Inspiratory Muscle Training in Exercise-Induced Paradoxical Vocal Fold Motion

*Barbara A. Mathers-Schmidt and †L. R. Brilla

Bellingham, Washington

Summary: The purpose of the study was to determine if inspiratory muscle training (IMT) would result in increased inspiratory muscle strength, reduced perception of exertional dyspnea, and improved measures of maximal exercise effort in an athlete with exercise-induced paradoxical vocal fold motion (PVFM). The participant, an 18-year-old woman, had a 2-year history of acute dyspnea with exertion during soccer games. Spirometry, transnasal flexible laryngoscopy, and patient history supported a PVFM diagnosis. The ABAB within-subject withdrawal design study comprised IMT treatment and withdrawal phases, each lasting 5 weeks. The participant trained 5 days per week, completing five sets of 12 breaths at 75% maximum inspiratory pressure (MIP) per session. Data consisted of MIP, exertional dyspnea ratings, and maximal exercise measures. IMT resulted in increased MIP and decreased dyspnea ratings across both treatment phases. No change in MIP or dyspnea ratings occurred in response to treatment withdrawal. The maximal exercise test revealed minimal changes across phases. At end of the study, the participant reported experiencing no PVFM symptoms when performing the outcome measurement tasks and when playing soccer. Transnasal flexible laryngoscopy, after strenuous exercise and during rapid breathing and phonation tasks, revealed normal laryngeal findings. The findings suggest that IMT may be a promising treatment approach for athletes with exercise-induced PVFM.

Key Words: Paradoxical vocal fold motion—Vocal cord dysfunction—Inspiratory muscle training—Sensation of dyspnea.

INTRODUCTION

Paradoxical vocal fold motion (PVFM) is a laryngeal disorder that interferes with normal respiration. In PVFM, the vocal folds adduct during inhalation and/or exhalation, which thereby restricts the airway opening.[1–3] The typical patient presents with episodic or recurrent symptoms of dyspnea and/or stridor. Frequently the patient complains of tightness localized to the laryngeal area. Most patients report that the dyspnea is precipitated by a trigger such as airway irritants (including gastric reflux, postnasal drip associated with sinusitis, and airborne particle/pollutants), emotional stressors, extreme temperatures, or exercise. Although the

Accepted for publication March 3, 2005.

From the *Department of Communication Sciences and Disorders and the †Department of Physical Education, Health, and Recreation, Western Washington University, Bellingham, Washington.

Address correspondence and reprint requests to Barbara Mathers-Schmidt, Department of Communication Sciences and Disorders, Western Washington University, 516 High Street, Bellingham, WA 98225. E-mail: barbara.mathers-schmidt@wwu.edu

Journal of Voice, Vol. 19, No. 4, pp. 635–644
0892-1997/$30.00

doi:10.1016/j.jvoice.2005.03.005

pathophysiology of PVFM is poorly understood, several causes have been proposed. These causes include psychological conditions, upper airway sensitivity to laryngeal irritants, and a form of laryngeal dystonia.[4] Recent interest in PVFM has yielded numerous articles addressing the challenges of describing, classifying, differentially diagnosing, and managing this disorder.[4–9] The literature supports the notion that PVFM is a "complex, heterogeneous disorder in both its etiology and expression."[4]

Exercise-induced PVFM is becoming increasingly recognized in athletes who frequently have been misdiagnosed with refractory exercise-induced asthma (EIA). Sports competition, practice, or merely strenuous activity can trigger the PVFM attacks.[10,11] Symptom onset with PVFM is sudden and occurs within a few minutes of the increased level of physical activity; symptom resolution usually is rapid as well.[12] Many athletes with exercise-induced PVFM report that they limit their physical activity in an effort to minimize the frequency and severity of PVFM episodes. When the athlete does engage in increased physical activity, which thereby elicits PVFM symptoms, a maladaptive pattern emerges—the individual shifts into tense, shallow breathing with excessive tension and struggle focused at the laryngeal level. The perceived dyspnea contributes to additional struggle behavior.

