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Acute Effects of Velopharyngeal Resistance Training on Aerodynamic Patterns: A Pilot Study on Healthy Individuals

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Abstract

Objective: The use of continuous positive airway pressure (CPAP) as a form of resistance training has shown some promise in strengthening velopharyngeal (VP) closure muscles in individuals with hypernasality (i.e., excessive nasal resonance perceived in speech). Systematic research on appropriate dosage, however, is largely lacking in the literature. The present pilot study explored the effects of an individual session on the speakers' VP function.

Methods: Healthy volunteers with normal VP function participated in the study. The Exercise group (n=6) underwent 10 individual CPAP sessions with varying intranasal pressure levels as overload during speech. The Control group (n=6) underwent one session with no exercise. VP measurements including aerodynamic and pressure-flow timing variables were obtained immediately before (Pre) and after (Post) each individual CPAP session.

Results: No statistically significant differences were found between groups (Exercise *vs.* Control) or across varying overload levels (5, 6, 7, 8, and 9 cmH_2O) in Pre-Post mean changes of the VP measurements. Data rather illustrated that, despite the short-term disturbance caused by resistance training, the participants were able to maintain consistency in the control of VP function, which appeared to be further augmented by exercise with sufficient overload.

Conclusion: Results provided pilot data on the acute effects of VP resistance training on healthy speakers' VP function using the range of overload typically employed in the established CPAP therapy protocol. Future clinical research may provide insights into adequate selection of exercise intensity for individual patients based on their working capacities of the VP mechanism.

Keywords: Resistance training; CPAP exercise; Hypernasality; Pressure-flow; Aerodynamic patterns

Introduction

Speech production is a complex motor behaviour involving coordinated physiologic interactions among the respiratory, laryngeal, and supralaryngeal mechanisms [1]. The velopharyngeal (VP) port, constructed by the velum (i.e., soft palate) and surrounding pharyngeal walls, is an important valving mechanism responsible for directing sound energy and airflow adequately through the oral and/or nasal cavities [2]. Inadequate control of the VP port likely results in excessive nasal resonance (i.e., hypernasality) and inadequate nasal airflow (i.e., nasal air emission), which negatively affect the speaker's overall speech/resonance quality. While physical management (e.g., surgery) is considered the first-line treatment, a behavioral therapy protocol that utilizes continuous positive airway pressure (CPAP) has shown some promise for treating hypernasality secondary to cleft palate or neurologic injury/dysfunction [3-5].

Positive pressure delivered from the CPAP device to the speaker's nasal passages has conventionally served as a means to treat individuals with obstructive sleep apnea through preventing airway collapse during sleep [6]. Unlike the conventional use, CPAP therapy for treating hypernasality instructs patients to produce speech against air pressure delivered from the CPAP device to the nasal passages. The regimented CPAP therapy protocol [4] consists of an 8-week CPAP home practice program for 6 days per week with progressively increasing pressure (4 through 8.5 cmH_2O) and session duration (10 through 24 min). Positive intranasal air pressure acts as a source of resistance (i.e., overload) against which the VP closure muscles should work in order to adequately close off the VP port for proper speech and resonance. The CPAP therapy regimen indeed has its basis in resistance training principles established in the exercise physiology literature [3,7,8]. In brief, CPAP therapy targets exercising specific speech movement patterns during speech activities (specificity) against resistance (overload) which progressively increases (progression). Accordingly, VP closure muscles are expected to undergo adaptational changes (i.e., neural adaptations, hypertrophy, and strength increases) in response to progressive overload along the course of the CPAP therapy program. In fact, a recent study [9] provided preliminary evidence for CPAP therapy-induced VP morphologic changes using magnetic resonance imaging (MRI), primarily related to thickness measures of the velum and the levator veli palatini (LVP) muscle (i.e., the primary elevator of the velum).

