Effect of Inspiratory Muscle Training on Exercise Tolerance in Asthmatic Individuals

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[1]Department of Kinesiology, Indiana University, Bloomington, IN; [2]Department of Sport and Exercise Sciences, Northumbria University, Newcastle upon Tyne, UNITED KINGDOM; [3]Centre for Sport Medicine and Human Performance, Brunel University, Uxbridge, Middlesex, UNITED KINGDOM; [4]Health and Human Performance, Nebraska Wesleyan University, Lincoln, NE; and [5]School of Sport, Exercise and Health Sciences, Loughborough University, Loughborough, Leicestershire, UNITED KINGDOM

ABSTRACT

TURNER, L. A., T. D. MICKLEBOROUGH, A. K. MCCONNELL, J. M. STAGER, S. TECKLENBURG-LUND, and M. R. LINDLEY. Effect of Inspiratory Muscle Training on Exercise Tolerance in Asthmatic Individuals. *Med. Sci. Sports Exerc.*, Vol. 43, No. 11, pp. 2031–2038, 2011. **Purpose**: The aim of this study was to determine the effects of inspiratory muscle training (IMT) on exercise tolerance, inspiratory muscle fatigue, and the perception of dyspnea in asthmatic individuals. **Methods**: Using a matched double-blind placebo-controlled design, 15 clinically diagnosed asthmatic individuals underwent either 6 wk of IMT ($n = 7$) consisting of 30 breaths twice daily at 50% maximum inspiratory pressure (PI_{max}) or sham-IMT (placebo; PLA, $n = 8$) consisting of 60 breaths daily at 15% PI_{max}. Time to the limit of exercise tolerance (Tlim) was assessed using constant–power output (70% peak power) cycle ergometry. Inspiratory muscle fatigue was determined by comparing the pre- to postexercise reduction in PI_{max}. Dyspnea during the Tlim test was evaluated at 2-min intervals using the Borg CR-10 scale. **Results**: There were no significant changes ($P > 0.05$) in Tlim, inspiratory muscle fatigue, or perception of dyspnea in the PLA group after the intervention. In contrast, in the IMT group, PI_{max} increased by 28%, and Tlim increased by 16% ($P < 0.05$). Dyspnea during exercise was also reduced significantly by 16% ($P < 0.05$). The exercise-induced fall in PI_{max} was reduced from 10% before IMT to 6% after IMT ($P < 0.05$), despite the longer Tlim. Pulmonary function remained unchanged in both the IMT and PLA groups. **Conclusions**: These data suggest that IMT attenuates inspiratory muscle fatigue, reduces the perception of dyspnea, and increases exercise tolerance. These findings suggest that IMT may be a helpful adjunct to asthma management that has the potential to improve participation and adherence to exercise training in this group. However, the perception of breathlessness is also an important signal of bronchoconstriction, and thus, caution should be exercised if this symptom is abnormally low. **Key Words:** RESPIRATORY MUSCLE, DYSPNEA, ASTHMA, EXERCISE

Asthma is a disease associated with intermittent narrowing of the airways in which an individual experiences symptoms such as wheezing, chest tightness, coughing, and dyspnea (34). A characteristic of chronic asthma is that airway narrowing can alter airway function during exercise and reduce the maximal expiratory flow–volume curve (15), which can result in airflow limitation and reduced exercise capacity (18).

Airway obstruction during exercise in asthmatic individuals is associated with increased inspiratory muscle work because of resistance to airflow and dynamic hyperinflation of the lung (increased end-expiratory lung volume [EELV]) (15). The elevated work of breathing due to dynamic hyperinflation has been shown to be an important predictor of dyspnea in individuals with asthma (19), which is intensified by impairment in the contractile properties of the respiratory muscles (22). Increased work of breathing may also contribute to an increased risk of inspiratory muscle fatigue (39), which may exacerbate dyspnea and reduce exercise tolerance (24). Accordingly, it is reasonable to suggest that increasing the strength of the inspiratory muscles in people with asthma who experience increased inspiratory muscle work may reduce the intensity of dyspnea and improve exercise tolerance.

Inspiratory muscle training (IMT) has been shown to improve inspiratory muscle strength, exertional dyspnea, and exercise tolerance in both healthy people and patients with chronic obstructive pulmonary disease (10,21). A limited number of studies have evaluated the effect of IMT in people with asthma and have all demonstrated improved inspiratory muscle strength and reduced asthma symptoms, as well as reductions in hospitalizations, absence from school or work, medication usage, dyspnea during loaded

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breathing, and improved pulmonary function (35–38). However, the influence of IMT upon exercise tolerance and exertional dyspnea in people with asthma currently remains unknown.

Therefore, the main aim of the present study was to investigate the effect of 6 wk of IMT on exercise tolerance and exertional dyspnea in asthmatic individuals. We hypothesized that IMT would 1) increase inspiratory muscle strength and attenuate respiratory muscle fatigue, 2) reduce the perception of dyspnea, and 3) increase exercise tolerance.

