Research Article

The Effect of Exercise on Respiratory Resistance in Athletes With and Without Paradoxical Vocal Fold Motion Disorder

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Purpose: An investigational, portable instrument was used to assess inspiratory (R_i) and expiratory (R_e) resistances during resting tidal breathing (RTB), postexercise breathing (PEB), and recovery breathing (RB) in athletes with and without paradoxical vocal fold motion disorder (PVFMD).

Method: Prospective, controlled, repeated measures within-subject and between-groups design. Twenty-four teenage female athletes, 12 with and 12 without PVFMD, breathed into the Airflow Perturbation Device for baseline measures of respiratory resistance and for two successive 1-min trials after treadmill running for up to 12 min. Exercise duration and dyspnea ratings were collected and compared across groups.

Results: Athletes with PVFMD had lower than control R_i and R_e values during RTB that significantly increased at

PEB and decreased during RB. Control athletes' R_e decreased significantly from RTB to PEB but not from PEB to RB, whereas R_i did not change from RTB to PEB but decreased from PEB to RB. Athletes without PVFMD ran longer, providing lower dyspnea ratings.

Conclusion: Immediately following exercise, athletes with PVFMD experienced increased respiratory resistance that affected their exercise performance. The difference in resting respiratory resistances between groups is intriguing and could point to anatomical differences or neural adaptation in teenagers with PVFMD. The Airflow Perturbation Device appears to be a clinically feasible tool that can provide insight into PVFMD and objective data for tracking treatment progress.

In individuals with exercise-induced paradoxical vocal fold motion disorder (PVFMD), breathing is abnormally impeded at the level of the larynx during exercise. This leads to dyspnea and the reduced ability to continue strenuous activity. The label *PVFM* or *PVFMD* has largely replaced the more clinically common term *vocal cord dysfunction* in the literature and is used to describe narrowing of the laryngeal airway particularly during the inspiratory phase of breathing. Various mechanisms have been implicated for this disorder, the most common and

most literal of which is inappropriate adduction of the vocal folds (Andrianopoulos, Gallivan, & Gallivan, 2000; Mathers-Schmidt, 2001; Sandage & Zelazny, 2004). Another documented mechanism is prolapse of laryngeal structures into the airway, also known as *exercise-induced laryngomalacia* (Christopher & Morris, 2010; Heinle, Linton, & Chidekel, 2003; Hicks, Brugman, & Katial, 2008). In this article, the term *PVFMD* will be used regardless of mechanism to be consistent with current literature, although the results will be interpreted with consideration to specific mechanism.

During strenuous aerobic exercise, normal breathing involves high airflow with low airway resistance, resulting in efficient air exchange with minimized energy expenditure (England & Bartlett, 1982). This is accomplished in part through increased respiratory rate and greater air-volume exchange (England & Bartlett, 1982). Normal vocal fold posturing during exercise involves a wider glottis during both inspiration and expiration in comparison with its resting position (England & Bartlett, 1982; Hurbis & Schild, 1991).

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To confirm PVFMD, an examiner typically views the larynx using laryngoscopy once the symptomatic behavior is triggered (Balkissoon & Kenn, 2012; Christopher et al., 1983; Røksund et al., 2009; Tervonen et al., 2009). In previous publications, we demonstrated construct validity (Gallena et al., 2013) and test–retest reliability (Gallena, Solomon, Johnson, Vossoughi, & Tian, 2014) of a less intrusive method to infer laryngeal airway patency. This method involves a portable, handheld instrument, the Air-flow Perturbation Device (APD), that uses aerodynamic assessment to determine respiratory resistance (R_r) over time. The measurement incorporates resistances present throughout the lower and upper airways as well as resistances offered by structures that support the airways—namely, the chest wall (Johnson & Sahota, 2004)—and reports separate results for inspiratory (R_i) and expiratory (R_e) resistances as well as the average of both phases (R_r).

The larynx accounts for a significant part of total airway resistance during both rest and exercise (Ferris, Mead, & Opie, 1963; Røksund et al., 2009), and the APD has been used to noninvasively detect changes in laryngeal airway resistance (Gallena et al., 2013). Even during normal resting tidal breathing (RTB), the vocal folds abduct for inspiration and adduct slightly for expiration (Balkissoon, 2007; Beaty, Wilson, & Smith, 1999; England & Bartlett, 1982), which are changes that have been supported by APD measures (Johnson et al., 2007; Silverman, Johnson, Scott, & Koh, 2005). During exercise, airflow velocity increases, causing increased R_r (Ferris et al., 1963) that is mitigated in part by abducting the vocal folds, dilating the supraglottic area, and flattening the epiglottis during both inspiration and expiration (Beaty et al., 1999; England & Bartlett, 1982; Hurbis & Schild, 1991).

