The Effect of Preexercise Expiratory Muscle Loading on Exercise Tolerance in Healthy Men

TIM A. HARDY[1], STEPHEN C. HOW[2], and BRYAN J. TAYLOR[3,4]

[1]Faculty of Biological Sciences, School of Biomedical Sciences, University of Leeds, Leeds, UNITED KINGDOM; [2]School of Sport and Exercise, University of Gloucestershire, Gloucester, UNITED KINGDOM; [3]School of Sport and Health Sciences, College of Life and Environmental Sciences, University of Exeter, Exeter, UNITED KINGDOM; and [4]Department of Cardiovascular Medicine, Mayo Clinic Florida, Jacksonville, FL

ABSTRACT

HARDY, T. A., S. C. HOW, and B. J. TAYLOR. The Effect of Preexercise Expiratory Muscle Loading on Exercise Tolerance in Healthy Men. *Med. Sci. Sports Exerc.*, Vol. 53, No. 2, pp. 421–430, 2021. **Purpose:** Acute nonfatiguing inspiratory muscle loading transiently increases diaphragm excitability and global inspiratory muscle strength and may improve subsequent exercise performance. We investigated the effect of acute expiratory muscle loading on expiratory muscle function and exercise tolerance in healthy men. **Methods:** Ten males cycled at 90% of peak power output to the limit of tolerance (T_{LIM}) after 1) 2 × 30 expiratory efforts against a pressure-threshold load of 40% maximal expiratory gastric pressure (Pga_{MAX}) (EML-EX) and 2) 2 × 30 expiratory efforts against a pressure-threshold load of 10% Pga_{MAX} (SHAM-EX). Changes in expiratory muscle function were assessed by measuring the mouth pressure (PE_{MAX}) and Pga_{MAX} responses to maximal expulsive efforts and magnetically evoked (1 Hz) gastric twitch pressure (Pga_{tw}). **Results:** Expiratory loading at 40% of Pga_{MAX} increased PE_{MAX} (10% ± 5%, P = 0.001) and Pga_{MAX} (9% ± 5%, P = 0.004). Conversely, there was no change in PE_{MAX} (166 ± 40 vs 165 ± 35 cm H_2O, P = 1.000) or Pga_{MAX} (196 ± 38 vs 192 ± 39 cm H_2O, P = 0.215) from before to after expiratory loading at 10% of Pga_{MAX}. Exercise time was not different in EML-EX versus SHAM-EX (7.91 ± 1.96 vs 8.09 ± 1.77 min, 95% CI = −1.02 to 0.67, P = 0.651). Similarly, exercise-induced expiratory muscle fatigue was not different in EML-EX versus SHAM-EX (−28% ± 12% vs −26% ± 7% reduction in Pga_{tw} amplitude, P = 0.280). Perceptual ratings of dyspnea and leg discomfort were not different during EML-EX versus SHAM-EX. **Conclusion:** Acute expiratory muscle loading enhances expiratory muscle function but does not improve subsequent severe-intensity exercise tolerance in healthy men. **Key Words:** EXPIRATORY MUSCLE WARM-UP, GASTRIC TWITCH PRESSURE, EXPIRATORY MUSCLE FATIGUE, EXERTIONAL DYSPNEA AND LEG DISCOMFORT, EXERCISE PERFORMANCE

Severe-intensity whole-body exercise (≥85% of maximum O_2 uptake [$\dot{V}O_{2max}$]) sustained to volitional exhaustion elicits inspiratory muscle fatigue in healthy men and women (1,2). Such inspiratory muscle fatigue plays a role in limiting exercise tolerance, likely through an exacerbation in the perception of dyspnea and/or an increase in the severity of exercise-induced limb locomotor muscle fatigue secondary to a reflexively mediated reduction in leg blood flow (3–7). Accordingly, the identification of interventions that may overcome the aforementioned respiratory system limitation to exercise tolerance is of interest.

Acute, submaximal muscular contractions can elicit a phenomenon known as postactivation performance enhancement (PAPE), which is characterized by a transient improvement in the maximum voluntary force generating capacity of the target muscle(s) (8). Previously, it has been shown that a bout of inspiratory pressure-threshold loading (~60 breaths at ~40% of maximal inspiratory mouth pressure) can increase peripheral excitability of the diaphragm, improve the coordination of contraction between the diaphragm and the accessory intercostal muscles during maximal inspiratory efforts, and acutely enhance global inspiratory muscle strength (9–15). In addition to an apparent enhancement in pulmonary function (16), it has been reported in some (11–13,17,18) but not all previous studies (10,15,19) that submaximal inspiratory muscle loading, or an inspiratory muscle "warm-up," can facilitate a significant improvement in subsequent exercise performance. It has been suggested that any such inspiratory muscle warm-up induced improvement in exercise performance may be due to an attenuation in perceived respiratory effort during exercise (11,12,17), an improvement in locomotor muscle tissue oxygenation (20), and/or a decrease in the severity of exercise-induced inspiratory muscle fatigue (11).

Exhaustive severe-intensity exercise also causes fatigue of the expiratory abdominal muscles in healthy men, as evidenced by a significant pre- to postexercise reduction (~12%–25%) in gastric twitch pressure (21,22). Previously, we and others have shown that prior induction of expiratory muscle fatigue impairs

Address for correspondence: Bryan J. Taylor, Ph.D., Department of Cardiovascular Medicine, Mayo Clinic, Florida, Jacksonville, FL 32224; E-mail: taylor.bryan@mayo.edu.
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subsequent whole-body exercise tolerance in healthy adults, primarily via an increased severity of exercise-induced leg locomotor muscle fatigue and a heightened perception of leg discomfort (23,24). Interestingly, relative to control conditions, augmentation of expiratory muscle work during moderate- to heavy-intensity exercise is associated with a greater increase in muscle sympathetic nerve activity, an increase in intercostal muscle blood flow, and a decrease in quadriceps muscle blood flow (25,26). In combination, the aforementioned findings suggest that, as for the inspiratory muscles, elevated levels of expiratory muscle work and expiratory muscle fatigue limit exercise tolerance likely through a sympathetically mediated vasoconstrictor influence that impairs blood flow and oxygen delivery to the exercising limbs.

Despite the important contribution of the expiratory muscles to the hyperpnea of exercise (27,28), and their apparent role in limiting exercise tolerance (23,24), it is currently unknown whether prior submaximal expiratory threshold loading (i.e., an expiratory muscle "warm-up") affects subsequent exercise performance. Accordingly, the aim of the present study was to determine the effect of expiratory muscle loading relative to a sham condition on 1) expiratory muscle function, 2) exercise tolerance, 3) perceptual responses to exercise, and 4) severity of exercise-induced expiratory muscle fatigue in healthy men. We hypothesized that acute submaximal expiratory threshold loading at 40% of maximal expiratory gastric pressure (Pga_{MAX}) would enhance expiratory muscle function and improve subsequent exercise tolerance primarily through a reduction in the perception of dyspnea and/or a reduction in the severity of exercise-induced expiratory muscle fatigue.