Despite some success in the use of CPAP therapy to treat hypernasality, systematic research on appropriate exercise dosage (e.g.,

frequency, load, and session duration) is largely lacking in the literature. Exercise intensity should be carefully determined. Specifically, exercise intensity should be great enough to serve as proper stimuli to elicit performance adaptations of the VP closure muscles; in the meantime, exercise intensity should not exceed a level that may lead to muscle exhaustion [10,11] or injury [12]. Previous physiologic investigations provided some groundwork for establishing an overload/intensity guideline [10,11,13-15]. For example, the participants in the Kuehn et al. study [13] demonstrated decreased VP activities in response to excessive pressure levels employed (e.g., above 10 cmH_2O), suggesting an upper pressure limit that should be avoided during initial therapy sessions. While physiologic responses of the VP mechanism (e.g., VP closure force or LVP muscle activity) to overload have received some research attention [10,11,13,16,17], it is unclear to what extent a single bout of VP resistance exercise would affect underlying aerodynamic processes of the VP mechanism. To date, no studies have described the acute effects of VP resistance exercise on speech aerodynamic patterns. Relative to VP closure force or LVP muscle activity measures, aerodynamic characteristics of VP function are easily accessible with a minimally invasive procedure and have been widely used in research and clinical settings. If VP resistance exercise induces a measurable change in the VP aerodynamic patterns, such information may be useful in customizing exercise dosage for individual patients and monitoring therapeutic progresses.

Therefore, the present study aimed to explore and quantify the acute effects of individual CPAP sessions with varying overload on the speakers' VP function as manifested in the aerodynamic phenomena. The range of intranasal pressure that typically serves as overload in the established CPAP therapy protocol [4] was of particular interest in the present study. It was hypothesized that the Exercise group that received individual CPAP sessions with overload would have greater magnitudes of change in VP aerodynamic patterns compared to the Control group with no exercise. It was also hypothesized that the magnitude of change in VP aerodynamic patterns following an individual CPAP session would increase as a function of increasing intranasal pressure among speakers who received exercise.

Methods

Participants

This study was approved by the university Institutional Review Board and informed consents were acquired from the participants prior to their participation. Twelve healthy female speakers (mean: 24.1 years, range: 19-32) were recruited through convenience sampling. All participants were Caucasian and spoke American English as their first language. None of the participants had reported history of speech/language/hearing problems, tonsillectomy, adenoidectomy, or current/chronic middle ear infection. The participants were divided into two groups: Exercise (n=6) *vs.* Control (n=6).

Exercise protocol

Each participant from the Exercise group underwent 10 individual CPAP sessions. During each 10 min CPAP session, participants practiced exercise speech stimuli while receiving positive air pressure through the nasal mask (Comfort Gel Blue Nasal CPAP Mask, Philips-Respironics, Murrysville, PA) connected to the CPAP device (REMstar Auto A-Flex, Philips-Respironics, Murrysville, PA) at the following pressure levels: 5, 6, 7, 8, and 9 cmH_2O. These pressure levels were approximated those used in previous studies [4,5,9,18]. Each pressure level was repeated in two different sessions (5 pressure levels × 2 sessions=10 individual sessions), and the delivery order of pressure levels was randomized within each participant. Any two consecutive CPAP sessions were at least 48 h apart to reduce any possible residual effects from a previous session.

The same list of exercise speech stimuli suggested by Kuehn et al. [4], consisting of a series of nonsense words with phonetic makeup of vowel-nasal consonant-pressure consonant-vowel (VNCV) and 12 sentences, was adopted. In order to control for exercise dose within and between participants, pre-recorded stimuli were played through the participant's headphones, and the participant was instructed to repeat after each stimulus. During a 10 min individual CPAP session, participants typically completed production of about 250 nonsense words and 24 sentences. The Control group participants partook in one session during which no particular exercise was performed.

