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Research Article

Speech Rate Mediated Vowel and Stop Voicing Distinctiveness in Parkinson's Disease

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Purpose: The purpose of this study was to quantify changes in acoustic distinctiveness in two groups of talkers with Parkinson's disease as they modify across a wide range of speaking rates.
Method: People with Parkinson's disease with and without deep brain stimulation and older healthy controls read 24 carrier phrases at different speech rates. Target nonsense words in the carrier phrases were designed to elicit stop consonants and corner vowels. Participants spoke at seven self-selected speech rates from very slow to very fast, elicited via magnitude production. Speech rate was measured in absolute words per minute and as a proportion of each talker's habitual rate. Measures of segmental distinctiveness included a temporal consonant measure, namely, voice onset time, and a spectral vowel measure, namely, vowel articulation index.
Results: All talkers successfully modified their rate of speech from slow to fast. Talkers with Parkinson's disease and deep brain stimulation demonstrated greater baseline speech impairment and produced smaller proportional changes at the fast end of the continuum. Increasingly slower speaking rates were associated with increased temporal contrasts (voice onset time) but not spectral contrasts (vowel articulation). Faster speech was associated with decreased contrasts in both domains. Talkers with deep brain stimulation demonstrated more aberrant productions across all speaking rates.
Conclusions: Findings suggest that temporal and spectral segmental distinctiveness are asymmetrically affected by speaking rate modifications in Parkinson's disease. Talkers with deep brain stimulation warrant further investigation with regard to speech changes they make as they adjust their speaking rate.

Imprecise articulation is a prominent perceptual feature of dysarthria associated with Parkinson's disease (PD). Consonant and vowel imprecision is hypothesized to be related to *articulatory undershoot* in this dysarthria (Ackermann & Ziegler, 1991; Logemann & Fisher, 1981; M. J. McAuliffe et al., 2006a, 2006b; Weismer, 1984b), that is, articulatory positions that do not achieve the target placement due to restricted movement. One result of this undershoot may be less canonical articulatory postures during speech that result in greater overlap of segmental categories. This may affect the spectral integrity of the signal or the timing of finely coordinated speech movements.

Two documented examples of acoustic–phonetic imprecision in PD are vowel contrasts (Lam & Tjaden, 2016; Sapir et al., 2010; Skodda et al., 2011; Tjaden & Wilding, 2004; Whitfield, 2019; Whitfield & Goberman, 2014) and stop voicing distinctions (Whitfield et al., 2018). Given that speech features of dysarthria associated with PD include other dimensions of speech in addition to articulatory imprecision, such as impairments in vocal loudness, prosody, and rate of speech, behavioral speech interventions that are designed to be global, rather than targeted, are often recommended (Yorkston et al., 2007). Reducing rate of speech is one form of a global treatment strategy that is thought to indirectly facilitate greater articulatory precision by allowing more time for the speaker to reach canonical articulatory positions. To date, however, little is known about the impact of these treatments on acoustic–phonetic contrasts. Furthermore, there is no present evidence on the efficacy of rate reduction for talkers with PD and deep brain stimulation of the subthalamic nucleus (STN-DBS).

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Speech symptoms in PD, including articulatory features, are often further affected for those who undergo surgical treatment for PD. STN-DBS, an increasingly common adjunctive surgical treatment for the primary motor symptoms of PD, has been associated with more variable and often worsening speech symptoms (Aldridge et al., 2016), among them more severe imprecise articulation (Tripoliti et al., 2014; Tsuboi et al., 2017). Worse speech symptom severity in these cases has been associated with unwanted spread of electrical current to adjacent neural fiber tracts involved in speech motor control, such as the corticobulbar and cerebellothalamic fiber tracts (Fenoy et al., 2016; Tommasi et al., 2008). Factors such as suboptimal electrode positioning or electrical settings have been posited as potential factors that can lead to greater speech detriment (Knowles et al., 2018; Tripoliti et al., 2008, 2014). The precise mechanistic role of the STN in speech and the subsequent impact of DBS are not presently well understood.

Acoustic Distinctiveness in PD

Vowel Production

Relative to healthy adults, people with PD produce more centralized vowel formants, resulting in overall smaller acoustic vowel spaces (Lam & Tjaden, 2016; Lansford & Liss, 2014; McRae et al., 2002; Rusz et al., 2013; Skodda et al., 2011, 2012; Tjaden, Lam, & Wilding, 2013; Watson & Munson, 2008; Whitfield & Goberman, 2014). STN-DBS has also been linked to further impairment in vowel production, indexed by further reductions in vowel working space (Martel-Sauvageau et al., 2014, 2015; Sidtis et al., 2016; Skodda et al., 2014; Tanaka et al., 2016). Literature on the relationship between speech rate and acoustic distinctiveness largely supports the finding that slower speech is associated with increases in the acoustic distinctiveness of vowels, as evidenced by acoustic vowel expansion, in both healthy talkers (Fletcher et al., 2015; Fourakis, 1991; Tjaden & Wilding, 2004; Tsao & Iqbal, 2006; Turner et al., 1995; Weismer et al., 2000) and talkers with PD (Buccheri et al., 2014; McRae et al., 2002; Tjaden et al., 2005; Tjaden & Wilding, 2004).

In addition to reduced overall vowel space, individuals with hypokinetic dysarthria have also demonstrated reduced vowel contrasts in front and back vowels, as demonstrated by the ratio of the second formant (F2) in /i/ and /u/ (Rusz et al., 2013; Sapir et al., 2007). Reduced F2 transitions, which reflect the speed and extent of tongue movement, have also been found (Feenaughty et al., 2014; H. Kim et al., 2011; Y. Kim et al., 2009; Walsh & Smith, 2011; Yunusova et al., 2005), though this finding is not uniform across all individuals or test words (Y. Kim et al., 2009; Lam & Tjaden, 2016). Preceding consonant place of articulation can also influence the following vowel quality. For example, vowels following alveolar consonants tend to be more centralized in talkers with PD (Martel-Sauvageau et al., 2014), which may be related to more limited jaw opening in these contexts (Iskarous et al., 2010; Kawahara et al., 2014; Sussman et al., 1991).

In an effort to minimize potential confound effects of interspeaker variability and vocal tract sizes related to age and gender in acoustic vowel space (Yang, 1996), other indices have been developed to better capture vowel centralization in dysarthria. These include the vowel articulation index (VAI; Sapir et al., 2011) and its inverse, the formant centralization ratio (Karlsson & van Doorn, 2012; Martel-Sauvageau et al., 2014, 2015; Roy et al., 2009; Rusz et al., 2013; Sapir et al., 2010; Skodda et al., 2011), both of which are ratio measures that differentiate vowel formants that are expected to increase or decrease with centralization. The use of these metrics provides further evidence of increased vowel centralization in hypokinetic dysarthria compared to in healthy aging.

Stop Consonant Production

Voice onset time (VOT) is a temporal measure of stop consonant production, which reflects the timing between the onset of a stop consonant release and the onset of voicing of the following vowel. VOT is considered to be reflective of laryngeal and supralaryngeal coordination (Weismer, 2006). It is the primary acoustic and perceptual cue for stop consonant voicing, with voiceless stops characterized by longer VOT compared to voiced stops. VOT also systematically differs across distinct places of articulation, with more posterior placements associated with longer VOT. This pattern has been observed for both healthy and disordered talkers (Abramson & Whalen, 2017; Cho et al., 2019; Lisker & Abramson, 1964).

Reports of abnormalities in VOT in PD are inconsistent (Bunton & Weismer, 2002; Cushnie-Sparrow et al., 2016; Fischer & Goberman, 2010; Flint et al., 1992; Forrest et al., 1989; Lieberman et al., 1992; Miller et al., 1986; Weismer, 1984b). Forrest et al. (1989) found that voiced bilabial stops had longer average VOT for speakers with PD, making them more like voiceless stops, but did not find differences in voiceless bilabial VOT. On the other hand, other authors have found shorter *voiceless* VOT in talkers with PD (Flint et al., 1992; Weismer, 1984a). This has been attributed to stiffness in laryngeal musculature, causing the vocal folds to have reduced abduction and preventing them from staying open as long as would be expected for typical voiceless VOT production (Weismer, 1984a). Talkers with PD may exhibit more *overlap* between voiced and voiceless VOT, calculated based on the distributions of both voiced and voiceless stops (Hochstadt et al., 2006; Lieberman et al., 1992; Whitfield et al., 2018). Other studies have reported no differences in VOT in talkers with PD compared to healthy age matched controls (Bunton & Weismer, 2002; Cushnie-Sparrow et al., 2016), even when speech rate was controlled for (Fischer & Goberman, 2010; Ravizza, 2003). As is the case for vowel space, further detriment to stop production has also been reported for people with PD following DBS surgery (Chenausky et al., 2011). Specifically, talkers with PD and STN-DBS demonstrate longer and more variable VOT, characterized by leaky,

incomplete stop closure (Chenausky et al., 2011; Dromey & Bjarnason, 2011; Eklund et al., 2014; Karlsson et al., 2014).

Rate Modification and Acoustic Distinctiveness

In general, findings suggest that slower rates of speech are associated with greater acoustic distinctiveness for talkers with hypokinetic dysarthria (Adams, 1994; McRae et al., 2002; Tjaden et al., 2005; Tjaden & Wilding, 2004). Slower speech is a common treatment target in PD and may allow speakers with dysarthria more time to reach articulatory positions needed to produce more canonical productions and, ultimately, more intelligible speech. This pattern of increased acoustic distinctiveness in slow speech tends to be true in healthy talkers as well, both across variations at habitually slower rates of speech as well as when speech rate is intentionally modified (Adams, 1994; Bradlow et al., 1996; Miller et al., 1986; Tjaden & Weismer, 1998; Tsao et al., 2006). Research from healthy talkers also suggests this relationship persists in a predictable fashion when talkers produce faster rates of speech. That is, they produce less acoustic contrast when speaking more quickly. Since faster speech is unlikely to be a treatment target in speech intervention for people with PD, this relationship has received little attention. However, recent reports suggest that people with PD do not always show predictable declines in intelligibility at faster rates of speech (Knowles et al., 2021; Kuo et al., 2014). This finding suggests that further investigations in the acoustic elements of speech production at faster rates of speech are warranted in order to better understand the mechanisms behind rate modification in PD in general.

In general, vowel space has been found to vary with rate of speech in dysarthria, with larger vowel spaces produced at slower rates (Buccheri et al., 2014; McRae et al., 2002; Tjaden et al., 2005; Tjaden & Wilding, 2004). However, despite being readily observed, this pattern is often not robustly supported. For example, Tjaden and Wilding (2004) found an overall expansion in vowel space when a group of talkers with PD and dysarthria were cued to speak more slowly, but this trend did not reach thresholds of statistical significance. There is evidence that this relationship may be stronger in dysarthria secondary to other etiologies such as multiple sclerosis (Tjaden & Wilding, 2004), amyotrophic lateral sclerosis (Turner et al., 1995; Weismer et al., 2000), or cerebral palsy (Hustad & Lee, 2008). It could be the case that individuals with PD need to reach an even slower rate than has previously been tested in order to achieve sufficient articulatory positions that lead to measurably increased vowel space.

The effect of rate of speech on VOT has not been explicitly studied in previous reports of PD. Evidence from neurologically healthy talkers, though, suggests that there is a predictable, robust, inverse relationship between VOT and speech rate (Kessinger & Blumstein, 1997, 1998; Miller et al., 1997; Summerfield, 1981; Volaitis & Miller, 1992), with asymmetric lengthening for voiceless compared to voiced VOT at slower rates. The consequence of this asymmetry is increased voicing contrasts at slower rates, as evidenced by greater change in voiceless stop production, and reduced contrastiveness at faster rates (e.g., voiced and voiceless VOT become more or less similar, respective to slow and fast rates).

Previous research suggests differences in acoustic–phonetic distinctiveness are a function of speaker sex and/or gender as well. While some measures, such as the VAI, are designed to account for anatomical and physiological differences across speakers, such as differences in male and female vocal tract lengths, differences may also be linked to differences in behavior or in the progression of parkinsonian symptoms. Anatomical and physiological differences may affect both source and filter characteristics of speech. For example, male talkers have a lower phonation threshold due to greater vocal fold mass and, thus, may phonate earlier than female talkers, resulting in potential shorter VOT (Koenig, 2000). Previous evidence also suggests that PD differentially impacts the progression of dysarthria in men and women, which has been suggested to, at least in part, be due to differences in anatomical laryngeal sizes (Hertrich & Ackermann, 1995). Sociolinguistic variables may also account for differences. Compared to female talkers, male talkers have been found to produce greater amounts of phonological reduction or hypoarticulation (e.g., Byrd, 1994) and speak at faster rates (Swartz, 1992, but see Bradlow et al., 1996), which may be related to lower overall intelligibility, even in healthy talkers (Bradlow et al., 1996). These differences may result in male talkers presenting with smaller vowel spaces (Bradlow et al., 1996), shorter overall VOT durations (Swartz, 1992; Whiteside & Irving, 1997), or smaller VOT differences across voicing categories (Scharf & Masur, 2012).