METHODS

Subjects. Fifteen subjects (seven males, eight females; mean ± SEM: age = 24 ± 1 yr, stature = 1.74 ± 0.02 m, body mass = 76 ± 4 kg) with physician-diagnosed asthma were recruited into the study. All had mild to moderate persistent asthma, with a resting forced expiratory volume in 1 s (FEV_1) of >70% predicted, and were taking physician-prescribed maintenance medications. All subjects continued with their daily maintenance medications (stable dose) throughout the duration of the study (i.e., leukotriene receptor antagonists (n = 5 montelukast [Singulair®, Merck & Co. Inc, Whitehouse Station, NJ]), longacting A_2-agonists (n = 4, fluticasone propionate [Flovent®, GlaxoSmithKline, Research Triangle Park, NC] and salmeterol [Advair®, GlaxoSmithKline, Research park, NC]), and long-acting β_2-agonists and leukotriene receptor antagonists (n = 5 salmeterol [Advair®] and montelukast [Singulair®]), although short-acting β_2-agonists were discontinued 12 h before exercise testing; all medication use throughout the course of the study was recorded. Subjects were asked to refrain from exercising 24 h before testing and to avoid caffeine and alcohol intake during this period. Subjects were asked to maintain a normal diet throughout the duration of the study and to avoid consuming food 2 h before exercise. Before testing, each subject completed a health screening questionnaire and provided written informed consent. All testing procedures and informed consent were approved by the Indiana University Human Subjects Review Board.

Study design. Subjects reported to the laboratory on four separate occasions. During session 1, they performed a maximal incremental cycling test to determine peak power output (PPO). Sessions 2 and 3 were conducted before intervention and consisted of a constant-power cycle ergometer test to the limit of tolerance (Tlim) at ~70% PPO (session 2 was for familiarization purposes). Session 4 was a repeat of sessions 2 and 3 and was conducted after the 6-wk intervention period. All tests were separated by at least 48 h and were conducted at a similar time of day and under similar environmental conditions.

After baseline measurements, subjects were assigned in a double-blind manner to either an inspiratory muscle training (IMT, n = 7) or sham-IMT (placebo; PLA, n = 8) group and were matched across groups according each subjects' maximal inspiratory pressure (PI_{max}).

Pulmonary function and maximal inspiratory pressure measurements. Pulmonary function was assessed using a calibrated computerized pneumotachograph spirometer (Vmax 22; SensorMedics, Yorba Linda, CA) according to the American Thoracic Society's recommendations (1), which require each subject to perform three acceptable spirograms, of which values of forced vital capacity (FVC), FEV_1, and forced expiratory flow during 25%–75% of vital capacity may not vary by more than 10%. The largest value is reported.

Maximal inspiratory pressure (PI_{max}) was measured using a portable handheld mouth pressure meter (Micro Medical, Ltd., Kent, United Kingdom) and was used as an index of inspiratory muscle strength. All maneuvers were initiated from residual lung volume and were conducted in an upright-seated position. A minimum of five maneuvers at 30-s intervals were conducted, with the variability between the best three measurements less than 5% or within 5 cm H_2O (40). Measurements of pre- and postexercise PI_{max} were conducted before and after the intervention.

Exercise testing. PPO was determined using a continuous maximal incremental ramp test (6 W for 15 s) on an electromagnetically braked cycle ergometer (Lode Excalibur; Groningen, The Netherlands). The test was terminated when the subject reached the limit of tolerance or failed to maintain a pedaling cadence above 50 rpm. The power at which the subjects terminated exercise was defined as the PPO.

Metabolic and ventilatory data were measured continuously using breath-by-breath indirect calorimetry (Vmax 22; SensorMedics). HR was monitored using an F1 Polar HR Monitor (Polar, Helsinki, Finland). Arterial oxygen saturation was estimated using ear oximetry (model 47201A; Hewlett-Packard Company, Palo Alto, CA). The oximeter was calibrated using an internal protocol before each test, according to the manufacturer's instructions.

The data generated from the maximal incremental ramp test (PPO) was used to determine the power output for the Tlim. The Tlim test was performed at a workload of ~70% PPO until volitional fatigue. During the last 30 s of each minute, two maximum inspiratory capacity maneuvers (inspiration to maximal lung capacity) were performed. EELV was calculated by subtracting the maximum inspiratory capacity from the resting FVC, with values expressed as a percentage of FVC. EILV was determined as the sum of EELV and tidal volume (V_T) (average of 10–20 breaths). Dyspnea was assessed using the Borg scale (CR-10 scale) at 2-min intervals during the Tlim test.

Training intervention. Inspiratory muscle training was completed using a pressure threshold training device (POWERbreathe®; HaB International, Ltd., Southam, United Kingdom). The IMT group performed 30 dynamic inspiratory efforts twice daily at a pressure threshold load of 50% of PI_{max} for 6 wk. This specific protocol has been shown to elicit several changes in inspiratory muscle function, including strength, power, and endurance (3,27–29). The PLA group performed the training intervention once daily

for 6 wk at a pressure threshold load equivalent to 15% of PI_{max}; this protocol has been shown to exhibit no changes in inspiratory muscle function (3,28). Each breath was initiated from residual lung volume, with breathing frequency decreased to minimize hyperventilation-induced hypocapnia.

