disrupts both automatic and controlled processing of action verbs. Brain Lang. 127 (1), 65–74. [PubMed: 22910144]

Foley JA, Foltynie T, Zrinzo L, Hyam JA, Limousin P, Cipolotti L, 2017. Apathy and reduced speed of processing underlie decline in verbal fluency following DBS. Behav. Neurol 2017, 7348101. [PubMed: 28408788]

Forsgren E, Antonsson M, Saldert C, 2013. Training conversation partners of persons with communication disorders related to Parkinson's disease—a protocol and a pilot study. Logoped. Phoniatr. Vocol 38 (2), 82–90. [PubMed: 23072445]

Garrett MF, 1989. Processes in language production. In: Linguistics: The Cambridge Survey, pp. 69–96.

Gauvin HS, Hartsuiker RJ, 2020. Towards a new model of verbal monitoring. J. Cogn 3 (1), 17. 10.5334/joc.81. [PubMed: 32944680]

Gauvin HS, Mertens J, Mariën P, Santens P, Pickut BA, Hartsuiker RJ, 2017. Verbal monitoring in Parkinson's disease: a comparison between internal and external monitoring. PLoS One 12 (8), e0182159. [PubMed: 28832595]

Goberman AM, Blomgren M, Metzger E, 2010. Characteristics of speech disfluency in Parkinson disease. J. Neurolinguistics 23 (5), 470–478.

Golfinopoulos E, Tourville JA, Guenther FH, 2010. The integration of large-scale neural network modeling and functional brain imaging in speech motor control. Neuroimage 52 (3), 862–874. [PubMed: 19837177]

Gomez-Gomez LF, Morales A, Fierrez J, Orozco-Arroyave JR, 2020. Exploring Facial Expressions and Affective Domains for Parkinson Detection. (arXiv preprint arXiv:2012.06563).

Griffiths S, 2013. Managing Everyday Participation in Parkinson's Disease: A Conversation Analytic Study. Dissertation, Exeter and Plymouth Peninsula Medical School.

Griffiths S, Barnes R, Britten N, Wilkinson R, 2012. Potential causes and consequences of overlap in talk between speakers with Parkinson's disease and their familiar conversation partners. Semin. Speech Lang 33 (1), 27–43. [PubMed: 22362322]

Griffiths S, Barnes R, Britten N, Wilkinson R, 2015. Multiple repair sequences in everyday conversations involving people with Parkinson's disease. Int. J. Lang. Commun. Disord 50 (6), 814–829. [PubMed: 26152654]

Guenther FH, Hickok G, 2015. Role of the auditory system in speech production. Handb. Clin. Neurol 129, 161–175. [PubMed: 25726268]

Guo Z, Wu X, Li W, Jones JA, Yan N, Sheft S, Liu H, 2017. Top-down modulation of auditory-motor integration during speech production: the role of working memory. J. Neurosci 37 (43), 10323–10333. [PubMed: 28951450]

Han EY, Yun JY, Chong HJ, Choi K-G, 2018. Individual therapeutic singing program for vocal quality and depression in Parkinson's disease. J. Mov. Disord 11 (3), 121–128. [PubMed: 30086617]

Hanna-Pladdy B, Pahwa R, Lyons KE, 2015. Paradoxical effect of dopamine medication on cognition in Parkinson's disease: relationship to side of motor onset. J. Int. Neuropsychol. Soc 21 (4), 259–270. [PubMed: 25923830]

Herrera E, Cuetos F, 2012. Action naming in Parkinson's disease patients on/off dopamine. Neurosci. Lett 513 (2), 219–222. [PubMed: 22387157]

Hickok G, 2012. Computational neuroanatomy of speech production. Nat. Rev. Neurosci 13 (2), 135–145. [PubMed: 22218206]

Higgins AN, Richardson KC, 2019. The effects of a choral singing intervention on speech characteristics in individuals with Parkinson's disease: an exploratory study. Commun. Disord. Q 40 (4), 195–205.