Summary and Purpose

In a review of the literature assessing rate, loudness, and prosody-based interventions for motor speech disorders, Yorkston et al. (2007) identified a need for a better understanding of speaker candidacy for rate-reduction interventions, as well as better descriptions of how optimal rates are selected. A more complete understanding of the precise speech outcomes resulting from a wide range of rate adjustments across speech tasks and speaker profiles is needed in order to (a) implement such findings in treatment and (b) better characterize the consequences of rate modifications on speech motor control. The purpose of this study was to examine the effects of rate modification along a broad range of speaking rates to quantify changes in acoustic distinctiveness in talkers with PD. Two metrics of acoustic distinctiveness were considered for this study: vowel distinctiveness, quantified with a composite spectral measure of vowel centralization, and stop voicing distinctiveness, quantified by VOT. A secondary aim of this study was to identify differences in talkers with PD with and without STN-DBS. Given previous reports of worsening severity with regard to articulatory precision and

control in people with PD and STN-DBS, it is reasonable to anticipate differences across these two groups. People with PD and STN-DBS have historically been excluded from studies of rate adjustment, leaving the nature of these differences an open question.

In this study, we address the following research questions:

1. How do talkers with PD (with and without STN-DBS) differ from healthy, older talkers in the magnitude of speech rate adjustments from very slow to very fast, compared to their habitual rates of speech?
2. What is the effect of speech rate modifications for each of these groups at both slower and faster rates on articulatory–acoustic distinctiveness along
   a. a spectral domain (vowel centralization) and
   b. a temporal domain (stop voicing distinctiveness)?

Method

This study was approved by the Health Sciences Research Ethics Board at Western University and the Lawson Health Research Institute.

Participants

Three participant groups were included in the final study for a grand total of 51 speakers: (a) older healthy control participants (OC; $n = 17$, 56–82 years of age, 11 men and six women), (b) people with PD and dysarthria who were receiving standard pharmaceutical interventions (PD-Med; $n = 22$, 18 men and four women), and (c) people with PD who had undergone bilateral STN-DBS surgery (PD-DBS; $n = 12$, 10 men and two women).[1] Participant information for the PD groups is reported in Tables 1 and 2. These participants have been described elsewhere (Knowles et al., 2021). OC participants were recruited from the community via flyers and word of mouth. All PD participants were recruited through the Movement Disorders Centre at University Hospital in London, Ontario (directed by neurologist M. J.). All participants were native or near-native speakers of North American English[2] and had self-reported adequate vision or corrected vision for reading print. Hearing and cognitive status were not exclusionary criteria for this study, though all participants underwent a 40 dB SPL hearing screening at 0.5, 1, 2, and 4 kHz unless they wore hearing aids and all completed the Montreal Cognitive Assessment (MoCA). Two OC, five PD-Med, and three PD-DBS participants reported a hearing aid prescription, though five of these PD participants reported that they did not wear them on a regular basis (PD-Med 08, PD-Med 12, PD-Med 14, PD-Med 19, and PD-DBS 02). MoCA scores for the PD groups are in Tables 1 and 2. Within the OC group, the majority received a score of 26 or higher (the suggested cutoff for mild cognitive impairment; Dalrymple-Alford et al., 2010). Two OC participants received a score of 25 (OC 03 and OC 16), and one received a score of 21 (OC 06), which is representative of the overall prevalence of mild cognitive impairment in the general aging population (Petersen et al., 2010). Two OC, four PD-Med, and two PD-DBS participants reported wearing dentures.

Participants in the two PD cohorts were deemed eligible if they (a) had received a diagnosis of PD at least 1 year prior by a neurologist with expertise in movement disorders (M. J.) using current diagnostic criteria (Postuma et al., 2015) and (b) were stabilized on anti parkinsonian medication and/or via surgical STN-DBS settings. PD-Med participants were also recruited on the basis of evidence of at least mild dysarthria, as identified by a neurologist on the Unified Parkinson's Disease Rating Scale present in their patient chart history. Because of a smaller number of potential PD-DBS participants (compared to the PD-Med group), PD-DBS participants were not recruited on the basis of speech symptoms and represented a convenience sample of STN-DBS patients. Deviant perceptual characteristics listed in Tables 1 and 2 below were determined by consensus by the first two authors (T. K. and S. G. A.).

Procedure

Audio Recordings

Recordings were made in an audiometric booth (IAC Acoustics) using a 2017 15-in. Dell laptop computer (Inspiron 15). Participants wore a headset microphone (AKG C520), positioned 6 cm from the mouth and connected to the laptop via a preamplifier and digitizing unit (M-Audio MobilePre) attached via a USB port. The headset was positioned so as to allow hearing aids and glasses to remain in place. Stimulus presentation and audio capture during the experiment were done through a customized MATLAB script (MathWorks, Inc., 2018) adapted from the McGill ProsodyLab template (Wagner, 2018) by the first author. Audio signals were digitized at 44.1 kHz and 16 bits. Practice periods (described below) were also recorded but were excluded in the final analyses. These were recorded on a 2014 MacBook Air in Praat (Boersma & Weenink, 2011) using a lapel microphone connected to a Focusrite amplifier.

Experimental stimuli were presented via text on the monitor. Participants were encouraged to read the text silently to themselves. The text then turned red, indicating that they could begin speaking aloud.

[1] PD-DBS participants were also taking titrated doses of anti parkinsonian medication.

[2] Two participants (PD-Med 10 and PD-Med 20) were native Dutch speakers and had moved to Canada in early childhood; they reported speaking Canadian English as their dominant language since childhood. One participant (PD-Med 01) was born in Canada but reported speaking Spanish at home until preschool. One participant (PD-Med 16) grew up speaking Trinidadian English and reported moving to Canada in his 20s. These speakers were not excluded because (a) their native languages include two-way stop contrast systems and contain similar vowel quality to Standard Canadian English, (b) the task elicited nonsense words unfamiliar to all talkers, and (c) the focal point of the analyses was acoustic distinctiveness; baseline dialectal differences in acoustic–phonetic productions were not of concern because the primary analyses focused on acoustic *distinctiveness*.

Table 1. Demographic data for the PD-Med group.

ID	Sex	Age	MoCA	Years post diagnosis	PD medications	LEDD (mg)	Deviant perceptual characteristics
01	m	60	29	12	Levodopa	400	Monopitch, mild hypophonia, short rushes
02	m	65	18	14	Apo-Levocarb	1,200	Monopitch, moderate hypophonia, imprecise consonants
03	m	65	23	12	Levodopa	532	Repeated phonemes, imprecise consonants, short rushes
04	m	66	28	35	Levodopa	NA	Harsh voice, monopitch, short rushes, imprecise consonants
05	m	73	27	7	Levodopa	NA	Hypophonia, short phrases, short rushes
06	f	67	30	10	Levodopa, Mirapex	700	Short rushes, fast rate, breathy voice
07	m	72	29	9	Levodopa, Amantadine	NA	Imprecise consonants, breathy voice, increased pitch
08	m	85	24	4	Levodopa	400	Harsh voice, imprecise consonants, short rushes
09	m	56	28	25	Levodopa, Amantadine	NA	Strained–strangled voice, imprecise consonants, short rushes of speech, phoneme repetitions
10	m	71	25	5	Levodopa	800	Imprecise consonants, distorted vowels, high pitch, hyponasality
11	m	68	25	8.5	Pramipexole, Levodopa	300	Strained voice, hoarse voice, hypophonia
12	m	72	24	15	Levodopa, Pramipexole	1,300	Hypernasality, monopitch, low pitch
13	m	62	26	3	Apo-Levocarb	800	Hoarse voice, imprecise consonants, short rushes
14	m	90	24	10	NA	NA	Hypernasality, high pitch, imprecise consonants, harsh voice
15	m	70	28	2	Levodopa	900	Moderate hypophonia, short rushes, imprecise consonants, high pitch
16	m	73	23	10	Levodopa	800	Moderate hypophonia, hoarse voice, imprecise consonants, monopitch
17	f	71	26	5	Levodopa	NA	Hoarse voice
18	m	64	28	6	Levodopa	600	Imprecise consonants, short rushes, monopitch, moderate hypophonia
19	f	68	28	18	Duodopa	NA	Mild hypophonia, breathy voice, imprecise consonants, short rushes
20	f	73	25	30	Levodopa, Mirapex, Amantadine, Apo-Gabapentin	1,200	Imprecise consonants, short rushes, audible inhalations
21	m	64	28	8	Mirapex	450	Mild hypophonia, monopitch, imprecise consonants
22	m	71	25	10	Levodopa, Pramipexole	900	Imprecise consonants, harsh voice

Note. One PD-Med participant (PD14) was unsure of their current medication list, which is listed here as NA. Deviant perceptual characteristics for the PD-Med and PD-DBS groups correspond to features noted during the habitual monologue speech samples. PD-Med = people with Parkinson's disease who were receiving standard pharmaceutical interventions; MoCA = Montreal Cognitive Assessment; PD = Parkinson's disease; LEDD = Levodopa equivalent daily dose; m = male; f = female; PD-DBS = people with Parkinson's disease who had undergone bilateral deep brain stimulation of the subthalamic nucleus surgery.

Speech Stimuli

Participants read a series of carrier phrases containing nonsense words, "Please say ___ again." All target words were disyllabic of the form /əCVd/, where C was always a stop consonant (/p/, /t/, /k/, /b/, /d/, or /g/) and V was always a corner vowel (/i/, /æ/, /ɑ/, or /u/), resulting in 24 phrases of interest in total. The preceding /ə/ context was chosen to provide a neutral intervocalic position that would preserve contextual cues to consonant identity in running speech (Cheesman & Jamieson, 1996) while allowing the words to be presented as true nonsense words. The experimental speech stimuli presented here were part of a larger battery of speech tasks.[3] Breaks were offered as needed.

Participants were presented with the word list before the experiment began and read the words aloud to ensure they pronounced them as intended and so that the first experimental condition would not be the first time they encountered the novel words (as in Vogel et al., 2017). During the experiment, stimuli were presented once per condition and were interspersed by other speech stimuli of interest (including other target words, sentences, and spontaneous speech prompts). In order to minimize the likelihood of mispronunciations due to orthographic ambiguity, consistent spelling conventions were used in the nonsense words.

Speech Rate Elicitation

Seven speech rate conditions were elicited: habitual rate, three slower rates, and three faster rates. Throughout the text, these rates are referred to as H1 (habitual), S2, S3, and S4 (slower), and F2, F3, F4 (faster). All speech tasks were elicited for each condition. Habitual speech was elicited first, followed by the modified rates that were presented in blocks (faster, slower) using magnitude production. Magnitude production techniques are considered to elicit more natural speaking rate adjustments (Adams et al., 1993; Turner et al., 1995) and have been used in several studies of dysarthric speakers (e.g., Clark et al., 2014; Hall, 2013; Kuo et al., 2014; McRae et al., 2002; Tjaden, Richards, et al., 2013; Tjaden & Wilding, 2004; Turner et al., 1995). Within each block, progressively faster and slower rates were elicited

[3]The full list of nonsense words contained 52 words including other manners of articulation. Additional speech tasks not reported here included sentence reading, picture description, and a monologue task.

Table 2. Demographic data for the PD-DBS group.

ID	Sex	Age	MoCA	Years post diagnosis	Years since DBS surgery	PD medications	LEDD (mg)	Deviant perceptual characteristics
01	m	60	24	12	2	Levodopa, Amantadine	300	Hoarse, breathy voice; monopitch; imprecise consonants; prolonged intervals
02	f	71	16	25	9	Levodopa	50	Hoarse, breathy voice; imprecise consonants; short rushes; fast rate
03	m	63	24	18	9	Amantadine, Levodopa	430	Mild hypophonia, imprecise consonants, short rushes, high pitch
04	m	73	20	12	4	Levodopa	NA	Strained–strangled voice, imprecise consonants, prolonged phonemes, slow rate
05	m	56	27	16	6	Levodopa	NA	Harsh voice, imprecise consonants
06	m	59	16	13	5	Levodopa, Amantadine, Sinemet	NA	Mild hypophonia, imprecise consonants, high pitch
07	f	69	25	16	3	Levodopa	550	Moderate hypophonia, strained–strangled voice, audible inspirations, voice breaks
08	m	66	28	14	6	Levodopa	NA	Mild hypophonia, strained–strangled voice, pitch breaks, imprecise consonants
09	m	55	28	8	1	Levodopa	500	Imprecise consonants, hoarse voice, short rushes, fast rate
10	m	66	23	4	3	Levodopa	150	High pitch, hypernasality, imprecise consonants, short rushes
11	m	60	25	12	4	Levodopa, Ropinirole	NA	Harsh, breathy voice; imprecise consonants; audible inspirations
12	m	66	28	14	7	Levodopa	500	Mild hypophonia, imprecise consonants, short rushes, fast rate
13	m	72	22	15	4	Levodopa	600	Imprecise consonants, breathy voice

Note. Deviant perceptual characteristics for the PD-Med and PD-DBS groups correspond to features noted during the habitual monologue speech samples. PD-DBS = people with Parkinson's disease who had undergone bilateral deep brain stimulation of the subthalamic nucleus surgery; MoCA = Montreal Cognitive Assessment; DBS = deep brain stimulation; PD = Parkinson's disease; LEDD = Levodopa equivalent daily dose; m = male; f = female; NA = not applicable; PD-Med = people with Parkinson's disease who were receiving standard pharmaceutical interventions.

in order of magnitude (e.g., progressively faster or slower). The order of the rate blocks was counterbalanced across participants, and stimuli were randomized within each condition for all participants.

For each modified rate condition, participants were instructed "For this next part, please speak at a rate that feels [2x/3x/4x] [slower/faster] than your normal speaking rate." For slower blocks, they were encouraged to slow their speech down by stretching out their speech rather than pausing in between words (McHenry, 2003; Tjaden et al., 2014). For faster blocks, they were encouraged to increase their rate while still saying all the words. In addition to verbal instructions, participants had constant access to a visual prompt comprised of a curved, numbered line (designed to look like a speedometer) and a movable arrow pointing to the target rate.

