

## Research Article

# Aerodynamic and Acoustic Characteristics of Nasal Airflow in Parkinson's Disease

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## ABSTRACT

**Purpose:** Velopharyngeal incompetence may contribute to speech difficulties in Parkinson's disease (PD) but has been minimally studied. This study investigated the acoustic and aerodynamic characteristics of nasal airflow in people with and without PD.

**Method:** Twenty adults diagnosed with idiopathic PD and 20 age- and sex-matched controls produced consonant–vowel speech stimuli while wearing a nasal airflow mask and oral microphone. Mean nasal airflow was measured during the 25-ms period immediately preceding consonant release (“burst airflow”) and over the central 100 ms of each vowel (“vowel airflow”). Vocal intensity (dB SPL) was also measured over the center of each vowel.

**Results:** The PD group exhibited significantly higher burst airflow than the control group (7.7 vs. 1.9 cc/s), though vowel airflow did not differ significantly between groups. Vocal intensity was positively associated with burst and vowel nasal airflow only in the PD group, despite comparable mean intensity levels between groups. Within the PD group, disease duration and speech-specific motor scores were significantly correlated with burst airflow, and voice-related quality of life was correlated with vowel airflow.

**Conclusions:** Velopharyngeal dysfunction in PD was more pronounced during rapid motor sequences (stop consonant bursts) than vowel production and showed dynamic motor deterioration under increasing vocal intensities. The intensity–airflow relationship observed in PD suggests compromised velopharyngeal closure during higher vocal demands. Measures of velopharyngeal dysfunction may be useful markers of axial motor symptom severity, which has a large impact on quality of life and prognosis in people with PD.

Parkinson's disease (PD) is the second most common neurodegenerative disease worldwide, affecting over 6 million people globally (Chaudhuri et al., 2013). The neurodegenerative effects of PD can lead to motor symptoms such as tremor, rigidity, bradykinesia, and postural instability, as well as nonmotor symptoms including mood disorders, cognitive problems, and sleep disturbances. More than 90% of individuals with PD also develop hypokinetic dysarthria, a motor

speech disorder associated with decreased range of motion in the muscles associated with speech production (Chaudhuri et al., 2024; Duffy, 2019; Ho et al., 1999). It can appear at any stage of the disease, usually worsening as the disease progresses (Atalar et al., 2023; Gison et al., 2025), and is commonly characterized by reduced loudness, monotone pitch, and limited vowel space (Darley, 1975; Kuruvilla-Dugdale & Mefferd, 2022; Ramig et al., 2008; Roland et al., 2023; Skodda et al., 2012).

The mechanisms underlying speech impairment in PD are complex and reflect impaired coordination across speech subsystems (Broadfoot et al., 2019). Prior work shows

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that—relative to typical speakers—individuals with PD often rely on elevated subglottal pressure to achieve comparable vocal intensity levels (Jiang et al., 1999) and demonstrate greater variability in transglottal (Panzone & Watts, 2020) and transnasal (Andelman-Gur et al., 2024) airflow during phonation. These airflow abnormalities may reflect velopharyngeal dysfunction, as both laryngeal and velopharyngeal systems require precise timing for efficient speech. Specifically, velopharyngeal control depends on rapid, coordinated movements of the velum and pharyngeal walls during speech production. The neural circuits governing these movements—including cortical motor areas, basal ganglia, and brainstem nuclei—overlap substantially with those affected in PD (Jankovic, 2008). Given that PD-related bradykinesia and rigidity compromise other rapid, sequential motor tasks (Berardelli et al., 2001), velopharyngeal control during speech production represents a motor subsystem likely vulnerable to PD-related dysfunction.

Velopharyngeal incompetence (VIC) is an aspect of neurodegenerative diseases that likely results from impaired neuromotor control of the levator palatine muscle and subsequent decreased closure of the velopharyngeal seal (Folkins, 1988). When the velopharyngeal port is insufficiently sealed, increased leakage of air from the nasal pathways occurs (Hoodin & Gilbert, 1989b; Mazzoni et al., 2012), preventing adequate buildup of intraoral air pressure during obstruent production (Thompson & Hixon, 1979). Consequently, aerodynamic measures of nasal airflow serve as sensitive indicators of velopharyngeal functionality (Thompson & Hixon, 1979; Warren, 1967).

Impaired velopharyngeal control can result in hypernasality due to excessive nasal airflow leakage (Thompson & Hixon, 1979; Warren, 1967). Hypernasal speech, typically assessed through auditory-perceptual judgments (Hoit et al., 1994; Kuehn & Moller, 2000), can decrease speech intelligibility and naturalness while proving distracting to listeners (Baylis et al., 2015). Because the reliability of judgments can be affected by factors such as stimulus type (Bradford et al., 1964) or presence of articulatory deficits (Counihan & Cullinan, 1970), research has pivoted toward the development of more objective measures of nasality to supplement current clinical assessment. Yet, studies comparing perceptual hypernasality measures with acoustic measures of energy (e.g., nasalance; Fletcher & Daly, 1976) have yielded mixed results. For instance, Novotný et al. (2016) showed a notable discrepancy between perceptual and acoustic measures of hypernasality in PD: Perceptual analyses revealed nearly 3 times higher occurrence rates in people with PD compared to controls (65% vs. 22%), whereas acoustic analyses showed more comparable rates between groups (27% vs. 19%). Other investigations have demonstrated significant relationships between perceptions of hypernasality and acoustic

estimates of nasalance among those with PD (Chenery et al., 1988; Theodoros et al., 1995).

Although hypernasality may not be the most prominent perceptual feature of PD speech, velopharyngeal dysfunction could contribute to reduced speech intelligibility and communicative effectiveness in this population. These divergent findings highlight the need for a more granular analysis of speech-related nasal airflow patterns, which can provide a clearer picture of the underlying mechanisms of VIC in PD. Aerodynamic measures of nasal airflow directly quantify the physiological processes underlying velopharyngeal closure during speech production (Thompson & Hixon, 1979; Warren, 1967), potentially offering more sensitive detection of dysfunction than perceptual or acoustic approaches alone. This approach may reveal subclinical velopharyngeal impairments and provide insights into how factors such as phonetic context or disease severity influence nasal airflow patterns in individuals with PD.