Ho AK, Iansek R, Marigliani C, Bradshaw JL, Gates S, 1998. Speech impairment in a large sample of patients with Parkinson's disease. Behav. Neurol 11 (3), 131–137. [PubMed: 11568413]

Ho AK, Bradshaw JL, Iansek R, 2008. For better or worse: the effect of levodopa on speech in Parkinson's disease. Mov. Disord 23 (4), 574–580. [PubMed: 18163453]

Hochstadt J, 2009. Set-shifting and the on-line processing of relative clauses in Parkinson's disease: results from a novel eye-tracking method. Cortex 45 (8), 991–1011. [PubMed: 19473654]

Huber JE, Darling-White M, 2017. Longitudinal changes in speech breathing in older adults with and without Parkinson's disease. Semin. Speech Lang 38 (3), 200–209. [PubMed: 28618443]

Huber J, Stathopoulos E, Sussman J, Richardson K, Matheron D, Snyder S, 2019. Changes to speech production after SpeechVive treatment in individuals with Parkinson's disease. Mov. Disord 34 (Suppl. 2).

Illes J, Metter E, Hanson W, Iritani S, 1988. Language production in Parkinson’s disease: acoustic and linguistic considerations. Brain Lang. 33 (1), 146–160. [PubMed: 3342316]

Im H, Adams S, Abeyesekera A, Pieterman M, Gilmore G, Jog M, 2019. Effect of Levodopa on speech dysfluency in Parkinson’s disease. Mov. Disord. Clin. Pract 6 (2), 150–154. [PubMed: 30838314]

Johari K, Behroozmand R, 2018. Functional dissociation of temporal processing mechanisms during speech production and hand movement: an ERP study. Behav. Brain Res 347, 281–291. [PubMed: 29577960]

Johari K, Walenski M, Reifegerste J, Ashrafi F, Behroozmand R, Daemi M, Ullman MT, 2019. A dissociation between syntactic and lexical processing in Parkinson’s disease. J. Neurolinguistics 51, 221–235. [PubMed: 31777416]

Jones C, Jahanshahi M, 2014. Motor and perceptual timing in Parkinson’s disease. In: Merchant H, de Laufuente V (Eds.), Neurobiology of Interval Timing. Autophagy: Biology and Diseases, pp. 265–290. 10.1007/978-1-4939-1782-2_14.

Kendi AK, Lehericy S, Luciana M, Ugurbil K, Tuite P, 2008. Altered diffusion in the frontal lobe in Parkinson disease. Am. J. Neuroradiol 29 (3), 501–505. [PubMed: 18202242]

Klostermann F, Ehlen F, Vesper J, Nubel K, Gross M, Marzinzik F, Sappok T, 2008. Effects of subthalamic deep brain stimulation on dysarthrophonia in Parkinson’s disease. J. Neurol. Neurosurg. Psychiatry 79 (5), 522–529. [PubMed: 17766427]

Knowles T, Adams S, Abeyesekera A, Mancinelli C, Gilmore G, Jog M, 2018. Deep brain stimulation of the subthalamic nucleus parameter optimization for vowel acoustics and speech intelligibility in Parkinson’s disease. J. Speech Lang. Hear. Res 61 (3), 510–524.

Lam J, Tjaden K, 2016. Clear speech variants: an acoustic study in Parkinson’s disease. J. Speech Lang. Hear. Res 59 (4), 631–646. [PubMed: 27355431]

Lam J, Tjaden K, Wilding G, 2012. Acoustics of Clear Speech: Effect of Instruction.

Lee J, Huber J, Jenkins J, Fredrick J, 2019. Language planning and pauses in story retell: evidence from aging and Parkinson’s disease. J. Commun. Disord 79, 1–10. [PubMed: 30844602]

Levelt WJ, 1980. On-line processing constraints on the properties of signed and spoken language. In: Signed and Spoken Language: Biological Constraints on Linguistic Form. Verlag Chemie, pp. 141–160.

Levitt JS, Walker-Batson D, 2018. The effects of the “speak with intent” instruction for individuals with Parkinson’s disease. Assist. Technol 1, 1–15.

