

## Research Article

# The Impact of Expiratory Muscle Strength Training on Speech Breathing in Individuals With Parkinson's Disease: A Preliminary Study

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**Purpose:** The purpose of this study was to examine the impact of expiratory muscle strength training on speech breathing and functional speech outcomes in individuals with Parkinson's disease (PD).

**Method:** Twelve individuals with PD were seen once a week for 8 weeks: 4 pretraining (baseline) sessions followed by a 4-week training period. Posttraining data were collected at the end of the 4th week of training. Maximum expiratory pressure, an indicator of expiratory muscle strength, and lung volume at speech initiation were the primary outcome measures. Secondary outcomes included lung volume at speech termination, lung volume excursion, utterance length, and vocal intensity. Data were collected during a

spontaneous speech sample. Individual effect sizes  $> 1$  were considered significant.

**Results:** Maximum expiratory pressure increased in a majority of participants after training. Training resulted in 2 main respiratory patterns: increasing or decreasing lung volume initiation. Lung volume termination and excursion, utterance length, and vocal loudness were not consistently altered by training.

**Conclusions:** Preliminary evidence suggests that the direct physiologic intervention of the respiratory system via expiratory muscle strength training improves speech breathing in individuals with PD, with participants using more typical lung volumes for speech following treatment.

Speech breathing is a highly coordinative effort, requiring a delicate balance between passive recoil and active muscle forces in order to maintain steady and adequate pressure for speech. Individuals with Parkinson's disease (PD) experience significant respiratory physiologic changes as a result of both aging and disease that affect speech breathing, including greater than normal increases in chest wall rigidity (Sabate, Gonzalez, Ruperez, & Rodriguez, 1996; Solomon & Hixon, 1993) and decreases in respiratory muscle strength and coordination (De Bruin, De Bruin, Lees, & Pride, 1993; Hovestadt, Bogaard, Meerwaldt, van der

Meché, & Stigt, 1989). These physiologic impairments affect the balance of passive recoil and active muscle forces, often contributing to an overreliance on active muscle forces, making speech less effective and more effortful and fatiguing.

As a result of these deficits, individuals with PD demonstrate two different types of respiratory patterns: (a) initiating and terminating speech at higher than normal lung volumes (Huber & Darling, 2011) and (b) initiating and terminating speech at lower than normal lung volumes (Bunton, 2005). Both patterns increase the need to utilize active muscle forces. Initiating and terminating speech at higher than normal lung volumes increases the need to utilize active inspiratory muscle forces in order to slow the descent of the rib cage in the face of high passive recoil forces. Initiating and terminating speech at lower than normal lung volumes almost exclusively relies on active expiratory muscle forces because the expiratory muscles are the primary source of pressure generation for speech at low lung volumes. Overreliance on active muscle forces for speech is problematic for people with PD, not only because it increases the work of speech breathing but also because reductions in respiratory muscle strength and

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control as a result of PD may lead to difficulties in sustaining and modulating pressure for speech.

Developing and validating interventions designed to improve speech breathing in individuals with PD should be a priority in the field. When speech is effortful and fatiguing, individuals are less likely to seek experiences that require significant speech demands (i.e., speaking in group situations), leading to social isolation and reduced quality of life. In addition, deficits in speech breathing lead directly to many of the hallmark characteristics of speech impairment in individuals with PD, including reduced vocal intensity and short utterances. The evidence that current intervention techniques, focused on improving specific speech deficits (i.e., vocal intensity), improve speech breathing is mixed (Huber, Stathopoulos, Ramig, & Lancaster, 2003; Stathopoulos et al., 2014). In fact, focusing on specific speech deficits without directly addressing speech breathing may lead to maladaptive respiratory behavior (Sadagopan & Huber, 2007).

The expiratory muscles are a likely target for respiratory intervention because they provide a base of support for the rib cage and diaphragm, allowing for quick, forceful contractions during inspiration, and help to modulate pressure for speech. Improving expiratory muscle strength in individuals with PD has the potential to affect both speech breathing and functional speech outcomes by improving pressure generation and control. Expiratory muscle strength training (EMST) has been shown to improve cough production (Pitts et al., 2009) and swallow function (Troche et al., 2010) in individuals with PD. However, changes in speech breathing following EMST in individuals with PD have yet to be studied.