METHODS

Subjects. Ten healthy, nonsmoking, recreationally active men participated in the study (mean ± SD; age, 26 ± 5 yr; stature, 175 ± 5 cm; body mass, 72.8 ± 10.8 kg). The subjects had resting pulmonary function within normal limits (% predicted ± SD: forced vital capacity [FVC], 113% ± 18%; forced expiratory volume in 1 s [FEV_1], 105% ± 15%; FEV_1/FVC, 95% ± 7%; maximum voluntary ventilation [MVV], 108% ± 7%]. Each subject provided written informed consent to the study procedures, which were approved by the University of Exeter Sport and Health Sciences Research Ethics Committee (application reference no. 160217/B/03).

Experimental procedures. The experimental procedures were conducted at the School of Sport and Health Sciences at the University of Exeter. Each subject visited the laboratory on three separate occasions that were each separated by at least 48 h but not longer than 1 wk. The subjects abstained from food for 3 h, caffeine for 12 h, and exercise for 24 h before each visit. At the first visit, maximal dynamic pulmonary function (FVC, FEV_1, and MVV) was determined according to standard procedures (29) before the subjects performed maximal incremental exercise (35 W every 3 min, starting at 95 W) on an electromagnetically braked cycle ergometer (Excalibur; Lode, Groningen, The Netherlands) to determine peak work rate (W_{peak}) and the associated cardiopulmonary responses. Subjects were then familiarized with the magnetic nerve stimulation technique and the expiratory pressure-threshold loading protocols, described below. During the next two visits, using a randomized, single-blind (subjects), placebo-controlled design, the subjects performed constant-load cycle exercise at 90% of W_{peak} preceded by either 1) 2 × 30 expiratory efforts against a pressure-threshold load of 40% maximal expiratory gastric pressure (Pga_{MAX}) (EML-EX) or 2) 2 × 30 expiratory efforts against a pressure-threshold load of 10% Pga_{MAX} (SHAM-EX). Exercise time to the limit of tolerance (T_{LIM}) was recorded for each trial. Expiratory muscle function was assessed before and immediately after each bout of expiratory pressure-threshold loading (EML and SHAM) and at 5 min after each of the exercise trials (EML-EX and SHAM-EX).

Magnetic nerve stimulation. Gastric pressure (Pga) and esophageal pressure (Pes) were measured using two balloon-tipped catheters (Ackrad Laboratories, Cooper Surgical, Berlin, Germany) that were positioned and filled as described previously (23,30). Each catheter was connected to a differential pressure transducer (Validyne DP45, Northridge, CA; range of ±229 cm H_2O) that was calibrated across the physiological range using a digital differential manometer (model TPI 621; JMW Digital Limited, Harlow, UK). With subjects sat facing an inclined bench with hips flexed and chest supported, magnetic stimuli (1 Hz) were delivered to the thoracic nerve roots between the 8th (T8) and the 11th (T11) thoracic vertebrae via a double 70-mm coil powered by a magnetic stimulator (Magstim 200^2; Magstim Company Ltd., Whitland, UK), as described before (23,30,31). The area of stimulation that evoked the greatest Pga_{tw} was located and marked for use for all subsequent stimulations. All stimulations were delivered at 100% of the stimulator's power output and at relaxed end expiration. End-expiratory Pes and Pga were not different across time for either EML-EX or SHAM-EX, indicating that all stimulations were delivered at the same lung volume and at the same abdominal muscle length throughout the study (Table 1). To determine whether the depolarization of the thoracic nerves in response to magnetic stimulation was supramaximal, three single twitches were obtained at 50%, 60%, 70%, 80%, 85%, 90%, 95%, and 100% of the stimulator's maximum power output. Despite a tendency for the group mean response to level off, a clear plateau in Pga_{tw} was not evident with increasing stimulator power output (Fig. 1). That is, the depolarization of the thoracic nerve roots in response to 1-Hz magnetic stimulation at 100% of the stimulator's power output was likely submaximal.

Expiratory abdominal muscle electromyography. EMG was recorded from the rectus abdominis (RA) and external oblique (EO) muscles using active bipolar bar skin-surface electrodes with single differential configuration (DE-2.1; DELSYS Inc., Boston, MA). A pair of electrodes was placed over the belly of each muscle on the right-hand side of the abdomen. The pairs of electrodes were positioned within 2 cm superior and 2–4 cm lateral to the umbilicus for RA and 4–6 cm medial to the iliac crest for EO. A ground electrode was placed on the bony process of the anterior superior iliac

TABLE 1. Measurements of expiratory muscle function before and after EML and SHAM, and at 5 min after EML-EX and SHAM-EX.

	EML-EX			SHAM-EX		
	Pre-EML	Post-EML	5-Min Postexercise	Pre-SHAM	Post-SHAM	5-Min Postexercise
Pga_{tw}, cm H_2O	39.0 ± 6.2	41.3 ± 6.4	29.4 ± 6.0**	39.5 ± 5.8	37.7 ± 4.1	27.7 ± 4.0**
EE Pes, cm H_2O	–5.4 ± 3.6	–5.5 ± 2.2	–5.2 ± 2.1	–5.6 ± 2.8	–5.7 ± 2.6	–6.1 ± 3.8
EE Pga, cm H_2O	15.3 ± 9.5	14.1 ± 9.2	11.9 ± 8.8	13.1 ± 6.2	13.0 ± 6.6	10.4 ± 6.4
CT, ms	153 ± 15	146 ± 16	141 ± 13	147 ± 17	142 ± 18	136 ± 14
MRPD/Pga_{tw}, s	11.4 ± 1.3	11.3 ± 1.5	12.6 ± 2.4	12.2 ± 1.5	12.2 ± 2.2	13.5 ± 1.8
MRR/Pga_{tw}, s	–4.2 ± 0.8	–4.3 ± 0.9	–4.5 ± 1.4	–4.4 ± 0.5	–4.0 ± 0.7*	–4.7 ± 1.1
PE_{MAX}, cm H_2O	163 ± 37	178 ± 42*	146 ± 32**	166 ± 40	165 ± 35	145 ± 25**
Pga_{MAX}, cm H_2O	180 ± 31	196 ± 32*	165 ± 39**	196 ± 38	192 ± 39	162 ± 30**
RA RMS, μV	274 ± 197	282 ± 191	–	280 ± 100	286 ± 83	–
EO RMS, μV	513 ± 305	515 ± 243	–	579 ± 374	600 ± 353	–