Compliance to training was monitored using a pressure sensor suspended within the main body of the inspiratory muscle trainer (28). The generation of a negative pressure exceeding the set point on the pressure switch caused a count to be registered. The cumulative number of pressure threshold changes were recorded and computed into total number of breaths. Physical activity diaries were used throughout the duration of the study to monitor training volume and intensity.

Data analysis. Data were analyzed using SPSS version 17.0 statistical software (Chicago, IL). The data were assessed for normality using the Kolmogorov–Smirnov test, and the Levene test was used to test for homogeneity of variance between tests. A one-way repeated-measures ANOVA was used to analyze within-group changes over time during the Tlim test. A Mauchly test was conducted to determine whether sphericity was violated. In cases where sphericity was violated, a Geiser–Greenhouse correction was applied. Within- and between-group interactions were analyzed using a two-way ANOVA with repeated measures. A Tukey *post hoc* analysis was performed to determine significant differences. Statistical significance was set at $P < 0.05$. Values are reported as mean ± SEM.

RESULTS

Baseline measurements of pulmonary function (Table 1) were within predicted normal limits and were not significantly different ($P > 0.05$) between the IMT and PLA groups. All parameters remained unchanged in both groups after the intervention. PPO achieved during the maximal incremental cycle test was not significantly different ($P > 0.05$) between the IMT (250 ± 21 W) and PLA (255 ± 24 W) groups. In addition, peak aerobic capacity was not significantly different ($P > 0.05$) between the IMT and PLA groups ($\dot{V}O_{2peak}$ = 42.6 ± 2.9 and 40.2 ± 2.2 $mL \cdot kg^{-1} \cdot min^{-1}$, respectively). Subjects adhered well to the training intervention with a compliance of 94% and 92% in the IMT and PLA groups, respectively. Daily physical activity remained unchanged throughout the intervention period as reported by their physical activity logs.

Maximal inspiratory pressure. Table 1 shows the mean pre- and postexercise values for PI_{max}, before and after the training intervention. Baseline values for PI_{max} were not significantly different ($P > 0.05$) between the IMT and PLA groups. After 6 wk of training, the IMT group demonstrated a significant increase ($P < 0.05$) of 27.7% ± 3.2% in PI_{max}, whereas no change ($P > 0.05$) in PI_{max} occurred in the PLA group.

Before the intervention, PI_{max} decreased significantly ($P < 0.05$) by 9.6% ± 3.9% and 10.6% ± 3.5% from before to after exercise (index of inspiratory muscle fatigue) in both the IMT and PLA groups, respectively. After IMT, the postexercise decrease in PI_{max} was significantly reduced ($P < 0.05$) to 6.2% ± 4.2% in the IMT group. There was no significant change ($P > 0.05$) in inspiratory muscle fatigue in the PLA group after the intervention.

Exercise tolerance. Before the training intervention, there was no significant difference ($P > 0.05$) between groups in the time to the limit of exercise tolerance (Tlim) (8.5 ± 0.9 min for the IMT group and 9.1 ± 0.9 min for the PLA group). After IMT, exercise Tlim increased significantly ($P < 0.05$) by 22% to 10.6 ± 1.5 min in the IMT group. No significant change ($P > 0.05$) in the exercise Tlim was observed in the PLA group (after PLA = 8.5 ± 1.3 min).

Metabolic and ventilatory responses during exercise. The metabolic and ventilatory data from the constant-power Tlim test are shown in Table 2. All data are reported in the range of 0–5 min because all subjects sustained exercise for a minimum of 5 min both before and after training intervention. In addition, comparisons were made at an end exercise point and at a time point during the posttraining Tlim test that corresponded to the pretraining

TABLE 1. Pulmonary function and inspiratory muscle strength.

	IMT		PLA	
	Before	After	Before	After
Resting pulmonary function				
FVC (L)	4.33 ± 0.26	4.34 ± 0.25	4.58 ± 0.36	4.63 ± 0.36
% predicted	95 ± 5	95 ± 5	97 ± 5	98 ± 5
FEV_1 (L)	3.61 ± 0.22	3.63 ± 0.21	3.71 ± 0.26	3.69 ± 0.30
% predicted	93 ± 3	93 ± 3	96 ± 3	95 ± 4
PEFR (L)	7.19 ± 0.50	7.25 ± 0.42	7.40 ± 0.30	7.47 ± 0.38
% predicted	89 ± 4	90 ± 4	93 ± 5	92 ± 3
$FEF_{25\%–75\%}$ (L)	3.58 ± 0.35	3.74 ± 0.39	3.91 ± 0.44	3.94 ± 0.53
% predicted	87 ± 9	91 ± 10	93 ± 9	93 ± 11
Inspiratory muscle strength				
Preexercise PI_{max} (cm H_2O)	114.6 ± 10.1	145.4 ± 11.7**	114.3 ± 10.3	120.6 ± 9.2
Postexercise PI_{max} (cm H_2O)	101.8 ± 6.8*	134.1 ± 8.0*	103.5 ± 11.4*	109.0 ± 8.6*

Values are mean ± SEM.
* Significant difference from before to after exercise ($P < 0.05$).
** Significant difference from before to after training ($P < 0.05$).
$FEF_{25\%–75\%}$, forced expiratory flow during 25%–75% of vital capacity.