Changes in R_r with exercise have been investigated with the APD in two previous studies, both involving healthy adults (ages 18–40 years) and continuous monitoring with the APD for at least a portion of the studies (Johnson, Chapain, Slaughter, Gallena, & Vossoughi, 2013; Silverman et al., 2005). Immediately following moderate exercise, R_i and R_e decreased from baseline levels in one study (Silverman et al., 2005) and did not change significantly in the other (Johnson et al., 2013), but both studies demonstrated decreases in R_i and R_e after 30–45 s of rest that continued for 1–2 min into the recovery period. Results from these studies are not directly comparable because Silverman et al. (2005) only analyzed pre- and postexercise trials that were matched for airflow rates, whereas Johnson et al. (2013) allowed airflow to vary naturally with exercise.

Although continuous monitoring of R_r with the APD is informative for instantaneous changes, it may be impractical for clinical applications. The most common demographic group for exercise-induced PVFMD comprises teenage girls who participate in competitive sports (Hicks et al., 2008; Landwehr, Wood, Blager, & Milgrom, 1996; Mathers-Schmidt, 2001). Previously, we demonstrated that 1-min trials with the APD are feasible and reliable before and after exercise (Gallena et al., 2014). Twenty-four female teenage athletes, 12 with and 12 without PVFMD,

ran on a treadmill according to a challenging, well-controlled exercise protocol. APD 1-min trials were scheduled three times before and three times after exercise. In addition, the athletes without PVFMD repeated the study over three sessions. APD measures were stable during RTB for both groups within a session, although control participants' R_i during RTB decreased significantly but slightly (approximately 5%) across sessions. Following exercise, the APD measures from the control group were examined for reliability. These were highly correlated across separate sessions when matched for recovery time. Thus, the APD appears to be a reliable instrument for assessing respiratory resistance under resting as well as exercise conditions. That report did not address exercise effects for athletes with PVFMD or between-group differences.

Once confirming that the APD is a feasible tool for providing quantitative measures of respiratory resistance during RTB in young athletes diagnosed with exercise-induced PVFMD, the next step along this line of inquiry is to use the APD to examine exercise-related changes in R_i and R_e . This study investigated the direction and extent of changes in R_i and R_e in the same 24 female teenage athletes (12 with and 12 without PVFMD) studied previously (Gallena et al., 2014). In addition, the study examined R_i and R_e for changes over a 3-min postexercise recovery period within and between the two groups of athletes. Finally, ratings of dyspnea and duration of exercise were compared between athletes with PVFMD and control athletes.

Based on previous literature and clinical experience, athletes with PVFMD were expected to exercise for shorter durations and have higher dyspnea ratings than athletes without PVFMD. Healthy athletes without PVFMD were predicted to experience a decrease in APD-determined R_i and R_e immediately following exercise, whereas athletes with PVFMD were expected to experience an increase in R_i and possibly R_e . By the second and third minutes after exercise, both groups were predicted to demonstrate a progressive recovery of R_i and R_e toward RTB baseline levels. The two matched groups were expected to have comparable R_i and R_e values during RTB because athletes with PVFMD do not report dyspnea during RTB. Successful execution of this protocol will also demonstrate the feasibility of the APD as a clinically useful instrument for quantitatively assessing respiratory resistance associated with exercise-induced PVFMD in teenage girls.

Method

Participants

The experimental and control groups each comprised 12 female athletes who have been described previously (Gallena et al., 2014). The groups did not differ significantly in age ($M = 14.5$ years vs. 14.8 years), height ($M = 162.8$ cm vs. 160.5 cm), or weight ($M = 54.5$ kg vs. 52.0 kg). Twenty-two participants were White and non-Hispanic, and two (sisters) identified as Pacific Asian Islanders, although all races and ethnicities were eligible to participate. All athletes

were involved in aerobic sports for, at minimum, two seasons per year. The most frequently played sports were field hockey ($n = 6$), soccer ($n = 6$), and lacrosse ($n = 6$).

The participants composing the experimental group were suspected of having PVFMD by their referring pulmonologist, immunologist, or pediatrician and were referred to the Loyola Clinical Center for further management after given medical clearance to participate in the exercise challenge. The teens assented and their parents provided informed consent, following the regulations of the university institutional review board. The otolaryngologist and speech-language pathologist (SLP) at the Loyola Clinical Center together confirmed the diagnosis of PVFMD via a postexercise laryngeal examination documenting adductory vocal fold motion and/or prolapse of the supraglottal structures during inspiration. Candidates disqualified if they answered affirmatively to a coexisting diagnosis of asthma or current use of asthma medications.