Values are presented as group mean ± SD for 10 subjects.
*$P < 0.05$, significantly different versus Pre-EML or Pre-SHAM.
**$P < 0.05$, significantly different versus Post-EML or Post-SHAM.
EML-EX, exercise following acute expiratory muscle loading at 40% of maximal expiratory gastric pressure (Pga_{MAX}); SHAM-EX, exercise following acute expiratory muscle loading at 10% of Pga_{MAX}; EML, acute expiratory muscle loading at 40% of Pga_{MAX}; SHAM, acute expiratory muscle loading at 10% of Pga_{MAX}; Pga_{tw}, gastric twitch pressure; EE Pes, end-expiratory esophageal pressure at the initiation of Pga_{tw}; EE Pga, end-expiratory gastric pressure at the initiation of Pga_{tw}; CT, contraction time; MRR, maximal relaxation rate; PE_{MAX}, maximal expiratory mouth pressure during a maximal expulsive effort; Pga_{MAX}, maximal expiratory gastric pressure during a maximal expulsive effort; RMS, root mean square amplitude of surface electromyographic signal.

crest. After the verification of correct electrode positioning (via the EMG response to a forced exhalation), the electrodes were secured in place using double-sided adhesive interfaces and hypoallergenic medical tape. The position of each electrode was marked with indelible ink to ensure that it was placed in the same location at subsequent visits.

Data acquisition. The Pga and Pes signals were passed through a carrier demodulator (Validyne Model CD15, Northridge, CA) and digitized at 150 Hz (Micro 1401-3; Cambridge Electronic Design, Cambridge, UK). The EMG signals were preamplified (×1000), band-pass filtered (20–450 Hz) (Bagnoli-8 Desktop EMG System, DELSYS Inc.), and digitized at a sampling rate of 2 kHz (Micro 1401-3, Cambridge Electronic Design). The pressure and the EMG signals were acquired and analyzed using commercially available software (Spike 2 version 8.1, Cambridge Electronic Design).

Expiratory muscle function. Expiratory muscle function was assessed before and immediately after each bout of expiratory pressure-threshold loading (EML and SHAM) and at 5 min after each of the exercise trials. At each assessment time point, subjects performed six maximal expulsive maneuvers. The maneuvers were initiated from total lung capacity, lasted ~5 s, and were separated by ~20 s. Potentiated Pga_{tw} was determined by measuring the Pga_{tw} response to 1-Hz stimuli delivered ~5 s after each maximal expulsive effort. The degree of potentiation was slightly smaller after the first and, to a lesser extent, the second expulsive maneuver. Accordingly, the first two measures of potentiated Pga_{tw} were discarded for each assessment of expiratory muscle function. Any twitch response that was initiated from an unstable end-expiratory Pes and/or Pga or in the presence of subject "bracing" (evidenced by RA and/or EO EMG activity immediately before the stimulation) was excluded from subsequent analysis.

Each gastric twitch pressure response was assessed for amplitude (baseline to peak), maximal rate of pressure development (MRPD), maximal relaxation rate, and contraction time (CT). Expiratory mouth pressure during each expulsive effort was measured using a hand-held mouth pressure meter (Micro RPM; Vyaire Medical Products Ltd., Hampshire, UK), and PE_{MAX} was defined as the highest value recorded of those that varied by ≤5%. Similarly, the peak Pga response (across 1 s) to each maximal expulsive effort was identified, and Pga_{MAX} was recorded as the highest Pga value of those that varied by ≤5%. The RA and the EO EMG signals recorded during the 1-s periods in which peak Pga was identified were analyzed in the time domain as root mean square amplitude with a time constant of 0.25 ms.

The coefficient of variation and the intraclass correlation coefficient for MRPD corrected for Pga_{tw} (MRPD/Pga_{tw}) were 8.4% and 0.226, respectively; all other within-day between-occasion reproducibility coefficients for our measures of expiratory muscle function were ≤6.3% for coefficient of variation and ≥0.555 for intraclass correlation coefficient, which is similar to previous reports (21,23).

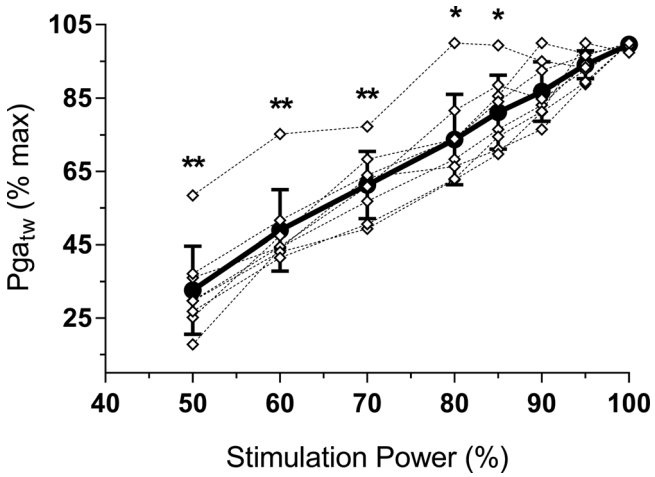

FIGURE 1—Unpotentiated gastric twitch pressure (Pga_{tw}) during magnetic stimulation of the thoracic nerve roots (1 Hz) at different power outputs of the magnetic stimulator. Pga_{tw} is expressed as a percentage of the values generated at 100% power output (% max). Values are presented as mean ± SD for eight subjects. *$P < 0.05$ and **$P < 0.01$, significantly different versus at 100% of stimulator power output (repeated-measures ANOVA with Bonferroni correction).

Expiratory pressure-threshold loading. The expiratory pressure-threshold loading consisted of two sets of 30 breaths at either 1) 40% of Pga_{MAX} (EML) or 2) 10% of Pga_{MAX} (SHAM) (PowerLung Trainer; PowerLung Inc., Houston, TX). A 1-min rest was allowed between each set of loaded breaths. We ensured that inspiration was unimpeded by removing the inspiratory valve tensioning spring from the device. The target expiratory pressure was displayed on a computer screen, and the subjects were instructed to perform dynamic expiratory efforts starting near total lung capacity and terminating toward residual volume for each breath. No additional instructions related to breathing pattern during the pressure-threshold loading were given.