TABLE 2. Metabolic and ventilatory responses to constant-power cycling.

	Before Training				After Training				
	Rest	2 min	4 min	End	Rest	2 min	4 min	T_{base}	End
IMT									
$\dot{V}O_2$ (L·min^{-1})	0.30 ± 0.04	1.94 ± 0.18	2.45 ± 0.26	2.93 ± 0.34	0.32 ± 0.03	1.85 ± 0.17*	2.28 ± 0.21*	2.32 ± 0.25*	2.58 ± 0.24*
$\dot{V}O_2$ (mL·kg^{-1}·min^{-1})	4.4 ± 0.3	27.7 ± 2.1	34.8 ± 2.8	41.7 ± 3.9	4.8 ± 0.3	26.5 ± 2.1	32.6 ± 2.4*	36.1 ± 3.1*	36.7 ± 2.9*
$\dot{V}CO_2$ (L·min^{-1})	0.28 ± 0.04	1.94 ± 0.19	2.82 ± 0.30	3.04 ± 0.35	0.31 ± 0.03	1.85 ± 0.20	2.56 0.26*	2.66 0.28*	2.69 0.28*
$\dot{V}_E/\dot{V}O_2$	48.0 ± 4.6	29.2 ± 2.3	34.1 ± 2.6	35.4 ± 2.6	44.3 ± 3.1	30.5 ± 1.9	36.1 ± 2.1*	38.8 ± 1.7	39.1 ± 1.9
$\dot{V}_E/\dot{V}CO_2$	48.6 ± 3.4	29.2 ± 1.8	29.7 ± 2.2	34.1 ± 2.4	47.2 ± 2.6	30.8 ± 1.3	32.3 ± 1.6	37.0 ± 1.5	37.7 ± 1.7
$\dot{V}_E$ (L·min^{-1})	13.9 ± 1.7	55.1 ± 3.6	80.7 ± 5.5	100.7 ± 9.4	14.0 ± 1.3	56.0 ± 4.9	81.5 ± 7.1	97.8 ± 9.9	100.6 ± 9.8
V_T (L)	0.78 ± 0.10	2.08 ± 0.20	2.47 ± 0.25	2.23 ± 0.22	1.04 ± 0.15	1.94 ± 0.14	2.41 ± 0.21	2.21 ± 0.21	2.18 ± 0.22
F_R (breaths per minute)	18 ± 2	28 ± 2	36 ± 2	46 ± 3	17 ± 3	30 ± 2	34 ± 2	45 ± 3	47 ± 2
Ti/TT (%)	44.6 ± 2.9	46.4 ± 1.0	47.3 ± 1.0	47.7 ± 1.1	40.9 ± 1.9	46.4 ± 1.0	47.6 ± 1.0	47.6 ± 0.6	48.0 ± 0.5
RER	0.92 ± 0.04	0.99 ± 0.03	1.15 ± 0.02	1.04 ± 0.01	0.98 ± 0.04	0.98 ± 0.05	1.12 ± 0.02	1.05 ± 0.02	1.04 ± 0.02
HR (bpm)	91.0 ± 4.3	147.7 ± 5.1	162.0 ± 5.1	179.3 ± 4.3	85.3 ± 2.0	147.4 ± 4.4	161.0 ± 4.0	172.9 ± 5.8	175.1 ± 5.1
SaO_2 (%)	95.2 ± 0.6	94.3 ± 0.7	94.6 ± 0.72	94.0 ± 0.5	95.3 ± 0.3	94.8 ± 0.3	93.9 ± 0.5	95.1 ± 0.7	94.4 ± 0.6
Control									
$\dot{V}O_2$ (L·min^{-1})	0.32 ± 0.06	1.84 ± 0.20	2.45 ± 0.18	2.86 ± 0.23	0.32 ± 0.04	1.83 ± 0.18	2.38 ± 0.19	2.73 ± 0.21	2.74 ± 0.22
$\dot{V}O_2$ (mL·kg^{-1}·min^{-1})	3.5 0.4	24.3 ± 1.67	30.6 ± 2.0	35.5 ± 2.0	3.6 ± 0.5	23.7 ± 1.6	29.6 ± 1.9	33.3 ± 2.0	33.7 ± 1.9
$\dot{V}CO_2$ (L·min^{-1})	0.28 ± 0.05	1.88 ± 0.21	2.80 ± 0.19	2.88 ± 0.26	0.29 0.04	1.96 ± 0.18	2.71 ± 0.20	2.91 ± 0.21	2.93 ± 0.21
$\dot{V}_E/\dot{V}O_2$	41.9 ± 3.0	29.7 ± 2.3	33.7 ± 1.3	35.8 ± 0.8	44.5 ± 3.8	30.6 ± 1.5	35.0 ± 1.2	38.7 ± 1.1	37.1 ± 0.6
$\dot{V}_E/\dot{V}CO_2$	47.4 ± 3.1	27.2 ± 2.1	29.8 ± 0.9	36.0 ± 1.5	49.3 ± 4.6	29.8 ± 1.8	31.1 ± 0.8	36.1 ± 1.1	37.3 ± 0.7
$\dot{V}_E$ (L·min^{-1})	12.6 ± 1.6	50.4 ± 5.9	81.5 ± 3.9	101.9 ± 7.3	13.6 ± 1.1	57.2 ± 3.7	83.2 ± 4.0	104.6 ± 6.6	106.4 ± 7.6
V_T (L)	0.88 ± 0.14	2.18 ± 0.15	2.53 ± 0.14	2.42 ± 0.18	0.86 ± 0.09	2.14 ± 0.16	2.49 ± 0.15	2.31 ± 0.11	2.27 ± 0.12
F_R (breaths per minute)	16 ± 1	27 ± 1	34 ± 1	44 ± 2	18 ± 2	28 ± 1	34 ± 1	45 ± 3	47 ± 2
Ti/TT (%)	42.3 ± 2.9	45.4 ± 1.5	47.8 ± 1.0	49.0 ± 1.1	40.9 ± 2.0	46.8 ± 0.6	47.8 ± 1.0	48.8 ± 1.2	48.3 ± 1.3
RER	0.90 ± 0.02	1.02 ± 0.05	1.15 ± 0.03	1.04 ± 0.02	0.91 ± 0.03	1.03 ± 0.04	1.15 ± 0.03	1.08 ± 0.02	1.07 ± 0.02
HR (bpm)	78.2 ± 4.7	148.9 ± 6.8	162.4 ± 6.2	178.4 ± 5.2	85.0 ± 3.3	149.4 ± 5.6	163.9 ± 5.8	176.6 ± 5.3	177.9 ± 4.9
SaO_2 (%)	94.7 ± 0.9	93.9 ± 0.9	93.7 ± 1.1	93.4 ± 1.3	95.0 ± 0.6	93.9 ± 1.2	93.9 ± 1.0	94.1 ± 0.8	94.4 ± 1.0