Data collection

Each participant's VP function was assessed using a pressure-flow technique (PERCI-SAR, Microtronics, Inc., Chapel Hill, NC), immediately before (Pre) and after (Post) an individual CPAP session. The pressure-flow technique provides information on the dynamics of VP function including the VP orifice estimate based on measurements of differential intraoral and nasal air pressure and volume rate of nasal airflow [19]. Intraoral air pressure was measured by the PERCI-SAR oral pressure transducer via a plastic catheter of 2.67 mm diameter (Rüsch, Teleflex Medical, Durham, NC). A foam tip (E-A-RLINK, 3M, Maplewood, MN), directly attached to a nasal pressure catheter and connected to the PERCI-SAR nasal pressure transducer, was inserted into the participant's least patent nostril to detect nasal air pressure. A plastic tube, connected in series to a heated pneumotachometer (HR7319-FP Pneumotachograph, Microtronics, Inc., Chapel Hill, NC), was inserted into the participant's most patent nostril to detect nasal airflow. Aerodynamic data were acquired during repeated productions of "hamper." The /mp/ sequence requiring a rapid shift of the VP status, from an open to a closed one, has widely been used in previous aerodynamic studies [20-22]. The participants were instructed to produce the speech sample at their comfortable rate and loudness level.

Data analysis

Figure 1 illustrates nasal airflow and intraoral air pressure traces for a sample of "hamper." The time point and time segment notations used in the present study are consistent with previous aerodynamic studies [20,21]. Based on the middle five repetitions of "hamper," the following measurements were obtained: velopharyngeal (VP) orifice estimate (mm^2), peak nasal airflow (ml/s), peak intraoral air pressure (cmH_2O), and 5 pressure-flow timing variables (msec). The VP orifice estimate was measured during production of /p/ in "hamper." Peak nasal airflow and peak intraoral air pressure measurements were obtained during production of /m/ and /p/ in "hamper," respectively (Figure 1).

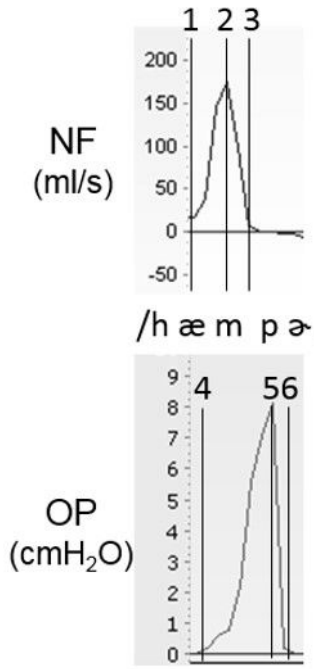

Figure 1: Nasal airflow (NF) and intraoral air pressure (OP) traces for a sample of "hamper": 1=begin flow for /m/, 2=peak flow for /m/, 3=end flow for /m/, 4=begin pressure for /p/, 5=peak pressure for /p/, and 6=end pressure for /p/.

Table 1 provides a summary of pressure-flow timing variables of interest in reference to Figure 1. Arbitrary airflow and air pressure thresholds of 0.2 cmH_2O and 20 ml/s were applied to determine the onset and offset points, while controlling for signal artifacts [22].

Reliability

Reliability was assessed by comparing two raters' measurements on a randomly selected set comprising 14% of the entire data using percent agreement rates. Two raters agreed on 100% of measurements within 0.01 mm^2 for the VP orifice estimate, 0.11 ml/s for peak nasal airflow, and 0.1 cmH_2O for intraoral air pressure. With regard to the pressure-flow timing variables, two raters agreed on their measurements within 30 msec difference at the following rates: Segment 1-3 (100%), Segment 4-6 (98%), Segment 2-3 (100%), Segment 4-5 (97%), and Segment 1-6 (99%).

Pressure-flow timing variable	Description
Segment 1-3	from begin flow to end flow
Segment 4-6	from begin pressure to end pressure
Segment 2-3	from peak flow to end flow
Segment 4-5	from begin pressure to peak pressure
Segment 1-6	from begin flow to end pressure

Table 1: Summary of pressure-flow timing variables obtained in the study in reference to Figure 1.