This preliminary study sought to examine the impact of EMST on (a) speech breathing (i.e., lung volume initiation, termination, and excursion) and (b) functional speech outcomes (i.e., vocal intensity and utterance length) in individuals with PD. We hypothesized that, following EMST, individuals with PD would use more efficient respiratory strategies for speech breathing and that vocal intensity and utterance length would increase.

## Method

### Participants

Twelve individuals (three women, nine men) diagnosed with idiopathic PD by a neurologist participated in this study. The age range of participants was 66–82 years ( $M = 75$  years,  $SD = 5$  years). Demographic information for the participants is provided in Table 1. All procedures were approved by the Purdue University Institutional Review Board (Protocol 1105010869).

Inclusionary criteria included the following: (a) speech characteristics indicative of speech breathing deficits (i.e., vocal intensity, short utterances, inappropriate pausing, and reduced prosody) based on the perceptual judgment of the authors, both certified speech-language pathologists; (b) no severe depression as indicated by the Beck Depression

Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961); (c) no neurological diseases except PD; (d) no hypertension; (e) no respiratory illness; (f) no head, neck, or chest surgery except for implantation of a deep brain stimulator; (g) nonsmoking; and (h) free of infections, colds, or allergies on testing days. Participants were given the Cognitive Linguistic Quick Test (Helm-Estabrooks, 2001) to assess cognitive function, but no criterion score was required to participate. Participants did not engage in speech therapy during the study. Participants demonstrated mild–moderate speech deficits based on the perceptual judgments of the authors. Participants were between Stages 1 and 3 of the Hoehn and Yahr Scale (Hoehn & Yahr, 1967).

### Research Design

Participants were seen once a week for 8 weeks: four pretraining (baseline) sessions followed by a 4-week training period. The 4-week pretraining phase was used to obtain a stable baseline level of functioning in order to determine if improvements resulted from training or from a non-training-related effect such as repeated testing or disease variability. Pretraining data collection sessions lasted approximately 1.5 hr and included the measurement of maximum expiratory pressure (MEP) and the collection of acoustic data. In addition, respiratory data were collected in Pretraining Sessions 1 and 4. These data were not collected in all pretraining sessions to reduce the travel burden on participants. Participants were required to come to the Speech Physiology Laboratory at Purdue University to participate in respiratory data collection, but could be seen in their homes otherwise. During the training period, participants were seen each week for measurement of MEP and device threshold adjustments. These sessions lasted approximately 30 min. Posttraining data were collected at the end of the fourth week of training. This session was identical to Pretraining Sessions 1 and 4. Participants were tested at similar times in their drug cycles across sessions.

### Training Protocol

Participants used a calibrated pressure threshold trainer, the EMST 150 (Aspire Products), that consisted of a mouthpiece and a one-way spring-loaded valve (Sapienza, 2008). Participants inspired deeply and then forcefully expired into the mouthpiece. When the threshold pressure was reached and maintained, the valve opened and air flowed through the trainer (Baker, Davenport, & Sapienza, 2005; Kim & Sapienza, 2005; Pitts et al., 2009; Saleem, Sapienza, & Okun, 2005; Sapienza, Davenport, & Martin, 2002; Troche et al., 2010). Participants were given the EMST 150 at the end of Pretraining Session 4. The threshold pressure of the EMST 150 was set to 75% of the participant's MEP, an indirect measure of expiratory muscle strength, or the highest level at which the participant could successfully complete the training. MEP was measured once a week during training, and the threshold level on the trainer was adjusted accordingly. The participants

**Table 1.** Participant characteristics and demographic information.

Participant	Age (years)	Time since diagnosis	CLQT composite score	Deep brain stimulation	Medications for Parkinson's disease
F01	79	4 years	WNL	No	Mirapex
M02	66	13 years	WNL	Yes	Sinemet
M03	71	11 years	WNL	No	Amantadine, Mirapex
F04	71	7 years	WNL	Yes	Carbidopa-Levodopa, Comtan, Mirapex
M05	82	8 years	Mild	No	Sinemet
M06	76	3 years	WNL	No	Carbidopa-Levodopa, Ropinirole
M07	75	8 years	WNL	No	Sinemet
M08	76	4 years	Mild	No	Requip XL
M09	69	6 years	WNL	No	Requip XL, Sinemet
M10	73	16 years	WNL	No	Carbidopa-Levodopa, Comtan, Requip XL
F11	83	6 years	WNL	No	Carbidopa-Levodopa, Requip XL
M12	74	4 years	WNL	No	Requip XL

Note. CLQT = Cognitive Linguistic Quick Test; WNL = within normal limits.

performed five sets of five breaths 5 days a week for 4 weeks (Baker et al., 2005; Pitts et al., 2009; Saleem et al., 2005; Troche et al., 2010). Participants used a training log to document their training sessions and were considered compliant if they completed at least 4 days of training per week. All participants were compliant with the training. Two participants, M03 and M06, trained for 5 weeks due to travel schedules.