The assessment and treatment of PVFM have been reviewed extensively elsewhere.[4–6] Differential diagnosis of PVFM is based on clinical history, pulmonary function and laboratory test results, laryngoscopic findings, speech pathology evaluation, and in some cases, psychological evaluation. The diagnosis of PVFM may be extremely difficult, and it is often one of exclusion.[4,5] The failure to recognize PVFM has led to prolonged and unnecessary drug use (such as anti-inflammatory agents, inhaled or oral corticosteroids, leukotriene modifier agents, and bronchodilators), hospitalization, intubation, and even tracheostomy.[12–16] Medical intervention for PVFM typically involves patient education, termination of unnecessary medications, and prescription of medications to treat contributory conditions. For example, in cases in which the condition of a patient has been misdiagnosed as asthmatic or has been overmedicated for a coexisting asthma, drugs such as corticosteroids will be tapered. When airway irritants such as gastric reflux, rhinosinusitis, or allergens are identified as triggering PVFM, pharmacological therapy and lifestyle modifications will be initiated. For many patients, the behavioral intervention that is provided by the speech-language pathologist (SLP) contributes to alleviating the PVFM symptoms. Customary SLP intervention consists of additional patient education, supportive counseling, instruction in tension identification and control, and instruction in some type of optimal, or "relaxed-throat," breathing.[1,2,4–6] Few, if any, prospective experimental studies have been conducted to provide research-based clinical guidance for treating dyspnea in the PVFM patient.

As one considers the pathophysiology of dyspnea, it is important to recognize that more than one mechanism usually contributes to perceived effortful breathing.[17] The medical section of the American Lung Association defined dyspnea as "a subjective experience of breathing discomfort that derives from interactions among multiple physiologic, psychological, social, and environmental factors."[18] Research has revealed a direct relationship between inspiratory muscle fatigue and estimation of respiratory effort in healthy subjects.[19] Other studies have shown an inverse relationship between inspiratory muscle strength and perceived dyspnea/respiratory effort in patients with chronic obstructive pulmonary disease (COPD),[20,21] in healthy subjects,[22,23] and in highly trained athletes.[24,25] Although the typical patient with exercise-induced PVFM associates dyspnea with tightness localized to the laryngeal area,[10–12] it could be that the patient perceives effortful breathing related to relative weakness in the respiratory muscles, as well.

Inspiratory muscle weakness[24,26] and fatigue[19,22] also have been linked with rapid, shallow breathing (tachypnea) during heavy exercise. In addition, increases in respiratory load have been related to abdominal paradox.[27] Such abnormal respiratory patterns have been reported by PVFM patients and observed in at least one study[28] in which episodes of PVFM were associated with rapid, shallow breathing and abnormal, asynchronous thoraco-abdominal wall motion. It is possible that these abnormal respiratory behaviors are exacerbated by the anxiety or panic experienced at the first signs of dyspnea. One also

might speculate that in patients with exercise-induced PVFM, inspiratory muscle weakness and perceived increases in breathing resistance might play a role in a tendency to develop and persist in abnormal and maladaptive respiratory patterns especially notable during exercise.

The significance of respiratory muscle fitness and the effectiveness of respiratory/ventilatory muscle training (RMT/VMT) have been explored for the past 30 years in patients with compromised respiratory function, in healthy sedentary participants, and in trained athletes.[20–26,29–32] In a review of the research literature, a joint committee of the American College of Chest Physicians and the American Association of Cardiovascular and Pulmonary Rehabilitation (ACCP/AACVPR)[20] determined that there is sufficient evidence to support VMT as a component of pulmonary rehabilitation for COPD patients: "Selected randomized, controlled trials that included adequate training loads (i.e., an intensity of at least 30% of maximum inspiratory pressure) and measured clinical outcomes have shown improvements in dyspnea and/or exercise tolerance with VMT." Specific inspiratory muscle training (IMT), which involves resistive breathing maneuvers, likewise improved inspiratory muscle strength and endurance and stabilized vital capacity in patients with neuromuscular disorders.[29] Similarly, IMT involving inspiratory pressure threshold breathing maneuvers resulted in increased inspiratory muscle strength and decreased dyspnea during exercise and speech in a patient with decreased glottal area[30] and in two patients with bilateral abductor vocal fold paralysis.[31,32]