Several factors are known to influence nasal airflow patterns during speech production. Phonetic context plays a crucial role, with place of articulation affecting velopharyngeal opening and magnitude. In typical speakers, bilabial consonants (e.g., /p/, /b/) result in higher nasal airflow compared to alveolar (e.g., /t/, /d/) and velar (e.g., /k/, /g/) consonants (Dotevall et al., 2002; Emanuel & Counihan, 1970). Vowel context further modulates these patterns: In consonant–vowel (CV) sequences, consonants paired with low vowels (i.e., those produced with a lowered tongue position, such as /i/) generate higher nasal airflow than those with high vowels (i.e., those produced with an elevated tongue position; Emanuel & Counihan, 1970). These effects likely reflect biomechanical constraints on velopharyngeal closure related to articulatory positioning.

In individuals with PD, disease-related motor control deficits likely compound these typical patterns. The bradykinesia and rigidity symptoms that are characteristic of PD affect velopharyngeal movements (Hammer et al., 2011; Hoodin & Gilbert, 1989a) just as they affect other speech subsystems (Jankovic, 2008). One study found that individuals with moderate-to-severe PD demonstrated significantly higher nasal airflow rates compared to those with mild symptoms and those without PD (Hoodin & Gilbert, 1989a), suggesting progressive deterioration of velopharyngeal control with advancing disease. However, research specifically examining nasal airflow characteristics in PD remains limited. The single prior study analyzing nasal airflow in those with PD and controls (Hoodin & Gilbert, 1989a) used only alveolar consonants with the vowel /i/, preventing more nuanced analysis of how articulation points and vowel heights (i.e., factors known to affect nasal airflow) interact with

PD-related articulatory impairments. No prior studies have examined nasal airflow during a range of consonant and vowel combinations to elucidate how motor speech articulatory demands and PD interact to impact velopharyngeal physiology.

The role of age in velopharyngeal function remains controversial. Some research suggests decreased functionality in older adults (Hutchinson et al., 1978), whereas other studies show no age-related decline (Hoit et al., 1994; Zajac & Mayo, 1996). A recent systematic review suggests that age significantly influences acoustic nasalance measures, with moderate effect sizes observed between adults and younger age groups (Pereira et al., 2025). Understanding how aging interacts with PD-related motor changes could support the development of more appropriate diagnostic criteria.

In PD, the relationship between vocal intensity and velopharyngeal function may be particularly important to optimize therapeutic interventions. A leading treatment for speech deficits in PD, LSVT-LOUD, directly targets vocal intensity. This Cochrane-recommended approach (Herd et al., 2012) uses external behavioral cues to increase loudness and has been shown to yield lasting improvements for up to 2 years posttreatment (Ramig et al., 2001). The biomechanical effects of such intensity-focused interventions extend beyond laryngeal adjustments. Recent work demonstrated that louder speech in PD results in increased bilabial contact pressures, with differential effects based on manner of articulation; specifically, bilabial stop consonants (/p/, /b/) showed greater increases (47.7% and 35.7%, respectively) compared to the nasal consonant /m/ (27.4%; Searl, 2025).

Yet, little attention has been given to how these intensity-focused interventions might affect or interact with velopharyngeal function. When VIC allows excessive nasal airflow leakage, speakers with PD may need to increase respiratory efforts to achieve adequate vocal intensity levels, potentially creating a relationship between vocal intensity and nasal airflow (Thompson & Hixon, 1979; Warren, 1967). Additionally, the increased subglottal pressure needed for louder phonation may further compromise already weakened velopharyngeal closure in individuals with PD. As such, it is important to determine whether higher vocal intensities exacerbate nasal airflow leakage to inform speech therapy for PD; more advanced or personalized speech therapies could target vocal loudness while incorporating velopharyngeal considerations.

Thus, this study compared nasal airflow and acoustic measures from speakers with and without PD,

examining effects of phonetic context and age. We hypothesized the following:

- People with PD would exhibit significantly higher nasal airflow than those without PD due to impaired velopharyngeal closure.
- Bilabial stops would result in the greatest nasal airflow values, and low vowels would elicit greater nasal airflow values than high vowels.
- Vocal intensity (dB SPL) would positively correlate with nasal airflow measurements in both groups, with this relationship being stronger in the PD group due to compromised velopharyngeal closure.
- In people with PD, nasal airflow would increase with disease duration, motor severity, and diminished voice-related quality of life, potentially indicating progressive deterioration of velopharyngeal control mechanisms.

## Method

### Participants

Aerodynamic measures of nasal airflow and speech acoustics were measured from 40 participants, including a group of 20 individuals diagnosed with PD (12 men, eight women;  $M = 63.3$  years,  $SD = 6.1$  years, range: 49–71 years) and an age- and sex-matched control group consisting of 20 individuals without PD (12 men, eight women;  $M = 65.0$  years,  $SD = 5.7$  years, range: 50–75 years). All participants were fluent in English and completed written consent in compliance with the Boston University Institutional Review Board (Protocol #2625).

Participants in the PD group were diagnosed with idiopathic PD by a neurologist and were recorded while on their usual carbidopa/levodopa medication schedule to preserve typical vocal function. On average, participants were recorded 6.0 years after their PD diagnosis ( $SD = 3.7$ , range: 1–13; see Table 1). All participants filled out the Voice-Related Quality of Life (VRQOL) patient-reported instrument to provide an indication of how their voice impacts their day-to-day activities, with values ranging from 10 (“excellent”) to 30 (“fair;”  $M = 16.0$ ,  $SD = 5.0$ ). The Movement Disorder Society–Sponsored Revision of the Unified Parkinson’s Disease Rating Scale (MDS-UPDRS) Part III (speech item and total score) and Hoehn and Yahr staging were administered by a certified rater as a measure of motor symptom severity (Goetz et al., 2008). The severity of motor complications was, on average, 39.6 ( $SD = 11.5$ ) and ranged from mild to moderate (range: 15–58).