Exercise responses. The time between the completion of EML or SHAM and the start of exercise was standardized at 4 min. First, the subjects cycled for 2 min at 40%, 2 min at 50%, and 1 min at 60% of W_{peak} before the work rate was increased to 90% of W_{peak}. As such, the interval between the completion of EML or SHAM and the initiation of the criterion exercise was constant at 9 min. Each subject pedaled at a self-selected cadence and maintained this cadence throughout. The point of exercise intolerance was defined as the inability to maintain pedal cadence above 60 rpm. Ventilatory and pulmonary gas exchange indices (Metalyzer 3B; Cortex Biophysik GmbH, Leipzig, Germany) were obtained breath by breath and averaged over a 30-s period at rest, during the last 30 s of each full minute of exercise, and during the final 30 s of exercise. Similarly, HR (Polar Vantage NV; Polar Electro Oy, Kempele, Finland) was measured beat by beat and averaged over a 30-s period at rest, during the last 30 s of each full minute of exercise, and during the final 30 s of exercise. Capillary blood was sampled from an earlobe at rest, every 2 min during exercise, and within 10 s of exercise termination for the subsequent determination of hemolyzed blood lactate concentration (YSI 2300; Yellow Springs Instruments, Yellow Springs, OH). RPE (dyspnea and leg discomfort) were obtained at rest, at 1 min of exercise, every 2 min thereafter, and within 10 s of exercise termination using Borg's modified CR10 scale.

Statistical analyses. Changes in expiratory muscle function in response to acute expiratory pressure-threshold loading (EML and SHAM) and constant-load cycle exercise (EML-EX and SHAM-EX) were assessed using two-way repeated-measures ANOVA, with the time and interaction effects (intervention–time) inspected. When a significant effect of time was observed, a one-way repeated-measures ANOVA with Bonferroni correction was used to assess changes in expiratory muscle function across time within EML-EX and SHAM-EX (pre-EML or pre-SHAM vs post-EML or post-SHAM vs 5 min postexercise). Paired samples *t*-test was used to compare exercise time to the limit of tolerance (T_{LIM}) between trials (EML-EX vs SHAM-EX). Similarly, paired samples *t*-test was used to compare the pre- to postexercise percent changes in expiratory muscle function (i.e., the severity of exercise-induced expiratory muscle fatigue) between trials (EML-EX vs SHAM-EX). A two-way repeated-measures ANOVA was used to compare the absolute physiological responses to EML-EX versus SHAM-EX. When a significant interaction effect (intervention–time) was observed, paired samples *t*-test was used to compare the physiological responses to exercise at equivalent time points between the two trials. Changes in the perceptual response to exercise across time in EML-EX and SHAM-EX were assessed using the Friedman test. Wilcoxon signed rank test was used to compare the perceptual responses to exercise at equivalent time points between the two trials. The acceptable type I error was set at $P < 0.05$. Data are expressed as group mean ± SD. Statistical analyses were performed using SPSS version 22.0 for Windows (SPSS, Chicago, IL).

RESULTS

Maximal incremental exercise test. During maximal incremental exercise, peak $\dot{V}O_2$, power, $\dot{V}_E$, HR, and RER were 3.68 ± 0.58 L·min^{-1} (50.0 ± 3.4 mL·kg^{-1}·min^{-1}), 265 ± 41 W, 159 ± 22 L·min^{-1}, 187 ± 6 bpm, and 1.11 ± 0.08, respectively.

Effect of EML and SHAM on expiratory muscle function. There was a significant main effect of time for PE_{MAX} ($F = 40.0$, $P < 0.001$), Pga_{MAX} ($F = 59.5$, $P < 0.001$) and Pga_{tw} amplitude ($F = 102.0$, $P < 0.001$). There was also a significant interaction effect (intervention-time) for PE_{MAX} ($F = 7.0$, $P = 0.008$) and Pga_{MAX} ($F = 6.8$, $P = 0.022$), but not for Pga_{tw} amplitude ($F = 3.4$, $P = 0.058$). There was no interaction effect for contraction time or the pressure development and relaxation responses for the evoked twitches (all $F \leq 1.5$, $P \geq 0.166$).

There was no change in any measure of expiratory muscle function in response to SHAM (Fig. 2, Table 1). By contrast, there was a 10% ± 5% (95% CI = 7.72 to 23.49, $P = 0.001$) and a 9% ± 5% (95% CI = 7.61 to 23.59, $P = 0.004$) increase in group mean PE_{MAX} and Pga_{MAX}, respectively, from before to after EML (Fig. 2, Table 1). The group mean Pga_{tw} and the abdominal muscle EMG responses to maximal expulsive efforts were not different before versus after EML (Table 1).

Exercise tolerance. Individual subject T_{LIM} values for SHAM-EX and EML-EX are shown in Figure 3. Only 5 of the 10 subjects cycled longer in EML-EX versus SHAM-EX, and group mean T_{LIM} was not different in EML-EX versus SHAM-EX (7.91 ± 1.96 vs 8.09 ± 1.77 min, 95% CI = −1.02 to 0.67, $P = 0.651$). Importantly, the order in which each subject performed the exercise trials did not affect this finding. In subjects who performed EML-EX first, T_{LIM} was not different in EML-EX versus SHAM-EX (7.60 ± 0.89 vs 7.89 ± 1.94 min, 95% CI = −1.75 to 1.18, $P = 0.616$) (Fig. 3). Similarly, T_{LIM} was not different in EML-EX versus SHAM-EX in the subjects who performed SHAM-EX first (8.23 ± 2.76 vs 8.29 ± 1.78 min, 95% CI = −1.70 to 1.57, $P = 0.919$) (Fig. 3).

Exercise-induced expiratory muscle fatigue. In EML-EX, there was a significant reduction in Pga_{tw} from post-EML to 5 min after exercise (Fig. 4, Table 1). Similarly, PE_{MAX} and Pga_{MAX} during the maximal expulsive maneuvers decreased from post-EML to 5 min after exercise (Fig. 4, Table 1). In