### Equipment and Data Collection Procedure

MEP was obtained in accordance with the American Thoracic Society guidelines using a handheld digital pressure manometer (VacuMed Micro RPM). Participants inhaled to total lung capacity and then blew as hard and fast as possible into the digital pressure manometer while wearing nose clips. MEP was defined as the average of the three highest values that were within 5% of one another.

Acoustic data were collected using a high-quality head-mounted microphone (Shure WBH53). The mouth-to-microphone distance was a constant 6 cm. The acoustic signal was recorded via a digital audio recorder (Marantz PMD-671) at 44.1 kHz and resampled at 18 kHz with a low-pass filter at 9 kHz for antialiasing. Acoustic data were measured in Praat (Boersma & Weenink, 2010). The gain provided by the digital audio recorder was factored in when calibrating the acoustic signal for sound pressure level (SPL) measurements.

Respiratory data were collected via respiratory inductive plethysmography using Inductotracer (Ambulatory Monitoring Series 10.9000), and procedures described by Huber and colleagues (Huber, 2007; Huber, Chandrasekaran, & Wolstencroft, 2005; Huber & Darling, 2011). Rib cage (RC) movement was transduced with an elastic band placed around the RC, just under the axilla. Abdominal (AB) movement was transduced with an elastic band placed around the abdomen, over the belly button and below the last rib. Calibration maneuvers necessary to calibrate the sum signal (Huber, 2007; Huber & Darling, 2011; Huber et al., 2005) and determine vital capacity (VC; Hoit & Hixon, 1987) were performed prior to the speech tasks.

Lung volume was estimated from the sum of the RC and AB signals, calibrated against a spirometer (SP) signal (Chadha et al., 1982; Huber, 2007; Huber & Darling, 2011; Huber et al., 2005). The relative contribution of the RC and AB to lung volume was determined during two tasks: rest breathing and speech-like breathing. Participants breathed into a digital spirometer (VacuMed Universal Ventilation Meter) while wearing nose clips during these tasks. Rest breathing consisted of 1.5 min of quiet breathing. Speech-like breathing consisted of 1.5 min of the participant reading the sentence “you buy Bobby a puppy now if he wants one” silently to themselves one time per breath. Using a least squares technique (Moore–Penrose pseudoinverse function), the correction factor ( $k_1$ ) for the RC signal was determined using the following formula (Huber & Darling, 2011):

$$SP = k_1(RC) + 1(AB). \quad (1)$$

The correction factor was then used to estimate lung volume from the RC and AB signals during the speech tasks, using the following formula:

$$\text{Estimated Lung Volume} = k_1(RC) + 1(AB). \quad (2)$$

The average correction factor for the RC signal was 1.68.

To determine maximum lung capacity, participants performed at least three trials of a VC task. During the VC task, participants were asked to breathe in as much as possible and breathe out as much as possible. The correction factors previously determined were used to estimate lung volume during the VC task (Huber & Darling, 2011; Huber et al., 2005).

Each participant performed a spontaneous speech task in which they spoke about a topic of their choice for approximately 2 min. Participants were instructed to complete the task using a comfortable loudness level and pitch. A spontaneous speech task was chosen to maintain ecological validity. Huber and Darling (2011) demonstrated that

individuals with PD have difficulty coordinating language formulation with speech breathing patterns when compared with typical older adults, particularly during spontaneous speech tasks. Although a reading passage would have allowed for better control of cognitive–linguistic variables, this type of structured task would likely result in speech breathing patterns that were not representative of typical behavior.

All signals were recorded and time-locked via LabChart (ADInstruments LabChart Version 7.1.2). Sampling rates were 10 kHz for the microphone and 1 kHz for the SP and respiratory kinematic signals.