Prior to beginning any of the modified rate conditions, participants underwent a brief practice period in which they read a practice sentence aloud as many times as needed until they felt they achieved the rate they were aiming for. While the precise rate was irrelevant, the investigator confirmed (via inspection of utterance duration in Praat) that they were indeed faster/slower than the previous rate. The investigator extracted the most representative practice utterance and played it back to the participant approximately every 10 trials in the experiment to serve as a reference in order to help them maintain their target rate.

Acoustic Measures

Acoustic analysis was semi-automated and then manually checked. Each utterance was first manually segmented at the utterance boundaries and then force-aligned using the Montreal Forced Aligner (M. McAuliffe et al., 2017). Speech rate was calculated in two ways: (a) actual speech rate measured in words per minute[4] (WPM) for each utterance and (b) proportional rate of speech, which was calculated as follows. The average habitual rate of speech (from the habitual speech condition) was measured for each speaker. Proportional speech rate treated each speaker's mean as a value of 1, and actual speech rate was converted into a proportion of this habitual rate. For example, for a speaker with a mean habitual rate of 200 WPM, utterances with actual speech rates of 100 WPM, 200 WPM, and 300 WPM produced by this speaker would be converted into proportional rates of 0.5, 1, and 1.5, respectively. VOT was first automatically detected using AutoVOT (Keshet et al., 2014), and vowel formants were first measured using a custom Praat script; all measures (segment boundaries and first formant [F1] and F2) were then manually corrected using criteria defined below and facilitated by a custom Praat script.

[4]This is distinct from articulatory rate (e.g., Waito et al., 2021), as small pauses were not excluded from the rate measure.

Segmental Boundaries

In most cases, the onset of VOT was identifiable by a clear burst. In many cases, however, the onset was more ambiguous, as is often reported in studies of VOT in clinical speech (Auzou et al., 2000; Fischer & Goberman, 2010; Karlsson, Unger, Wahlgren, & van Doorn, 2011). Informed by previous studies and patterns observed in these data, the following criteria were established.

- Multiple bursts were marked at the onset of the initial burst (Fischer & Goberman, 2010; Parveen & Goberman, 2014; Wang et al., 2004).
- Stops with clear frication preceding the burst were marked at the onset of frication present in the signal that corresponded to "the transient with the strongest amplitude in the portion of the signal approximate to where an audible release was perceived" (Karlsson et al., 2014, p. 1181). These cases were also documented for later analysis, which will not be discussed here.
- Stops with no obvious frication and no obvious burst could not reliably be marked as containing VOT.

A small subset of the stops could not be reliably marked as having a clear VOT onset. These were divided into three cases:

- No VOT (removed from the data set): 311 observations (3.7% of the data) had no obvious frication or burst; that is, were unreleased (Özsancak et al., 2001).
- Completely omitted or glided (removed from the data set): In a very small number of cases (n = 37; < 1%), there was no evident closure or release at all; that is, the stop was unidentifiable. These cases were documented and removed from the analysis.
- Complete spirantization (retained in the data set): 172 cases (2.1%) had no closure but did have clear evidence of frication onset; that is, these stops were fully spirantized. In these cases, VOT onset was considered as the onset of the consonant and the offset of the preceding vowel.

It should be noted that the criteria above intentionally did not take into account voicing during closure and therefore should be considered a measure of positive (also known as lag) VOT (Chodroff & Wilson, 2017). There were very few observed cases of prevoicing that would contribute to negative VOT. More frequently seen was either no voicing, partial voicing into closure, or complete voicing through closure (Davidson, 2016). The above criteria also did not consider distinctions between other aspects of the stop release (i.e., transient duration, etc.).

VOT offset/vowel onset. VOT offset was determined as the onset of periodicity in the following vowel, marked on the part of the waveform crossing the x-axis going up. Three main causes of ambiguity were noted: quasiperiodicity in VOT, devoiced or breathy vowels, and voicing throughout closure. As such, the following criteria were followed:

- In the presence of quasiperiodicity, the onset of voicing was marked where there was an accompanying rise in amplitude in the signal. Praat's pulse detection was also used to supplement particularly ambiguous decisions.
- In the presence of breathy or devoiced vowels, the offset of VOT was marked as an obvious visual change in the waveform and spectrogram indicating quasiperiodicity and formant-like spectral energy.

Vowel offset. When possible, vowel offset was determined as the offset of vowel periodicity and the onset of closure of the word-final /d/. In many cases, /d/ was unreleased or omitted, in which case vowel offset was marked using a combination of (a) visual inspection for decreases in amplitude and waveform complexity, (b) changes in formant structure corresponding to a vocalic transition from the vowel of interest to the following schwa in "again," and (c) audio perceptual judgments.

Vowel formants. The first two vowel formants (F1 and F2) were measured from a 30-ms section occurring at the midpoint of the vowel, using the boundaries established in the boundary correction phase described above. Formant values were manually checked using the same custom Praat script described above. Formant settings in Praat were uniformally set to begin, then set for each individual speaker on a case-by-case basis. Whenever possible, the same formant settings were kept consistent for a given speaker. Ambiguous cases were documented. Vowel formants were then entered into the following formula in order to compute the quadrilateral vowel articulation index (QVAI; Knowles et al., 2018; Roy et al., 2009; Sapir et al., 2011):

$$\mathrm{QVAI} = \frac{\mathrm{F2i} + \mathrm{F2æ} + \mathrm{F1æ} + \mathrm{F1a}}{\mathrm{F1i} + \mathrm{F1u} + \mathrm{F2u} + \mathrm{F2a}}. \quad (1)$$

QVAI was calculated using the average F1 and F2 for vowels following each of the six stop consonants for each participant in each of the seven rate conditions. This resulted in approximately 42 tokens per participant. In the above formula, the numerator includes formant values that are expected to *decrease with centralization*, and the denominator includes formant values that are expected to *increase with centralization*. A larger QVAI thus reflects *less centralization* and greater expansion.

All boundaries and measurements were annotated by the first author (T. K.). A trained research assistant additionally annotated VOT for a random subset of approximately 1,000 stops across all participant groups and speech rates. Interrater reliability was calculated with the intraclass correlation coefficient (ICC; Koo & Li, 2016) using average consistency in separate two-way random models for VOT onset, offset, and overall duration (ICC 2, k). Average interrater reliability was considered excellent for all measurements (onsets: M = 0.999, CI = [0.9994, 0.9995]; offsets: M = 0.999, CI = [0.9993, 0.9994]; duration: 0.909, CI = [0.8995, 0.9185]). The average absolute VOT durational difference across raters was 8.3 ms, which is consistent with

previous reports of VOT measurement in PD (Fischer & Goberman, 2010).

Statistical Analyses

Four outcome measures were of interest: actual speech rate and proportional speech rate (Research Question 1), QVAI (Research Question 2a), and VOT (Research Question 2b). All outcome variables were modeled using linear mixed-effects regression with the *lme4* package (Bates et al., 2015) in R (R Core Team, 2020).

Speech Rate: Absolute and Proportional Adjustments

To answer Research Question 1 (what are the group differences in the magnitude of rate adjustments), two linear mixed models were built: one modeling actual rate of speech (in WPM) as the dependent variable and one modeling proportional rate of speech as defined above. The first model sought to quantify the degree of change in speech rates for talkers on a standard scale of WPM, while the second model focused on relative rate changes for each individual compared to their baseline speech. This dual-rate approach was chosen in order to account for expected variation in speaking rates across the groups while quantifying the degree of change. In subsequent models where rate of speech is treated as an independent variable, actual rate is used in habitual speech, while proportional rate is used to look at faster and slower rates.

Both models included fixed effects of group, speech rate condition, and their interaction, as well as by-participant random intercepts and by-participant random slopes for each rate contrast.[5] Speaker group was coded using reverse Helmert contrasts with three levels. Helmert contrasts allow the mean of each level to be compared to the overall mean of the subsequent levels. The contrast scheme for group may be interpreted in the following way: (a) healthy older controls versus PD groups (OC vs. PD-Med and PD-DBS combined) and (b) PD with and without DBS (PD-Med vs. PD-DBS). For Research Question 1, rate condition was contrasted using treatment contrasts, such that each rate condition was compared to the habitual rate. All subsequent models used proportional rate in WPM as the predictor and did not take into account the rate conditions.

QVAI

To answer Research Question 2a, three separate linear mixed-effects models were constructed to model QVAI as a function of rate: (a) habitual rates, (b) slower rates, and (c) faster rates. The modified rate models are of primary interest, and separate models, rather than a single unified model, were chosen in order to precisely characterize distinct patterns at relatively slower and faster rates. Habitual speech was included in order to describe baseline differences across the groups.

For the modified rate models, QVAI was modeled as a function of group, proportional rate of speech, and their interaction. Each model contained utterances elicited in the slow or fast conditions. Fixed effects of preceding consonant articulation and speaker gender were also included. Speaker group was coded using reverse Helmert contrasts as above. Proportional speech rate was treated as a continuous variable.[6] Speaker gender was sum-coded as a two-level category variable (female vs. male). Preceding consonant place of articulation was coded using reverse Helmert contrasts as a three-level variable, with contrasts interpreted as (a) bilabial versus alveolar and velar and (b) alveolar versus velar. The random effects structure included by-participant random intercepts and random slopes for consonant place of articulation.[7] The habitual rate model was constructed in the same way, but actual speech rate rather than proportional rate was included in order to look at baseline differences in vowel articulation as a factor of true speaking rate.

VOT

To address Research Question 2b, three VOT models (slow, fast, and habitual) were constructed in the same way as the QVAI models, with notable differences in the fixed effects structure. VOT was treated as a continuous variable,[8] and primary fixed effects of interest included group, proportional speech rate, and consonant voicing as well as all possible interactions. Consonant voicing was sum-coded. Additional fixed effects included consonant place of articulation and speaker gender (as above), as well as following vowel height and vowel backness (each sum-coded). Random effects structures included the by-participant random intercepts and slopes by consonant place of articulation (as above) as well as by-item random intercepts (nested by condition).

In order to look at within voicing effects (e.g., for voiced vs. voiceless stops), post hoc pairwise comparisons for the VOT models were computed using estimated marginal means (i.e., least squares means) from the emmeans package, with p values adjusted using the Tukey method (Lenth, 2020). The p values for the fixed effects terms in all

[5]The proportional WPM model excluded the rate contrast for the 2x faster rate condition in order to avoid nonconvergence.

[6]In order to observe a nonlinear effect of rate, a model was fit in which proportional speech rate was coded with a restricted cubic spline with three knots (i.e., one "bend") using the *rms* R package (Harrell, 2020). However, model comparison revealed this nonlinear term did not significantly improve the model fit for either slow or fast speech and so only the linear effect of rate was included in the final model. For ease of comparison, the VOT model also did not include nonlinear terms.

[7]Random slopes for rate were attempted but led to a singular model fit and were thus dropped.

[8]Log-transforming VOT is an increasingly common practice in order to account for normality of the residuals. In this study, VOT was left untransformed because (a) residual plots did not clearly indicate improved residual distributions and (b) previous studies of VOT in dysarthria report on untransformed VOT, which allowed for better comparisons. See further considerations of VOT in the discussion.

models were calculated using the Satterthwaite approximation from the lmerTest package (Kuznetsova et al., 2017).

Results

Speech Rate

Speech rate results are presented in Tables A1–A5 in the Appendix section and Figure 1. Habitual rates of speech are presented in Table 3. Both PD groups spoke at a faster rate than the healthy controls during this carrier phrase speech task. This finding was statistically supported for the OC versus PD-Med groups through a Welch t test ($p = .029$), but not for the OC versus PD-DBS groups ($p = .110$). The two PD groups did not significantly differ in their habitual rates ($p = .983$).

Regarding speech rate modifications, two linear mixed-effects models were built (as described in the Method section) to investigate the effects of speaker group and speech rate condition on (a) actual and (b) proportional rates of speech. Across all speech rate modifications, the PD groups produced a significantly overall faster actual rate of speech compared to the OC group (Group 1 contrast, OC vs. PD-Med and PD-DBS: $\hat{\beta} = -20.001$, $p = .026$). The two PD groups did not significantly differ from one another (Group 2 contrast: $\hat{\beta} = 0.082$, $p = .994$). There were no main group differences, however, when rate was measured as a proportion of each individual's mean habitual rate (OC vs. PDs: $\hat{\beta} = -0.004$, $p = .871$; PD-Med vs. PD-DBS: $\hat{\beta} = -0.005$, $p = .854$), indicating that overall, averaged across all rates, the groups modified their speaking rates to similar degrees, though differences emerged in the Group × Rate interactions detailed below.

Both raw and proportional speech rates increased and decreased with respect to habitual rate as expected for all rate conditions, and these changes were significant for all contrast levels. In Table A1, this is indicated by progressively larger differences between the modified rates compared to the habitual condition (e.g., S4 vs. H1: $\hat{\beta} = -92.388$, $p < .001$).

Group × Rate interactions indicate differences in the extent of speech rate modification across the groups. Significant Group × Rate interactions for the actual rate of speech model would indicate differences in raw change, measured in WPM, from habitual rates to modified rates. Significant interactions for the proportional rate model would indicate changes in the magnitude of adjustment compared to an individual's own baseline.

For slower speech, few Group × Rate Condition interactions were present for either model, indicating that, for the most part, all groups produced both similar actual rates of speech and slowed their speech by similar magnitudes in the slow conditions. An exception to this pattern in slow speech was at the 2x slower condition for the PD-Med versus PD-DBS contrast ($\hat{\beta} = -0.083$, $p < .001$), indicating that the PD-Med group spoke slower relative to their own baseline in the 2x slower condition compared to the PD-DBS group. In Figure 1B, this is visible in the S2 condition by a greater separation between the PD-Med and other groups.