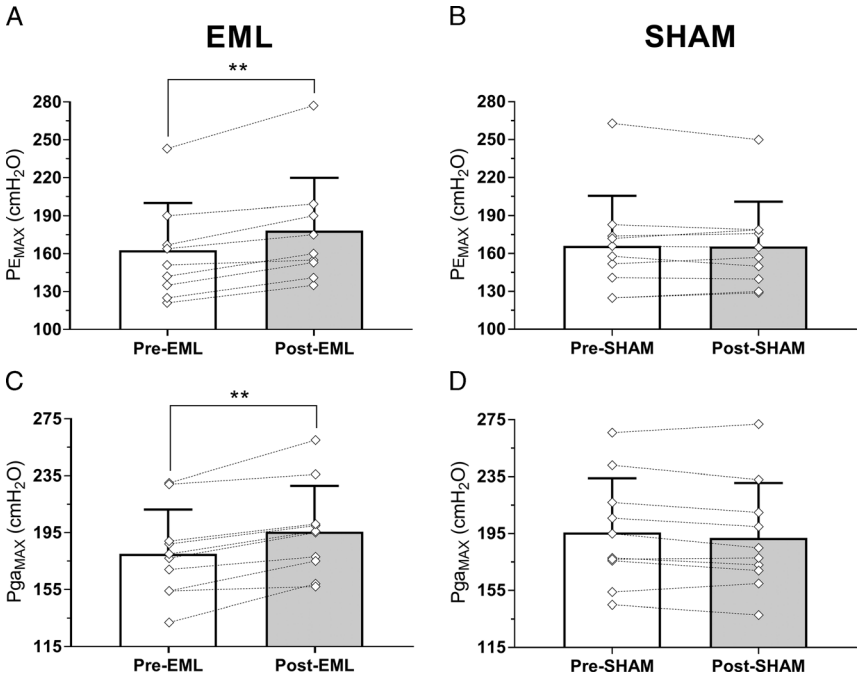

FIGURE 2—Individual subject (*dashed lines*) and group mean (*solid bars*) maximal expiratory mouth pressure (PE_{MAX}) and maximal expiratory gastric pressure (Pga_{MAX}) during the maximal expulsive efforts before and immediately after acute expiratory muscle loading at 40% of Pga_{MAX} (EML) (left-hand panels A and C) and acute expiratory muscle loading at 10% of Pga_{MAX} (SHAM) (right-hand panels B and D). Values are presented as group mean ± SD for 10 subjects. **$P < 0.01$, significantly different versus pre-EML.

SHAM-EX, Pga_{tw}, PE_{MAX}, and Pga_{MAX} were reduced from post-SHAM to 5 min after exercise (Fig. 4, Table 1).

The pre- to postexercise reductions in Pga_{tw}, PE_{MAX}, and Pga_{MAX} were not different for EML-EX versus SHAM-EX (Pga_{tw}: −28% ± 12% vs −26% ± 7%, 95% CI = −5.71 to 1.86, $P = 0.280$; PE_{MAX}: −18% ± 3% vs −12% ± 8%, 95% CI = −11.89 to 0.28, $P = 0.058$; Pga_{MAX}: −17% ± 7% vs −15% ± 7%, 95% CI = −8.50 to 5.09, $P = 0.581$). That is, the magnitude of exercise-induced expiratory muscle fatigue was not different between the two exercise trials.

Exercise responses. The cardiorespiratory and perceptual responses to exercise in EML-EX and SHAM-EX are shown in Figure 5. In EML-EX, $\dot{V}O_2$, $\dot{V}_E$, and f_R reached 97% ± 5%, 98% ± 5%, and 99% ± 4%, respectively, of maximum values obtained during the initial maximal incremental exercise test. Throughout exercise, no measure of cardiopulmonary function was different between EML-EX and SHAM-EX (Fig. 5). The perception of dyspnea and the perception of leg discomfort increased with time during both EML-EX and SHAM-EX (*all* $P < 0.001$). However, neither dyspnea nor leg discomfort was different between EML-EX versus SHAM-EX at any time point during exercise (Fig. 5).

DISCUSSION

Main Findings

In the present study, we investigated the effect of an expiratory muscle "warm-up" on subsequent exercise tolerance in healthy men. We hypothesized that acute expiratory muscle pressure-threshold loading (60 breaths) at 40% of maximal expiratory gastric pressure (EML) would enhance expiratory muscle function and improve subsequent exercise tolerance, primarily through a reduction in the perception of dyspnea and/or a reduction in the severity of exercise-induced expiratory

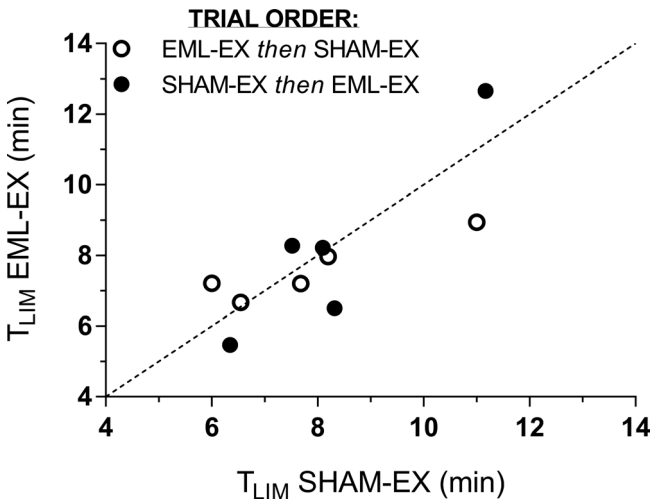

FIGURE 3—An identity plot showing individual subject exercise time to the limit of tolerance (T_{LIM}) for exercise following acute expiratory muscle loading at 40% of maximal expiratory gastric pressure (Pga_{MAX}) (EML-EX) and exercise following acute expiratory muscle loading at 10% of Pga_{MAX} (SHAM-EX). *Open circles* represent subjects who performed EML-EX (first trial) then SHAM-EX (second trial); *closed circles* represent subjects who performed SHAM-EX (first trial) then EML-EX (second trial).