The value of IMT also has been demonstrated in persons without known respiratory system impairment. Specific IMT resulted in significant increases in measures of inspiratory muscle strength in healthy, sedentary adults.[22,23,33,34] In studies in which IMT training periods were sufficiently long and intense, participants demonstrated reduced exertional dyspnea (or perception of the magnitude of resistive load)[22,23] and enhanced exercise performance.[22] Even highly trained athletes have responded to IMT, with decreased inspiratory fatigue,[26] increased inspiratory muscle strength,[24,35] improved perception of respiratory effort during training,[24] improved breathing pattern (deeper, rather than tachypneic) during exercise performance,[24,26] and improved sports performance.[24,26]

These findings suggest that IMT would benefit a patient with exercise-induced PVFM patient, where vocal fold adduction results in increased upper airway resistive load. It was hypothesized that a participant could maintain adaptive respiratory patterns (open glottis with reliance on increased respiratory muscle effort) while breathing against incrementally increased threshold load, with consequent increased inspiratory muscle strength and decreased dyspnea. There are several possible benefits. First, by developing increased strength in the muscles that need to be activated to support the increased level of activity, the athlete would be less likely to engage in the maladaptive excessive laryngeal tension (ie, vocal fold adduction associated with PVFM) during the trigger level of activity. Second, the decrease in perception of respiratory effort likely will translate into a decrease in the sense of panic and associated struggle behavior, which reportedly are experienced before and during a PVFM episode. Finally, in cases in which there is some degree of persistent PVFM, the increased inspiratory muscle strength may enable the person to generate sufficient pressure differentials to overcome laryngeal obstruction during an episode of PVFM.

The purpose of this study was to determine if inspiratory muscle training would result in increased inspiratory muscle strength, reduced perception of exertional dyspnea, and improved measures of maximal exercise effort in a young female athlete with exercise-induced paradoxical vocal fold motion.

PARTICIPANT AND METHODS

Participant

The participant was a nonsmoking 18-year-old woman (height: 157 cm; weight: 54.5 kg) with a 2-year history of acute dyspnea triggered by high-intensity exertion during soccer workouts and games. She had been treated for exercise-induced asthma, with a bronchodilator inhaler, with no improvement. The PVFM diagnosis was made by two physicians, an asthma and immunology specialist and an otolaryngologist. A speech-language pathologist (first author) confirmed that the history and symptoms of the patient were consistent with exercise-induced

PVFM. A methacholine challenge revealed mild bronchial hyperreactivity that did not meet the criteria for the diagnosis of asthma. The participant reported mild rhinoconjunctivitis during the pollen season. Skin testing for allergies revealed marked reactivity to grass and moderate to tree and cat. Otherwise, the participant was in good health. The participant maintained a regular workout routine throughout the study.

The Human Subjects Review Committee of Western Washington University approved the study. The study was explained to the participant, and written informed consent was obtained.

Study design

The ABAB within-subject withdrawal design study was conducted for 16 weeks. Preintervention assessment was conducted during the first week. Baseline data consisted of maximum inspiratory pressure (MIP) measurements (an indicator of inspiratory muscle strength[23]) recorded on 3 consecutive days, three dyspnea ratings during the symptom trigger level of activity (ie, shifting to a sprint when running) executed on 3 separate days, and one set of maximal exercise ventilatory measures during a maximal graded exercise test on a treadmill. Each treatment and withdrawal phase lasted 5 weeks. All measurements were completed at the end of each phase. In addition, MIP measurements and dyspnea ratings were completed at the end of the third week of each treatment phase. Before the IMT sessions, the participant met one time with the first author who provided patient education regarding the mechanics of respiration and instruction in a relaxed-throat breathing technique. Throughout the study, the patient was reminded to use primary inspiratory muscles during inhalation and to allow the face and throat muscles to remain relaxed. Three speech-language pathology graduate students were trained to make the MIP measurements and to conduct the IMT training sessions. Intertrainer reliability was demonstrated by repeated determination of MIP varying by only 1.5%.