### Measurements

All measurements were taken from the spontaneous speech tasks produced in Pretraining Sessions 1 and 4 as well as the posttraining session. Spontaneous speech tasks were transcribed orthographically using Praat. Respiratory data were analyzed using algorithms written in MATLAB. Respiratory data were measured relative to end expiratory level, the rest position of the lung–thorax unit; thus, positive values indicated volumes above end expiratory level, and negative values indicated volumes below end expiratory level. End expiratory level was measured as the average of the troughs of three rest breaths prior to the initiation of the speech task. Lung volume measures were expressed as a percentage of VC (%VC), the maximum amount of air that can be exchanged by the lungs.

Lung volume initiation and termination were measured as the points in the lung volume waveform where speech started and stopped, respectively, using the time-locked acoustic signal. Lung volume excursion was defined as the amount of lung volume used during one breath and was calculated by subtracting lung volume termination from lung volume initiation.

Utterance length was defined as the number of syllables produced during one breath. Speech rate was measured as an explanatory variable for changes in utterance length. The duration of each utterance was measured in seconds from the initiation of speech to the termination of speech. Speech rate was calculated for each breath group by dividing the number of syllables by the utterance duration.

All pauses of 0.15 s or longer were identified. SPL was measured as the average intensity across words between two pauses (Stathopoulos et al., 2014) using the acoustic waveform and corresponding spectrogram displayed in TF32 (Milenkovic, 2003).

### Statistics

Outlier analyses were performed on lung volume initiation, lung volume termination, and SPL within each session (Tukey, 1977). The percentages of data discarded for each measure across sessions were as follows: lung volume initiation = 0.10%; lung volume termination = 0.20%; and SPL = 0.13%.

Three participants were randomly chosen to be re-analyzed by a second trained measurer. Inter-measurer reliability was analyzed using *t* tests with an alpha level of  $p < .05$ . There were no significant differences, indicating that the measures were reliable: lung volume initiation,  $t(1243) = -0.07$ ,  $p = .943$ , mean difference =  $-0.09$  %VC; lung volume termination,  $t(1244) = 0.03$ ,  $p = .974$ , mean difference =  $0.03$  %VC; SPL,  $t(1089) = 0.71$ ,  $p = .480$ , mean difference =  $0.16$  dB; utterance length,  $t(1244) = 0.02$ ,  $p = .983$ , mean difference =  $0.01$  syllables; utterance duration,  $t(1244) = 0.20$ ,  $p = .845$ , mean difference =  $0.02$  s.

Effect sizes were calculated to characterize the degree of change from pre- to posttraining. Effect sizes are recommended for single-subject treatment research due to inherent flaws in the use of visual analysis (i.e., high rate of Type I error) and inferential statistics (i.e., autocorrelation) with single-subject data (Beeson & Robey, 2006). The *d* statistic was calculated by dividing the change in performance (i.e., posttraining mean – pretraining mean) by the standard deviation of pretraining (Busk & Serlin, 1992). Significant posttraining changes were operationally defined as effect sizes greater than  $\pm 1.00$  (Maas, Butalla, & Farinella, 2012; Maas & Farinella, 2012). Positive effect sizes indicated an increase in the variable from pre- to posttraining, whereas negative effect sizes indicated a decrease in the variable.

### Results

Individual effect sizes for all dependent variables are presented in Table 2. For MEP, there were significant positive effect sizes for nine participants and significant negative effect sizes for three participants. EMST successfully increased expiratory muscle strength in a majority of participants. Participants who demonstrated significant negative effect sizes for MEP were excluded from further analyses due to the lack of expiratory muscle strength increases after training. Respiratory data from M10 were not measured due to severe dyskinesias that interfered with the respiratory signal, but acoustic data from M10 were included in the SPL analyses. While the majority of participants demonstrated some sort of resting tremor and/or dyskinesia, these extraneous movements were only disruptive to the data analysis for M10.

Lung volume initiation was consistently altered after training. Seven of the eight participants had significant effect sizes, but the direction of change was not consistent. Three participants had significant positive effect sizes, and four participants had significant negative effect sizes.

Lung volume termination and excursion were not consistently altered after training. Four of the eight participants had significant effect sizes for each measure (two participants who had significant positive effect sizes and two participants who had significant negative effect sizes). Lung volume termination changed in concert with lung volume initiation.