At faster rates, the OC group demonstrated larger actual increases in WPM for the fastest condition (F4: $\hat{\beta} = 34.418$, $p = .005$) and larger proportional increases in WPM for both the F3 and F4 conditions (F3: $\hat{\beta} = 0.164$, $p = .041$; F4: $\hat{\beta} = 0.292$, $p = .004$). There were no significant differences for the 2x faster condition (S2) for either group contrast and no significant differences for the PD-Med versus PD-DBS contrast at any of the faster rate conditions.

The difference between the actual and proportional rate models is important to note because of the difference in baseline speech rates across the groups, with the OC

Figure 1. Speech rate for each group across all rate conditions. (A) Actual speech rate in words per minute [WPM]. (B) Proportional speech rate. OC = older healthy control participants; PD-Med = people with Parkinson's disease who were receiving standard pharmaceutical interventions; PD-DBS = people with Parkinson's disease who had undergone bilateral deep brain stimulation of the subthalamic nucleus surgery.

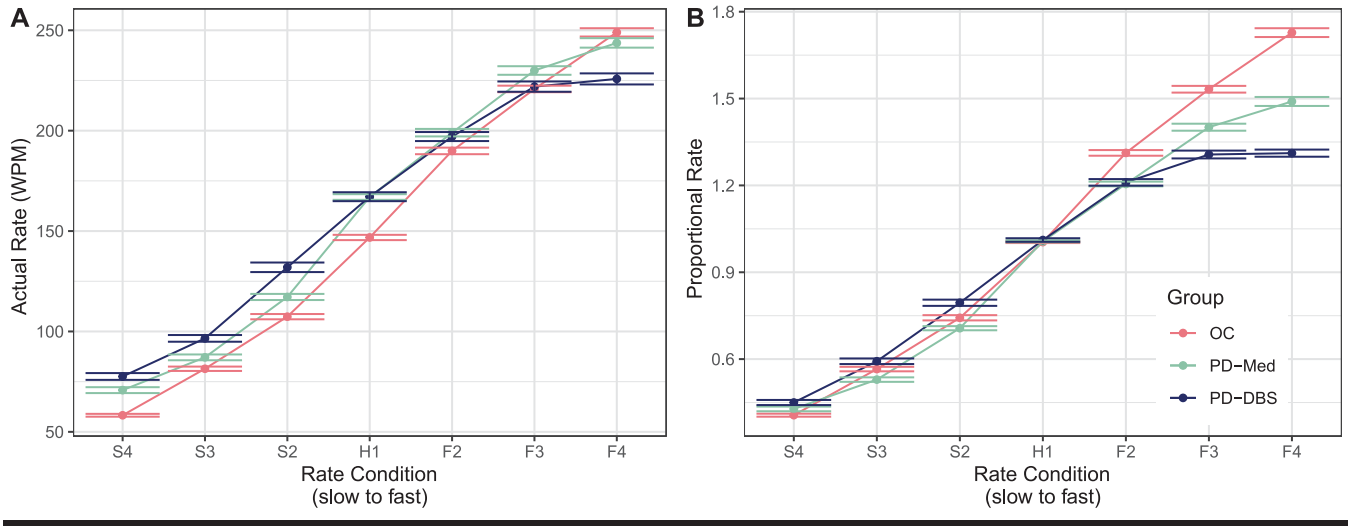


Table 3. Habitual rates of speech in words per minute (WPM) for all three groups.

Group	*n*	WPM	*SD*
OC	17	146.809	25.663
PD-Med	22	166.847	29.495
PD-DBS	12	166.765	35.204

Note. OC = older healthy control participants; PD-Med = people with Parkinson's disease and dysarthria who were receiving standard pharmaceutical interventions; PD-DBS = people with Parkinson's disease who had undergone bilateral deep brain stimulation of the subthalamic nucleus surgery.

group demonstrating a slower overall rate (see Table 3). The proportional rate captures subtle group differences at modified rates. For example, even though the PD groups achieved similar faster actual rates of speech as the healthy controls, relative to their own (faster) baselines, they made smaller relative magnitudes of adjustment at the fastest rates. This is visible in Figure 1B by a clear fanning-out of proportional speech rates across the groups in the faster speech. Proportional rate, rather than actual rate, is entered into the subsequent models looking at fast and slow speech, while actual rate of speech is used in the habitual models.

Acoustic Distinctiveness

Figure 2 reports QVAI and VOT across the whole speech rate continuum, and Figure 3 presents these metrics at habitual rates of speech. In the QVAI models, a significant main effect of rate may be interpreted as vowel distinctiveness varying as a function of speech rate. For the VOT models, where VOT is not a composite measure, the Rate × Voicing interaction indicates whether or not rate led to a change in voicing distinction.

To summarize the presentation of the following sections, the habitual rate models are presented first for both QVAI and VOT, which model vowel and stop production as a function of actual, not proportional, rate of speech. The slower and faster rate models are presented next, and these model the acoustic variables as a function of proportional rate.

Habitual Rate of Speech

QVAI

To reiterate, the final model included fixed effects of group, actual rate of speech, their interaction, as well as preceding consonant place of articulation and speaker gender. Random effects included random by-participant intercepts and random slopes for place of articulation. Random by-participant slopes for rate of speech were omitted due to a singular fit.

In the habitual (unmodified speech) condition, QVAI did not vary as a function of actual speech rate when all other predictors were held at their average values ($\hat{\beta} = 0$, $p = .297$). QVAI did differ by group, with the OC group producing the most expanded vowel space combined ($\hat{\beta} = 0.355$, $p = .018$) and the PD-Med producing a more expanded vowel space

Figure 2. (A) Quadrilateral vowel articulation index (QVAI) and (B) voice onset time (VOT) across the full continuum of speech rates for all three speaker groups. Dotted vertical line corresponds to each talker's mean habitual speech rate. Each individual line in gray represents a unique participant. Speech rate is treated as a proportion of each individual talker's habitual rate. In the bottom panel, the dotted lines correspond to voiceless stops, and the solid lines correspond to voiced stops. OC = older healthy control participants; PD-Med = people with Parkinson's disease who were receiving standard pharmaceutical interventions; PD-DBS = people with Parkinson's disease who had undergone bilateral deep brain stimulation of the subthalamic nucleus surgery.

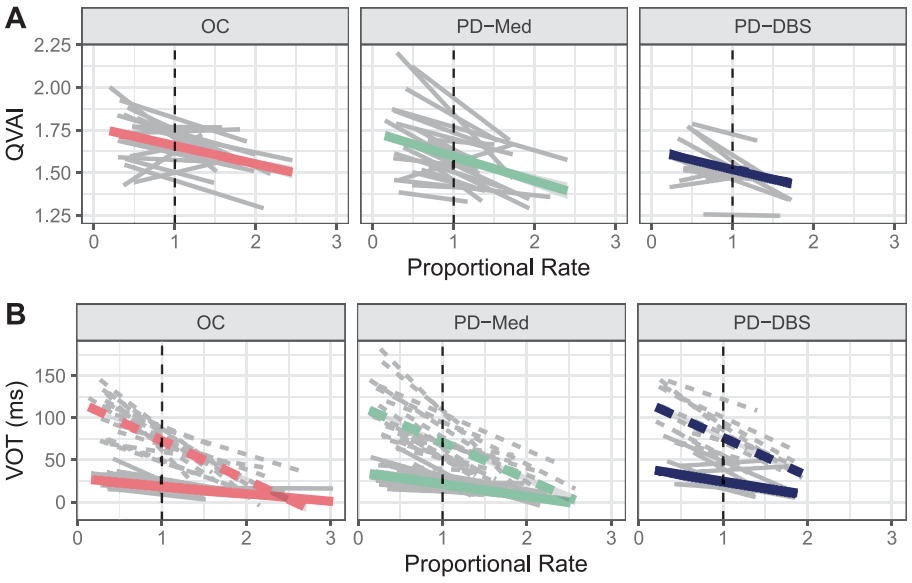
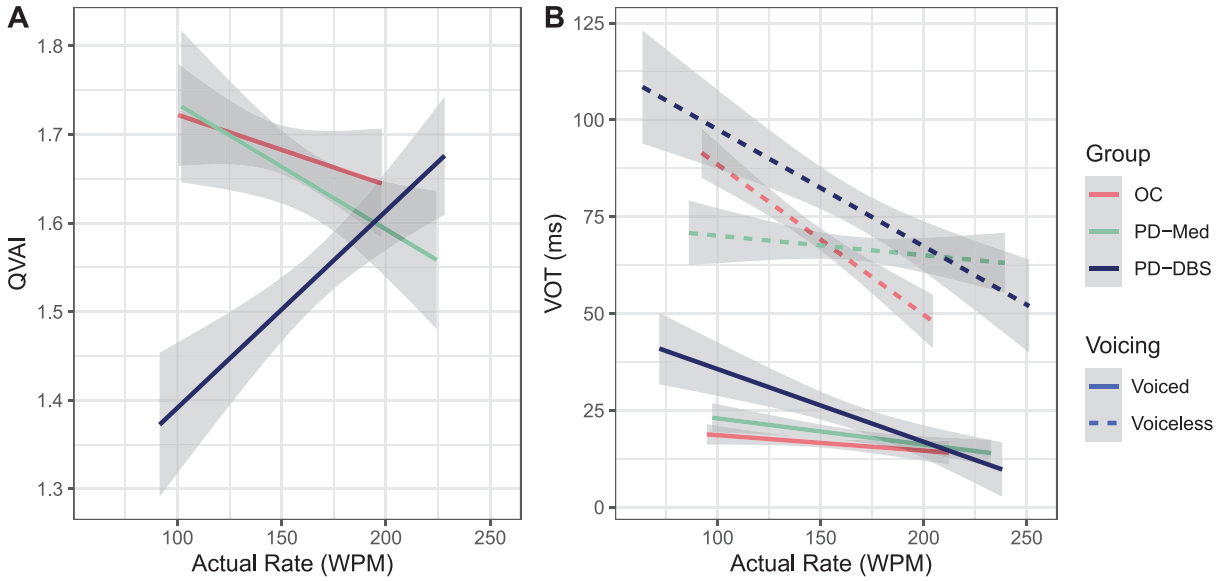


Figure 3. (A) Quadrilateral vowel articulation index (QVAI) and (B) voice onset time (VOT) by speech rate and speaker group during habitual speech. OC = older healthy control participants; PD-Med = people with Parkinson's disease who were receiving standard pharmaceutical interventions; PD-DBS = people with Parkinson's disease who had undergone bilateral deep brain stimulation of the subthalamic nucleus surgery; WPM = words per minute.



compared to the PD-DBS group ($\hat{\beta}$ = 0.425, p = .007). QVAI was associated with larger values at slower rates of speech for the OC and PD-Med talkers, but the opposite trend was apparent for the PD-DBS talkers; this was captured by significant Group × Rate interactions between speech rate and group for both levels of the group contrasts (OC vs. PD groups: $\hat{\beta}$ = −0.002, p = .049; PD-Med vs. PD-DBS groups: $\hat{\beta}$ = −0.002, p = .032). This pattern is visible in Figure 3. Females produced greater vowel expansion than males ($\hat{\beta}$ = 0.058, p < .001). Vowels that followed alveolar consonants were more centralized compared to other preceding consonants (bilabial vs. alveolar/velar $\hat{\beta}$ = 0.112, p < .001; alveolar vs. velar: $\hat{\beta}$ = −0.124, p < .001).

Two separate models were run to explore the effects of proportional rates of speech and QVAI. Speech produced during the slower and faster conditions was included, and speech produced during the habitual condition was excluded. Model results appear in the Appendix (see Tables A4 and A5).

VOT

In habitual speech, VOT showed a predictable pattern with rate of speech, such that (across groups and stop voicing) it was longer for slower actual rates of speech and shorter for faster rates ($\hat{\beta}$ = −0.15, p < .001). Habitual VOT was overall longer for the PD-DBS speakers, captured by a significant group effect for the PD-Med versus PD-DBS contrast ($\hat{\beta}$ = −19.384, p = .046) but not for the OC versus PD contrast ($\hat{\beta}$ = 2.436, p = .797). VOT varied in the expected directions for both rate of speech and consonant voicing, with longer VOTs at slow rates ($\hat{\beta}$ = −0.15, p < .001) and for voiceless consonants ($\hat{\beta}$ = −34.609, p < .001).

Across the groups, the PD talkers produced longer VOT intervals in voiced stops compared to the OC talkers, evident by a significant Group × Voicing interaction for the OC versus PD contrast ($\hat{\beta}$ = −21.769, p = .011). This was especially apparent for the PD-DBS talkers, though there was no significant difference for the PD-Med versus PD-DBS talkers ($\hat{\beta}$ = −6.592, p = .451). Estimated mean differences in voiced and voiceless VOT for the three groups demonstrated no measurable average differences in voiceless VOT for any of the groups and significantly longer voiced VOT for the PD-DBS group compared to the controls and PD-Med talkers. This trend is visible in Table 4. Averaged across both voiced and voiceless stops, the groups did not differ in the degree of VOT adjustments as their (habitual) rate of speech fluctuated (i.e., there were no two-way Group × Rate interactions; OC vs. PD: $\hat{\beta}$ = −0.055, p = .362; PD-Med vs. PD-DBS: $\hat{\beta}$ = 0.068, p = .215).