Laryngeal status

Before the study, the participant was referred to an otolaryngologist. The fiberlaryngoscopic evaluation was performed transnasally. After 5 minutes of strenuous patient exercise, laryngoscopy revealed moderate PVFM during inhalation; no adduction was observed on exhalation. The otolaryngologist rated the PVFM episode as 3.5 on a 6-point scale (0 = no vocal fold adduction; 1 = very slight adduction; 2 = minimal adduction; 3 = moderate adduction; 4 = almost complete adduction (perhaps with posterior diamond-shaped chink); 5 = complete adduction). No other laryngeal abnormalities were noted during rapid breathing and phonation tasks.

Spirometry

Before the study, the participant was referred to an allergy and immunology specialist for spirometric studies. Spirometry was performed with a full-function, computer-based spirometer (KoKo Spirometer; Ferraris Respiratory; Louisville, CO). Spirometry revealed a truncated inspiratory flow-volume loop, which supported a PVFM diagnosis. At mid-forced vital capacity, the inspiratory flow was 2.0 L/s, whereas the expiratory flow was 4.0 L/s. These values correspond to the maximal expiratory flow at 50% of the vital capacity (MEF50) and maximal inspiratory flow at 50% of the vital capacity (MIF50), and they yield an MEF50/MIF50 ratio of 2.0. An MEF50/MIF50 ratio exceeding 1.5 is indicative of extrathoracic airway obstruction.[4]

Tests

Respiratory muscle strength. MIP was measured at the mouth with a mouthpiece with a two-way valve connected via a vacuum hose to the electronics of the IMT device (described below). A calibrated modification to the IMT electronics allowed a scaled output to be displayed on the Fluke True-rms Multimeter (Model 110). Maximum inspiratory pressure was measured with the MIN–MAX function of the digital voltmeter. The experimenter instructed the participant by referring to a standard written MIP procedure adapted from Kellerman et al.[23] The participant stood, with noseclips occluding the nares, and placed her lips around the mouthpiece. She was instructed to breathe quietly for two to three cycles while the valve was open. During the third inhalation, the participant was instructed to exhale maximally (to residual volume), close the valve, and

breathe in as forcefully as possible for about 2 seconds. The participant then opened the valve, resting for 1 minute before repeating the MIP maneuver. Measurements were repeated until three were obtained within 5% variability. The largest of these three MIP values was recorded.

Exertional dyspnea. After jogging at a comfortable pace for 200 m on an outdoor track, the participant performed the PVFM trigger level of activity, shifting to a sprint for at least 100 m or until she experienced the PVFM symptoms. The experimenter instructed the participant to rate the dyspnea with a 4-point rating scale (0 = no dyspnea, 1 = mild dyspnea, 2 = moderate dyspnea, 3 = severe dyspnea).

Measures of maximal exercise effort. Measures of maximal exercise effort were conducted in an exercise physiology laboratory. Maximal graded exercise tests were completed with a Quintron treadmill and Parvo metabolic analysis system. We used a modified Balke protocol, 160 $m.min^{-1}$ with 2% grade increments every 3 minutes to volitional fatigue. The measures included the following: minute ventilation (V_E), oxygen consumption (VO_2), respiratory rate (RR), and respiratory exchange ratio (RER). Patient Borg scale ratings of perceived exertion (RPE) and perceived breathlessness or dyspnea (RPD) were assessed during the maximal graded exercise tests.

Inspiratory muscle training

The participant trained 5 days per week for 5 weeks, with a custom-made inspiratory muscle strengthening device (Figure 1) modeled after a device described by Sapienza et al.[30] The device consists of a mouthpiece and a solenoid valve that opens when a sensor detects a negative (inspiratory) pressure exceeding a threshold level. The trainer is calibrated, with a threshold pressure load ranging from 15 to 110 cm H_2O. The respiratory valve remains open for exhalation. The IMT device is ideal for research purposes because the inspiratory pressure load can be varied over a continuum of increments. Furthermore, the pressure-based threshold training allows specific training of inspiratory muscles without modifying airflow.[23]