Levitt JS, Chitnis S, Walker-Batson D, 2015. The effects of the “SPEAK OUT!®” and “LOUD crowd®” voice programs for Parkinson. Int. J. Health Sci 3 (2), 13–19.

Li Q, Millard K, Tetnowski J, Narayana S, Cannito M, 2021. Acoustic analysis of intonation in persons with Parkinson’s disease receiving transcranial magnetic stimulation and intensive voice treatment. J. Voice (Epub ahead of print).

Lieberman P, Kako E, Friedman J, Tajchman G, Feldman LS, Jiminez EB, 1992. Speech production, syntax comprehension, and cognitive deficits in Parkinson’s disease. Brain Lang. 43 (2), 169–189. [PubMed: 1393519]

Liu Y, Fan H, Li J, Jones JA, Liu P, Zhang B, Liu H, 2018. Auditory-motor control of vocal production during divided attention: behavioral and ERP correlates. Front. Neurosci 12, 113. [PubMed: 29535605]

Louis ED, Winfield L, Fahn S, Ford B, 2001. Speech dysfluency exacerbated by levodopa in Parkinson’s disease. Move. Disord 16 (3), 562–565.

Lowit A, Marchetti A, Corson S, Kuschmann A, 2018. Rhythmic performance in hypokinetic dysarthria: relationship between reading, spontaneous speech and diadochokinetic tasks. J. Commun. Disord 72, 26–39. [PubMed: 29471176]

MacDonald PA, Monchi O, 2011. Differential effects of dopaminergic therapies on dorsal and ventral striatum in Parkinson’s disease: implications for cognitive function. Parkinson’s Dis. 2011, 572743. [PubMed: 21437185]

Manes JL, Tjaden K, Parrish T, Simuni T, Roberts A, Greenlee JD, Kurani AS, 2018. Altered resting-state functional connectivity of the putamen and internal globus pallidus is related to speech impairment in Parkinson’s disease. Brain Behav. 8 (9), e01073. [PubMed: 30047249]

Martel-Sauvageau V, Tjaden K, 2017. Vocalic transitions as markers of speech acoustic changes with STN-DBS in Parkinson's disease. J. Commun. Disord 70, 1–11. [PubMed: 29032347]

McAuliffe MJ, Baylor CR, Yorkston KM, 2017. Variables associated with communicative participation in Parkinson's disease and its relationship to measures of health-related quality-of-life. Int. J. Speech Lang. Pathol 19 (4), 407–417. [PubMed: 27347713]

McDougle SD, Taylor JA, 2019. Dissociable cognitive strategies for sensorimotor learning. Nat. Commun 10 (1), 1–13. [PubMed: 30602773]

McGuigan S, Zhou S-H, Brosnan MB, Thyagarajan D, Bellgrove MA, Chong TT, 2019. Dopamine restores cognitive motivation in Parkinson's disease. Brain 142 (3), 719–732. [PubMed: 30689734]

McNamara P, Obler LK, Au R, Durso R, Albert ML, 1992. Speech monitoring skills in Alzheimer's disease, Parkinson's disease, and normal aging. Brain Lang. 42 (1), 38–51. [PubMed: 1547468]

Mikos A, Zahodne L, Okun MS, Foote K, Bowers D, 2010. Cognitive declines after unilateral deep brain stimulation surgery in Parkinson's disease: a controlled study using Reliable Change, part II. Clin. Neuropsychol. 24 (2), 235–245.

Miller N, 2017. Communication changes in Parkinson's disease. Pract. Neurol 17 (4), 266–274. [PubMed: 28687681]

Miller N, Noble E, Jones D, Burn D, 2006. Life with communication changes in Parkinson's disease. Age Ageing 35 (3), 235–239. [PubMed: 16540492]

Miller N, Allcock L, Jones D, Noble E, Hildreth AJ, Burn DJ, 2007. Prevalence and pattern of perceived intelligibility changes in Parkinson's disease. J. Neurol. Neurosurg. Psychiatry 78 (11), 1188–1190. [PubMed: 17400592]

Miller N, Noble E, Jones D, Deane KH, Gibb C, 2015. Survey of speech and language therapy provision for people with Parkinson's disease in the United Kingdom: patients' and carers' perspectives. Int. J. Lang. Commun. Disord, 1–10. [PubMed: 25069537]

Miller CA, Behroozmand R, Etler CP, Nourski KV, Reale RA, Oya H, Greenlee JD, 2021. Neural correlates of vocal auditory feedback processing: unique insights from electro-corticography recordings in a human cochlear implant user. Eneuro 8 (1).