However, a three-way Group × Voicing × Rate interaction for the OC versus PD contrast indicated a distinct relationship between speech rate and the voicing distinction for the OC and PD groups ($\hat{\beta}$ = 0.14, p = .01). Specifically, for the OC group, slower habitual rates of speech were associated with increased distinctiveness of voiced and voiceless VOT. This pattern was not as apparent for the PD groups, as is visible in Figure 3. There was no significant three-way interaction for the PD-Med versus PD-DBS contrast ($\hat{\beta}$ = 0.056, p = .264).

At habitual rates, VOT was also longer for velar stops (/k/ and /g/; labial vs. alveolar/velar: $\hat{\beta}$ = −13.665, p < .001;

Table 4. Estimated mean differences in voiced and voiceless voice onset time across the groups at habitual rates of speech.

Voicing	Contrast	Estimate	*SE*	*df*	*t* ratio	*p* value
Voiced	OC – PD-All	–2.491	2.102	59.323	–1.185	.467
	OC – PD-DBS	–8.691	2.447	60.693	–3.551	.002
	PD-Med – PD-DBS	–6.200	2.250	61.102	–2.756	.021
Voiceless	OC – PD-All	–1.720	5.483	59.360	–0.314	.947
	OC – PD-DBS	–12.555	6.362	58.409	–1.974	.128
	PD-Med – PD-DBS	–10.835	5.914	54.900	–1.832	.169

Note. OC = older healthy control participants; PD-All: PD-Med and PD-DBS groups combined; PD-Med = people with Parkinson's disease who were receiving standard pharmaceutical interventions; PD-DBS = people with Parkinson's disease who had undergone bilateral deep brain stimulation of the subthalamic nucleus surgery.

alveolar vs. velar: $\hat{\beta} = -9.124$, $p < .001$) and when preceding high vowels ($\hat{\beta} = 3.083$, $p = .002$). There was no effect of following vowel ($\hat{\beta} = -0.541$, $p = .548$) or speaker gender ($\hat{\beta} = 0.1$, $p = .913$). While variables of secondary interest such as speaker gender and consonant place of articulation were also included in the modified speech rate models in order to account for their variability, they will not be reported on.

Slow Speech

QVAI

The fixed effects structure for the slower speech models was identical to the habitual rate model with one exception: Rate of speech was modeled as proportional rate of speech instead of actual rate of speech. Random effects included by-participant intercepts and random slopes for place of articulation and proportional rate of speech.

At slower rates of speech, there were no significant group differences in vowel distinctiveness, though a nonsignificant trend suggested that QVAI was largest for the OC group and smallest for the PD-DBS group (OC vs. PD groups: $\hat{\beta} = 0.091$, $p = .169$; PD-Med vs. PD-DBS: $\hat{\beta} = 0.076$, $p = .346$). There was no effect of proportional rate of speech on QVAI ($\hat{\beta} = -0.012$, $p = .797$), nor was there an interaction between proportional rate and group for either contrast (OC vs. PD: $\hat{\beta} = -0.077$, $p = .399$; PD-Med vs. PD-DBS: $\hat{\beta} = 0.036$, $p = .747$). QVAI was mediated by speaker gender and preceding consonant place of articulation following the same pattern as at habitual rates (gender: $\hat{\beta} = 0.09$, $p < .001$; labial vs. alveolar/velar: $\hat{\beta} = 0.089$, $p < .001$; alveolar vs. velar: $\hat{\beta} = -0.1$, $p < .001$).

VOT

As with QVAI, the slow speech models for VOT were constructed in the same way as the habitual speech models, with the exception that proportional rather than actual rate was used. Across the groups, VOT was predictably longer for voiceless stops ($\hat{\beta} = -47.52$, $p < .001$) and lengthened the slower the speech became ($\hat{\beta} = -39.181$, $p < .001$). VOT in slow speech did not, however, vary by group for either contrast (OC vs. PD: $\hat{\beta} = 3.636$, $p = .499$; PD-Med vs. PD-DBS: $\hat{\beta} = 8.581$, $p = .185$). Significant two-way Group × Rate contrasts indicate that, collapsed across voiced and voiceless stops, all groups demonstrated differences in the degree of VOT adjustments, with the OC group demonstrating the steepest slope of change (OC vs. PD: $\hat{\beta} = -11.784$, $p = .021$) and the PD-DBS group demonstrating the shallowest (PD-Med vs. PD-DBS: $\hat{\beta} = -21.535$, $p = .001$). A two-way Group × Voicing interaction indicated that the OC group also produced a greater voiced–voiceless distinction, averaged across the slow rate spectrum ($\hat{\beta} = -9.942$, $p = .022$), though the two PD groups did not significantly differ from one another ($\hat{\beta} = -5.015$, $p = .339$).

This relationship was further mediated by proportional speech rate adjustments, evidenced by a two-way Rate × Voicing interaction indicating that slower rate was associated with increased voicing contrasts ($\hat{\beta} = 26.17$, $p < .001$). A significant three-way Group × Voicing × Rate interaction for the OC vs. PD groups indicated that this increase was largest for the controls ($\hat{\beta} = 11.734$, $p = .018$). There was no three-way Group × Rate × Voicing interaction for the PD-Med vs. PD-DBS groups ($\hat{\beta} = 9.484$, $p = .116$).

Fast Speech

QVAI

Fixed effects for speech produced in the faster conditions were identical to the slow speech models for QVAI and VOT. Random effects excluded by-participant slopes for proportional speech rate to avoid a singular model fit.

Unlike for slower speech, a significant group difference was found for QVAI at faster rates of speech, with the PD-DBS group showing the most vowel centralization compared to the others. This was captured by a significant effect of the PD-Med versus PD-DBS contrast ($\hat{\beta} = 0.208$, $p = .018$). The OC versus PD contrast was not significant ($\hat{\beta} = 0.095$, $p = .119$). Proportional rate of speech was negatively associated with a significant change in QVAI ($\hat{\beta} = -0.216$, $p < .001$), indicating greater vowel centralization as relative speech rates increased. There was no interaction between group and rate (OC vs. PD groups: $\hat{\beta} = 0.044$, $p = .247$; PD-Med vs. PD-DBS: $\hat{\beta} = -0.087$, $p = .153$). Effects

of speaker gender and preceding consonant place of articulation demonstrated the same predicted effects as the habitual and slow models (gender: $\hat{\beta} = 0.049$, $p = .003$; labial vs. alveolar/velar: $\hat{\beta} = 0.113$, $p < .001$; alveolar vs. velar: $\hat{\beta} = -0.091$, $p < .001$).

VOT

Overall, at faster rates, VOT was altered in predictable ways, that is, longer for voiceless versus voiced stops ($\hat{\beta} = -37.088$, $p < .001$) and shortest at the fastest proportional rates ($\hat{\beta} = -15.96$, $p < .001$). The PD-DBS group produced the longest VOTs overall, with a significant main effect of group for the PD-Med versus PD-DBS group contrast (PD-Med vs. PD-DBS: $\hat{\beta} = -11.714$, $p = .044$) but not for the OC versus PD group contrast ($\hat{\beta} = 5.367$, $p = .216$). Unlike in slower speech, at faster rates, there were no Group × Rate interactions for VOT for either contrast (OC vs. PD: $\hat{\beta} = -4.649$, $p = .065$; PD-Med vs. PD-DBS: $\hat{\beta} = 5.414$, $p = .158$).

In faster speech, the PD-DBS group produced overall less distinction between voiced and voiceless VOT compared to the other groups, indicated by a significant positive effect for the PD-Med versus PD-DBS group by voicing contrast ($\hat{\beta} = 12.051$, $p = .028$). This effect, which was not significant for the OC versus PD group contrast ($\hat{\beta} = -2.223$, $p = .582$), reflects a similar trend to what was observed in slow speech (though in slow speech, the negative estimate for the OC vs. PD group contrast reflected greater voicing distinctiveness for the OCs but no observable differences between the PD groups). As with the other rate blocks (viz., habitual and slower), relatively faster rate was associated with decreased voicing distinctiveness within the fast speech blocks ($\hat{\beta} = 12.936$, $p < .001$).

While the OC and PD-Med groups demonstrated similar degrees of more VOT voicing collapse as rate of speech increased, the PD-DBS group demonstrated a different pattern. Specifically, both voiced and voiceless VOT decreased as rate increased for the OC and PD-Med talkers, but voiceless VOT decreased by a greater extent, resulting in less voicing contrast at the fastest rates of speech. For the PD-DBS talkers, while voiceless VOT behaved in this same way, voiced VOT actually increased as speech rate increased. This is captured by a significant three-way Group × Rate × Voicing interaction for the PD-Med versus PD-DBS contrast ($\hat{\beta} = -7.992$, $p = .033$). The OC versus PD contrast was not significant, reflecting the similarity between the OC and PD-Med groups ($\hat{\beta} = 0.758$, $p = .759$). As can be seen in Figure 5, this pattern can be observed for the PD-DBS group in slow speech too, though it did not reach significance in the models.

In summary, at slower than normal rates of speech, the OC group demonstrated greater VOT voicing contrastiveness as rate continued to decrease. At faster than normal rates of speech, talkers produced a smaller VOT voicing contrast; this collapsed contrast was most apparent for the PD-DBS talkers (who actually increased voiced VOT at their fastest rates of speech).

Discussion

Acoustic–Phonetic Modifications at Habitual Rates of Speech

In summary, the results of this study demonstrated that increasingly slower speech resulted in temporal but not spectral phonetic strengthening, while increasingly faster speech was associated with reductions in both domains. People with PD both with and without STN-DBS demonstrated an ability to slow their speaking rate down to a similar degree as controls. While they were in general able to increase their rate as well, talkers with PD and STN-DBS made smaller magnitudes of change at the fastest rates. The OC group also demonstrated slower habitual rates of speech than both of the PD groups for this carrier phrase task.[9] To reiterate the interpretation of the results, acoustic–phonetic distinctiveness in this study was indexed by changes to vowel centralization (QVAI) and differences in voiced and voiceless VOT.

At habitual rates of speech, healthy control speakers, compared to the PD groups, demonstrated overall more expanded vowel articulation (larger QVAI) and shorter overall VOTs. Of the three groups, the PD-DBS group demonstrated the most vowel centralization and longest VOTs. Natural speaking rate fluctuation in habitual speech (i.e., not intentionally modified) demonstrated a predictable inverse relationship with both vowel centralization and VOT lengthening for the OC and PD-Med groups, but not for the PD-DBS (as depicted in Figure 3 and further discussed below). In other words, segmental strengthening was not uniformally found to be related to speech rate across all groups during habitual speech. This would have been captured by a main effect of rate for QVAI and a Rate × Voicing interaction for VOT.

QVAI at Habitual Rates

The overall vowel articulation patterns observed in this study largely support the current literature. Specifically, talkers with PD demonstrated smaller working vowel spaces (greater centralization) compared to controls (Lam & Tjaden, 2016; Lansford & Liss, 2014; McRae et al., 2002; Rusz et al., 2013; Skodda et al., 2011, 2012; Tjaden, Lam, & Wilding, 2013; Watson & Munson, 2008; Whitfield & Goberman, 2014) and more so for talkers with DBS (Sidtis et al., 2016; cf. Tanaka et al., 2016). Female talkers also demonstrated less centralization than male talkers (Byrd, 1994; Fletcher et al., 2017; Jacewicz et al., 2009; Neel, 2008). While vowel centralization did not vary by rate when averaged across the groups, this was driven by differences in the PD-DBS group. While the OC and PD-Med groups showed a similar degree of vowel centralization at faster habitual rates, the PD-DBS group demonstrated the opposite trend, with faster habitual rates associated with *less* centralization (see Figure 3). This finding indicates that PD-DBS talkers who habitually spoke

[9]Note, however, that this same group of talkers did not demonstrate habitual rate differences in a sentence reading task (Knowles et al., 2021).

at faster rates of speech had less vowel centralization than those who spoke at slower rates. This is a surprising finding. However, examining the speakers at the extreme ends of the PD-DBS group may be warranted. The PD-DBS talker with the slowest habitual rate (97 WPM) and most vowel centralization (QVAI = 1.29) was PD-DBS 04, whose speech was characterized by having a strained–strangled quality. Conversely, PD-DBS 12 had the fastest habitual rate of speech of the PD-DBS talkers (218 WPM) and the least amount of vowel centralization (QVAI = 1.72). Perceptually, his speech was characterized by mild hypophonia. These speakers also had relatively lower and higher sentence intelligibility, respectively, as reported in a previous study from this project (Knowles et al., 2021). Previous studies have suggested that some talkers with STN-DBS demonstrate mild spasticity due to the spread of the electrical current to nearby fiber tracts (Fenoy et al., 2016; Narayana et al., 2009; Tsuboi et al., 2017). It could be that slower rates within the PD-DBS group indicate a greater degree of spasticity, leading to greater restriction of articulatory movements. Further speculation, however, is beyond the scope of this article.

VOT at Habitual Rates

While all groups produced global lengthening of VOT at slower habitual rates, a similar degree of lengthening for both voiced and voiceless VOT for the PD groups (i.e., absence of a Voicing × Rate interaction) meant that this was not associated with segmental category strengthening. In contrast, the older controls demonstrated greater lengthening for voiceless VOTs, resulting in a greater voicing contrast at slower habitual rates. These findings are consistent with well-established patterns of VOT in healthy talkers demonstrating a clear relationship between speech rate and VOT (Kessinger & Blumstein, 1997; Miller et al., 1986, 1997; Summerfield, 1981).