Control athletes were recruited from area schools by contacting school personnel or the athletes and their parents directly. Athletes in the control group responded to a screening questionnaire determining that they were negative for asthma diagnosis and PVFMD symptoms and were not taking asthma medications.

Instrumentation

A commercial-grade treadmill (Nautilus NTR700) that displayed speed, incline, and time was used for the exercise challenges. It passed a calibration check according to the manufacturer's specifications prior to each session. Each participant wore a Polar FS2C heart rate (HR) monitor (Polar Electro Inc., Lake Success, NY) throughout exercise to ensure that her heart rate did not exceed predicted maximum for her age. Prior to and throughout exercise, the participants rated their perceived dyspnea with the Modified Borg Dyspnea Scale (Borg, 1998), with anchors from 0 (*nothing at all*) to 10 (*maximal*).

The APD, described and illustrated previously (Gallena et al., 2013; Lausted & Johnson, 1999; Pan, Saltos, Smith, Johnson, & Vossoughi, 2013; Silverman & Johnson, 2005), is a light, handheld, self-calibrating unit that operates on alternating current, with a rotating wheel containing open and screened segments that introduce periodic resistances that perturb airflow. Participants wear a noseclip and place a disposable air-filter mouthpiece (Model 758EW, Respitech Medical, Inc., Lancaster, PA) coupled with the APD. During breathing, R_r is calculated in $\text{cm H}_2\text{O/l/s}$ for each perturbation on the basis of measured changes in the ratio between differential air pressure and airflow across the source of perturbation. Data were transferred via the APD's digital output port to a laptop computer (ASUS, Eee PC, ASUSTeK Computer Inc., Taipei, Taiwan) installed with custom software (Hand Held APD Real-Time Display; Lausted, 1997; Silverman, 2007). Data displayed on the computer following each trial represent the mean values across all perturbations during inspiration (R_i) and expiration (R_e) as well as across the entire trial (R_r). To

guard against instrumental threats to validity, the same APD was used throughout the study.

Laryngoscopy for the athletes suspected of having PVFMD was conducted using a 70° rigid laryngoscope (JEDMED, St. Louis, MO), connected to the JEDMED Stroboscopy system (Strobocam II, JEDMED) and recorded onto DVD for further review. The university where the research was conducted is not affiliated with a medical institution, and at the time of this study, only rigid laryngoscopy was permitted. For each athlete, an experienced SLP or otolaryngologist passed the scope transorally without topical anesthesia.

Procedure

The athletes diagnosed with PVFMD participated in a single diagnostic session that included both laryngoscopy and APD measures before and after an exercise challenge. Control athletes participated in three separate sessions, each with an exercise challenge, to establish reliability of the APD (Gallena et al., 2014). For the purposes of this group-comparison study, data were used from the first session only. Prior to data collection, all participants received training on test procedures.

Pre-Exercise Baseline

Each participant wore the HR monitor, which recorded a baseline HR. She provided a baseline rating of dyspnea using the Modified Borg Dyspnea Scale (MBDS). While seated with the instructional picture in clear view and the noseclip in place, she breathed into the APD for three trials, each separated by approximately 5 s.

Exercise Challenge

Each participant was given the opportunity to stretch and to walk on the treadmill for 1–2 min prior to exercising. As described previously (Gallena et al., 2014), the exercise challenge involved a predetermined progressive incremental ramp protocol whereby the speed or incline of the treadmill was alternately increased at 1-min increments beginning at 4 miles/hr (mph) with 0% incline and ending at 7 mph with 4% incline. This was a challenging exercise protocol that could be completed in 12 min, the maximum exercise test duration for adults advised by the American College of Sports Medicine (Thompson, Gordon, & Pescatello, 2010) and for children by the American Heart Association (Fletcher et al., 2001; Paridon et al., 2006). While running, the participant did not hold onto the treadmill handrails (Thompson et al., 2010). Treadmill speed, incline settings, and incremental adjustments in exercise progression were decided on the basis of performance and feedback from female athletes without PVFMD who piloted the treadmill exercise challenge task. HR was monitored continuously throughout the exercise period. Participants rated dyspnea at 1-min intervals and any time they sensed a notable change in breathing effort (Mahler, Mejia-Alfaro, Ward, & Baird, 2001). Each participant's tolerance dictated the duration of the exercise challenge. Athletes were

encouraged to exercise for the entire 12-min duration but were instructed that they could stop sooner if needed and that they would be stopped by the researcher if their HR exceeded their predetermined maximum. Athletes in the experimental group were instructed to run until they reported a rating of 8 on the MBDS (between 7 = *very severe* and 9 = *very, very severe*; 8 has no specific descriptor; Brugman & Simons, 1998).