**Table 1.** Demographics for the group with Parkinson's disease.

ID	Sex	Age	YSD	MDS-UPDRS Part III Motor Score (max = 108)	H&Y Scale (max = 4)	MDS-UPDRS Part III Speech Score (max = 4)	VRQOL Score
P01	F	59	1	22	1	1	10
P02	F	60	13	35	4	4	30
P03	F	60	5	44	2	2	12
P04	F	65	6	47	3	2	12
P05	F	65	4	36	1	2	10
P06	F	61	4	15	1	0	10
P07	F	69	10	51	2	2	16
P08	F	70	10	54	2	2	11
P09	M	49	2	46	2	1	15
P10	M	56	4	35	2	1	14
P11	M	58	8	40	2	1	13
P12	M	58	2	30	2	0	15
P13	M	59	1	24	2	1	21
P14	M	60	2	47	2	2	18
P15	M	65	11	40	2	1	19
P16	M	69	6	41	2	1	17
P17	M	70	7	51	2	1	20
P18	M	70	3	36	2	1	21
P19	M	71	9	58	3	1	21
P20	M	71	11	39	2	1	15
Mean		63.3	6.0	39.6	2.1	1.4	16.0
SD		6.1	3.7	11.5	0.7	0.9	5.0

Note. YSD = years since diagnosis; MDS-UPDRS = Movement Disorder Society–Sponsored Revision of the Unified Parkinson's Disease Rating Scale; H&Y = Hoehn and Yahr; VRQOL = Voice-related Quality of Life.

## Procedure

The voiceless stop consonants /p/, /t/, /k/ were combined with the low vowel /ɑ/ and high vowel /i/ to produce six /CV/ stimuli tokens. The participants were instructed to produce six utterance strings of each stimuli token (i.e., /pɑ/, /pi/, /kɑ/, /ki/, /tɑ/, /ti/) at a comfortable pitch and loudness, with five repetitions of each token per utterance string, while wearing a nasal airflow mask and an oral microphone.

Nasal airflow was measured using a Phonatory Aerodynamic System (PAS; Model 6600; KayPentax) with a nasal airflow mask to ensure that only the airflow from the nose was captured (Hammer et al., 2011). Airflow was recorded at a 200-Hz sampling frequency after calibration, and zero setting of airflow signals was completed using a 1-L airflow calibrator syringe.

Speech acoustics were measured using a Shure headset microphone (Model SHR240; Shure) placed 7 cm from the mouth at a 45° angle from the midline. Speech samples were then recorded using *SONAR Artist* recording software (Cakewalk, Inc.). The acoustic signal was pre-amplified via an RME Quadmic II (RME Audio) and an external soundcard (UltraLite mk3 Hybrid; MOTU Audio) at 44.1 kHz.

The speech signal was also captured via an embedded microphone in the PAS, which was recorded at 22.025 kHz and up-sampled to 44.1 kHz.

During data collection, the nasal airflow mask was carefully monitored by the second author (T.S.M.) to ensure that there remained a tight seal for the entire duration of the session. If the mask shifted during the experimental session, data collection was stopped and repeated once the mask was resecured. The experimenter instructed participants to repeat any utterance strings in which mask seal integrity may have been compromised or token quality was uncertain. All valid tokens from each utterance string were retained for subsequent analysis ( $N = 1,322$ ).

## Measurements

Microphone signals from the oral microphone were first examined using Praat software (Boersma, 2001) by the first (J.V.) and second (T.S.M.) authors to identify the release of the voiceless stop consonant of each /CV/ token. If the release was not able to be clearly identified in either the time waveform or spectrogram, that /CV/ token was not transcribed ( $n = 39$ ).

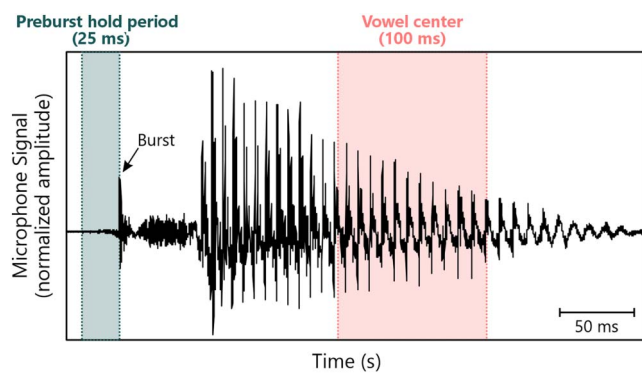
Mean nasal airflow was subsequently captured using custom scripts for MATLAB (Version 23.2; MathWorks) software. Specifically, the nasal airflow and oral microphone signals from each participant were first time-aligned using the oral headset and PAS-embedded microphone signals. The average nasal airflow was then calculated in cubic centimeters per second (cc/s) over the 25-ms hold period prior to the release of each stop consonant in the /CV/ tokens (see Figure 1). This time window was empirically chosen to ensure that burst airflow measurements for each /CV/ did not include airflow from previous /CV/ utterances. Although airflow measurements below 5–10 cc/s are often reported as 0 cc/s or excluded completely from analysis (e.g., Thompson & Hixon, 1979), these values were included in the current study because they represent meaningful variation within the naturally limited dynamic range of nasal airflow measurements during oral speech production (Hoodin & Gilbert, 1989a; Lubker & Moll, 1965; Thompson & Hixon, 1979).

Additionally, the mean nasal airflow over the center 100 ms of the vowel for each /CV/ utterance was calculated, as well as the sound pressure level (in dB SPL) at the center of the vowel. The first and last tokens were excluded from this analysis to avoid possible effects of speech onsets and offsets to the airflow measurements, respectively. This analysis resulted in two sets of mean nasal airflow values per participant: one set comprising airflow values for the 25-ms hold period prior to the burst release (“burst airflow”) and one set for the 100-ms center of the vowel (“vowel airflow”).

## Statistical Analyses

Linear mixed-effects models were fit to analyze burst and vowel nasal airflow data, with participant included as a random intercept to account for within-subject variance.

**Figure 1.** Acoustic waveform of a /pa/ token from one participant. Shaded regions indicate the two analysis windows: the preburst hold period (final 25 ms of closure) and the vowel center (central 100 ms of the vowel). The burst release is marked with an arrow.



Fixed effects included group (PD vs. control), consonant (/p/, /t/, /k/), and vowel (/a/, /i/), as well as age and sound pressure level (dB SPL) as covariates, with all two-way interactions involving group tested (Group × Consonant, Group × Vowel, Group × Age, Group × dB SPL). For significant main effects involving categorical variables, Tukey simultaneous post hoc tests were conducted to examine all pairwise comparisons. For significant Group × Covariate interactions, a simple slope analysis was conducted to examine the relationship between the continuous predictor and nasal airflow within each group, with individual slopes tested against zero and between-group differences in slopes evaluated using *t* tests. An additional multiple regression model was constructed to evaluate the association between nasal airflow, disease duration, motor severity, Hoehn and Yahr stage, VRQOL rating, and sound pressure level (dB SPL) in participants with PD.