Values are mean ± SEM.

* Significantly different from before training ($P < 0.05$).

End, end of exercise; SaO_2, arterial oxygen saturation; F_R, breathing frequency; Ti/TT, ratio of inspiratory time to total time; $\dot{V}_E$, minute ventilation; $\dot{V}O_2$, peak oxygen consumption.

end point (T_{base}). Before training, there were no significant differences ($P > 0.05$) between the IMT and PLA groups for any of the measured metabolic and ventilatory variables during exercise.

After the training intervention, the PLA group exhibited no significant change ($P > 0.05$) in any of the measured metabolic and ventilatory variables. However, significant changes ($P < 0.05$) in $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}_E/\dot{V}O_2$ ($P < 0.05$) were observed after the training intervention in the IMT group (Table 2). $\dot{V}O_2$ decreased significantly by 6% at minute 4 and by 12% at the end point of exercise (Fig. 1). Similar changes were also noted for $\dot{V}CO_2$, which was significantly lower ($P < 0.05$) at minute 4 (9%) and at the end of exercise (11%) in the IMT group after the training intervention. All other metabolic and ventilatory variables remained unchanged ($P > 0.05$) after training in the IMT group.

Figure 2 shows the end-inspiratory lung volume (EILV) and EELV responses to exercise, before and after intervention for both the IMT (Fig. 2A) and PLA (Fig. 2B) groups. There was no significant change ($P > 0.05$) in EILV, from before to after intervention, in either group at any time point during exercise. However, there was a significant decrease ($P < 0.05$) in EELV at minute 5 of exercise in the IMT group after the intervention. EELV remained unchanged in the PLA group. The exercising expiratory flow

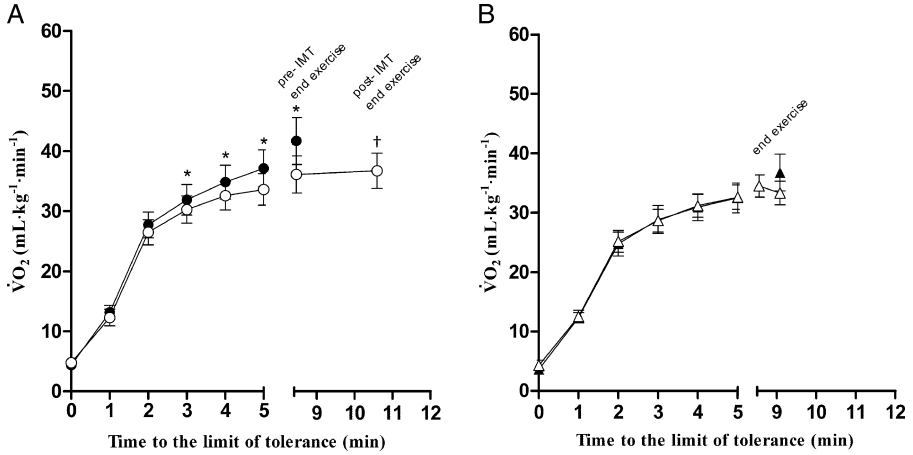

FIGURE 1—$\dot{V}O_2$ response to constant-power cycle ergometry to the limit of tolerance for both IMT (Fig. 1A) and PLA (Fig. 1B) before and after intervention (mean ± SEM). *Shaded circles* indicate before IMT; *open circles* indicate after IMT; *shaded triangles* indicate before PLA; *open triangles* indicate after PLA. *Significant difference from before intervention ($P < 0.05$); †significant difference from T_{base} ($P < 0.05$).