Mossner JM, Chou KL, Maher AH, Persad CC, Patil PG, 2020. Localization of motor and verbal fluency effects in subthalamic DBS for Parkinson's disease. Parkinsonism Relat. Disord 79, 55–59. [PubMed: 32866879]

Murray LL, 2000. Spoken language production in Huntington's and Parkinson's diseases. J. Speech Lang. Hear. Res 43 (6), 1350–1366. [PubMed: 11193957]

Natsopoulos D, Grouios G, Bostantzopoulou S, Mentenopoulos G, Katsarou Z, Logothetis J, 1993. Algorithmic and heuristic strategies in comprehension of complement clauses by patients with Parkinson's disease. Neuropsychologia 31 (9), 951–964. [PubMed: 8232851]

Nozari N, Novick J, 2017. Monitoring and control in language production. Curr. Dir. Psychol. Sci 26 (5), 403–410.

Palmeri R, Lo Buono V, Corallo F, Foti M, Di Lorenzo G, Bramanti P, Marino S, 2017. Nonmotor symptoms in Parkinson disease: a descriptive review on social cognition ability. J. Geriatr. Psychiatry Neurol 30 (2), 109–121. [PubMed: 28073327]

Parveen S, 2020. Group-based intervention of participants with Parkinson disease: findings from a 6-month LOUD Crowd® program. Clin. Arch. Commun. Disord 5 (2), 96–105.

Pell MD, Monetta L, Rothermich K, Kotz SA, Cheang HS, McDonald S, 2014. Social perception in adults with Parkinson's disease. Neuropsychology 28 (6), 905–916. [PubMed: 24885448]

Péran P, Rascol O, Démonet J-F, Celsis P, Nespoulous J-L, Dubois B, Cardebat D, 2003. Deficit of verb generation in nondemented patients with Parkinson's disease. Mov. Disord 18 (2), 150–156. [PubMed: 12539207]

Pinho P, Monteiro L, de Paula Soares MF, Tourinho L, Melo A, Nóbrega AC, 2018. Impact of levodopa treatment in the voice pattern of Parkinson's disease patients: a systematic review and meta-analysis. Codas 30 (5), e20170200. [PubMed: 30304100]

Plowman-Prine E, Okun M, Sapienza C, Shrivastav R, Fernandez H, Foote K, Rosenbek J, 2009. Perceptual characteristics of Parkinsonian speech: a comparison of the pharmacological effects of levodopa across speech and non-speech motor systems. NeuroRehabilitation 24 (2), 131–144. [PubMed: 19339752]

Postma A, 2000. Detection of errors during speech production: a review of speech monitoring models. Cognition 77 (2), 97–132. [PubMed: 10986364]

Postuma RB, Aarsland D, Barone P, Burn DJ, Hawkes CH, Oertel W, Ziemssen T, 2012. Identifying prodromal Parkinson's disease: pre-motor disorders in Parkinson's disease. Mov. Disord 27 (5), 617–626. [PubMed: 22508280]

Prenger M, Madray R, Van Hedger K, Anello M, MacDonald PA, 2020. Social symptoms of Parkinson's disease. Parkinson's Dis 2020, 8846544. [PubMed: 33489081]

Ramig LO, Sapir S, Countryman S, Pawlas AA, O'Brien C, Hoehn M, Thompson LL, 2001. Intensive voice treatment (LSVT$^{\circledR}$) for patients with Parkinson's disease: a 2 year follow up. J. Neurol. Neurosurg. Psychiatry 71 (4), 493–498. [PubMed: 11561033]