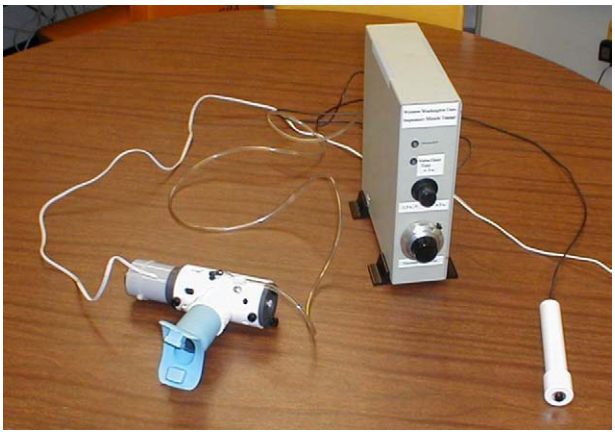

FIGURE 1. Inspiratory muscle trainer.

The participant was trained according to a protocol adapted from Kellerman et al.[23] During each IMT session, she completed 5 sets of 12 training breaths at the designated threshold level. A noseclip precluded breathing through the nose. The participant was instructed to breathe quietly for two to three breaths. On the third exhalation, the instructor told her to prolong exhalation. The intake valve was then closed, so that the participant inhaled against resistance, with the valve opening when she achieved the designated threshold. Each set of 12 training breaths was followed by a 1-minute rest period. The daily training sessions lasted approximately 30 minutes each.

The baseline MIP measurements determined the initial IMT threshold levels. The participant had two introductory sessions, training for 2 days at 25% MIP. During the next two sessions, she trained at 50% MIP. For the remaining sessions, she trained at 75% MIP. The IMT threshold level was adjusted so that the participant trained at 75% of the changing MIP, which was determined by repeated measurement across phases—at the end of the third week of the first training phase, at the beginning of the second training phase, and at the end of the third week of the second training phase.

RESULTS

Respiratory muscle strength

MIP increased substantially across both IMT treatment phases (Table 1; Figure 2). During the baseline period, MIP was relatively stable over 3 days, with a mean MIP of 68.2 ± 1.2 cm H_2O (mean ± SD). By the end of the third week of phase one IMT, the MIP had increased to 85.85 ± 4.45 cm

TABLE 1. *Maximum Inspiratory Pressure in Centimeters of Water*

Baseline (A phase)	Treatment (B phase)	Treatment withdrawal (A phase)	Treatment (B phase)
66.4; 68.5; 69.7	Post-wk 3: 81.4; 90.3		Post-wk 3: 120.4; 107.5
	Post-wk 5: 94.6; 101	Post-wk 5: 103; 102.6	Post-wk 5: 121.6; 119.8

H_2O, or 25.9% (compared with baseline mean). By the end of the fifth week of phase one IMT, the MIP increased to 97.8 ± 3.2 cm H_2O, or 43.4%. By the end of the third week of phase two IMT, the MIP increased to 113.95 ± 6.45 cm H_2O, or 67.1%. By the end of the fifth week of phase two IMT, the MIP increased to 120.7 ± 0.9 cm H_2O, or 77%. There was essentially no change in MIP after the no treatment period. By the end of the IMT withdrawal phase, the MIP was 102.8 ± 0.2 cm H_2O, for an increase of 1.8 cm H_2O, or 1.8%, when compared with the final MIP of the preceding treatment phase.

Exertional dyspnea

During exercise that previously triggered PVFM symptoms, participant ratings of dyspnea decreased after IMT (Table 2). The ratings decreased from 2.5 at baseline to 1.3 by the end of the second treatment phase. There was no change in dyspnea rating after the IMT withdrawal phase.

Measures of maximal exercise effort

The measures of maximal effort during the graded exercise test revealed minimal changes across treatment and no-treatment phases, with the exception

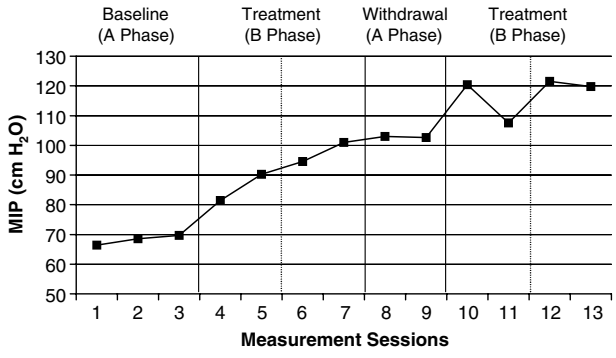

FIGURE 2. Maximum inspiratory pressure in centimeters of water.