Statistical treatment

A series of nonparametric Mann-Whitney U tests were performed to test differences between groups (Exercise *vs.* Control) in Pre-Post changes of all VP measurements. Based on a subset of the acquired data from the Exercise group only, the effects of varying intranasal pressure on Pre-Post changes of all VP measurements were tested using a series of nonparametric Friedman tests. All statistical tests were performed at a significance level of $p<0.05$.

Results

While the magnitudes of Pre-Post mean changes appeared greater for the Control group than the Exercise group, results showed no statistically significant group differences in any of the VP measurements, as summarized in Table 2.

	Exercise (n=6)		Control (n=6)		U statistics	p
	Pre	Post	Pre	Post		
VPO (mm^2)	0.3 (0.2)	0.2 (0.1)	0.2 (0.1)	0.2 (0.1)	15.5	0.69
NF (ml/s)	144.8 (36.6)	149.2 (32.9)	154.2 (26.4)	146.6 (42.6)	12	0.34
OP (cmH_2O)	6.2 (1.2)	6.4 (1.4)	5.4 (1.0)	5.4 (0.9)	18	1
Segment 1-3	137 (32)	140 (32)	132 (17)	141 (34)	18	1
Segment 4-6	207 (38)	206 (38)	222 (40)	230 (49)	17	0.94
Segment 2-3	62 (7)	62 (10)	55 (12)	70 (22)	17	0.94
Segment 4-5	148 (41)	150 (41)	161 (33)	173 (58)	11	0.31

Segment 1-6	235 (49)	246 (64)	252 (33)	274 (72)	17	0.94

Table 2: Means (standard deviations) of velopharyngeal orifice estimate (VPO), peak nasal airflow (NF), peak intraoral pressure (OP), and pressure-flow timing variables for Exercise and Control groups before (Pre) and after (Post) individual sessions. The Mann-Whitney U test statistics with corresponding p-values are listed.

A subsequent analysis with the data only from the Exercise group further revealed that varying intranasal pressure, ranging from 5 to 9 cmH_2O, did not have statistically significant effects on Pre-Post changes in the VP measurements (Table 3). Taken together, the exercise condition or intranasal pressure overload employed in the study failed to elicit significant Pre-Post mean changes of the VP measurements.

Overload	**5 cmH_2O**	**6 cmH_2O**	**7 cmH_2O**	**8 cmH_2O**	**9 cmH_2O**	**Friedman Tests**
VPO (mm^2)	0.4 (0.5)	0.1 (0.1)	0.3 (0.2)	0.2 (0.2)	0.1 (0.1)	$\chi^2_{(4)}$=4.1; p=0.40
NF (ml/s)	149.2 (40.4)	144.6 (38.3)	148.0 (42.5)	135.6 (43.1)	157.4 (26.3)	$\chi^2_{(4)}$=4.4; p=0.36
OP (cmH_2O)	6.3 (1.1)	6.3 (1.6)	6.4 (1.1)	6.4 (1.3)	6.2 (1.4)	$\chi^2_{(4)}$=2.8; p=0.59
Segment 1-3	141 (29)	136 (39)	140 (34)	139 (30)	136 (33)	$\chi^2_{(4)}$=4.2; p=0.38
Segment 4-6	194 (36)	212 (53)	213 (33)	207 (36)	208 (40)	$\chi^2_{(4)}$=5.9; p=0.21
Segment 2-3	64 (10)	61 (12)	60 (10)	66 (12)	59 (13)	$\chi^2_{(4)}$=1.9; p=0.75
Segment 4-5	138 (41)	155 (53)	154 (39)	147 (36)	151 (42)	$\chi^2_{(4)}$= 3.8; p=0.44
Segment 1-6	225 (41)	253 (91)	238 (46)	245 (59)	240 (53)	$\chi^2_{(4)}$=6.0; p=0.20

Table 3: Means (standard deviations) of velopharyngeal orifice estimate (VPO), peak nasal airflow (NF), peak intraoral pressure (OP) and pressure-flow timing variables for the Exercise group across varying intranasal pressure levels. The Friedman test results with corresponding p-values are listed.