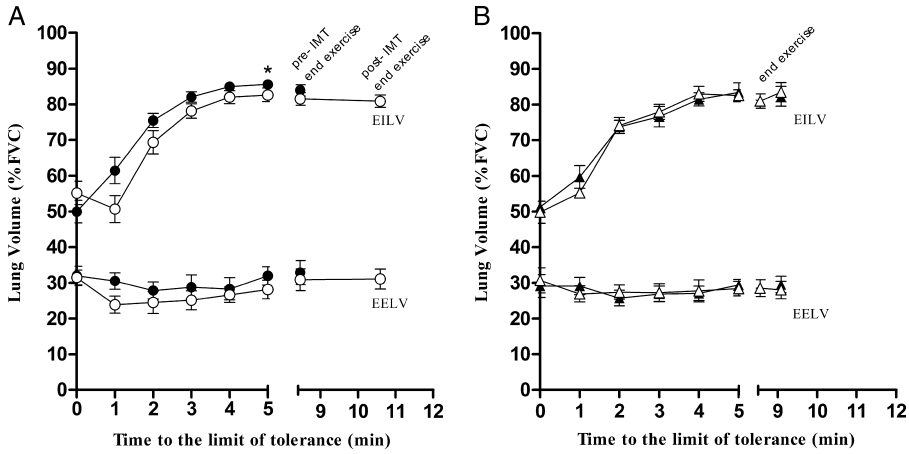

FIGURE 2—EILV and EELV responses to constant-power cycle ergometry to the limit of tolerance for both IMT (Fig. 2A) and PLA (Fig. 2B) before and after intervention (mean ± SEM). *Shaded circles* indicate before IMT; *open circles* indicate after IMT; *shaded triangles* indicate before PLA; *open triangles* indicate after PLA. *Significant difference from before intervention ($P < 0.05$).

rates of both groups are shown in Figure 3. Peak expiratory flow rate (PEFR) and mid-expiratory flow rate remained unchanged ($P > 0.05$) in both the IMT and PLA groups after intervention.

Dyspnea. The perception of dyspnea during exercise in the IMT and PLA groups before the training intervention was not significantly different ($P > 0.05$) (Fig. 4). Before to after IMT, dyspnea was significantly reduced at minute 4 (from 3.7 ± 0.7 to 3.0 ± 0.5) and at the end of exercise (from 5.9 ± 0.8 to 5.1 ± 0.9). No significant differences ($P < 0.05$) in dyspnea ratings were observed in the PLA group from before to after intervention at any time point during exercise.

DISCUSSION

The main findings of the present study are that asthmatic individuals performing 6 wk of IMT 1) increased inspiratory muscle strength and showed attenuation of exercise-induced inspiratory muscle fatigue and 2) reduced oxygen consumption and perception of dyspnea during exercise and 3) increased Tlim during constant-power exercise.

Inspiratory muscle strength increased by 27% in the present study, which is similar in magnitude to that reported previously using pressure threshold IMT in patients with asthma (35,36). An increase in inspiratory muscle strength after IMT has been shown to be associated with an increase in diaphragm thickness (6,8) and hypertrophy of Type I and Type II muscle fibers of the external intercostal muscles (25). Thus, changes in strength reflect structural remodeling within the inspiratory musculature.

Impaired inspiratory muscle strength before exercise has been shown to intensify exertional dyspnea in individuals with asthma (17) and may reduce exercise tolerance in these individuals. (24). Consistent with the results of this study, IMT has previously been shown to reduce the perception of dyspnea during exercise in healthy individuals (28,29,32) and during pressure threshold inspiratory loading in asthmatic individuals (36). The increased inspiratory muscle work associated with increased airway resistance and dynamic hyperinflation is an important predictor of dyspnea during bronchoconstriction (19) and may be related to several factors such as reduced strength of the inspiratory muscles due to their shortened operating length (16), recruitment of additional accessory respiratory muscles (33), and increased inspiratory activity during expiration (20). Furthermore, the intensity of dyspnea during exercise in asthmatic individuals is associated ($r = 0.72$) with the degree of bronchoconstriction, diffusing capacity of the lung, and inspiratory muscle strength at rest (17).

Pulmonary function (FEV_1 and FVC) at rest has previously been shown to increase by ~12% after 3–6 months of IMT in individuals with moderate to severe asthma (35,36). In contrast, neither FEV_1 nor FVC changed after 6 wk of IMT in the present study, which may be because of the duration of the training intervention being insufficient and/or the use of individuals with mild asthma because previous studies have used subjects with moderate to severe asthma (35,36). Therefore, our data suggest that the reduction in the perception of dyspnea that we observed was most likely due to the increase in inspiratory muscle strength after IMT and not due to changes in lung function. Further, this study supports the presence of a negative correlation between change in inspiratory muscle strength and the intensity of dyspnea during an inspiratory loading task after IMT (36). Specifically, increasing inspiratory muscle strength after IMT would require a smaller percentage of maximal force generating capacity to produce the required pressure for a given change in volume, decreasing the central motor command and thus reducing the perception of effort (7).