TABLE 2. *Dyspnea Ratings (0-3) and Time to Symptom Onset (s) During Exercise*

Baseline (A phase)	Treatment (B phase)	Treatment withdrawal (A)	Treatment (B phase)
2.5 (21 s)	Post-wk 3:		Post-wk 3:
2.4 (23 s)	1.5 (29 s)		1.5 (41 s) (rained out)
2.2 (22 s)	1.5 (26 s)		
	Post-wk 5:	Post-wk 5:	Post-wk 5:
	1.7 (35 s) (ill for next 2 days)	1.7-2.0 (29 s)	1.3 (28 s)
		1.7 (25 s)	1.3 (30 s)

of a 10% increase in VO_2 at the end of the first treatment phase and a slight increase in V_E during treatment phases (Table 3).

Post-IMT laryngeal status

The otolaryngologist reported a normal laryngeal examination, with no evidence of vocal fold adduction during inhalation or exhalation after a period of exercise or during rapid breathing. Movement was normal during and after phonation tasks as well.

Post-IMT spirometry

Spirometry continued to show a truncated inspiratory limb on the flow-volume loop. At mid-forced vital capacity, the inspiratory flow was 3.0 L/s, whereas the expiratory flow was 4.0 L/s. These values yield an MEF50/MIF50 ratio of 1.3. Even though the inspiratory limb was somewhat truncated, the post-IMT MEF50/MIF50 ratio did not exceed

TABLE 3. *Measures of Maximal Exercise Effort*

	Baseline (A)	Treatment (B)	Withdrawal (A)	Treatment (B)
HR (per min)	211	214	207	211
RPE	18	20	17	18
RPD	3	3	3	3
VO_2 (mL/kg/min)	50.7	55	52	53.6
V_E (L/min)	78	79.4	75.5	82.2
RR (per min)	48	47	46	47
RER	1.13	1.09	1.06	1.11

Abbreviations: HR, heart rate; RPE, ratings of perceived exertion; RPD, ratings of perceived breathlessness or dyspnea; VO_2, oxygen consumption; V_E, minute ventilation; RR, respiratory rate; RER, respiratory exchange ratio.

1.5, the criterion for indicating extrathoracic airway obstruction in symptomatic patients.[10]

DISCUSSION

This study showed that specific inspiratory muscle training improved both the inspiratory muscle strength and the sensation of exertional dyspnea experienced by an athlete with exercise-induced PVFM. As hypothesized, the participant maintained an open airway against incrementally increased resistance, while relying on adaptive respiratory patterns. She experienced no PVFM episodes throughout the training period. The post-IMT laryngeal examination confirmed the absence of exercise-related PVFM. Although post-IMT spirometry continued to elicit a somewhat truncated inspiratory limb on the flow volume loop, performance on this task changed in the desired direction. It could be that her abnormal inspiratory loop is simply an artifact of the test, and it is not that helpful in monitoring her PVFM status.

The IMT training effect carried over to actual sports performance. During each baseline trial, the participant experienced a PVFM episode within 22 seconds of sprinting. The episode was characterized by moderate-to-severe dyspnea and inspiratory stridor. By the end of the first 3 weeks of training, the dyspnea rating declined to mild to moderate. At that time, she described the sensation in terms of laryngeally localized airway resistance; she also evidenced mild stridor. Symptoms resolved within 5 minutes of rest. By the end of the fifth week of phase one IMT, the participant sprinted at a faster pace and for a longer period of time before eliciting a PVFM episode that she rated as mild to moderate. She maintained this level of performance until the second treatment phase. By the end of the third week of phase two IMT, the participant was sprinting even faster and maintaining the pace for 41 seconds before experiencing milder dyspnea. It is important to note that during this second treatment phase, the participant did not evidence stridor or PVFM-related dyspnea. She vaguely described the dyspnea as feeling "farther back" by the end of the third week phase two IMT. By the end of the fifth week of phase two IMT, she no longer associated the milder dyspnea with throat tension, even though she was sprinting faster and running farther than in any previous sessions. In addition, although we did not collect objective respiratory pattern data during the sprint task, apparent changes in respiratory patterns were noted. During baseline sprints, the participant evidenced a tachypneic breathing pattern, along with elevated shoulders and obvious use of accessory muscles. This tendency diminished somewhat throughout phase one IMT sprinting. By phase two IMT, she appeared to be maintaining more optimal respiratory behaviors throughout the sprint.