Postexercise Data Collection

Exercise duration to the nearest second, HR, and MBDS rating were noted immediately after the exercise challenge. Participants quickly transferred to a chair 60 cm from the treadmill, placed the noseclip, and immediately started the first APD trial. Approximately 10 s elapsed between ceasing exercise and starting the APD trial. All 12 control athletes and five of the experimental athletes performed a total of three consecutive postexercise breathing trials. The other seven athletes with PVFMD declined to perform the third APD trial, citing anxiety with the remaining procedures. Due to these missing data, the third postexercise trial was not included in data analysis; instead, the second postexercise trial was used to indicate recovery breathing (RB).

Following the postexercise R_i trials, athletes suspected of having PVFMD immediately returned to the treadmill for several minutes of sprinting to again trigger PVFMD symptoms for purposes of laryngoscopic evidence. Athletes ran until providing an MBDS rating of 8 or until the SLP or otolaryngologist heard stridor, at which time they were assisted to the examination chair, the rigid laryngoscope was placed, and evidence of PVFMD during several breathing cycles was viewed and recorded.

Data Analysis

All analyses were conducted using SPSS, Version 21 (IBM Corporation, Armonk, NY). A repeated measures multivariate analysis of variance (RM-MANOVA) with group (with and without PVFMD) as the between-subjects factor and RTB as the within-subject factor was conducted to determine whether the three RTB trials could be collapsed for subsequent analyses. To examine the effects of exercise on R_i and R_e , another RM-MANOVA was used with group as the between-subjects factor and condition (RTB, PEB, and RB) as the within-subjects factor. Data distributions met MANOVA test assumptions according to Box's M plot; variables that did not meet the assumption for sphericity necessitated the use of the Greenhouse–Geisser statistic. Post hoc testing used ANOVAs and independent-samples t tests for between-groups differences and related-samples t tests for within-group differences. An independent-samples t test examined between-groups differences for exercise duration, and the nonparametric test Mann-Whitney U, tested between-groups differences in dyspnea ratings. The α levels for significance were .05 for each MANOVA, adjusted to .025 for ANOVAs with two dependent variables and .017 for t tests with three contrasts.

Results

Figure 1 illustrates the means for R_i and R_e for each participant group (PVFMD and control) across the study conditions: three 1-min RTB trials, one trial following the exercise challenge (PEB), and one trial during the recovery process (RB). Table 1 lists RM-MANOVA results for the analysis of RTB data for the two groups. Pre-exercise RTB measures of R_i and R_e differed significantly between the participant groups, $F(2, 21) = 7.18, p = .004$, such that resistance values were greater for the control group. Consistency across the three RTB trials for each group and lack of a trial-by-group interaction are apparent from Figure 1 and were supported by RM-MANOVA (see Table 1). Given these results, the R_i and R_e data for each group were averaged across RTB trials to provide a single pre-exercise value for comparison with the PEB and RB conditions.

Table 2 lists summary statistics for the variables and conditions included in the RM-MANOVA presented in Table 3 for group (PVFMD and control) and condition (RTB, PEB, RB), as well as between-groups MANOVA results. Resistance values for the two groups did not differ when data were collapsed across condition, $F(2, 21) = 1.95, p = .168$ (see Table 3); therefore, separate analyses for R_i and R_e were not warranted. There was a significant main effect for condition when collapsed for group, $F(4, 19) = 15.92, p < .001$, with R_i contributing significantly to the difference, $F(2, 44) = 28.93, p < .001$. A significant Group $\times$ Condition interaction was detected when both resistance variables were included in the analysis, $F(4, 19) = 16.51, p < .001$, as well as when R_i and R_e were analyzed separately, $F(2, 44) = 46.14, p < .001$, and $F(1.3, 31.9) = 18.07, p < .001$, respectively. The interaction was characterized by an overall increase in respiratory resistances from pre- to postexercise in the PVFMD group and a decrease in respiratory resistances in the control group. Examination of Figure 1 and post hoc analysis of paired conditions (see Table 4) revealed that R_i increased substantially and significantly from RTB to PEB for the PVFMD group, $t(11) = -12.44, p < .001$, but that R_i did not change significantly with exercise for the control group. The increase in R_e for the PVFMD group and decrease in R_e for the control group after exercise both met criteria for statistical significance, $t(11) = -337, p = .006$, and $t(11) = 3.46, p = .005$, respectively.

APD trials taken during the first 2 min after exercise provided an indication of respiratory resistance at near-maximal exertion and during recovery from exercise. R_i decreased significantly from PEB to RB for both groups, PVFMD: $t(11) = 5.85, p < .001$; control: $t(11) = 3.01, p = .012$, whereas R_e did not differ significantly between PEB and RB for either group. R_i did not return to pre-exercise resting levels (RTB) after the second minute of recovery for either group, as indicated by statistically significant differences between RTB and RB, PVFMD: $t(11) = -5.44, p < .001$; control: $t(11) = 4.04, p = .002$. R_e at RB tended to remain higher than baseline for the PVFMD group and continued to differ from baseline for the control group, PVFMD: $t(11) = -2.40, p = .035$; control: $t(11) = 3.90, p = .002$.