Significance for these analyses was set a priori to  $p < .05$ , and all statistical analyses were conducted in R (Version 2025.09.0). Participant sex was not included in the statistical models based on preliminary analyses that revealed no significant Sex × Group interactions for either burst airflow or vowel airflow measures (all  $p$  values  $> .05$ ). Given the absence of differential sex effects between groups and the focus on group-specific airflow patterns, sex was excluded from subsequent analyses to maintain statistical power. Effect sizes for significant effects for the mixed-effects and multiple linear regression models were computed using partial eta-squared ( $\eta_p^2$ ).

## Results

Table 2 summarizes the mixed-effects model examining factors influencing nasal airflow across participants, and Figure 2 shows group descriptive statistics for burst and vowel airflow. The model for burst airflow revealed a significant main effect of group,  $F(1, 60.2) = 5.964$ ,  $p = .018$ , with individuals with PD demonstrating higher burst airflows than controls. Vowel context also significantly influenced burst airflow,  $F(1, 185.4) = 10.626$ ,  $p = .001$ , whereas consonant place of articulation was not significant ( $p = .057$ ). Among the covariates, vocal intensity emerged as a significant predictor,  $F(1, 216.4) = 15.491$ ,  $p < .001$ , whereas age did not reach significance,  $F(1, 36.2) = 0.211$ ,  $p = .649$ . Notably, the Group × Vocal Intensity interaction was significant,  $F(1, 216.4) = 7.620$ ,  $p = .006$ .

The model for vowel airflow showed a different pattern, with no significant main effect of group,  $F(1, 68.3) = 3.664$ ,  $p = .060$ , indicating comparable vowel airflow levels between PD and control participants after accounting for other model factors. However, vowel context remained a significant predictor,  $F(1, 189.0) = 10.221$ ,  $p = .002$ , and

**Table 2.** Linear mixed-effects analyses of burst and vowel airflow.

Model	Effect	df	F	p	$\eta_p^2$	Effect size interpretation
Burst Airflow	<b>Group</b>	<b>1</b>	<b>5.964</b>	<b>.018</b>	<b>.09</b>	<b>Medium</b>
	Consonant	2	2.909	.057	.03	Small
	<b>Vowel</b>	<b>1</b>	<b>10.626</b>	<b>.001</b>	<b>.05</b>	<b>Small</b>
	Age	1	0.211	.649	.01	Small
	<b>Vocal Intensity</b>	<b>1</b>	<b>15.491</b>	<b>&lt; .001</b>	<b>.07</b>	<b>Medium</b>
	Group × Consonant	2	0.180	.836	.00	Negligible
	Group × Vowel	1	0.326	.569	.00	Negligible
	Group × Age	1	0.591	.447	.02	Small
	<b>Group × Vocal Intensity</b>	<b>1</b>	<b>7.620</b>	<b>.006</b>	<b>.03</b>	<b>Small</b>
Vowel Airflow	Group	1	3.664	.060	.05	Small
	Consonant	2	1.415	.246	.02	Small
	<b>Vowel</b>	<b>1</b>	<b>10.221</b>	<b>.002</b>	<b>.05</b>	<b>Small</b>
	Age	1	0.741	.395	.02	Small
	<b>Vocal Intensity</b>	<b>1</b>	<b>4.128</b>	<b>.044</b>	<b>.02</b>	<b>Small</b>
	Group × Consonant	2	0.416	.660	.00	Negligible
	Group × Vowel	1	1.936	.166	.01	Small
	Group × Age	1	0.072	.790	.00	Small
	<b>Group × Vocal Intensity</b>	<b>1</b>	<b>6.761</b>	<b>.010</b>	<b>.04</b>	<b>Small</b>

Note. Effect size interpretations based on Cohen (1988). Bolded rows indicate significant effects ( $p < .05$ ).

consonant effects remained nonsignificant ( $p = .246$ ). Similar to the burst airflow model, vocal intensity was significantly associated with vowel airflow,  $F(1, 176.6) = 4.128$ ,  $p = .044$ , and the Group × Vocal Intensity interaction was also significant,  $F(1, 176.6) = 6.761$ ,  $p = .010$ , indicating differential relationships between vocal intensity and airflow across groups.

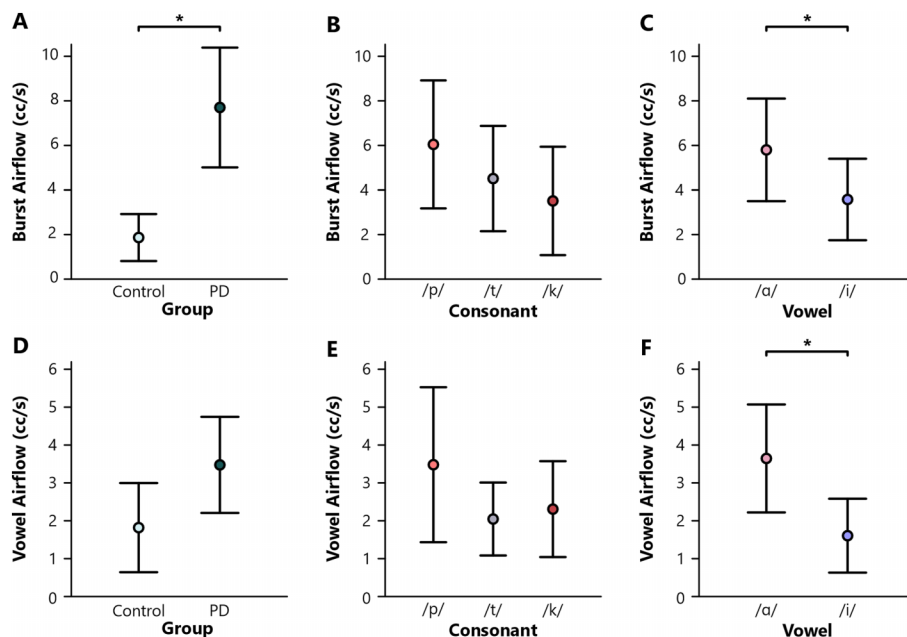
Nasal airflow values were, on average, higher in participants with PD compared to the control group for both measures. The PD group exhibited significantly higher ( $t = -2.159$ ,  $p = .038$ ) mean burst airflows ( $7.7 \pm 14.7$  cc/s) compared to the control group ( $1.9 \pm 5.8$  cc/s; see Figure 2A). Although not statistically significant, a similar pattern emerged for vowel airflow, wherein participants with PD demonstrated mean values of 3.5 cc/s ( $SD = 6.9$ ) versus 1.8 cc/s ( $SD = 6.5$ ) for controls (see Figure 2C). Average airflow patterns showed trends across consonant place of articulation that did not reach statistical significance (see Figures 2B and 2D). The bilabial /p/ elicited the highest mean values (burst:  $6.1 \pm 12.9$  cc/s; vowel:  $3.5 \pm 9.2$  cc/s), followed by the alveolar /t/ (burst:  $4.5 \pm 10.5$  cc/s; vowel:  $2.0 \pm 4.3$  cc/s), and the velar /k/ (burst:  $3.5 \pm 10.7$  cc/s; vowel:  $2.3 \pm 5.6$  cc/s). Vowel height also showed average differences across burst (see Figure 2C) and vowel (see Figure 2F) airflow measurements. The low vowel /a/ generated significantly higher burst airflow than the high vowel /i/ ( $5.8 \pm 12.6$  vs.  $3.6 \pm 10.0$  cc/s,  $t = 3.257$ ,  $p = .001$ ; see Figure 2C). Likewise, vowel airflow was significantly higher for /a/ than /i/ ( $3.6 \pm 7.7$  vs.  $1.6 \pm 5.3$  cc/s,  $t = 3.194$ ,  $p = .002$ ; see Figure 2F).