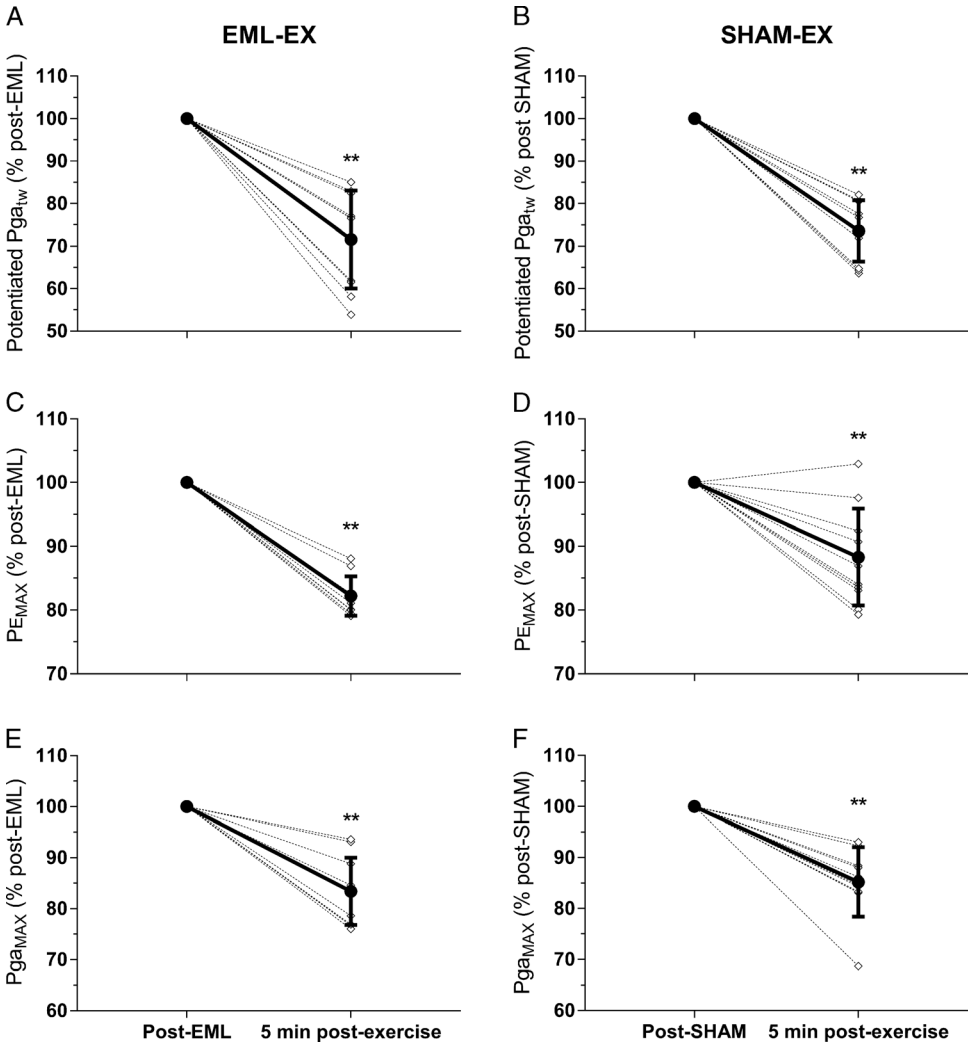

FIGURE 4—Individual subject (*dashed lines*) and group mean (*solid lines*) potentiated gastric twitch pressure (Pga_{tw}, A and B), maximal expiratory mouth pressure during a maximal expulsive effort (PE_{MAX}, C and D), and maximal expiratory gastric pressure during a maximal expulsive effort (Pga_{MAX}, E and F) before and 5 min after exercise in EML-EX (left-hand panels) and SHAM-EX (right-hand panels). Pga_{tw}, PE_{MAX}, and Pga_{MAX} are expressed as a percentage of the values measured before exercise. Before exercise measures represent measures made immediately after acute expiratory muscle loading at 40% of Pga_{MAX} (post-EML) or 10% of Pga_{MAX} (post-SHAM). Values are presented as group mean ± SD for 10 subjects. **$P < 0.01$, significantly different versus before exercise.

muscle fatigue. We found that, relative to a sham condition, EML increased maximal expiratory mouth pressure (PE_{MAX}) and maximal expiratory gastric pressure (Pga_{MAX}) by ~9%–10% but did not affect subsequent exercise time to the limit of tolerance (T_{LIM}), the perceptions of dyspnea or leg discomfort during exercise, or the severity of exercise-induced expiratory muscle fatigue. In combination, these findings suggest that acute expiratory muscle loading (i.e., an expiratory muscle warm-up) can transiently enhance expiratory muscle function but does not improve subsequent exercise tolerance in healthy men.

Technical Considerations

Did a lack of subject familiarization affect our outcome measure T_{LIM}? One concern is that we did not familiarize the subjects with our T_{LIM} protocol before investigating the effect of EML on exercise tolerance. As such, it is conceivable that the test-to-test variability and/or any learning effect from the first to the second exercise test may have exceeded, and thus masked, any improvement in T_{LIM} that resulted from the EML intervention. To counteract any potential learning effect, we randomized and counterbalanced the order of EML-EX and SHAM-EX trials between subjects. More importantly, we show no evidence of a systematic improvement in T_{LIM} in the second-performed exercise trial compared with the first-performed exercise trial across all subjects (see Fig. 3). Indeed, T_{LIM} for SHAM-EX was not systematically longer in the subjects who performed EML-EX first. Perhaps even more tellingly, in subjects who performed EML-EX second, where there was the potential for a combined ergogenic and learning effect on exercise tolerance, T_{LIM} was still not different between EML-EX and SHAM-EX. Moreover, of the five subjects