Exertional dyspnea, which is one of the symptoms of asthma that is associated with impaired exercise tolerance (24), has been shown to be reduced by aerobic training (13,26) and after bronchodilator administration (18). The novel finding of this study is that IMT can improve exercise

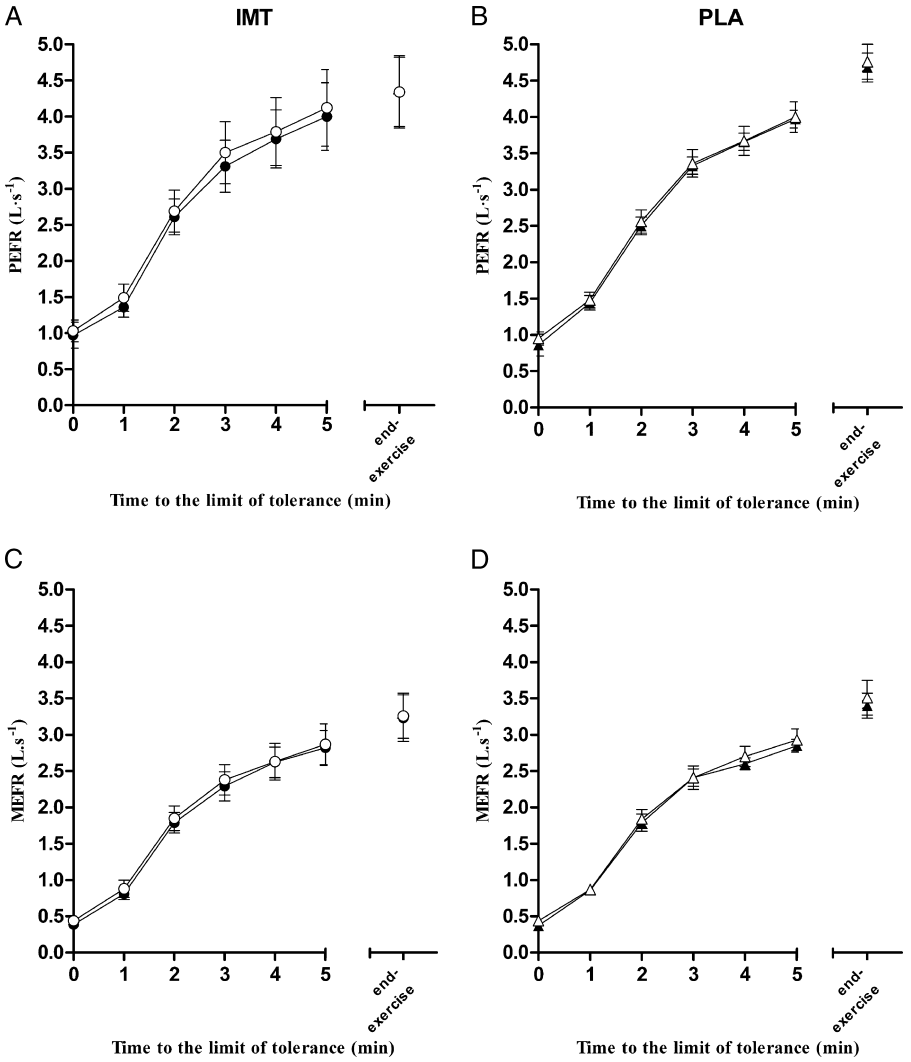

FIGURE 3—PEFR (Fig. 3A and 3B) and mid-expiratory flow rate (Fig. 3C and 3D) responses to constant-power cycle ergometry to the limit of tolerance for both IMT and PLA before and after intervention (mean ± SEM). *Shaded circles* indicate before IMT; *open circles* indicate after IMT; *shaded triangles* indicate before PLA; *open triangles* indicate after PLA.

tolerance and reduce the perception of dyspnea in recreationally active asthmatic individuals with mild asthma, suggesting a potential role for IMT as a complementary intervention in this population. However, it should also be noted that a reduction in the perception of dyspnea may be potentially dangerous in the small population of asthmatic individuals who have a low perception of dyspnea and may cause them to underestimate the severity of asthmatic exacerbations.

Exercise tolerance may be reduced in asthmatic individuals in association with expiratory flow limitation and dynamic hyperinflation (increased EELV) during exercise (18). Participants in the present study demonstrated a progressive increase in EELV with increasing exercise duration; however, participants in the IMT group showed a tendency toward a reduction in the degree of hyperinflation during the first 5 min of exercise after IMT and a significant attenuation at 5 min. There was a similar tendency for EILV to be reduced and for V_T to remain unchanged in the IMT group, after intervention. Previous research has shown that an increase in EELV during voluntary hyperpnea increased the elastic work of breathing and elevated the oxygen cost of ventilation (5). Thus, the reduction in the extent of hyperinflation demonstrated in the present study may be associated with a lower level of inspiratory muscle work and is consistent with the reduction in the oxygen consumption during exercise that was observed in the IMT group after intervention.