Posttraining changes in functional speech outcomes were mixed as well. For SPL, four of the nine participants (M10 included) had significant effect sizes: two with positive

**Table 2.** Effect sizes by participant.

Participant	MEP	Lung volume initiation	Lung volume termination	Lung volume excursion	Utterance length	SPL
F01	12.17 <sup>a</sup>	1.73 <sup>a</sup>	0.60	7.38 <sup>a</sup>	0.41	0.07
M02	-1.74 <sup>a</sup>	—	—	—	—	—
M03	-3.98 <sup>a</sup>	—	—	—	—	—
F04	6.12 <sup>a</sup>	-1.99 <sup>a</sup>	-18.37 <sup>a</sup>	2.33 <sup>a</sup>	7.14 <sup>a</sup>	1.87 <sup>a</sup>
M05	-3.42 <sup>a</sup>	—	—	—	—	—
M06	14.86 <sup>a</sup>	1.30 <sup>a</sup>	1.13 <sup>a</sup>	-0.94	0.77	8.79 <sup>a</sup>
M07	13.04 <sup>a</sup>	-4.29 <sup>a</sup>	-2.87 <sup>a</sup>	-0.51	0.71	-0.03
M08	19.20 <sup>a</sup>	-0.25	0.18	-21.28 <sup>a</sup>	-10.88 <sup>a</sup>	-0.43
M09	11.62 <sup>a</sup>	1.74 <sup>a</sup>	1.59 <sup>a</sup>	-0.96	10.09 <sup>a</sup>	-1.95 <sup>a</sup>
M10	6.19 <sup>a</sup>	—	—	—	—	-0.69
F11	10.96 <sup>a</sup>	-1.74 <sup>a</sup>	0.16	-3.64 <sup>a</sup>	0.42	4.85 <sup>a</sup>
M12	4.14 <sup>a</sup>	-37.44 <sup>a</sup>	-0.84	0.31	19.50 <sup>a</sup>	0.01

Note. Em dashes indicate data not available. MEP = maximum expiratory pressure; SPL = sound pressure level.

<sup>a</sup>Indicates a significant effect size.

effect sizes and two with negative effect sizes. Four of the eight participants had significant changes in utterance length: three with positive effect sizes and one with a negative effect size. Increased speech rate was not the primary mechanism for increasing utterance length as only one participant had a positive effect size for both utterance length and speech rate.

## Discussion

This preliminary study sought to examine the impact of increased expiratory muscle strength on speech breathing and functional speech outcomes. Twelve individuals with PD participated in 4 weeks of an EMST protocol, designed to increase expiratory muscle strength. Consistent with previous studies (Pitts et al., 2009; Saleem et al., 2005), the majority of participants demonstrated significantly increased expiratory muscle strength after training.

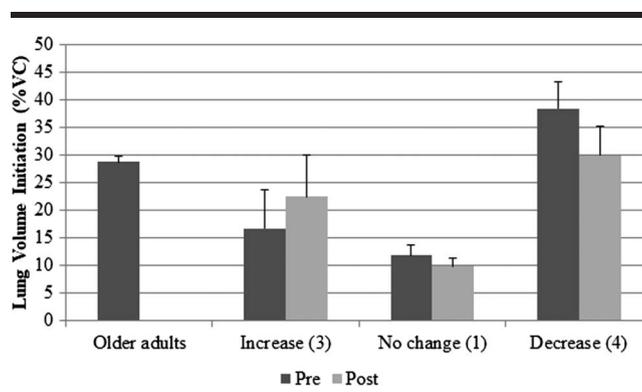
Speech breathing, particularly lung volume initiation, was altered in a majority of participants. Two subgroups emerged from the individual data: a subgroup that increased lung volume initiation and a subgroup that decreased lung volume termination after training. Regardless of whether lung volume initiation increased or decreased after training, all participants demonstrated lung volume initiations that more closely resembled previously published data from typically aging adults (Huber & Darling, 2011; see Figure 1). Thus, increased expiratory muscle strength served to normalize respiratory patterns in individuals with PD.

Alterations in lung volume initiation can be linked to increased expiratory muscle strength. Increased expiratory muscle strength was associated with increased lung volume initiation for three participants. These participants demonstrated pretraining mean lung volume initiations that were lower than previously reported data for control participants (Huber & Darling, 2011). Initiating speech at a lower than normal volume results in reliance on active expiratory forces to generate pressure for speech, increasing the work of breathing. It is possible that increased expiratory muscle

strength served to reduce abdominal volume, moving the abdominal contents in and the diaphragm to rest length (the length at which it can generate the most forceful and quick contraction; Druz & Sharp, 1981; Goldman & Mead, 1973; Hixon, Goldman, & Mead, 1973). Having the diaphragm closer to rest length could result in increased lung volume initiation because it would be less effortful to inspire to higher lung volumes. Initiating speech at higher lung volumes allowed participants to utilize passive recoil forces to a greater extent and decreased reliance on expiratory muscle forces to produce speech.