Figure 1. Mean inspiratory (circles) and expiratory (squares) resistances for 12 athletes with (black) and 12 without (gray) paradoxical vocal fold motion disorder (PVFMD) for resting tidal breathing (RTB), postexercise breathing (PEB), and after 1 min of rest (recovery breathing, RB).

There were no statistically significant between-groups differences for RB for either R_i or R_e .

Exercise duration and MBDS ratings for each participant according to group are plotted in Figure 2. Data were ranked on the basis of exercise duration and secondarily on MBDS ratings for the purpose of illustration. Independent-samples t test (one-tailed) revealed a statistically significant between-groups difference, $t(22) = 3.502$, $p = .001$, for exercise duration. Athletes with PVFMD exercised from 5–10.5 min and athletes without PVFMD exercised from 9–12 min; on average, the PVFMD group discontinued the exercise challenge test 1.9 min sooner than the control athletes. Dyspnea ratings differed significantly between groups, Mann–Whitney $U = 5.00$, $z = -3.93$, $p < .001$. MBDS ratings for athletes without PVFMD ranged from 0.5 to 6 and averaged 3.6 (*moderate*). Athletes with PVFMD rated dyspnea from 5 to 9, with an average of 7.4 (*severe*), at the time they discontinued the exercise challenge. Correlations

between exercise duration and exertion ratings were weak and nonsignificant for the control group, $r_s = -.217$, $p = .498$, and the PVFMD group, $r_s = -.391$, $p = .209$.

Discussion

This study investigated respiratory resistance as determined by the APD, exercise duration, and ratings of dyspnea in athletes with and without PVFMD over repeated trials before and after a rigorous, customized exercise challenge. The strenuousness of the exercise challenge was validated by the results for the perception of dyspnea and its correspondence to running duration. On average, the athletes with PVFMD stopped running approximately 1.5 min sooner than the control athletes and rated their dyspnea twice as severely (*severe* for athletes with PVFMD vs. *moderate* for control athletes). This result indicates that the

Table 1. Results from repeated measures multivariate analysis of variance (RM-MANOVA) for inspiratory and expiratory resistance (R_i and R_e , respectively) for two groups of athletes (with and without paradoxical vocal fold motion disorder) for resting tidal breathing across three trials.

Variable	df	F	η^2	p	Power
Group	2, 21	7.18	.406	.004*	.893
R_i	1, 22	15.04	.406	.001*	.959
R_e	1, 22	10.37	.320	.004*	.868
Trial	4, 19	8.98	.159	.484	.232
Trial $\times$ Group	4, 19	1.44	.670	.259	.362

* $p < .05$ for RM-MANOVA results and $p < .025$ for RM-ANOVA results.

Table 2. Means (SD) for inspiratory and expiratory resistances (R_i and R_e , in cm H₂O/L/s) during resting tidal breathing (RTB, averaged across three trials), postexercise breathing (PEB), and after 1 min of rest (recovery breathing, RB) for 12 athletes without (control) and 12 with paradoxical vocal fold motion disorder (PVFMD); between-groups analysis of variance results for each variable and condition.

Condition	Variable	Control	PVFMD	F(1, 22)	p
RTB	R_i	3.91 (0.60)	2.94 (0.65)	15.07	.001*
	R_e	4.39 (1.14)	3.13 (0.78)	10.37	.004*
PEB	R_i	3.70 (0.52)	4.36 (0.72)	6.65	.017*
	R_e	3.35 (0.37)	3.61 (0.68)	1.41	.248
RB	R_i	3.45 (0.35)	3.59 (0.51)	0.65	.427
	R_e	3.44 (0.47)	3.45 (0.41)	0.004	.948

* $p < .025$.

Table 3. Results from repeated measures multivariate analysis of variance (RM-MANOVA) for inspiratory and expiratory resistances (R_i and R_e , respectively) for two groups of athletes (with and without paradoxical vocal fold motion disorder) during rest, after exercise, and after 1 min of rest.

Variable	df	F	η^2	p	Power
Group	2, 21	1.95	.156	.168	.357
Condition	4, 19	15.92	.770	< .001*	1.000
R_i	2, 44	28.93	.568	< .001*	1.000
R_e^a	1.3, 31.9	3.32	.131	.063	.505
Condition $\times$ Group	4, 19	16.51	.777	< .001*	1.000
R_i	2, 44	46.14	.677	< .001*	1.000
R_e	1.3, 31.9	18.07	.451	< .001*	.997

^aGreenhouse–Geisser test statistic reported for all R_e .