The patterns of VOT asymmetry are most apparent for the control speakers and are consistent with previous studies: VOT varies by speech rate, but this is largely driven by changes to voiceless rather than voiced VOT (Kessinger & Blumstein, 1997; Miller et al., 1986, 1997; Summerfield, 1981). In Figure 3, this is visible as a steeper regression line for voiceless compared to voiced VOT across habitual speech rate variations in the control speakers. This pattern is much less robust in the two PD groups, though for different reasons. In particular, voiced and voiceless VOTs are modified along much more seemingly parallel trajectories. For the PD-Med group, the rate of change is fairly flat for both voiced and voiceless VOT. This mimics the pattern of change for voiced VOT for the control speakers. For the PD-DBS group, the rate of change for both voiced and voiceless VOT is much steeper, with both categories resembling the pattern observed for the control's voiceless VOT. Previous reports of VOT in talkers with PD have exclusively reported on habitual speech, with inconsistent findings across voiced and voiceless stops.

The results of this study are consistent with previous findings of overall reduced distinctiveness between voiced and voiceless stops in PD (Hochstadt et al., 2006; Lieberman et al., 1992; Whitfield et al., 2018). No differences were observed in voiceless VOT between the groups, which is consistent with several previous reports (Bunton & Weismer, 2002; Connor et al., 1989; Cushnie-Sparrow et al., 2016; Fischer & Goberman, 2010; Flint et al., 1992; Forrest et al., 1989; Ravizza, 2003; cf. Tjaden, 2000). People with PD have been reported to produce longer voiced VOT (Forrest et al., 1989; Tjaden, 2000), though voiced VOT has received less focus in the literature. Findings for the PD-Med group were inconsistent with this pattern, but the PD-DBS group did demonstrate longer voiced VOT. Despite similarities in VOT, even at habitual rates of speech, there is a striking difference in the relationship between rate and voiced and voiceless VOT across all three groups.

Slow Speech

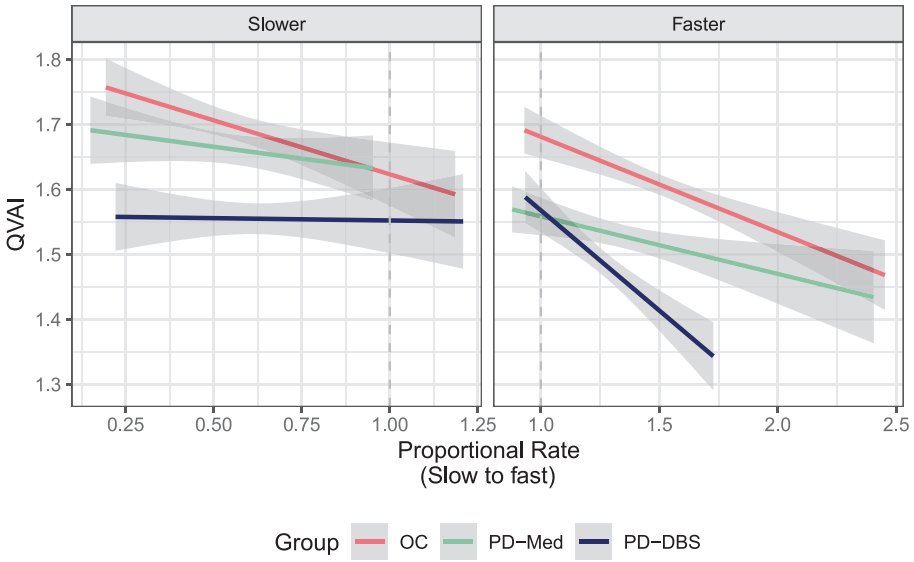
To reiterate, analyses for modified rates included speech rate characterized as a proportion of each individual speaker's baseline speaking rate. QVAI and VOT were modeled *within* each rate block; that is, the comparison is within slower than normal or faster than normal speech and *not* with habitual. Figure 2 demonstrates the overall trends across all rates, while the left panels in Figures 4 and 5 display the slow rates alone. Slower than normal speech was not associated with increased vowel distinctiveness but was associated with increased stop voicing distinctiveness for all groups.

QVAI

In slower than normal speech, vowel centralization did not increase as speech rates decreased (absence of main effect of rate). This was the case for all three groups, which was evidenced by a lack of Group × Rate interactions. Figure 2 shows that while there was an overall trend for less centralization (higher QVAI) at slower rates, this was not supported by the model results. QVAI did not robustly vary as talkers continued to decrease their rate across the slow speech conditions. This is apparent in Figure 4 as relatively flat slopes of change for QVAI in slow speech. While this would appear to contradict previous findings of vowel expansion at slower rates of speech (Buccheri et al., 2014; McRae et al., 2002; Tjaden et al., 2005; Tjaden & Wilding, 2004), previous studies have often reported nonsignificant trends in talkers with PD. For example, Tjaden and Wilding (2004) found that in a group of speakers with dysarthria secondary to PD or to multiple sclerosis, while slow speech was associated with greater QVAI, this difference was not statistically supported for the PD talker group. Changes in vowel space have been found to be more strongly related to rate reductions in other dysarthric groups such as speakers with amyotrophic lateral sclerosis (Turner et al., 1995; Weismer et al., 2000) and cerebral palsy (Hustad & Lee, 2008). It is worth noting that the current results capture change within slower and faster than normal speech, and not a direct comparison with habitual speech.

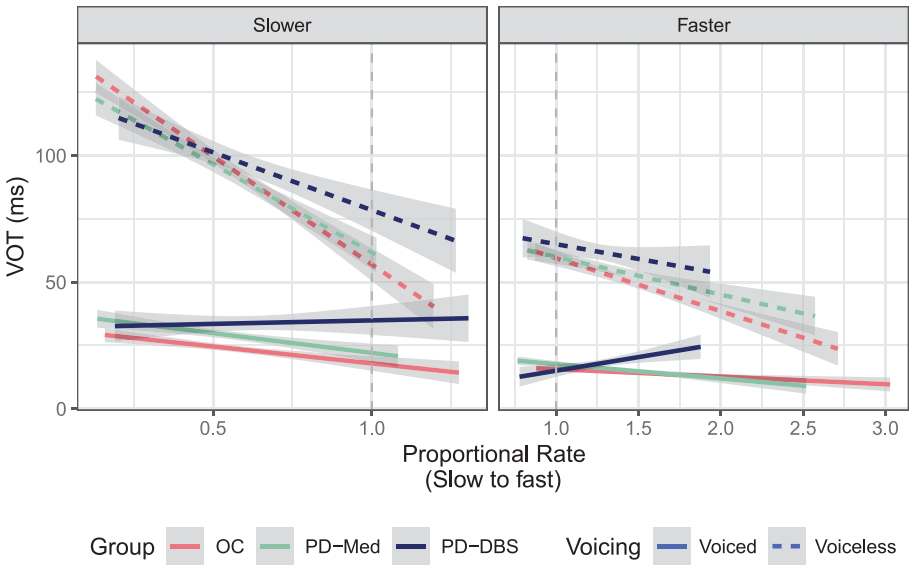
Tjaden and colleagues (Tjaden, Lam, & Wilding, 2013; Tjaden & Wilding, 2004) have demonstrated that

Figure 4. Quadrilateral vowel articulation index (QVAI) in slower and faster rate blocks for each group. Dotted vertical line at 1 represents each talker's average habitual rate of speech. OC = older healthy control participants; PD-Med = people with Parkinson's disease who were receiving standard pharmaceutical interventions; PD-DBS = people with Parkinson's disease who had undergone bilateral deep brain stimulation of the subthalamic nucleus surgery.



while enhancements in acoustic vowel measures do occur with slow speech, other speaking methods such as clear and loud speech may bring about greater changes. Tjaden, Lam, and Wilding (2013) found that *clear* speech led to overall greater differences in tense and lax vowel space and vowel distinctiveness (measured by dispersion and vowel lambdas) when compared to habitual, loud, and slow speech conditions. Buccheri (2013) found that acoustic distances between front and back vowels /i/ and /a/, as well as measures of vowel dispersion, increased in both clear and slow speech. In this study, neither the PD groups nor the healthy controls showed a change in vowel centralization at slower rates. This

Figure 5. Voice onset time (VOT; in milliseconds) for voiced and voiceless stops in slower and faster rate blocks for each group. Dotted vertical line at 1 represents each talker's average habitual rate of speech. OC = older healthy control participants; PD-Med = people with Parkinson's disease who were receiving standard pharmaceutical interventions; PD-DBS = people with Parkinson's disease who had undergone bilateral deep brain stimulation of the subthalamic nucleus surgery.



finding is inconsistent with previous reports of this behavior in healthy talkers (Fletcher et al., 2015; Fourakis, 1991; Tjaden & Wilding, 2004; Tsao & Iqbal, 2006; Turner et al., 1995; Weismer et al., 2000). This could be task specific. In this study, participants read aloud nonsense words in carrier phrases. It is possible that the controls were already hyperarticulating their speech even at their habitual rates. Less common words are also known to be produced with greater vowel space than high-frequency words for individuals with and without PD (Munson & Solomon, 2004; Watson & Munson, 2008; Wright, 2004). Future extensions of this work should explore changes in vowel production of the spontaneous speech samples.

VOT

While vowel centralization did not demonstrate a clear pattern of change at slower rates, VOT distinctiveness increased as slower than normal speech became even slower overall. This finding was strongest for the older healthy control group. In other words, slower speech was associated with temporal strengthening of phonemic contrasts (VOT) but did not yield sufficient changes in articulatory posturing to result in spectral strengthening (acoustic vowel space expansion). The increase in voicing distinctiveness was attenuated in the talkers with PD. While VOT as a function of speech rate modifications has not before been studied in people with PD, the findings of this study are consistent with previous literature in both healthy and disordered populations. In healthy talkers, VOT increases as rate decreases, with larger magnitudes of change for voiceless stops (Diehl et al., 1980; Miller, 1981; Miller et al., 1986; Summerfield, 1981). However, aberrant manifestations of this pattern have been observed in talkers with communication deficits. Baum and Ryan (1993) found that while people with aphasia produced longer VOT in slow speech similarly to healthy controls, the magnitude of voiced and voiceless VOT adjustment did not lead to increases in voicing or place distinctiveness. The pattern observed in the PD group here mirrors these observations: While the voicing contrast was maintained across groups, it was not magnified in slow speech for the PD groups to the same extent as for the healthy older controls.

Fast Speech

In contrast to slow speech, faster than normal speech was associated with changes in both spectral and temporal domains, with greater vowel centralization and reduced VOT contrasts at increasingly faster rates for all groups. At baseline (i.e., during habitual speech), the PD groups produced more centralized vowel space and more overlap in voiced and voiceless VOT, and this was most apparent in the PD-DBS group. The PD-DBS group in particular also produced overall longer voiced and voiceless VOT than the other two groups. All three groups produced similar degrees of vowel centralization across the modified rates, indicated by a lack of two-way Group × Rate interactions for both slower and faster speech conditions. This was not the case for stop voicing, however. Overall, the voicing contrast did not benefit from slower speech for either PD group but did for the controls. In faster speech, the voicing contrast collapsed for all three groups but did so to a lesser degree for the PD groups and, in particular, the PD-DBS group. This was largely due to a flatter slope of change for voiced stops and, at least for some individuals with STN-DBS, unexpected voiced VOT *lengthening* at faster rates.

Increased spatiotemporal variability at modified rates of speech may also explain this finding. Specifically, people with PD have been shown to demonstrate greater speech motor variability at slower speaking rates (Kleinow et al., 2001). This observation has been couched as a cautionary tale for clinicians training clients to use slower speaking rates (Smith et al., 1995). Furthermore, talkers with relatively slower habitual rates of speech have also been found to have greater variability in their vowel space (Tsao et al., 2006), and acoustic variability has been shown to increase at slower rates in general (Kleinow et al., 2001; McHenry, 2003).

There are also reports of both healthy talkers and talkers with PD demonstrating increased spatiotemporal variability at slower *and* faster rates (Chu et al., 2020; Kleinow et al., 2001). In other words, the speech motor control required to make adjustments to habitual speech patterns may induce increased variability regardless of the direction of rate changes.

With regard to VOT adjustments, it is paramount to discuss how trends in VOT measurement may have a bearing on the implications of stop production. All previous studies examining VOT as a function of rate adjustments, to the authors' knowledge, have used raw (untransformed) VOT. Given voiced VOT is inherently smaller than voiceless VOT, this often results in skewed distributions. Furthermore, reports of greater magnitudes of change in voiceless VOT relative to voiced VOT and the implications on how the voicing contrast is preserved or adjusted may also be driven by these inherent temporal differences. Some more recent work on VOT has modeled VOT on the log scale as an appropriate strategy to mitigate the large differences in voicing categories as well as in order to account for the nonlinear perception of temporal stop voicing categories (Sonderegger, 2015; Sonderegger et al., 2019; Stuart-Smith et al., 2015; Volaitis & Miller, 1992). Choosing one approach over the other has been reported to yield changes in patterns of the results (Chodroff & Wilson, 2017). Previous work in VOT in PD has used untransformed VOT. The decision to do so in this study was made intentionally to be able to compare with previous findings and because there is no current consensus on the best way to model VOT. Future work should explore these discrepancies further.

While the focus of the current study was on acoustic modifications resulting from changes in speech rate, a discussion of the potential bearings on speech intelligibility is warranted. From a clinical perspective, slowed speech rate is a behavioral intervention typically chosen in order to effect change in speech intelligibility. As stated previously, slowed speech does not always result in improvements in

intelligibility (Kuo et al., 2014; Van Nuffelen et al., 2010), and the present findings may point toward avenues of investigation to better understand why this is so. For example, there is an established correlation in the literature between vowel space and intelligibility (Feenaughty et al., 2014; H. Kim et al., 2011; Y. Kim & Choi, 2017; Lansford & Liss, 2014; McRae et al., 2002; Tjaden & Wilding, 2004), and vowel centralization was not found to systematically change in slow speech in this study. Furthermore, the PD group did not increase VOT voicing distinctions to the same degree as controls in slow speech, which could have implications on listener expectations of voicing categories.