Group descriptive statistics for vocal intensity are shown in Figure 3. Both groups produced similar vocal intensity levels, averaging 65.0 dB SPL ( $SD = 3.7$ ) for participants with PD and 64.4 dB SPL ( $SD = 4.8$ ) for controls (see Figure 3A). Vocal intensity was also similar across consonants (/p/:  $64.6 \pm 4.5$  dB SPL; /t/:  $64.2 \pm 4.3$  dB SPL; /k/:  $65.3 \pm 4.1$  dB SPL; see Figure 3B) and vowels (/a/:  $64.3 \pm 4.8$  dB SPL; /i/:  $65.1 \pm 3.7$  dB SPL; see Figure 3C).

Simple slopes post hoc analyses demonstrated distinct patterns of vocal intensity effects between groups (see Figure 4). For burst airflow, the control group showed no significant relationship between dB SPL and nasal airflow (slope = 0.208,  $SE = 0.238$ ,  $p = .334$ ), whereas the PD group exhibited a strong positive relationship (slope = 1.184,  $SE = 0.267$ ,  $p < .001$ ; see Figure 4A). Similarly, for vowel airflow, controls showed no significant relationship (slope =  $-0.069$ ,  $SE = 0.158$ ,  $p = .664$ ), whereas the PD group demonstrated a significant positive relationship between vocal intensity and nasal airflow (slope = 0.56,  $SE = 0.188$ ,  $p = .003$ ; see Figure 4B). The slope differences between groups were statistically significant for both burst airflow ( $t = -2.732$ ,  $p = .007$ ) and vowel airflow ( $t = -2.562$ ,  $p = .011$ ), indicating different vocal intensity–nasal airflow relationships between individuals with and without PD.

Table 3 summarizes the multiple regression model examining the effects of PD-specific characteristics on nasal airflow measures in the group with PD. Years since

**Figure 2.** Mean burst (top row) and vowel (bottom row) nasal airflow values for (A, D) group, (B, E) consonant place of articulation, and (C, F) vowel. Error bars indicate 95% confidence intervals. \* $p < .05$ . PD = Parkinson's disease.



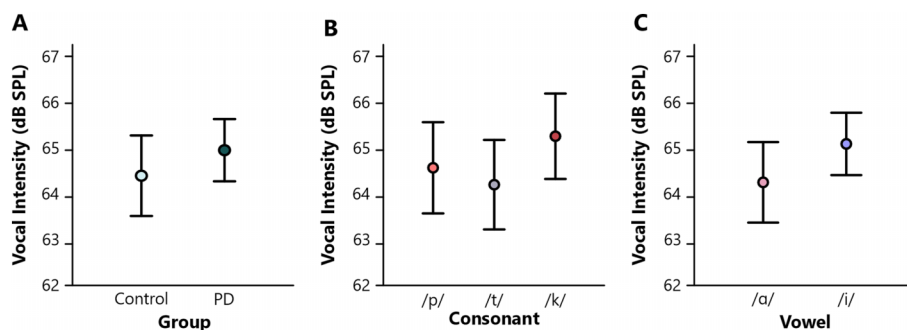
diagnosis exhibited a significant effect on burst airflow ( $F = 5.608$ ,  $p = .033$ ) but did not significantly affect vowel airflow ( $p = .070$ ). Burst airflow was significantly related to MDS-UPDRS-III speech scores ( $F = 5.565$ ,  $p = .033$ ) and vocal intensity ( $F = 19.206$ ,  $p < .001$ ). For vowel airflow, VRQOL scores showed a significant effect ( $F = 7.193$ ,  $p = .018$ ). MDS-UPDRS-III motor scores did not significantly affect either measure of nasal airflow ( $p > .05$ ).

## Discussion

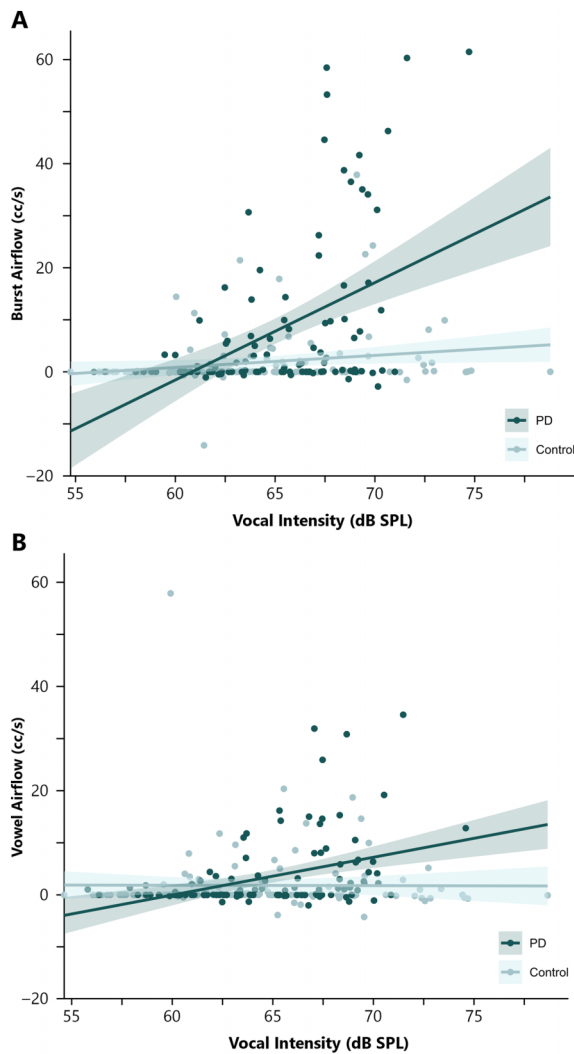
This study examined nasal airflow as an indicator of velopharyngeal function in PD to characterize the