* $p < .05$ for RM-MANOVA results and $p < .025$ for RM-ANOVA results.

average female athlete with PVFMD requires more breaks during exercise due to dyspnea but that the dyspnea will begin to diminish in a relatively short period of time when exercise ends. Despite careful attempts to control exercise conditions in research studies, exercise duration is necessarily shorter for athletes with PVFMD. The present study operationally defined the point at which participants would stop exercising, yet several athletes still requested stopping earlier than expected. Despite these procedural issues, the differential effects of exercise on respiratory resistance were revealed successfully with APD measures.

It was hypothesized that athletes with PVFMD would demonstrate higher R_i following exercise, but a change in R_e was less certain because most athletes report more difficulty inhaling than exhaling when symptomatic (Christopher & Morris, 2010; Divi, Hawkshaw, & Sataloff, 2008; Hicks et al., 2008). As expected, the athletes with PVFMD exhibited a marked increase in R_i and a smaller but significant increase in R_e immediately after exercise, suggesting biphasic PVFMD (Newman, Mason, & Schmalzing, 1995). The

Table 4. Post hoc within-groups related-samples t tests for significant repeated measures multivariate analysis of variance comparing inspiratory (R_i) and expiratory (R_e) resistance across resting tidal breathing (RTB), postexercise breathing (PEB), and recovery breathing (RB).

Contrasts	Control		PVFMD	
	$t(11)$	p	$t(11)$	p
R_i				
RTB and PEB	1.28	.226	−12.44	<.001*
RTB and RB	4.04	.002*	−5.44	<.001*
PEB and RB	3.01	.012*	5.85	<.001*
R_e				
RTB and PEB	3.46	.005*	−3.37	.006*
RTB and RB	3.90	.002*	−2.40	.035
PEB and RB	−0.92	.379	1.06	.312

Note. PVFMD = paradoxical vocal fold motion disorder.

* $p < .017$.

Figure 2. Modified Borg Dyspnea Scale (MBDS) Ratings (left axis, gray bars) and exercise duration (right axis, black diamonds, in min) for 12 athletes without paradoxical vocal fold motion disorder (PVFMD; control, top graph) and 12 athletes with PVFMD (bottom graph). Data sorted for decreasing exercise duration and, when tied, for increasing MBDR.

presumed primary source of the increased resistance is within the laryngeal airway (because none of the athletes had a diagnosis of lower airway disease), which is detectable by the APD (Gallena et al., 2013). These results are congruent with reports from direct observation of changes in glottal area during exercise among athletes with PVFMD (Beatty et al., 1999; Heinle et al., 2003; Røksund et al., 2009; Tervonen et al., 2009).

For control athletes, R_e decreased significantly immediately after exercise and stayed at a similar level after an additional minute of postexercise rest. R_i did not change significantly right after exercising but was significantly lower than baseline and postexercise levels during RB 1 min later. The decrease in R_e with exercise was predicted on the basis of previous evidence of a less resistant laryngeal airway after exercising by healthy athletes (England & Bartlett, 1982; Hurbis & Schild, 1991), but the delay of a postexercise reduction in R_i was not expected. Also surprising was the finding that neither R_i nor R_e returned to baseline levels after 2 min of recovery breathing. Nonetheless, these appear to be robust findings. Gallena et al. (2014) previously reported results from these same healthy athletes over three separate sessions, and the data indicate similar pre- to postexercise changes as well as the same patterns of recovery in R_i and R_e for all three exercise challenges. Overall, the data suggest that adaptation to exercise in

healthy teenage girl athletes occurs more quickly during the expiratory than inspiratory phases of breathing and that respiratory resistance remains lower than baseline for at least 2 min after exercising.

The reduction in R_i from the first to the second postexercise APD trial was substantial for the group with PVFMD but remained significantly higher than its pre-exercise level. Additional APD measures during the recovery period were collected in anticipation of a longer recovery, but these data were not analyzed due to missing trials by participants in the PVFMD group; only five athletes in this group agreed to provide a third APD trial during recovery. However, informal inspection of these data revealed that R_i returned to baseline levels (within 1 SD of the mean) for each of these five athletes. All 12 athletes in the control group provided a third APD trial during recovery (R_i : $M = 3.44$, $SD = 0.399$; R_e : $M = 3.55$, $SD = 0.623$). These data strengthen the observation that R_i and R_e did not return to pre-exercise levels even after 3 min of recovery. Overall, recovery patterns appear to differ between the groups, and there is a general delay for respiratory resistance to return to baseline levels. Similarly, the 15 healthy young adult athletes studied with continuous monitoring of respiratory resistance by Johnson et al. (2013) demonstrated an approximate 30-s delay before a gradual 2-min decrease in R_i and R_e .