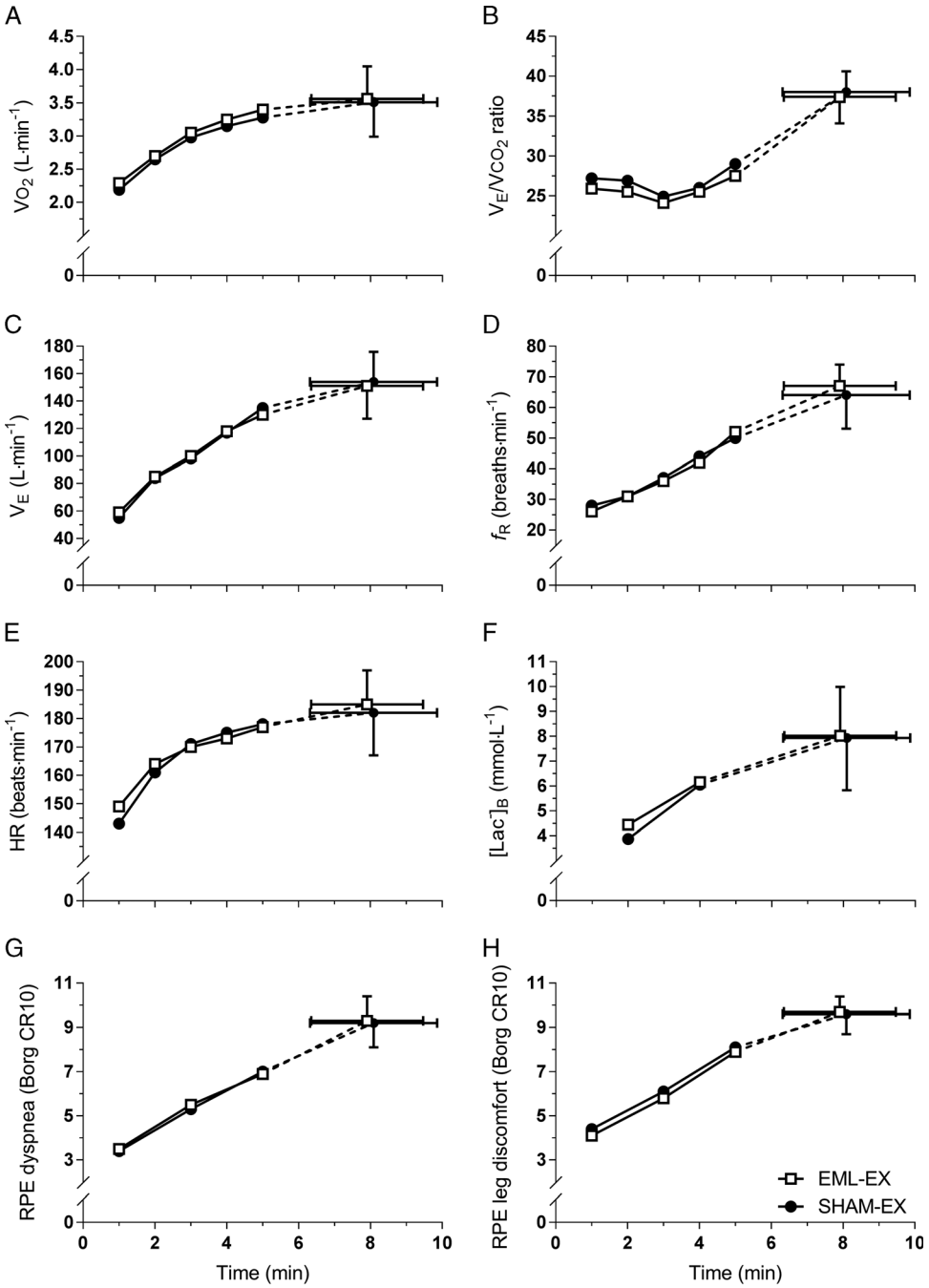

FIGURE 5—Cardiorespiratory, metabolic, and perceptual responses to exercise in EML-EX (*open squares*) and SHAM-EX (*closed circles*). $\dot{V}O_2$, oxygen uptake; $\dot{V}_E$, minute ventilation; $\dot{V}CO_2$, carbon dioxide production; f_R, breathing frequency; HR, heart rate; $[Lac^-]_B$, blood lactate concentration; RPE, rating of perceived exertion.

who "improved" T_{LIM} with EML (i.e., T_{LIM} was longer in EML-EX vs SHAM-EX), only three performed EML-EX second. Based on the aforementioned considerations, we remain confident in our conclusion that an expiratory muscle "warm-up" does not confer an ergogenic benefit in healthy men.

Submaximal depolarization of the thoracic nerve roots with magnetic stimulation. The depolarization of the thoracic nerve roots in response to magnetic stimulation at 100% of the stimulator's power output in the present study was likely submaximal, the technical considerations of which have been discussed in detail elsewhere (21,22,31). Briefly, although the stimulation of the thoracic nerve roots was not supramaximal, all stimulations were performed at 100% of the stimulator's maximum power output, the optimal coil position was marked in each subject before EML, SHAM, and exercise to ensure that the coil was repositioned in exactly the same location across all stimulations, and all stimulations were initiated at the same lung volume and expiratory abdominal muscle length as judged by end-expiratory Pes and Pga, respectively. Although not measured in the present study,

we have previously documented that magnetically evoked M-waves from the RA are not different before compared with after exhaustive heavy-intensity exercise (21). Moreover, work by others has shown that the reliability of magnetically evoked Pga_{tw} is similar before compared with after fatiguing MVV or exercise (22,31). Based on the aforementioned considerations, we conclude that the depolarization of the thoracic nerve roots via magnetic stimulation was kept constant throughout the present study, that the decreases in Pga_{tw} following exercise were not due to derecruitment of muscle fibers or to transmission failure, and that the same proportion of the muscles was activated by each stimulation across time (i.e., from before to after EML, SHAM, and exercise). Accordingly, we are confident that the exercise-induced changes in Pga_{tw} observed in the present study were the result of changes in the contractile function of the expiratory abdominal muscles.

Did we inadvertently "warm-up" the expiratory muscles in the sham trial? We considered it possible that the maneuvers we used to asses changes in expiratory muscle function (i.e., PE_{MAX} and Pga_{MAX}) across time may themselves have had a warm-up–like effect on the expiratory muscles. Although not directly reported in this manuscript, the maximal expulsive maneuvers potentiated Pga_{tw} by 30% ± 24% (relative to unpotentiated Pga_{tw}) across all subjects. The subjects performed these maximal expulsive efforts before and after EML and SHAM. That is, each subject performed six maximal expulsive efforts 9 min before the criterion exercise test in EML-EX and SHAM-EX; as such, we wondered whether both exercise trials were initiated with some degree of prior expiratory muscle "warm-up." Despite this concern, we are confident that any warm-up–like effect of performing six maximal expulsive efforts on expiratory muscle function and on subsequent exercise tolerance was negligible for the following two reasons. First, as documented throughout the manuscript (Fig. 2, Table 1), EML but not SHAM induced a transient increase in PE_{MAX} and Pga_{MAX}. That is, in the SHAM condition, performing six maximal expulsive efforts (pre-SHAM) followed by 2 × 30 breaths at 10% Pga_{MAX} did not facilitate an acute increase in the maximum voluntary force generating capacity of the expiratory muscles. This suggests that performing the maximal expulsive maneuvers themselves had negligible-to-no effect on PE_{MAX} and Pga_{MAX}. Second, the aforementioned increase in Pga_{tw} following the six maximal expulsive efforts represents postactivation potentiation (PAP), which is mechanistically underpinned by an increase in myosin light chain phosphorylation in Type II muscle fibers. However, PAP has a very short half-life (~28 s), and it has been observed that the potentiation of the evoked twitch response of the target muscle(s) dissipates almost completely by ~4–5 min after the conditioning activity (8,32). In the present study, the criterion exercise in EML-EX and SHAM-EX was initiated ~9 min after the post-EML and post-SHAM assessments of expiratory muscle function. Based on these considerations, we consider it highly unlikely that any substantial PAP remained present at the start of either exercise trial.

Comparison to Previous Studies

The effect of prior acute inspiratory muscle loading (i.e., an inspiratory muscle warm-up) on inspiratory muscle function and subsequent exercise performance has been investigated extensively. Acute inspiratory loading targeting ~40% of maximal inspiratory pressure (MIP) has been shown to increase peripheral excitability of the diaphragm, improve coordination of contraction between the diaphragm and the accessory intercostal muscles during maximal inspiratory efforts, and consistently elicit an ~7%–10% increase in maximum volitional inspiratory muscle strength and maximal pressure development rate (9–15,33). However, the ergogenic benefit of prior acute inspiratory muscle loading remains debated, with some (11–13,17,18,33) but not all previous reports (10,15,19,20,34–36) demonstrating that an inspiratory muscle warm-up confers a significant improvement on subsequent exercise performance. For example, Volianitis et al. (11) reported that, relative to a rowing-specific warm-up alone, the addition of an inspiratory muscle warm-up (2 × 30 breaths at 40% of MIP) to a rowing-specific warm-up increased mean power output (+1.2%) and distance covered (+7 m) during a 6-min all-out row. The authors suggested that this enhancement in rowing performance after an inspiratory muscle warm-up relative to control conditions was, at least in part, due to a 0.6 unit (Borg CR10) reduction in exertional dyspnea during the all-out row and a ~50% reduction in exercise-induced inspiratory muscle fatigue in response to the all-out row. By contrast, Johnson et al. (15) found that 10-km cycling time-trial performance was not different after a cycling warm-up with, compared to without, the addition of an inspiratory warm-up (14.70 ± 0.75 vs 14.75 ± 0.79 min). Moreover, the addition of an inspiratory warm-up did not alter either the ventilatory and pulmonary gas exchange or the perceptual responses to the subsequent exercise (15). The exact reason(s) for this divergence among previous findings regarding the ergogenic benefit of a specific inspiratory muscle warm-up is unclear and likely multifactorial, but it may in part be attributable to differences in the modality of the criterion exercise, whether the inspiratory muscle warm-up was in addition to an active whole-body warm-up, and the duration between the inspiratory muscle warm-up and the exercise task.