physiological mechanisms underlying speech impairment and inform clinical assessment approaches. Individuals with PD produced significantly higher nasal airflow during stop consonant bursts, whereas vowel airflow—although elevated on average—did not differ significantly from controls. Vocal intensity levels were comparable across groups, yet nasal airflow increased with vocal intensity only in the PD group, suggesting that velopharyngeal closure deteriorates under higher phonatory demands. Disease duration and speech-specific motor symptoms were related to airflow dysfunction, indicating that velopharyngeal control declines along a speech-specific trajectory rather than following global motor deterioration.

**Figure 3.** Mean vocal intensity values (dB SPL) shown by (A) group, (B) consonant place of articulation, and (C) vowel. Error bars show 95% confidence intervals. PD = Parkinson's disease.



**Figure 4.** Relationship of vocal intensity (dB SPL) with (A) burst and (B) vowel airflow in people with Parkinson's disease (PD) and matched controls. Lines show linear regression fits with 95% confidence intervals.



### Selective Vulnerability During Rapid Motor Sequences

Burst airflow was, on average, fourfold higher in the PD group (7.7 cc/s) than the control group (1.9 cc/s). Vowel airflow also showed higher mean values in the PD group (3.5 vs. 1.8 cc/s), though this effect did not reach statistical significance. This pattern suggests that the motor control deficits associated with PD differentially affect speech tasks based on their temporal and coordinative demands.

Stop consonants require precise, rapid coordination of several articulators (Masapollo et al., 2025; Munhall et al., 1994), with movements occurring within as little as 10 ms (Kent & Moll, 1975; Löfqvist & Yoshioka, 1981).

Complete velopharyngeal closure is essential to build sufficient intraoral pressure for the burst release. In contrast, vowels are produced with continuous airflow and do not rely on high oral pressure (Thompson & Hixon, 1979; Warren, 1967). The bradykinesia and rigidity that characterize PD may impair the speed and precision of velar elevation required for consonant bursts while having less impact on the slower, more gradual movements of vowel production.

This selective vulnerability aligns with broader evidence that PD preferentially affects rapid, sequential movements across multiple motor domains (Berardelli et al., 2001; Gracco & Abbs, 1988). This finding extends our understanding of hypokinetic dysarthria by demonstrating that temporal demands—not just articulatory complexity—determine which speech gestures are most compromised. Future research using higher sampling rates and kinematic measures could elucidate whether the burst airflow elevation reflects delayed velar elevation onset, reduced peak velar height, or both.

### Phonetic Context Effects

Our hypotheses regarding phonetic context were partially supported. Vowel height significantly influenced both burst and vowel airflow, with the low vowel /*a*/ generating higher airflow than the high vowel /*i*/ in both groups. This replicates prior findings in typical speakers (Emanuel & Counihan, 1970) and likely reflects biomechanical constraints, wherein lower jaw positions associated with low vowels reduce mechanical coupling efficiency for velopharyngeal closure, thereby increasing nasal airflow. No significant Group  $\times$  Vowel interaction emerged, indicating that the biomechanical relationship between jaw position and velopharyngeal closure remains intact in PD.

Contrary to our hypothesis and previous research in typical speakers (Dotevall et al., 2002; Emanuel & Counihan, 1970), however, consonant place of articulation was not significantly associated with nasal airflow. Although descriptive patterns showed the expected ordering (/p/: 6.1 cc/s, /t/: 4.5 cc/s, /k/: 3.5 cc/s), this effect remained nonsignificant. Several methodological and population factors may explain this null finding. Large within-subject variability in nasal airflow measurements may have required a larger sample to detect modest consonantal effects. Finally, the 25-ms measurement window for burst airflow—empirically chosen to avoid token overlap—may not have optimally captured the temporal dynamics of consonant-specific airflow patterns. Because different consonants may reach peak airflow at different timepoints relative to the release burst, potentially consonant-specific measurement windows may be needed to aptly detect place effects.

**Table 3.** Multiple regression analyses of burst and vowel airflow in people with Parkinson's disease.

Model	Effect	df	F	p	$\eta_p^2$	Effect size interpretation
Burst Airflow	MDS UPDRS-III Motor Score	1	1.289	.275	.08	Medium
	<b>MDS UPDRS-III Speech Score</b>	<b>1</b>	<b>5.565</b>	<b>.033</b>	<b>.28</b>	<b>Large</b>
	<b>Years Since Diagnosis</b>	<b>1</b>	<b>5.608</b>	<b>.033</b>	<b>.29</b>	<b>Large</b>
	VRQOL	1	1.292	.275	.08	Medium
	<b>Vocal Intensity</b>	<b>1</b>	<b>19.206</b>	<b>&lt; .001</b>	<b>.58</b>	<b>Large</b>
Vowel Airflow	<b>MDS UPDRS-III Motor Score</b>	<b>1</b>	<b>7.702</b>	<b>.015</b>	<b>.35</b>	<b>Small</b>
	MDS UPDRS-III Speech Score	1	1.011	.332	.07	Medium
	Years Since Diagnosis	1	3.844	.070	.22	Large
	<b>VRQOL</b>	<b>1</b>	<b>7.193</b>	<b>.018</b>	<b>.34</b>	<b>Large</b>
	Vocal Intensity	1	0.005	.946	.00	Negligible

Note. Effect size interpretations based on Cohen (1988). Bolded rows indicate significant effects ( $p < .05$ ). MDS-UPDRS = Movement Disorder Society–Sponsored Revision of the Unified Parkinson's Disease Rating Scale; VRQOL = Voice-related Quality of Life.