An intriguing and unexpected finding from the present study was that during RTB, R_i and R_e were significantly lower for athletes with PVFMD than for those without PVFMD. Both groups were expected to have similar R_r values when not exercising because the two groups were well matched for sex, age, body size, and athletic experience. Resistances during RTB for the athletes without PVFMD are commensurate with those obtained for 102 participants (ages 12–18 years) by Johnson and colleagues (2007).

A possible explanation for the differences in resistance measures between athletes without and with PVFMD during RTB involves anatomical differences in the laryngeal or respiratory systems. According to Sapienza, Hoffman-Ruddy, and Baker (2004), aspects of the larynx such as vocal fold length may not reach full maturity until the later teen years. Thus, incomplete laryngeal maturation may be a contributing factor to both constriction and expansion of the laryngeal airway because the laryngeal cartilages of the pediatric larynx are more pliable and the arytenoid cartilages are proportionately larger than the other laryngeal cartilages (Sapienza et al., 2004). It is possible that teenage girls who are susceptible to PVFMD have less mature laryngeal structures that could affect laryngeal airway resistance. Presumably, malleable laryngeal structures might serve to enlarge the laryngeal airway, thus contributing to the lower respiratory resistance values for the athletes with PVFMD during RTB.

As mentioned in the introduction, exercise-induced laryngomalacia is considered one cause of dyspnea (Christopher & Morris, 2010; Dion, Eller, & Thomas, 2012; Heinle et al., 2003; Richter, Rutter, deAlarcon, Orvidas, & Thompson, 2008; Røksund et al., 2009; Tervonen et al., 2009). To examine the present data for laryngomalacia, the primary investigator retrospectively reviewed laryngoscopic

examinations of the athletes with PVFMD after the exercise challenge to determine whether symptoms were related to prolapsed laryngeal and supralaryngeal structures. Four of the 12 girls appeared to demonstrate exercise-induced laryngomalacia only, six appeared to demonstrate exercise-induced laryngomalacia followed by vocal fold adductory motion, and two appeared to demonstrate PVFMD without signs of laryngomalacia. These impressions should be interpreted with caution because an orally placed rigid laryngoscope can influence laryngeal posture and can potentially change the static appearance of the supraglottis (Powell et al., 2000). However, these findings mirror those of Røksund et al. (2009), who examined 113 athletes with exercise-induced dyspnea during exercise and identified 21 who demonstrated laryngomalacia only, 88 with laryngomalacia followed by vocal fold adduction, and four with vocal fold adductory motion only. For those athletes with laryngomalacia during exercise, high airflow rates in conjunction with the negative pressure associated with the Bernoulli effect appeared to contribute to prolapse of the laryngeal structures, thus constricting the laryngeal airway (Dion et al., 2012). Interviews with athletes with PVFMD often reveal that they experienced occasional episodes suggestive of PVFMD at much younger ages when on the playground or in physical education class. Typically, however, it is not until their early teens that they experience a consistent PVFMD symptom pattern. This coincides with participating in sports more frequently, at higher levels of competition, and playing with faster (and sometimes older) athletes, often during a period of peak physical growth.

Another possible mechanism for lower R_i and R_e values during RTB among athletes with PVFMD involves neural adaptation. A feed-forward mechanism exists that regulates arterial partial pressure of carbon dioxide when breathing transitions from rest to mild-moderate exercise; this mechanism is followed by a chemoreceptor feedback mechanism that senses a lack of blood-gas homeostasis and adjusts the ventilatory response (Mitchell & Babb, 2006). This results from activation of multiple sensorineural pathways carrying information from the periphery to the central nervous system. The respiratory system is capable of adapting such that altered ventilatory homeostasis causes changes at the neuronal level. Adaptation can be short term, whereby the changes reverse quickly, or long term, also referred to as *adaptive plasticity*, where the adaptation occurs slowly in response to persistent or intense afferent stimuli and is maintained, resulting in structural or functional changes in the system (Mitchell & Babb, 2006; Pearson, 2000; Turner et al., 1997). Both short- and long-term adaptations have been observed in animal and human models for rhythmic motor behaviors such as walking and breathing (Mitchell & Babb, 2006; Pearson, 2000; Turner et al., 1997). It is possible that athletes with PVFMD experience long-term adaptation that influences their breathing at rest. According to Pearson (2000), the neuronal networks that generate rhythmic patterns are highly responsive to alterations in sensory feedback that in turn produce motor patterns “appropriate for the mechanical requirements of different forms of a behavior”

(p. 723). It is feasible that hypoxia or the fear and anxiety associated with dyspnea caused by recurring PVFM episodes could cause adaptive changes in the airway that would lower resting respiratory resistance. The airway, being on constant alert for air hunger, may have “learned” a ventilatory response adopting a posture for unobstructed airflow that is effective until taxed by high-level exercise.