As depicted in Figure 2, data points from the Exercise group appeared to cluster nearby the diagonal line of no change from Pre to Post (Post=Pre) in the scatterplots. This visual inspection led to supplemental analyses, testing the strengths of the linear associations between Pre and Post VP measurements across varying intranasal pressure using Pearson product-moment correlation coefficients (Table 4).

Group	**Exercise (n=6)**					**Control (n=6)**
Overload	**5 cmH_2O**	**6 cmH_2O**	**7 cmH_2O**	**8 cmH_2O**	**9 cmH_2O**	**No pressure**
VPO	0.17	0.92**	0.90*	0.94**	0.85*	0.55
NF	0.77	0.83*	0.94**	0.87*	0.14	0.30
OP	0.95**	0.99**	0.84*	0.91*	0.97**	0.82*
Segment 1-3	0.81	0.98**	0.95**	0.84*	0.94**	0.57
Segment 4-6	0.64	0.91*	0.89*	0.87*	0.77	0.69
Segment 2-3	-0.29	0.70	0.50	0.80	0.82*	0.44
Segment 4-5	0.58	0.92**	0.93**	0.79	0.91*	0.51
Segment 1-6	0.91*	0.99**	0.85*	0.87*	0.81*	0.74

Table 4: Pearson product-moment correlation coefficients between Pre and Post velopharyngeal (VP) measurements, including velopharyngeal orifice estimate (VPO), peak nasal airflow (NF), peak intraoral pressure (OP), and pressure-flow timing variables: Exercise group with varying intranasal pressure levels and Control group with no pressure (*p<.05; **p<0.01).

For the Control group, peak intraoral pressure was the only VP measurement that had statistically significant linear relationship between Pre and Post measurements. A similar pattern of peak intraoral pressure was observed in the Exercise group with significant

linear associations between Pre and Post measurements across varying intranasal pressure levels. These results suggest that peak intraoral pressure during pressure consonant productions remain robustly consistent within a given speaker regardless of the exercise condition or varying overload. Table 4 also showed that VP timing, particularly the interval of the entire /mp/ sequence (Segment 1-6), was found to have strong linear associations between Pre and Post measurements across varying intranasal pressure levels. Considerably higher correlation coefficients in the Exercise group than in the Control group across VP measurements were the most marked group difference. Within the Exercise group, most VP measurements illustrated this strong Pre-Post correspondence with intranasal pressure from 6 through 9 cmH_2O, but not so much with 5 cmH_2O as overload.