It is important to consider that the present study revealed nasal airflow values during stop consonant production that differ in magnitude from the only other published PD study examining this measure. The stop consonant /t/ was analyzed in both the present study and in the study by Hoodin and Gilbert (1989a), which reported a mean nasal airflow value of 27 cc/s across all speaking rates and groups (control: 9.7 cc/s, mild PD: 9 cc/s; moderate PD: 61.7 cc/s). In the current study, we observed a mean value of 4.5 cc/s across groups for /t/ (control: 2.1 cc/s, PD: 6.9 cc/s). These substantial differences most likely reflect differences in the temporal measurement window, as Hoodin and Gilbert measured peak nasal airflows during consonant production, whereas we measured mean airflow during the 25-ms period immediately preceding burst release. Additional methodological variations—such as instrumentation (Fleisch pneumotachometer vs. KayPentax Phonatory Aerodynamic System Model 6600), speech tasks (syllable repetitions at controlled rates vs. /CV/ tokens at comfortable pitch and loudness), and participant characteristics—may also contribute to the observed discrepancies. Standardized measurement protocols are needed to enable meaningful comparisons of velopharyngeal function across PD studies.

### **Disease Progression and Speech-Specific Motor Decline**

Age was not significantly associated with nasal airflow differences between groups. Within the PD group, however, disease duration was significantly associated with burst airflow, suggesting progressive disease-related deterioration of velopharyngeal control. If age-related structural changes to the velopharyngeal anatomy or muscle properties (i.e., sarcopenia) drove the elevation in nasal airflow, age should be associated with airflow across both groups. Instead, the disease-specific relationship between duration

and airflow suggests progressive deterioration of neural control circuits governing velopharyngeal function.

It is also important to note that MDS-UPDRS (Part III) speech scores were significantly related to burst airflow, whereas MDS-UPDRS (Part III) total motor scores were not. This divergence suggests that velopharyngeal dysfunction may align more closely with speech-specific motor impairment than with global motor severity. The MDS-UPDRS motor scale encompasses multiple domains including axial, facial, and limb functions yet may lack sensitivity to detect subtle bulbar changes that directly impact speech aerodynamics. It is worth noting that the Part III speech item has limitations as a single clinician-rated measure that was not designed for validity when used in isolation. Nevertheless, burst airflow may capture aspects of speech-related motor decline that progress along a trajectory somewhat distinct from generalized motor impairments.

This finding supports emerging evidence that motor deterioration in PD follows distinct trajectories across different motor subsystems rather than a uniform pattern of decline (Rusz et al., 2021; Skodda et al., 2012). From a clinical perspective, these findings highlight the need for speech-focused measures in both clinical assessment and disease monitoring. Objective markers may enhance sensitivity to speech-motor decline beyond what is captured by subjective clinical scales alone. Given the high individual variability in speech symptom onset and severity across disease stages, such measures may be particularly valuable for tracking speech-specific changes that do not follow the more linear progression typical of global motor symptoms.

An unexpected finding emerged regarding the predictors of vowel airflow versus burst airflow in PD: MDS-UPDRS (Part III) speech scores were related to burst airflow, whereas VRQOL scores were significantly related to vowel airflow. This pattern may reflect differences in what

clinicians using the MDS-UPDRS versus individuals with PD attend to during speech assessment, though it is important to note that the MDS-UPDRS (Part III) speech item is a single auditory-perceptual rating that may not capture the full complexity of speech dysfunction. Alternatively, vowel airflow dysfunction may emerge later in disease progression or reflect different underlying neural substrates. This finding suggests that clinician ratings and patient self-reports capture distinct aspects of velopharyngeal dysfunction and provide complementary information about speech motor decline.

MDS-UPDRS (Part III) speech scores were significantly related to burst airflow, whereas VRQOL scores were significantly related to vowel airflow. This dissociation may reflect both perceptual and methodological factors. Burst airflow dysfunction, which was related to clinician ratings, may be more perceptually salient during speech evaluations, as consonant production deficits may be more readily apparent to trained clinicians during brief assessments. In contrast, vowel airflow—related to patient-reported quality of life—may reflect aspects of speech production such as effortfulness or fatigue that impact daily communication but are less apparent during brief clinical assessments. Yet, methodological differences between these measures should also be considered. The MDS-UPDRS (Part III) speech item is based on a single clinician rating (0–4), whereas the VRQOL aggregates responses across multiple items (each rated 1–5), yielding a composite score with broader range and potentially greater sensitivity to subtle variations in speech function. Taken together, these findings suggest that clinician ratings and patient self-reports capture complementary aspects of speech dysfunction in PD, with different aerodynamic measures corresponding to these distinct evaluation approaches.

### ***Dynamic Motor Deterioration Under Increased Vocal Intensities***

The observed interaction between group and vocal intensity revealed that increases in sound pressure level affect velopharyngeal function differently in individuals with PD compared to controls. Whereas controls maintained stable nasal airflow across intensity levels, individuals with PD showed strong positive relationships between dB SPL and both nasal airflow measures. Notably, overall dB SPL levels were nearly identical between groups, indicating that the airflow increase in PD reflects an inability to maintain velopharyngeal closure under increasing vocal demands rather than simply producing louder speech.

This pattern suggests that the velopharyngeal system in PD can appear relatively intact during typical conversational speech but progressively fails as phonatory demands increase. Several non-mutually exclusive mechanisms may

contribute to this breakdown. One possibility is that increased vocal intensity requires elevated subglottal pressure (Stathopoulos & Sapienza, 1997), which may overwhelm already weakened velopharyngeal closure in PD. Speakers with PD rely on higher subglottal pressure to achieve vocal intensity levels comparable to controls (Jiang et al., 1999, p. 199), potentially creating excessive pressure demands on a compromised velopharyngeal system. The challenge of managing these elevated pressures while maintaining articulatory precision is evident in oral motor adjustments, as loud speech in PD necessitates greater lip contact forces, with the magnitude of increase inversely related to velopharyngeal port patency: highest for oral stops, lowest for nasals (Searl, 2025). Another factor may involve the coordination between respiratory drive, laryngeal valving, and velopharyngeal closure. Effective voice production requires precise temporal coordination of these subsystems (Hixon et al., 1976). The greater respiratory effort needed for louder phonation may exceed the capacity of the motor system for maintaining such coordination, consistent with evidence of impaired subsystem integration in PD (e.g., Broadfoot et al., 2019). Finally, compensatory strategies used to increase vocal intensity in PD, such as increased glottal resistance or laryngeal tension, may indirectly impair velopharyngeal function by altering biomechanical constraints on the vocal tract. The observation that vocal intensity was the strongest predictor of nasal airflow in the PD-specific regression model (even more so than disease duration or motor severity) underscores the central role of phonatory demands in revealing velopharyngeal dysfunction.