Study Limitations

This study is limited by its sample size, although the statistical power and effect sizes for these results were strong due to the clear differential effects of exercise for the two groups. As such, a larger sample size did not appear to be necessary to support the study’s primary hypothesis. However, these results may not be applicable to all demographic groups because only teenage girls participated. In addition, control athletes’ airway health was screened by self-report; laryngeal exams and pulmonary function testing would have enhanced the accuracy of their selection.

Two potential procedural limitations are noteworthy. First, this study did not capture the instantaneous effects of real-time exercise because APD data were averaged across 1-min epochs. Continuous R_r monitoring beginning prior to exercise and extending into recovery would have provided more detailed results but was deemed impractical in this fragile population. Second, the MBDS, which was selected for this study due to its common use to rate dyspnea in older children and adolescents with respiratory problems (Wilson & Jones, 1989), may have lacked clarity to some athletes. Although Gros Lambert and Mahon (2006) reported that children between the ages of 8 and 12 years can perceive two to four intensities of dyspnea and can isolate dyspnea from other competing sensations during exertion, the present athletes may have factored in other sensations such as leg muscle fatigue. Including a separate fatigue scale and additional training on the meaning of *dyspnea* are recommended for future studies.

Second, the use of rigid oral endoscopy rather than flexible nasoendoscopy to diagnose participants with PVFMD is not best practice because the rigid scope placement and procedure has been reported to cause laryngeal and vocal fold postural changes (Powell et al., 2000). To compensate in part for this limitation, each PVFMD participant underwent a laryngeal examination prior to the exercise challenge in order to familiarize them with the procedure and to obtain laryngeal images during RTB. It is noteworthy that Powell and colleagues (2000) reported a high correlation between oral endoscopy and posterior positioning of the epiglottis in their juvenile participants with PVFMD, yet they did not associate their participants’ dynamic supraglottic abnormalities with oral endoscopy.

Future Research and Clinical Implications

Replication of this study is needed in a larger and more diverse group of participants who are subgrouped by

demographics and type of glottal closure (paradoxical adductory motion of the vocal folds and/or laryngomalacia). Likewise, longitudinal studies that follow young athletes with either vocal fold adduction or laryngomalacia into their late teens and early adulthood will allow us to predict the typical course of PVFMD among this population. Clearly defining the disorder by observing and describing the type of laryngeal dysfunction, identifying various triggering stimuli, and elucidating associated pathophysiology will further our understanding of the clinical presentation for subgroups of athletes with PVFMD, positively affect treatment decisions, and compel evidence-based treatment techniques in the future.

A new generation of APD instruments is planned that will improve the device’s portability and ease of use. It will be battery powered, capable of data storage, and have an enhanced graphic interface. These features will allow data collection to be conducted in the environments and conditions where the athletes practice and compete in order to better understand the contributing factors for PVFMD. The present study successfully elicited PVFMD symptoms through a customized rigorous treadmill exercise protocol, yet no laboratory test can replicate the physical, cognitive, and emotional demands of competitive play or the physical environment in which sports are played.

Finally, treatment outcome studies are needed for PVFMD that are supported by objective data. Although continuous laryngoscopy is possible for this purpose (Røksund et al., 2009), the APD is more likely to be tolerated, especially under challenging breathing circumstances. Thus, the APD holds promise as a useful tool for monitoring progress and providing quantitative outcome data with treatment.

Conclusions

This study demonstrated the potential of the APD to provide quantitative measures of inspiratory and expiratory resistance as a function of exercise in 24 female teenage athletes, half with and half without PVFMD. The exercise protocol used in the present study was appropriately challenging and successfully differentiated the two groups. The athletes with PVFMD sensed more severe dyspnea and were unable to run as long as the athletes without PVFMD. Inspiratory resistance increased markedly with exercise for the athletes with PVFMD and decreased toward resting level during the 2 minutes following exercise. We find it interesting that respiratory resistance at rest was lower in the athletes with PVFMD than in the control athletes; this observation could have implications for predicting risk factors or selecting treatment approaches for PVFM.

The APD appears to be an effective tool for investigating respiratory resistance changes associated with PVFMD because it separately measures inspiratory and expiratory resistances that can reflect glottal area changes. As a supplement to indirect laryngoscopy, the APD can add scalable data to assessments, and, due to its ease of operation, it is feasible for repeated use throughout PVFMD treatment.

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