The participant also reported a carryover of treatment gains to soccer games and other exercise. Toward the end of the second phase of treatment, she played in several highly competitive soccer games. She experienced dyspnea in only one of five games, a game she described as being extremely tough and one in which the dyspnea likely was not PVFM-related but simply associated with prolonged maximal effort. One-month after the study, she reported one episode of mild PVFM dyspnea that she could control when sprinting uphill; otherwise, she experienced no PVFM symptoms as she resumed a regular exercise routine. At 9 and 10 months after the study, she reported being back in soccer practice with only minor episodes of PVFM-like dyspnea having occurred during the post-IMT interim. She reported maintaining optimal breathing patterns and shifting to more conscious monitoring of relatively relaxed open-throat breathing, as she experienced or anticipated difficulty breathing. She stated that she was not concerned about these relatively minor dyspnea experiences, because she could control them. She also reported occasionally "pushing through" the breathing difficulty.

Her improvement in MIP was consistent with that reported in other studies. In a case report,[36] which was published after the completion of this study, researchers reported that a teenage male rower with presumed exercise-induced PVFM achieved a 93% increase in MIP after 5 weeks of inspiratory muscle strength training. These exceptionally large gains (from a baseline of 73 cm H_2O to 166 cm H_2O) may be explained by the moving of the athlete from the earlier compromised performance level associated with PVFM to a level more consistent with that of a healthy male rower. Volianitis et al[24] reported a 45.3% improvement in MIP in elite athletes, after

11 weeks of training (from a mean of 104 cm H_2O to a mean of 148 cm H_2O). In this study, the participant was an athlete who had curtailed her exercise for 2 years because of PVFM. She was a varsity soccer player and not an elite athlete, or rower, as in the Volianitis et al study, which likely explains the lower initial MIP of 68.2 cm H_2O (baseline mean) that improved to 120.7 cm H_2O, once she no longer experienced PVFM and was benefiting from the specific inspiratory muscle training. Hence, in this study, the MIP of the participant increased by 43.4% after the first phase of treatment (essentially getting her up to normal for a healthy person) and then increased again by 17.4% as she responded to the second phase of treatment. This result is also somewhat consistent with Kellerman et al's[23] study of healthy young adults in which MIP improved approximately 60% after 4 weeks of IMT (from a mean of 87.2 cm H_2O to a mean of 138.5 cm H_2O). As one would expect, a study involving chronic obstructive pulmonary disease patients revealed smaller gains, with patients improving about 25% after 3 months of IMT (from a mean of 48 cm H_2O to a mean of 60 cm H_2O). In these group studies, the placebo subjects did not show similar gains. In this study, experimental control was demonstrated by lack of change during the treatment withdrawal period.

Although direct comparisons are complicated by the nature of contributory factors, IMT-related changes in dyspnea and exercise performance in this study are consistent with other research findings.[21–24,26,30,37] The mechanism by which this change occurs has not been fully explained. In this case, it is likely that dyspnea decreased in response to several factors. First, the participant began using adaptive respiratory behaviors (relying on primary inspiratory muscles) that precluded her tendency to tighten in the laryngeal area, the PVFM behavior that formerly created airway restriction. Second, she no longer lapsed into tachypneic breathing, but she maintained more desirable respiratory patterns, a behavior that promotes adequate diaphragm relaxation and mechanical efficiency.[26] She also reported less anxiety associated with breathing during increased level of exercise. This psychological factor likely played a role in a reduced perception of difficulty breathing and a reduced likelihood of engaging in maladaptive struggle behaviors. Finally, with increased inspiratory muscle strength, the participant could better "push on through" when she sensed dyspnea from any source, including slight laryngeal airway restriction.