Relatedly, while faster speech is rarely a therapeutic target for dysarthria, an increasing body of literature suggests that not all individuals demonstrate predicted declines in intelligibility at faster rates and some may even show modest increases (Knowles et al., 2021; Kuo et al., 2014). Conclusions from this study cannot necessarily account for this discrepancy but, again, may point toward avenues of future study. For example, the PD groups showed less of a collapse of voicing contrasts at faster rates, which has the potential to mitigate perceptual confusion of voicing categories from listeners. Follow-up work from this study is currently ongoing to investigate these trends.

Listeners likely rely on a combination of acoustic cues provided by a speaker when perceiving contrasts. This may be especially important for resolving contrasts reliant on temporal information such as VOT as speech rate varies. Aberrantly lengthened voiced VOT at slow speech rates, as observed in the PD groups, could present challenges for listener perception if accompanied by more typical acoustic adjustment of other features (e.g., vowel lengthening). Prosodic information may also play a critical role in a listener's ability to correctly perceive a given phonetic category (S. Kim & Cho, 2013; Mitterer et al., 2016; Steffman, 2019). A criticism of some rate control methods is the potential for disrupted prosody and consequent deterioration of speech naturalness (Yorkston et al., 1990). Yorkston et al. (1990) found impaired naturalness to be especially apparent for healthy controls at slower rates. However, the authors also found that while sentence-level intelligibility improved at slowed rates in talkers with dysarthria, in most cases, phoneme intelligibility did not. Further consideration of how prosodic structure and acoustic–phonetic contrasts interact at modified rates to impact intelligibility is warranted.

Talkers With STN-DBS

In this study, talkers with PD and STN-DBS demonstrated the most aberrant productions along the speech rate continuum. While they were able to modify their speech rate in both directions (slower and faster), they produced a more restricted range of proportional rate adjustments than the OC and PD-Med groups. At their habitual rates of speech, they produced more centralized vowels and longer VOT than the other two groups and more overlap in VOT voicing categories than the healthy controls (the two PD groups did not differ in this regard, given the absence of the relevant Group × Voicing contrast). The vowel production findings are consistent with previous reports of speech impairments following STN-DBS including greater vowel centralization (Chenausky et al., 2011; Martel-Sauvageau et al., 2014, 2015; Sidtis et al., 2016; Skodda et al., 2014) and that standard STN-DBS settings may be suboptimal for vowel production (Knowles et al., 2018). Previous reports of stop production in talkers with STN-DBS do not point to as clear a trend.

Regarding the patterns observed in VOT, previous research suggests that, generally, STN-DBS stimulation is associated with shorter, less variable VOT compared to when stimulation is off (Hoffman-Ruddy et al., 2001; Putzer et al., 2008) or presurgery (Åkesson et al., 2010) and with longer, more variable VOT compared to healthy controls and when stimulation is turned on (Chenausky et al., 2011; Karlsson et al., 2014). Other neural targets besides the STN may be more vulnerable to articulatory impairments compared to the STN, for example, caudal zona incerta (Eklund et al., 2014; Karlsson et al., 2014; Karlsson, Unger, Wahlgren, & van Doorn, 2011). Results of the current study are thus not directly analogous, but comparisons may still be drawn and considered in directions for future acoustic studies of talkers with DBS.

Spirantization (i.e., incomplete stop closure allowing for a leakage of air, making the stop more fricative-like) has been reported with greater frequency in individuals with DBS (Chenausky et al., 2011; Dromey & Bjarnason, 2011; Eklund et al., 2014; Karlsson et al., 2014) and could be related to the finding of longer VOT in this study. Karlsson et al. (2014) found that individuals with STN-DBS or caudal zona incerta exhibited greater degrees of spirantization compared to their preoperative speech and when DBS was off during passage reading. Interestingly, however, they also found that these talkers produced more prominent stop releases, attributable to a *stronger* stop occlusion. These findings would appear to contradict one another, but Karlsson et al. suggested that while these individuals were able to generate sufficient energy during speech to produce a distinctive plosive release (compared to when DBS was off), a consequence of this was premature stop consonant frication. Many of the stops in the current data were noted as having partial spirantization, but this was not categorically measured. Future work should explore the relationship between VOT, spirantization, and spectral stop moments and intensity in PD, especially in those with DBS.

Related to spirantization, another consideration is the presence of VOTs that could not be measured. In the current study, stops that had a clear release throughout the entire stop but had no clear closure were coded as having measurable VOT, but in these instances, the duration of VOT was equal to that of the entire stop consonant (i.e., no or little closure). These would likely be cases of longer VOT. These instances were kept for the VOT analysis because their release was still measurable. However, Karlsson, Unger, Wahlgren, Blomstedt, et al. (2011) considered these cases a form of unmeasurable VOT and noted that these types of instances were more common in individuals with DBS. While these extreme cases were

uncommon in the present data, accounting for less than 3% of the data, they were more common in individuals with PD and DBS (OC: < 1%; PD: 2.5%; DBS: 3.4%). Exploratory analyses with these data points removed did not change the pattern of the results. The proportion of VOT to closure duration would be another metric worth considering in order to explore these potential effects (Whitfield et al., 2018).

Another type of deviant stop production is "unreleased" stops, that is, stops produced with no obvious burst. These were excluded from the VOT analysis. In this study, 3.7% of stops overall were unreleased. This amounted to 1.86% for controls, 3.85% for PDs, and 7.81% for the DBS group. This demonstrates a similar but attenuated pattern to that reported by Özsancak et al. (2001), in which 19% of stops in talkers with hypokinetic dysarthria could not be measured due to the absence of a clear burst, compared to 7% in controls. Exploring more measures of aberrant stop production in combination with VOT measures may be a promising avenue for determining underlying acoustic and physiological underpinnings of differences in laryngeal–supralaryngeal coordination impairments in PD and especially in characterizing differences related to DBS.

As stated in the introduction, no previous studies have undertaken investigations of speech rate modification for talkers with DBS. In this study, the PD-DBS talkers demonstrated greater impairment and markedly different patterns of acoustic adjustments at both ends of the modified rate continuum compared to the other two speaker groups. These results suggest that future inclusion of people with PD and DBS is necessary to (a) understand potential differing treatment effects and (b) better describe the mechanisms of speech motor control following DBS surgery.

Limitations

Certain limitations warrant cautious interpretation of the findings presented above. Firstly, inclusion criteria for the speaker group were intentionally lenient to include a representative sample of speech deficits in PD. Furthermore, unequal distributions of talkers with DBS and of speaker gender limit the generalizability of the findings. Future studies would benefit from additional clinical and speaker-specific factors (such as dysarthria severity) included in the analyses of rate modifications.

Two limitations regarding decisions in the acoustic analysis bear discussion. Vowel formants were measured at the 30-ms midpoint to ensure consistency across measures. However, this may not be the most sensitive point of measurement. Recent evidence suggests that formant measurement taken from specific articulatory points more reflective of the expected steady-state vowel productions may be more adept at predicting perceptual ratings of dysarthric speech (Fletcher et al., 2017). Regarding VOT, as mentioned in the Method section, this study used a definition of positive VOT to distinguish voicing during closure (Chodroff & Wilson, 2017; Davidson, 2016) and did not consider elements of the stop release that could be used to describe spirantization (Karlsson et al., 2014). These decisions were made to present an overall characterization of the temporal components of plosive release across varied speaking rates, but these additional metrics of stop production would add value in understanding these effects more clearly. QVAI was also measured as a composite index, which required it to be averaged across multiple productions in the models. The VOT models, on the other hand, considered each individual data point (and thus were fit with more observations).

The speech task itself was designed to elicit controlled connected speech, but the nature of it may also limit the generalizability of the findings. While speakers were asked to read aloud the list of words prior to beginning the experiment, the fact that the stimuli were comprised of nonsense words may have elicited more hyperarticulation than may have been seen in more familiar words (Chiu & Forrest, 2017).

Conclusions

Findings from this study demonstrate that temporal but not spectral distinctiveness increases as talkers progressively slow their speaking rate. Conversely, both temporal and spectral contrasts diminish as talkers increase their rates. Healthy control speakers demonstrated overall greater degrees of phonetic strengthening at slow rates and more pronounced collapse at faster rates, while talkers with PD and STN-DBS demonstrated the smallest magnitude of change, despite overall successfully modifying their rate of speech to similar degrees.

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Individual Participant Results and Model Output

Figure A1. Individual quadrilateral vowel articulation index (QVAI) results. OC = older healthy control participants; PD-Med = people with Parkinson's disease who were receiving standard pharmaceutical interventions; PD-DBS = people with Parkinson's disease who had undergone bilateral deep brain stimulation of the subthalamic nucleus surgery.

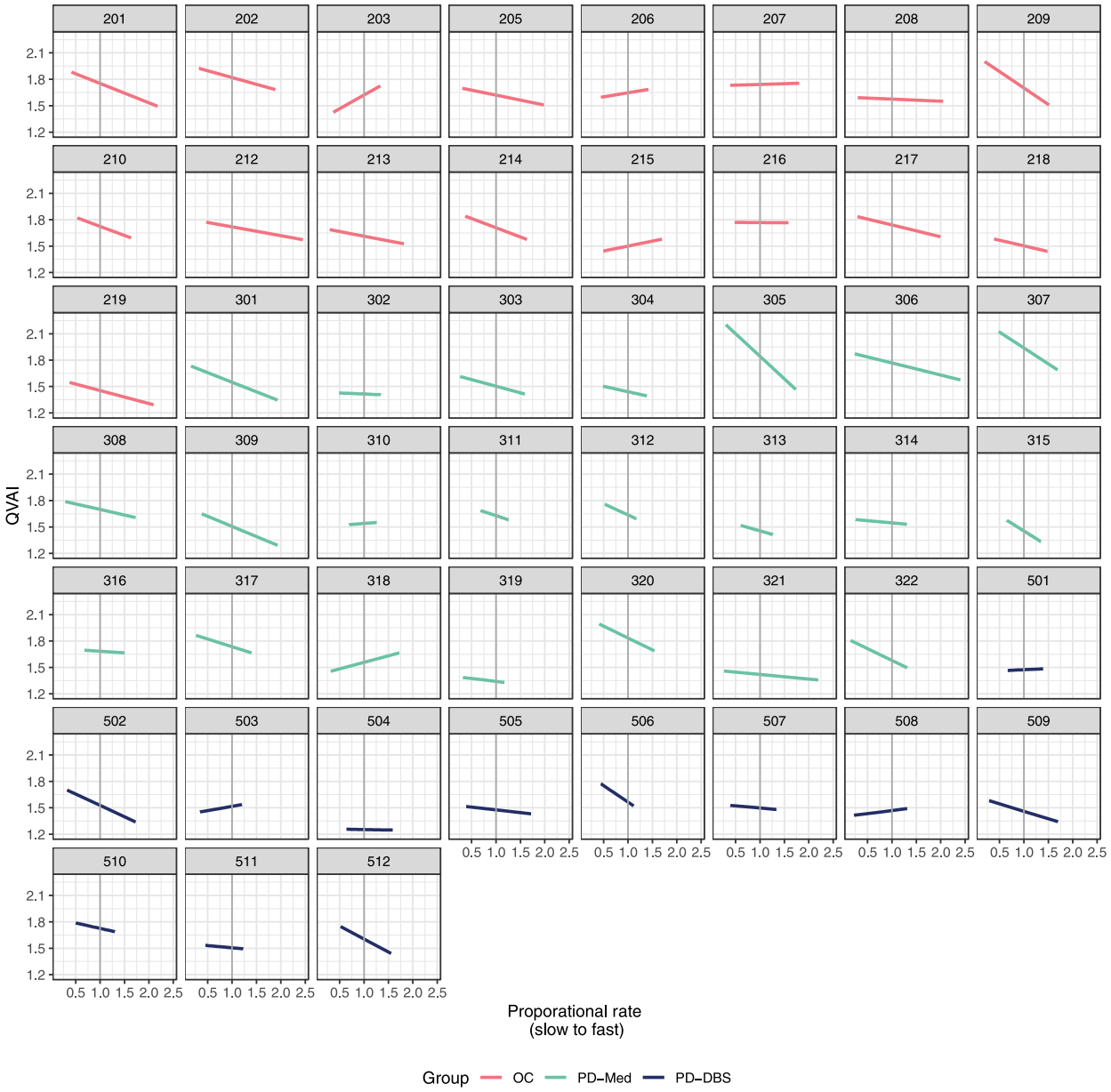

Figure A2. Individual voice onset time (VOT) results. OC = older healthy control participants; PD-Med = people with Parkinson's disease who were receiving standard pharmaceutical interventions; PD-DBS = people with Parkinson's disease who had undergone bilateral deep brain stimulation of the subthalamic nucleus surgery.