Ramig LO, Fox C, Sapir S, 2008. Speech treatment for Parkinson's disease. Expert Rev. Neurother 8 (2), 297–309. [PubMed: 18271714]

Ramig L, Halpern A, Spielman J, Fox C, Freeman K, 2018. Speech treatment in Parkinson's disease: randomized controlled trial (RCT). Mov. Disord 33 (11), 1777–1791. [PubMed: 30264896]

Reyes A, Castillo A, Castillo J, Cornejo I, Cruickshank T, 2020. The effects of respiratory muscle training on phonatory measures in individuals with Parkinson's disease. J. Voice 34 (6), 894–902. [PubMed: 31155431]

Richardson K, Sussman JE, Stathopoulos ET, Huber JE, 2014. The effect of increased vocal intensity on interarticulator timing in speakers with Parkinson's disease: a preliminary analysis. J. Commun. Disord 52, 44–64. [PubMed: 25459460]

Rinne N, Roberts R, 2019. Examining Conversation Breakdowns in Parkinson's Disease Versus Alzheimer's Dementia. Thesis, Northwestern University.

Roberts AC, 2014. Characterizing Spoken Discourse in Individuals With Parkinson's Disease Without Dementia. Health and Rehabilitation Sciences, The University of Western Ontario. Doctor of Philosophy.

Roberts A, Orange JB, 2013. Discourse in Lowy body spectrum disorder. In: Davis B, Guendouzi J (Eds.), Pragmatics in Dementia Discourse. Cambridge Scholars Publishing, Newcastle upon Tyne, pp. 153–211.

Roberts A, Post D, 2018. Information content and efficiency in the spoken discourse of individuals with Parkinson's disease. J. Speech Lang. Hear. Res 61 (9), 2259–2274. [PubMed: 30208482]

Roberts A, Nguyen P, Orange JB, Jog M, Nisbet KA, McRae K, 2017. Differential impairments of upper and lower limb movements influence action verb processing in Parkinson disease. Cortex 97, 49–59. [PubMed: 29080416]

Roberts AC, Rafferty MR, Wu SS, Miao G, Cubillos F, Simuni T, Neault M, 2021. Patterns and predictors of referrals to allied health services for individuals with Parkinson's disease: a Parkinson's foundation (PF) QII study. Parkinsonism Relat. Disord 83, 115–122. [PubMed: 33339716]

Roca M, Torralva T, Gleichgerrcht E, Chade A, Ar evalo GG, Gershanik O, Manes F, 2010. Impairments in social cognition in early medicated and unmedicated Parkinson disease. Cogn. Behav. Neurol 23 (3), 152–158. [PubMed: 20829664]

Rodríguez MÁ, Crespo I, Del Valle M, Olmedillas H, 2020. Should respiratory muscle training be part of the treatment of Parkinson's disease? A systematic review of randomized controlled trials. Clin. Rehabil 34 (4), 429–437. [PubMed: 31875689]

Rusz J, Tykalová T, Klempí r J, Cmejla R, Růži cka E, 2016. Effects of dopaminergic replacement therapy on motor speech disorders in Parkinson's disease: longitudinal follow-up study on previously untreated patients. J. Neural Transm 123 (4), 379–387. [PubMed: 26843071]

Saldert C, Bauer M, 2017. Multifaceted communication problems in everyday conversations involving people with Parkinson's disease. Brain Sci. 7 (10), 123. [PubMed: 28946714]

Saldert C, Ferm U, Bloch S, 2014. Semantic trouble sources and their repair in conversations affected by Parkinson's disease. Int. J. Lang. Commun. Disord 49 (6), 710–721. [PubMed: 24934292]

Salmazo-Silva H, Parente MA, Rocha MS, Baradel RR, Cravo AM, Sato JR, Carthery-Goulart MT, 2017. Lexical-retrieval and semantic memory in Parkinson's disease: the question of noun and verb dissociation. Brain Lang. 165, 10–20. [PubMed: 27912072]