Exercise in individuals with chronic asthma can induce airway narrowing that can modify airway function during exercise, thereby reducing the maximal flow–volume loop and, consequently, the ventilatory capacity and ventilatory reserve (15). Emerging evidence suggests that the paradoxical bronchoconstriction occurring in airways of people with asthma after deep inhalation (DI) is normalized when the DI occurs against a pressure threshold load (14). The modification of airway caliber after DI may be explained by the slow-rate crossbridge cycling in airway smooth muscle, termed the "latched state," which results in increased

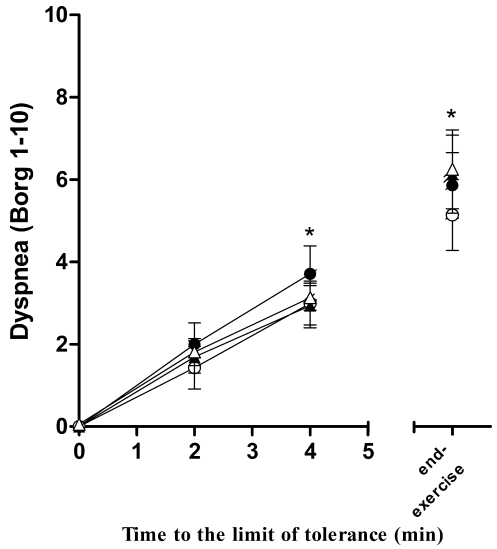

FIGURE 4—Dyspnea ratings during constant-power cycle ergometry to the limit of tolerance for both IMT and PLA before and after intervention (mean ± SEM). *Shaded circles* indicate before IMT; *open circles* indicate after IMT; *shaded triangles* indicate before PLA; *open triangles* indicate after PLA. *Significant difference from before intervention ($P < 0.05$).

smooth muscle stiffness (9,12). It has been suggested that the chronic application of pressure threshold loading may exert a beneficial influence upon airway smooth muscle by releasing it from its latched state (14). Thus, it is possible that IMT may elicit subtle improvements in airway diameter and airflow limitation during exercise, thereby reducing dynamic hyperinflation and increasing exercise capacity. The fact that peak and mean inspiratory flow rates were unchanged after IMT (Fig. 3), yet EELV was slightly reduced (Fig. 2), supports this notion, but further investigation is necessary to confirm this hypothesis.

The finding that the oxygen cost of constant-power submaximal cycling exercise at ~70% $\dot{V}O_{2max}$ was reduced after IMT is consistent with previous studies that have also shown a reduction in $\dot{V}O_2$ during running (23), cycling (2), and in hypoxia (6) after IMT in healthy individuals. A reduced $\dot{V}O_2$ may relate in part to improvements in respiratory muscle efficiency, which can be quantified as the additional oxygen consumption required for a given increase in ventilatory work (4). During incremental inspiratory loading, respiratory muscle efficiency has been shown to be lower in asthmatic individuals compared with healthy individuals and improved after the administration of a bronchodilator, which resulted in a reduction in hyperinflation and a corresponding increase of inspiratory muscle strength (39). Specifically, an increase in inspiratory muscle strength after IMT may decrease the relative intensity for a given amount of work, resulting in recruitment of fewer muscle fibers or delaying the recruitment of accessory respiratory muscles, thereby lowering the O_2 requirement of the respiratory musculature. Thus, the reduced whole-body $\dot{V}O_2$ after IMT in the present study may relate to changes in inspiratory muscle strength and/or changes in flow-resistive work due to altered operating lung volumes during exercise.

Consistent with previous studies of IMT in healthy people, we observed an attenuation of exercise-induced inspiratory muscle fatigue, despite an increase in the time to the limit of tolerance (11,30,32). There are two possible explanations for this: 1) stronger inspiratory muscles operate at a lower relative intensity after exercise, which improves their fatigue resistance, and 2) IMT attenuates the functional weakening of the inspiratory muscles that arises because of dynamic hyperinflation, which would otherwise lead to the recruitment of high-force–generating highly fatigable accessory muscle fibers, which exacerbate the development of respiratory muscle fatigue (33). The attenuation of inspiratory muscle fatigue may also have contributed to the improvement in exercise Tlim by delaying the onset of the inspiratory muscle metaboreflex (31).

CONCLUSIONS

This study has shown that 6 wk of IMT in individuals with mild to moderate asthma significantly increased inspiratory muscle strength, reduced inspiratory muscle fatigue, improved exercise tolerance, and reduced the perception of dyspnea during cycling exercise at ~70% $\dot{V}O_{2max}$ to the limit of tolerance. These data suggest that IMT may be a helpful adjunct to asthma management and has the potential to improve participation and adherence to exercise training in this group. However, it should also be noted that the perception of breathlessness is also an important signal of bronchoconstriction, and thus, caution should be exercised if this symptom is abnormally low.

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