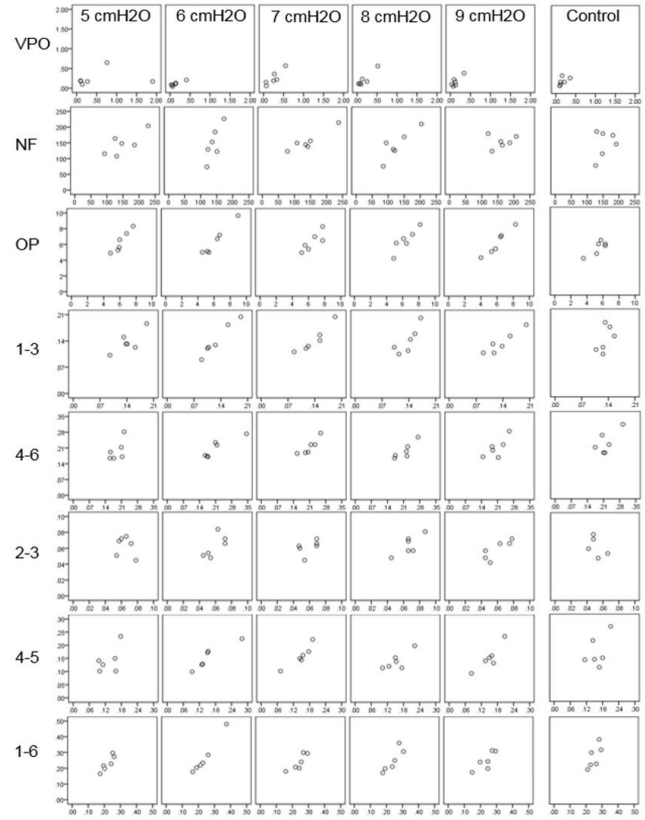

Figure 2: Scatterplots of Pre (x-axis) and Post (y-axis) measurements of velopharyngeal orifice estimate (VPO in mm_2), peak nasal airflow (NF in ml/s), peak intraoral pressure (OP in cmH_2O) and timing variables (Segments 1-3, 4-6, 2-3, 4-5, and 1-6 in s) across varying intranasal pressure levels. As a comparison, scatterplots of Pre and Post VP measurements from the Control group are presented in the right-most column.

Discussion

In limb muscle training, exercise load is generally determined in reference to a given individual's maximum strength (e.g., one-repetition maximum or 1 RM), which allows for prescribing similar training loads for people with varying strength capabilities [23]. To date, no systematic studies have been published on the appropriate dose of velopharyngeal resistance training. Difficulties in accessing the targeted VP muscle group and assessing the direct functional outcome (e.g., VP closure force or strength measure) have posed a challenge to evaluating the effects of VP resistance training. As an initial effort to determine appropriate exercise dosage, we monitored the acute effects of the individual CPAP session on the speakers' VP function as manifested in the aerodynamic characteristics using a non-invasive procedure. Contrary to a-priori hypotheses, the results showed that neither the group assignment (Exercise *vs.* Control) nor the varying intranasal pressure levels (5, 6, 7, 8 and 9 cmH_2O) had statistically significant effects on Pre-Post mean changes of the VP measurements. Data from the present study rather highlighted the speakers' high degree of consistency in the control of VP function evidenced in the aerodynamic patterns despite the short-term disturbance caused by overload. Supplemental analyses further demonstrated that the degree of linear associations between Pre and Post VP measurements for the Exercise group was much greater than for the Control group. Within the Exercise group, the strong linear correspondence between Pre and Post VP measurements was more evident with 6 cmH_2O and above than with the 5 cmH_2O as overload.

It is worthwhile to discuss the task and exercise intensity in relation to physiologic fatigue of the VP mechanism [10,11,14,15]. The Exercise group in the current study was involved in speech activities with overload. The speech task and the range of overload employed in this study resembled those employed in a typical individual CPAP session for treating hypernasality [4]. Fatigue-inducing activities with no overload in previous research involved having the participants sustain an effortful blowing task for 10 s [14] or repeat a consonant-vowel (CV) sequence over 50 times [15]. These tasks elicited declination in the LVP muscle activity, suggestive of physiologic fatigue of the VP mechanism, from both cleft and non-cleft groups with varying fatigue rates. Kuehn and Moon [11] employed a speech task with overload, during which the participants repeated a CV sequence over 100 times against intranasal air pressure at 0, 5, 15, 25, and 35 cmH_2O. Their findings showed that VP fatigability increased as a function of increasing intranasal pressure as overload in individuals without a history of cleft palate. The declining pattern of VP closure force was evidenced in 7 out of 10 participants with overload of 5 cmH_2O and 15 cmH_2O. Thus, it is reasonable to assume a certain level of physiologic fatigue of the VP mechanism experienced during an individual CPAP session in the Exercise group. Indeed, the participants of the Exercise group verbally expressed their perceived effort level, particularly after completion of CPAP sessions with upper pressure levels (e.g., 8 or 9 cmH_2O), which informally ensured that intranasal air pressure overload served as sufficient stimuli. That being said, non-significant mean changes from Pre to Post in VP measurements may represent the highly resilient nature of the VP mechanism in response to short-term disturbance or physiologic fatigue presumably induced by the range of overload that is typically prescribed in CPAP therapy.