This dynamic pattern of VIC has important implications for understanding inconsistent findings in prior research. Previous studies comparing perceptual and acoustic measures of hypernasality in PD have yielded mixed results (Chenery et al., 1988; Novotný et al., 2016; Theodoros et al., 1995). Our findings suggest a potential explanation: Velopharyngeal function in PD deteriorates progressively as vocal intensity increases, even within the comfortable speaking range where both groups produced similar dB SPL levels. If perceptual ratings are obtained across naturalistic speaking contexts with varying intensity demands, they may detect dysfunction that acoustic or aerodynamic measures obtained at controlled, comfortable intensity levels miss.

Our finding that burst airflow was significantly elevated in the PD group while vowel airflow showed only nonsignificant elevation may further contribute to understanding perceptual-acoustic discrepancies. Stop consonant production requires complete velopharyngeal closure to build sufficient intraoral pressure, and elevated nasal airflow during these segments indicates incomplete closure. In contrast, many acoustic measures (e.g., nasalance) and prior aerodynamic studies have focused primarily on

sustained vowels or nasal consonants, which may not capture dysfunction that is more pronounced during consonant production. Perceptual assessments conducted during connected speech naturally sample both consonants and vowels across varying intensity levels, potentially explaining why perceptual judgments of hypernasality in PD sometimes exceed rates detected by acoustic or sustained vowel-based aerodynamic measures. Future work directly comparing aerodynamic, acoustic, and perceptual measures within the same speakers while systematically manipulating vocal intensity and sampling both consonant and vowel contexts could clarify how these different assessment modalities capture velopharyngeal dysfunction in PD.

### **Clinical Implications and Treatment Considerations**

These findings have implications for voice treatment approaches in PD. Intensive voice therapy programs such as LSVT-LOUD focus on increasing vocal intensity and maintaining elevated loudness during daily communication, with demonstrated improvements in vocal intensity and speech intelligibility (Bryans et al., 2021; Campbell et al., 2022; Herd et al., 2012; Ramig et al., 2001, 2008, 2018). The significant positive relationship between vocal intensity and nasal airflow observed in our PD group warrants consideration in the context of these intensity-focused interventions.

These findings should not be interpreted as contradicting the robust evidence supporting intensity-focused treatments. Rather, they suggest that the intensity–airflow relationship may reflect meaningful individual differences in speech motor control that could inform treatment planning and monitoring. The measurement of nasal airflow across natural variations in vocal intensity may serve as a sensitive marker for individual differences in velopharyngeal control in PD. Unlike perceptual measures of hypernasality, which show inconsistent relationships with objective measures (Novotný et al., 2016), direct aerodynamic measurement provides objective, continuous data. However, it requires specialized equipment not needed for perceptual assessment. The finding that nasal airflow systematically relates to vocal intensity in PD—even within the limited range of comfortable conversational speech—suggests that this relationship may reflect meaningful individual differences in speech motor control. In particular, those with strong intensity–airflow coupling during baseline assessment might benefit from modified treatment approaches or additional velopharyngeal monitoring. Prospective studies tracking individuals through intensive voice therapy could determine whether baseline intensity–airflow relationships predict treatment outcomes or emergence of hypernasality.

It is important to note that although individuals with PD demonstrated significantly elevated nasal airflow compared to controls, the absolute magnitudes (mean burst airflow: 7.7 cc/s) remained relatively small and likely below the threshold for perceptually salient hypernasality or nasal emission. This suggests that instrumental measures of velopharyngeal function may detect subclinical motor control deficits that precede the emergence of perceptually obvious speech symptoms, potentially offering a sensitive marker for early speech motor decline in PD. However, the relationship between instrumental aerodynamic measures and perceptual ratings of hypernasality in PD warrants further investigation.

### **Limitations and Future Directions**

Several limitations should be noted. First, we had incomplete speech therapy history for our PD participants (available for only 10 of 20 participants); of these, one participant had received Lee Silverman Voice Treatment (LSVT) prior to the study. Hypophonia, or reduced loudness, is a common characteristic of PD speech. The similar mean vocal intensity levels observed in this study between individuals with (65.0 dB SPL) and without (64.4 dB SPL) PD may reflect either successful therapeutic intervention in participants with therapy history, relatively mild hypophonia in our sample, or compensatory strategies employed during the study. Without complete therapy histories, however, we cannot determine whether our nasal airflow findings represent untreated PD speech patterns or speech that has been modified through intervention. Future studies should systematically document speech therapy history and consider comparing treated versus untreated PD speakers to understand how interventions such as LSVT, which focus on vocal intensity, may influence respiratory and articulatory coordination during speech production.

Second, our sample included only individuals with mild-to-moderate PD, limiting insight into how velopharyngeal dysfunction evolves across the full disease spectrum. Nasal airflow was measured only during isolated /CV/ syllables at comfortable intensity, which may not capture patterns in connected speech or under systematically varied vocal demands. Additionally, our study measured nasal airflow but did not assess oral airflow or other aerodynamic parameters, limiting our ability to determine whether elevated nasal airflow in PD reflects globally increased airflow or nasal-specific dysfunction. Future physiologic studies incorporating simultaneous oral and nasal airflow measures, along with imaging of velopharyngeal structures during speech, could confirm whether VIC is the primary source of elevated nasal airflow in this population.

Future work should track individuals longitudinally across disease stages and examine nasal airflow during naturalistic speech tasks of varying linguistic and motor complexity.

Integrating aerodynamic measures with perceptual ratings and intelligibility assessments would clarify their functional significance. Intervention studies could determine whether velopharyngeal-focused exercises mitigate intensity-related airflow increases, and neuroimaging could reveal whether dysfunction stems from impaired cortical planning, altered basal ganglia output, or compensatory motor network changes.

## Conclusions

This study demonstrates that velopharyngeal dysfunction in PD is more pronounced during rapid motor sequences and increases systematically with vocal intensity, even at comfortable loudness levels. The finding that speech-specific motor symptoms and disease duration predicted dysfunction more strongly than overall motor severity indicates that speech-motor decline follows a partially independent trajectory in PD. These findings highlight the clinical utility of aerodynamic assessment and underscore the importance of monitoring velopharyngeal function during intensity-focused voice therapy.

## Data Availability Statement

The data sets analyzed during the current study are not publicly available due to identifiable information.

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