Others have suggested explanations for decreased dyspnea and increased exercise performance with specific IMT. As breathing efficiency increases in response to IMT, there may be a lower ventilatory workload and thereby reduced inspiratory muscle utilization of metabolic and blood flow resources. Thus, IMT would indirectly impact exercise by improving blood flow distribution to limb muscles.[24,26] Similarly, Boutellier[22] suggested that the inspiratory muscles consume less oxygen after IMT, which again frees up resources for the limb muscles. Another explanation focuses on neural adaptation; with increased inspiratory muscle strength, there is reduced motor output to respiratory muscles and hence a reduced perception of respiratory effort.[24] Reduced ventilatory work also would reduce sensory input to the central nervous system, which results in a reduced sensation of effortful breathing.[26] Ruddy et al[36] hypothesized that decreases in laryngeal airway resistance and dyspnea may be attributable to the synergistic training effect of the diaphragm and the posterior cricoarytenoid (PCA) muscle. They presumed that with IMT-related increased activation of the diaphragm, PCA activation increases as well, which thereby increases the glottal aperture size. This intriguing explanation warrants further consideration as we try to determine why exercise-induced PVFM patients benefit from inspiratory pressure threshold training.

The exercise performance measures were within normal limits, and most did not change in response to IMT, which is consistent with other studies in which healthy subjects and athletes were engaged in IMT.[22,25,35,38] The only exception was the minute ventilation (V_E), which showed a tendency to change in response to IMT here and in another IMT study involving elite athletes.[38] The increase in V_E during treatment phases, and the V_E decline after the 5-week IMT withdrawal, indicate that increased inspiratory muscle strength contributed to the ability of the participant to increase performance output or move more air. Our participant, however, did not

show decreased respiratory frequency, another indicator of increased inspiratory muscle strength seen elsewhere.[38] The VO_2 measures of our participant were superior and fluctuated in the anticipated direction during treatment and nontreatment phases. Nevertheless, the VO_2 change was negligible. According to Amonette and Dupler,[38] this result is not surprising given the short duration of the exercise performance task and the short training period. Furthermore, sustained increases in exercise involving large muscle groups, rather than the small groups of ventilatory muscles, would be required to increase cardiac output and, hence, VO_2max.[39] It may be that there simply is not a large functional transfer from IMT to standard exercise performance measures, such as a maximal treadmill test.

The generalizability of these findings to other patients with exercise-induced PVFM must be demonstrated. In future research, laryngeal tension, ventilatory patterns, and specific ventilatory behaviors should be measured to determine more precisely what contributes to improvement in PVFM status. Other performance measures may be necessary to validate IMT, including a precise tracking of PVFM episodes during trigger level activity, performance speed, and performance endurance. Finally, other laboratory-based exercise performance measures should be considered. Exercise testing with higher intensity anaerobic testing, such as a Wingate 30-second cycle sprint or "beep" sprinting test (repeat, shortened time intervals to cover 20 m until inability to complete the distance in the allotted time), seems warranted from the field exercise performance observations in this study.

A few precautionary remarks are warranted regarding future research or clinical application of these findings. First, one must be certain that a careful differential diagnosis has supported the fact that the dyspnea is caused by exercise-induced PVFM rather than one of the many other conditions that can produce dyspnea and airway obstruction.[4] In addition, this research does not support undertaking IMT in cases in which there are rarely occurring neurogenic etiologies such as brain stem compression, cortical or upper motor neuron injury, nuclear or lower motor neuron injury, and movement disorders.[9] Finally, in cases in which multiple factors may play a role in precipitating PVFM, each contributory factor must be managed to ascertain the best treatment approach.[4,6] For example, in some patients with exercise-induced PVFM, it may be that gastric reflux during exercise is the primary trigger of PVFM. Aggressive treatment of the reflux condition may result in symptom resolution, which thereby precludes other behavioral intervention such as IMT.

CONCLUSIONS

These findings suggest that IMT may be a promising treatment approach for athletes with exercise-induced PVFM. The athlete in this study learned to maintain an open airway against incrementally increased inspiratory resistance, with a subsequent increase in inspiratory muscle strength and a reduction in episodic dyspnea during trigger level of activity.

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