Resilience may further relate to the participant characteristics. As VP closure for speech requires low-level muscular effort in individuals with normal VP function [11,23], the range of overload (5 through 9 cmH_2O) might have been effectively tolerated within the participants' working capacity of the VP mechanism, without necessarily altering the aerodynamic patterns of VP function. Individuals with repaired cleft palate have been reported to have higher fatigability and greater VP closure force required for speech than individuals without cleft palate [10,14,15]. Thus, special caution should be exercised when prescribing exercise intensity for speakers with a history of cleft palate; that is, exercise intensity should be large enough to elicit performance adaptations but should be within the speaker's intrinsically limited

working capacity of the VP mechanism. Despite physiologic evidence suggesting high fatigability of the VP mechanism among speakers with cleft palate, little is understood regarding the effects of induced physiologic fatigue on aerodynamic or acoustic characteristics representing VP dynamics. Future clinical research examining the extent to which an individual CPAP therapy session with overload imposes physiologic fatigue onto the VP mechanism and subsequent aerodynamic and acoustic pattern changes is warranted.

Supplemental analyses showed consistently strong Pre-Post linear dependence for peak intraoral pressure in both groups and across varying pressure levels. Limited variance or stability of peak intraoral pressure during the /p/ production corroborates previous findings that highlight the propensity of intraoral pressure to remain fairly stable during consonant production in the presence of perturbation due to experimental manipulations (e.g., auditory masking or airway leaks) or secondary to VP inadequacy [24-28]. Along with peak intraoral pressure, Segment 1-6 also had consistently high correlation coefficients between Pre and Post measurements in both groups and across varying pressure levels, although the coefficient of the Control group did not reach statistical significance. This finding lends additional support to the view [22] of VP timing aspects that are stringently controlled in healthy adult individuals. Particularly, data from the present study highlight that overall /mp/ sequence duration is also highly reliable within speakers in addition to the intraoral pressure pulse duration which has been of major focus in previous studies [21,27].

One interesting finding from supplemental analyses was the strong linear correspondence between Pre and Post VP measurements, particularly within the Exercise group. In contrast, no such pattern was present in the Control group, suggesting that the strong correspondence may be attributable to some properties pertinent to Exercise. Although the source of the strong linear dependence cannot be ascertained by the current study, it is plausible that contraction of the VP closure muscles against heightened intranasal pressure might have altered the speakers' proprioceptive sensitivity and subsequent motoric control of the VP region. Further supporting this hypothesis is the presence of muscle spindles, a type of mechanosensory receptors responsible for proprioceptive sensation, in some VP muscles [29,30]. Unfortunately, neurophysiologic underpinnings of CPAP therapy-induced adaptations remain largely unknown, which should be systematically explored in future investigations.

Given the differences in fatigability between individuals with and without adequate VP function, the extent of aerodynamic manifestations observed within an individual CPAP session may also vary depending on the speaker' VP status. No comparative clinical data on the acute effects of individual CPAP sessions on VP aerodynamic patterns are yet available. From a clinical perspective, such data may provide important insights into adequate selection of exercise intensity for individual patients based on their working capacities of the VP mechanism. To that end, the results from the present study may serve as useful references for future clinical studies to characterize VP aerodynamic adjustments in response to an individual VP resistance exercise session.

Limitations

One of the major limitations of this study relates to relatively low-level overload employed, which might not have taxed VP closure muscles enough to elicit appreciable Pre-Post changes in the aerodynamic patterns of healthy individuals. It is also possible that an individual session-induced VP closure force or LVP muscle activity changes might have not been reflected in the VP aerodynamic patterns. The lack of a direct VP strength measure remains a clear limitation of the present study. Designing and developing a VP closure force measure, similar to the one used in previous research [31] are underway in our laboratory.

Conclusion

Results from this pilot study showed that individual CPAP sessions with varying positive intranasal pressure did not elicit significant changes in the healthy speakers' VP aerodynamic patterns. Our data rather illustrated the healthy speakers' high degree of consistency in the control of VP function, which appeared to be further augmented by individual CPAP sessions with sufficient overload. Future clinical research identifying the acute and long-term effects of CPAP therapy and corresponding neurophysiologic adaptational changes is necessary to have a better understanding on to what extent VP functional capacities can be altered through behavioural intervention program.

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