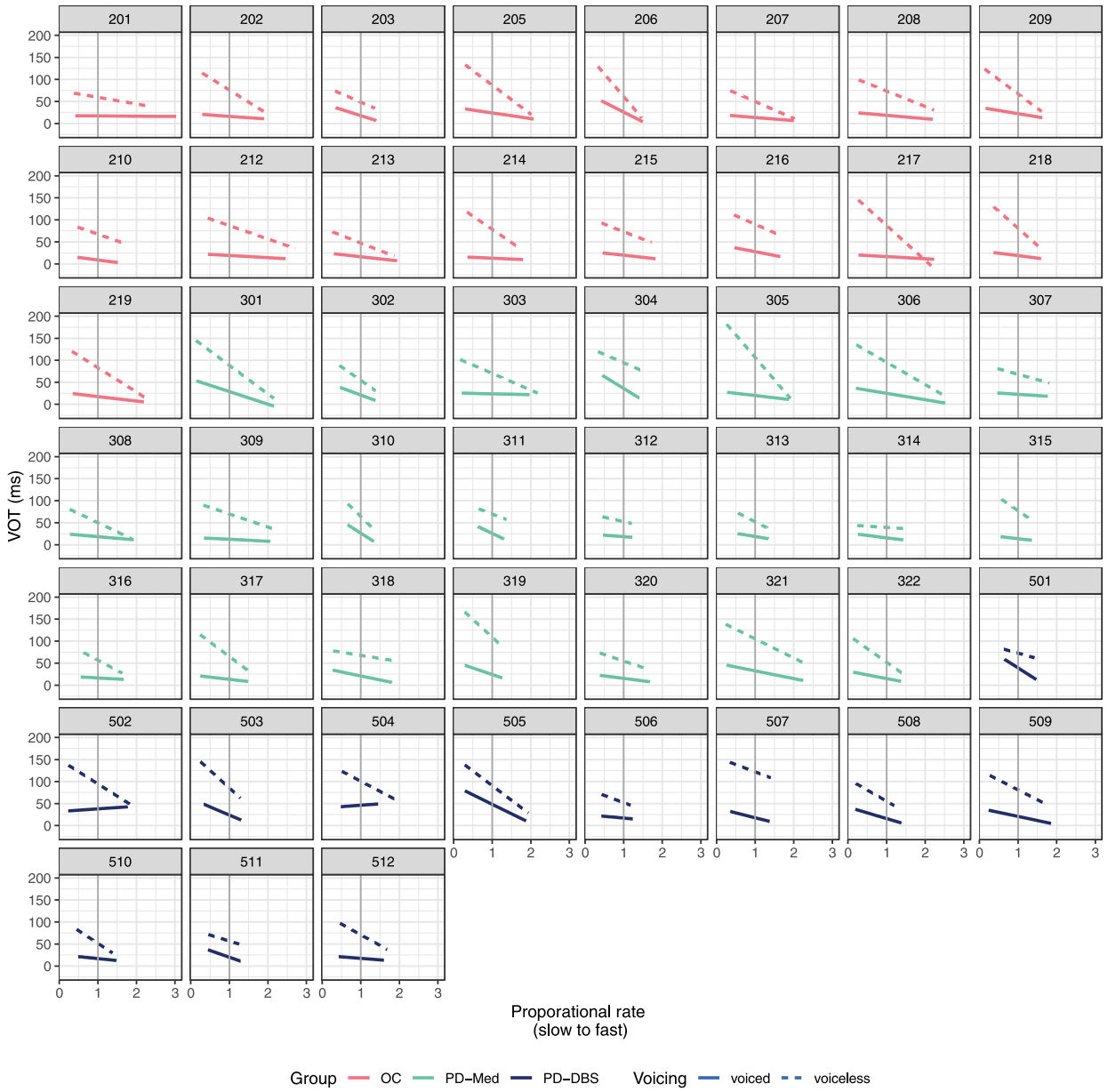

Table A1. Summary of model results for speech rate.

	Actual rate (WPM)					Proportional rate				
Contrast	**Estimate**	***SE***	***df***	***t* value**	**Pr(> |*t*|)**	**Estimate**	***SE***	***df***	***t* value**	**Pr(> |*t*|)**
(INTERCEPT)	160.143	4.165	51.000	38.448	.000	1.007	0.012	54.090	87.510	.000
GROUP1	–20.001	8.703	50.992	–2.298	.026	–0.004	0.024	54.035	–0.164	.871
GROUP1:F2	11.321	7.425	50.907	1.525	.134	0.103	0.058	51.417	1.777	.081
GROUP1:F3	16.727	9.822	51.079	1.703	.095	0.164	0.078	50.873	2.096	.041
GROUP1:F4	34.418	11.829	50.425	2.910	.005	0.292	0.095	50.005	3.065	.004
GROUP1:S2	3.248	8.000	51.002	0.406	.686	–0.004	0.008	8005.094	–0.447	.655
GROUP1:S3	9.722	9.261	50.997	1.050	.299	0.009	0.029	53.046	0.304	.762
GROUP1:S4	5.801	9.061	50.614	0.640	.525	–0.029	0.032	52.625	–0.895	.375
GROUP2	0.082	10.353	51.008	0.008	.994	–0.005	0.029	54.142	–0.184	.854
GROUP2:F2	2.527	8.835	50.963	0.286	.776	0.001	0.069	51.464	0.021	.983
GROUP2:F3	13.040	11.720	51.611	1.113	.271	0.077	0.093	51.353	0.824	.414
GROUP2:F4	17.924	14.212	51.855	1.261	.213	0.112	0.114	51.179	0.978	.333
GROUP2:S2	–14.351	9.517	51.033	–1.508	.138	–0.083	0.010	8005.663	–8.535	.000
GROUP2:S3	–9.528	11.017	51.013	–0.865	.391	–0.059	0.034	53.127	–1.709	.093
GROUP2:S4	–3.425	10.806	51.072	–0.317	.753	–0.017	0.039	53.671	–0.427	.671
F2	35.566	3.554	50.936	10.008	.000	0.240	0.028	51.441	8.671	.000
F3	62.825	4.708	51.353	13.344	.000	0.419	0.037	51.121	11.189	.000
F4	79.212	5.690	51.164	13.921	.000	0.530	0.046	50.611	11.604	.000
S2	–41.605	3.829	51.018	–10.866	.000	–0.259	0.004	8005.389	–66.445	.000
S3	–71.855	4.432	51.005	–16.212	.000	–0.444	0.014	53.088	–32.085	.000
S4	–92.388	4.342	50.850	–21.278	.000	–0.579	0.016	53.167	–37.212	.000

Note. Coefficient estimates, standard errors (*SE*), degrees of freedom (*df*), *t* values, and significances are reported. WPM = words per minute; GROUP1 = OC vs. PD-All; GROUP2 = PD-Med vs. PD-DBS; S2/S3/S4/F2/F3/F4 = rate conditions.

Table A2. Summary of model results for quadrilateral vowel articulation index (QVAI) at habitual rates of speech.

Contrast	**Estimate**	***SE***	***df***	***t* value**	**Pr(> |*t*|)**
(INTERCEPT)	1.711	0.068	90.628	25.223	.000
GROUP1	0.355	0.147	76.949	2.421	.018
GROUP2	0.425	0.156	100.786	2.730	.007
WPM	0.000	0.000	87.202	–1.050	.297
POA1	0.112	0.013	47.361	8.624	.000
POA2	–0.124	0.012	48.611	–10.467	.000
GENDER	0.058	0.016	49.930	3.735	.000
GROUP1:WPM	–0.002	0.001	77.736	–2.000	.049
GROUP2:WPM	–0.002	0.001	103.608	–2.171	.032

Note. Coefficient estimates, standard errors (*SE*), degrees of freedom (*df*), *t* values, and significances are reported. GROUP1 = OC vs. PD-All; GROUP2 = PD-Med vs. PD-DBS; WPM = words per minute; POA1 = bilabial vs. alveolar and velar place of articulation; POA2 = alveolar vs. velar place of articulation.

Table A3. Summary of model results for voice onset time (VOT) at habitual rates of speech.

Contrast	Estimate	*SE*	*df*	*t* value	Pr(> |*t*|)
(INTERCEPT)	69.378	4.299	247.044	16.138	.000
GROUP1	2.436	9.477	221.405	0.257	.797
GROUP2	–19.384	9.659	265.358	–2.007	.046
WPM	–0.150	0.026	297.778	–5.851	.000
VOICING	–34.609	3.876	206.163	–8.930	.000
POA1	–13.665	1.882	23.432	–7.262	.000
POA2	–9.124	2.170	23.305	–4.205	.000
V.BACKNESS	–0.541	0.887	23.401	–0.610	.548
V.HEIGHT	3.083	0.886	23.309	3.480	.002
GENDER	0.100	0.916	44.979	0.109	.913
GROUP1:WPM	–0.055	0.060	264.636	–0.913	.362
GROUP2:WPM	0.068	0.054	355.751	1.243	.215
GROUP1:VOICING	–21.769	8.449	179.959	–2.576	.011
GROUP2:VOICING	–6.592	8.732	213.695	–0.755	.451
WPM:VOICING	0.059	0.023	214.411	2.546	.012
GROUP1:WPM:VOICING	0.140	0.054	196.484	2.608	.010
GROUP2:WPM:VOICING	0.056	0.050	242.409	1.120	.264

Note. Coefficient estimates, standard errors (*SE*), degrees of freedom (*df*), *t* values, and significances are reported. GROUP1 = OC vs. PD-All; GROUP2 = PD-Med vs. PD-DBS; WPM = words per minute; POA1 = bilabial vs. alveolar and velar place of articulation; POA2 = alveolar vs. velar place of articulation; V.BACKNESS = vowel backness; V.HEIGHT = vowel height.

Table A4. Summary of model results for quadrilateral vowel articulation index (QVAI) at slower and faster rates of speech.

	Slow					Fast				
Contrast	Estimate	*SE*	*df*	*t* value	Pr(> |*t*|)	Estimate	*SE*	*df*	*t* value	Pr(> |*t*|)
(INTERCEPT)	1.678	0.039	53.846	43.482	.000	1.851	0.035	352.151	53.319	.000
GROUP1	0.091	0.065	47.397	1.396	.169	0.095	0.061	375.459	1.562	.119
GROUP2	0.076	0.080	49.309	0.951	.346	0.208	0.088	466.432	2.364	.018
PROP_RATE	–0.012	0.046	43.794	–0.259	.797	–0.216	0.022	741.370	–9.961	.000
POA1	0.089	0.011	50.378	7.749	.000	0.113	0.008	49.347	13.574	.000
POA2	–0.100	0.010	50.384	–9.621	.000	–0.091	0.009	50.148	–10.518	.000
GENDER	0.090	0.018	52.545	4.936	.000	0.049	0.016	50.858	3.137	.003
GROUP1:PROP_RATE	–0.077	0.091	39.161	–0.852	.399	0.044	0.038	748.898	1.159	.247
GROUP2:PROP_RATE	0.036	0.110	41.163	0.325	.747	–0.087	0.061	738.990	–1.430	.153

Note. Coefficient estimates, standard errors (*SE*), degrees of freedom (*df*), *t* values, and significances are reported. GROUP1 = OC vs. PD-All; GROUP2 = PD-Med vs. PD-DBS; PROP_RATE = proportional rate of speech; POA1 = bilabial vs. alveolar and velar place of articulation; POA2 = alveolar vs. velar place of articulation.

Table A5. Summary of model results for voice onset time (VOT) at slower and faster rates of speech.

	Slow					Fast				
Contrast	**Estimate**	***SE***	***df***	***t* value**	**Pr(> |*t*|)**	**Estimate**	***SE***	***df***	***t* value**	**Pr(> |*t*|)**
(INTERCEPT)	84.055	2.985	121.906	28.160	.000	58.943	2.707	406.206	21.773	.000
GROUP1	3.636	5.364	93.849	0.678	.499	5.367	4.329	335.683	1.240	.216
GROUP2	8.581	6.433	98.204	1.334	.185	−11.714	5.809	456.664	−2.017	.044
PROP_RATE	−39.181	3.158	305.770	−12.406	.000	−15.960	1.643	1351.036	−9.712	.000
VOICING	−47.520	2.384	181.758	−19.931	.000	−37.088	2.459	509.104	−15.082	.000
POA1	−16.542	1.531	68.254	−10.803	.000	−12.709	1.386	94.649	−9.168	.000
POA2	−13.722	2.326	71.306	−5.899	.000	−9.233	1.733	89.250	−5.327	.000
V.BACKNESS	−1.072	0.599	67.258	−1.790	.078	−0.257	0.518	69.948	−0.496	.621
V.HEIGHT	3.912	0.598	67.208	6.538	.000	3.046	0.518	70.033	5.878	.000
GENDER	−0.508	1.598	50.608	−0.318	.752	0.948	0.851	40.256	1.115	.272
GROUP1: PROP_RATE	−11.784	5.085	3056.203	−2.317	.021	−4.649	2.519	2102.802	−1.846	.065
GROUP2: PROP_RATE	−21.535	6.216	2936.494	−3.464	.001	5.414	3.832	1860.088	1.413	.158
GROUP1: VOICING	−9.942	4.304	153.256	−2.310	.022	−2.223	4.040	460.491	−0.550	.582
GROUP2: VOICING	−5.015	5.224	162.415	−0.960	.339	12.051	5.461	556.009	2.207	.028
PROP_RATE: VOICING	26.170	3.019	319.518	8.668	.000	12.936	1.595	931.797	8.111	.000
GROUP1: PROP_RATE: VOICING	11.734	4.961	2212.708	2.365	.018	0.758	2.470	1599.523	0.307	.759
GROUP2: PROP_RATE: VOICING	9.484	6.037	1944.222	1.571	.116	−7.992	3.736	1346.029	−2.139	.033

Note. Coefficient estimates, standard errors (*SE*), degrees of freedom (*df*), *t* values, and significances are reported. GROUP1 = OC vs. PD-All; GROUP2 = PD-Med vs. PD-DBS; PROP_RATE = proportional rate of speech; POA1 = bilabial vs. alveolar and velar place of articulation; POA2 = alveolar vs. velar place of articulation; V.BACKNESS = vowel backness; V.HEIGHT = vowel height.