Sanabria J, Ruiz PG, Gutierrez R, Marquez F, Escobar P, Gentil M, Cenjor C, 2001. The effect of levodopa on vocal function in Parkinson’s disease. Clin. Neuropharmacol 24 (2), 99–102. [PubMed: 11307045]

Schalling E, Johansson K, Hartelius L, 2017. Speech and communication changes reported by people with Parkinson’s disease. Folia Phoniatr. Logop 69 (3), 131–141. [PubMed: 29346787]

Schmitt F, Aarsland D, BrØnnick K, Meng X, Tekin S, Okin J, 2010. Evaluating rivastigmine in mild-to-moderate Parkinson’s disease dementia using ADAS-Cog items. Am. J. Alzheimer’s Dis. Other Dement 25 (5), 407–413.

Schwartz R, Pell MD, 2017. When emotion and expression diverge: the social costs of Parkinson’s disease. J. Clin. Exp. Neuropsychol 39 (3), 211–230. [PubMed: 27548715]

Scobie S, Jowett S, Lambe T, Patel S, Woolley R, Ives N, Sackley C, 2021. Lee Silverman voice treatment versus standard speech and language therapy versus control in Parkinson’s disease: preliminary cost-consequence analysis of the PD COMM pilot randomised controlled trial. Pilot Feasibility Stud. 7 (1), 154. [PubMed: 34372913]

Segawa J, Masapollo M, Tong M, Smith DJ, Guenther FH, 2019. Chunking of phonological units in speech sequencing. Brain Lang. 195, 104636. [PubMed: 31202179]

Singh A, Cole R, Espinoza A, Evans A, Cao S, Cavanagh J, Narayanan N, 2021. Timing variability and midfrontal Ȉ4 Hz rhythms correlate with cognition in Parkinson’s disease. NPJ Parkinsons Dis. 7 (1), 14. [PubMed: 33589640]

Skodda S, 2012. Effect of deep brain stimulation on speech performance in Parkinson’s disease. Parkinson’s Dis. 2012, 1–10.

Skodda S, Rinsche H, Schlegel U, 2009. Progression of dysprosody in Parkinson’s disease over time--a longitudinal study. Mov. Disord 24 (5), 716–722. [PubMed: 19117364]

Skodda S, Grönheit W, Schlegel U, 2011. Intonation and speech rate in Parkinson’s disease: general and dynamic aspects and responsiveness to levodopa admission. J. Voice 25 (4), e199–e205. [PubMed: 21051196]

Skodda S, Gronheit W, Mancinelli N, Schlegel U, 2013. Progression of voice and speech impairment in the course of Parkinson’s disease: a longitudinal study. Parkinsons Dis. 2013, 389195. [PubMed: 24386590]

Smith K, Caplan D, 2018. Communication impairment in Parkinson’s disease: impact of motor and cognitive symptoms on speech and language. Brain Lang. 185, 38–46. [PubMed: 30092448]

Stathopoulos ET, Huber JE, Richardson K, Kamphaus J, DeCicco D, Darling M, Sussman JE, 2014. Increased vocal intensity due to the Lombard effect in speakers with Parkinson’s disease: simultaneous laryngeal and respiratory strategies. J. Commun. Disord 48, 1–17. [PubMed: 24438910]

Tamplin J, Morris ME, Marigliani C, Baker FA, Vogel AP, 2019. ParkinSong: a controlled trial of singing-based therapy for Parkinson’s disease. Neurorehabil. Neural Repair 33 (6), 453–463. [PubMed: 31081485]

Tanaka Y, Tsuboi T, Watanabe H, Nakatsubo D, Maesawa S, Kato S, Sobue G, 2020. Longitudinal speech change after subthalamic nucleus deep brain stimulation in Parkinson’s disease patients: a 2-year prospective study. J. Parkinsons Dis 10 (1), 131–140. [PubMed: 31884493]

Thies T, Mucke D, Dano R, Barbe MT, 2021. Levodopa-based changes on vocalic speech movements during prosodic prominence marking. Brain Sci. 11 (5).