In contrast, four participants demonstrated decreased lung volume initiations after training. These participants demonstrated pretraining mean lung volume initiations that were higher than previously reported data for control participants (Huber & Darling, 2011). If these individuals with PD were unable to support speech at lower lung volumes due to decreased expiratory muscle strength, the natural compensation would be to initiate speech at higher lung

**Figure 1.** Means and standard errors for lung volume initiation. Participants are grouped according to outcomes of effect-size analysis (significant increases, decreases, or no significant change before and after training). Older adult data from Huber and Darling (2011). VC = vital capacity.



volumes to prevent the need for active expiratory forces. However, initiating speech at higher than normal lung volumes is inefficient because this requires increased effort from the inspiratory muscles to counteract high recoil forces and allow for a smooth, controlled expiration. Increased expiratory muscle strength allowed these participants to initiate speech in a range that did not rely as heavily on inspiratory muscle forces. Thus, both patterns, increasing and decreasing lung volume initiation, allowed for more efficient speech breathing.

Despite improvements in speech breathing, utterance length and vocal intensity did not consistently change. Participants in this study did not receive direct instruction regarding their speech breathing or their overall speech production. Given known deficits in self-monitoring (Zgaljardic, Borod, Foldi, & Mattis, 2003) as a result of PD and the highly habitual nature of respiratory patterns, participants may not have been able to spontaneously utilize their increased physiologic capacity after training. Pairing direct respiratory physiologic intervention with behavioral speech intervention may result in greater intervention outcomes than that of either treatment alone.

### **Study Limitations**

Though common in preliminary studies (Robey, 2004), this study did not include a control group or a sham treatment group. In order to interpret our results within a normative context, we utilized previously published data from typical older adults. Data from this group of older adults were collected in the same lab, using the same data collection techniques. In addition, it was not within the scope of this preliminary study to examine long-term treatment effects. At this time, it is unclear how long improvements in MEP will last following the cessation of treatment. Although some reduction in MEP values was observed in typical young adults following 8 weeks of detraining, MEP values were still significantly higher than the baseline (Baker et al., 2005). To our knowledge, this type of long-term follow-up has not been done in individuals with PD. Given the fact that PD is degenerative, it is likely that detraining in individuals with PD will occur more rapidly than in typical young or older adults. Therefore, it is important for future studies to examine not only long-term treatment effects following cessation of treatment but also long-term treatment effects with the addition of a maintenance program. Maintenance programs may help individuals with PD sustain treatment gains over a longer period of time.

Not all participants demonstrated improvements in MEP. Although this can be viewed as a limitation, it should be expected. No single treatment program will work for every individual. Both M02 and M05 had MEP values that were within normal limits for their age and sex before and after training (Black & Hyatt, 1969) and may not have been able to gain much more strength. Inclusionary criteria for this study were primarily based on perceptual speech characteristics related to speech breathing deficits. The fact that speech breathing deficits are not solely the result of

respiratory muscle weakness is interesting and suggests that individuals could also have impairments in their ability to appropriately scale their motor movements during speech production. Given that respiratory muscle weakness was not a primary contributor to their speech breathing deficits, M02 and M05 likely require a different type of intervention to ameliorate speech breathing deficits. Future studies should add reduced expiratory muscle strength as an inclusionary criterion to ensure that participants are receiving an intervention tailored to their physiologic needs and to increase the likelihood of successful intervention. Although M03's pretraining MEP was below normal limits for his age and sex (Black & Hyatt, 1969), his PD-related motor symptoms became noticeably worse during the course of the study and likely interfered with his ability to make gains.

Other potential limitations include the small number of participants and the limited range of speech severity. The majority of participants in this study had mild-moderate dysarthria. Individuals with more severe dysarthria may not exhibit the same training effects. Future work should include a wider range of severity and a larger number of participants to further determine which types of participants benefit the most from respiratory muscle strength training programs.

### **Conclusions**

This is the first study to demonstrate that EMST may improve speech breathing in individuals with PD. EMST resulted in speech breathing improvements, with participants moving toward lung volume initiations used by typical older adults. This study informs current clinical practice and supports the idea that the direct physiologic intervention of the respiratory system improves speech breathing. Further research is needed to fully examine the effectiveness of EMST as a method to improve speech breathing in individuals with PD.

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