In the present study, we report that relative to a sham condition, acute expiratory muscle loading (2 × 30 breaths) targeting 40% of Pga_{MAX} elicited a transient improvement in expiratory muscle function, as evidenced by an 10% ± 5% and a 9% ± 5% increase in PE_{MAX}, and Pga_{MAX}, respectively (Fig. 2). This EML-induced increase in maximal expiratory muscle strength is likely the function of a phenomenon known as PAPE, which has been mechanistically attributed to increases in muscle temperature, intramuscular fluid accumulation, increased neural drive and muscle activation, and an improved coordination of contraction between the muscles that contribute to maximal force or pressure generation (8). Crucially, in contrast to PAP, the enhancements in voluntary muscular force or pressure production secondary to PAPE

typically become substantive only after several minutes, and have a longer window of action (~5 to 13 min) (8). As stated above, the criterion exercise in EML-EX was initiated ~9 min after the expiratory muscle warm-up, at which time the degree of EML-induced PAPE was likely at or around maximal (8). However, this acute enhancement in expiratory muscle function did not translate into an improvement in subsequent exercise tolerance.

So why did prior acute expiratory muscle loading not improve subsequent exercise tolerance? We are unaware of any previous study that has examined the effect of acute submaximal expiratory muscle loading on subsequent exercise tolerance and/or performance. However, the sometimes reported ergogenic benefit of an inspiratory muscle warm-up has been attributed to a reduction in the fractional utilization of the maximum tension generating capacity of these muscles during exercise (11). That is, it is possible that owing to the transient increase in MIP after an acute bout of inspiratory muscle loading, inspiratory muscle pressure production per breath during subsequent exercise occurs at a lower percentage of this "new" maximal inspiratory muscle strength. Conceptually, this could lead to an improvement in exercise performance and/or tolerance via two primary mechanisms. First, it is possible that any such reduction in the fractional utilization of maximum inspiratory muscle tension generating capacity during exercise would somewhat alleviate perceived dyspnea during exercise, enhancing exercise capacity (37). Indeed, in several previous reports, the enhancement in exercise performance after an inspiratory warm-up was at least in part attributed to a reduction in the perception of breathlessness (11,12,33). Second, although speculative, it is possible that a lowering of inspiratory muscle pressure production relative to maximal inspiratory muscle strength could delay the onset of and/or reduce the severity of exercise-induced inspiratory muscle fatigue. Theoretically, this could in turn delay the triggering of a respiratory muscle metaboreflex, better sparing leg blood flow and oxygen delivery, reducing the severity of exercise-induced limb locomotor muscle fatigue and enhancing exercise tolerance (37,38). In support of this theory, it has been suggested previously that an inspiratory muscle warm-up can indeed lead to a ~50% reduction in exercise-induced inspiratory muscle fatigue (11) and an attenuation in limb locomotor muscle deoxygenation during subsequent exercise (20), although it is important to highlight that this is not a consistent finding (10,36).

Presently, we did not measure fractional utilization of the expiratory abdominal muscles during exercise in EML-EX and SHAM-EX, nor are we aware of any previous study that has measured changes in inspiratory muscle fractional utilization during exercise with versus without prior inspiratory muscle loading. However, in the context of the present study, we contest that any such reduction in the fractional utilization of the maximum tension generating capacity of the expiratory muscles during exercise would likely be very small and potentially therefore functionally unimportant. Although the evoked $\dot{V}_E$ response is lower compared with during lower-body exercise, the fractional utilization of the expiratory abdominal muscles ($\Delta Pga_{exp}/Pga_{MAX}$) during upper-body exercise is only ~14% (39,40). In addition, assuming a maximal within-breath expiratory gastric pressure of 34 cm H_2O during exhaustive lower-body exercise, which we have reported previously (23), then a ~9% increase in Pga_{MAX} (~180 to 196 cm H_2O) after EML as observed in the present study would only facilitate a ~1%–2% reduction in fractional utilization of the expiratory abdominal muscles during subsequent exercise [(34/180) × 100 = 18.9% vs (34/196) × 100 = 17.3% fractional utilization]. By comparison, while functionally significant, the purported alleviation of dyspnea, increase in limb blood flow, reduction in the severity of exercise-induced limb locomotor muscle fatigue, and improved exercise tolerance associated with a reduction in the inspiratory work of breathing during exercise is relatively small for a much larger manipulation of respiratory muscle pressure production. Indeed, it has been shown previously that a ~50% reduction in peak within-breath inspiratory esophageal pressure and a 40%–50% reduction in overall inspiratory work of breathing during exercise translates into a ~1.2 unit reduction in dyspnea, an ~11% increase in limb blood flow, an ~8% reduction in exercise-induced quadriceps muscle fatigue, and a ~14% improvement in T_{LIM} during high-intensity exhaustive exercise (3–5,7,41). To our minds, this is hard to reconcile. That is, for example, given that the substantial reduction in fractional utilization of the inspiratory muscles associated with a ~50% reduction in the inspiratory work of breathing and maximal within-breath inspiratory muscle pressure production elicits only a ~14% improvement in exercise tolerance, it is somewhat hard to conceive that a ~1%–2% reduction in fractional utilization of the expiratory or indeed the inspiratory muscles following acute respiratory muscle loading would have a mechanistic and/or functional significance in healthy adults.

CONCLUSION

Relative to a sham condition, acute submaximal expiratory muscle loading increased maximal expiratory mouth pressure (PE_{MAX}) and maximal expiratory gastric pressure (Pga_{MAX}) by ~9%–10% but did not affect subsequent exercise time to the limit of tolerance, the perceptions of dyspnea or leg discomfort during exercise, or the severity of exercise-induced expiratory muscle fatigue. As such, we conclude that acute expiratory muscle loading (i.e., an expiratory muscle warm-up) can enhance expiratory muscle function but does not improve subsequent exercise tolerance in healthy men.

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The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation and do not constitute endorsement by the American College of Sports Medicine.

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B. J. T. conceptualized and designed the work. T. A. H. and B. J. T. collected and analyzed data. T. A. H., S. C. H., and B. J. T. contributed to interpretation of data and revisions of intellectual content. All authors read and approved the final version of the manuscript.

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