Tiedt HO, Ehlen F, Wyrobnik M, Klostermann F, 2021. Thalamic but not subthalamic neuromodulation simplifies word use in spontaneous language. Front. Hum. Neurosci 15.

Tjaden K, 2008. Speech and swallowing in Parkinson’s disease. Top Geriatr Rehabil. 24 (2), 115–126. [PubMed: 19946386]

Tjaden K, Richards E, Kuo C, Wilding G, Sussman J, 2013. Acoustic and perceptual consequences of clear and loud speech. Folia Phoniatr. Logop 65 (4), 214–220. [PubMed: 24504015]

Tourville JA, Guenther FH, 2011. The DIVA model: a neural theory of speech acquisition and production. Lang. Cogn. Process 26 (7), 952–981. [PubMed: 23667281]

Tripoliti E, Zrinzo L, Martinez-Torres I, Tisch S, Frost E, Borrell E, Limousin P, 2008. Effects of contact location and voltage amplitude on speech and movement in bilateral subthalamic nucleus deep brain stimulation. Mov. Disord 23 (16), 2377–2383. [PubMed: 18785648]

Tripoliti E, Strong L, Hickey F, Foltynie T, Zrinzo L, Candelario J, Limousin P, 2011. Treatment of dysarthria following subthalamic nucleus deep brain stimulation for Parkinson’s disease. Mov. Disord 26 (13), 2434–2436. [PubMed: 21953693]

Tripoliti E, Limousin P, Foltynie T, Candelario J, Aviles-Olmos I, Hariz MI, Zrinzo L, 2014. Predictive factors of speech intelligibility following subthalamic nucleus stimulation in consecutive patients with Parkinson’s disease. Mov. Disord 29 (4), 532–538. [PubMed: 24532491]

Tsuboi T, Watanabe H, Tanaka Y, Ohdake R, Hattori M, Kawabata K, Sobue G, 2017. Early detection of speech and voice disorders in Parkinson’s disease patients treated with subthalamic nucleus deep brain stimulation: a 1-year follow-up study. J. Neural Transm. (Vienna) 124 (12), 1547–1556. [PubMed: 29098450]

Tykalová T, Rusz J, Cmejla R, Klempíř J, Růžičková H, Roth J, Růžička E, 2015. Effect of dopaminergic medication on speech dysfluency in Parkinson’s disease: a longitudinal study. J. Neural Transm 122 (8), 1135–1142. [PubMed: 25583417]

Wertheimer J, Gottuso AY, Nuno M, Walton C, Duboille A, Tuchman M, Ramig L, 2014. The impact of STN deep brain stimulation on speech in individuals with Parkinson’s disease: the patient’s perspective. Parkinsonism Relat. Disord 20 (10), 1065–1070. [PubMed: 25048615]

Whitworth A, Lesser R, McKeith I, 1999. Profiling conversation in Parkinson’s disease with cognitive impairment. Aphasiology 13 (4–5), 407–425.

Wolff L, Benge J, 2019. Everyday language difficulties in Parkinson’s disease: caregiver description and relationship with cognition, activities of daily living, and motor disability. Am. J. Speech Lang. Pathol 28 (1), 165–173. [PubMed: 31072160]

Wu AD, Fregni F, Simon DK, Deblieck C, Pascual-Leone A, 2008. Noninvasive brain stimulation for Parkinson’s disease and dystonia. Neurotherapeutics 5 (2), 345–361. [PubMed: 18394576]

Xu H, Bao Z, Liang D, Li M, Wei M, Ge X, Li J, 2020. Speech and language therapy for voice problems in Parkinson’s disease: a meta-analysis. J. Neuropsychiatry Clin. Neurosci 32 (4), 344–351. [PubMed: 32374650]

Zheng ZZ, Munhall KG, Johnsrude IS, 2010. Functional overlap between regions involved in speech perception and in monitoring one’s own voice during speech production. J. Cogn. Neurosci 22 (8), 1770–1781. [PubMed: 19642886]

Zollinger SA, Brumm H, 2011. The Lombard effect. Curr. Biol 21 (16), R614–R615. [PubMed: 21854996]