Inspiratory muscle fatigue in trained cyclists: effects of inspiratory muscle training

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ABSTRACT

ROMER, L. M., A. K. McCONNELL, and D. A. JONES. Inspiratory muscle fatigue in trained cyclists: effects of inspiratory muscle training. *Med. Sci. Sports Exerc.*, Vol. 34, No. 5, pp. 785–792, 2002. **Purpose:** This study evaluated the influence of simulated 20- and 40-km time trials upon postexercise inspiratory muscle function of trained competitive cyclists. In addition, we examined the influence of specific inspiratory muscle training (IMT) upon the responses observed. **Methods:** Using a double-blind placebo-controlled design, 16 male cyclists (mean ± SEM $\dot{V}O_{2max}$ 64 ± 2 $mL \cdot kg^{-1} \cdot min^{-1}$) were assigned randomly to either an experimental (IMT) or sham-training control (placebo) group. Maximum static and dynamic inspiratory muscle function was assessed immediately pre- and <2, 10, and 30 min post-simulated 20- and 40-km time trials before and after 6-wk of IMT or sham-IMT. **Results:** Maximum inspiratory mouth pressure (P_0) measured within 2 min of completing the 20- and 40-km time trial rides was reduced by 18% and 13%, respectively, and remained below preexercise values at 30 min. The 20- and 40-km time trials induced a reduction in inspiratory flow rate at 30% P_0 by 14% and 6% in the IMT group versus 13% and 7% for the placebo group, and also remained below preexercise values at 30 min. There was also a significant slowing of inspiratory muscle relaxation rate postexercise; these trends were almost completely reversed by 30 min postexercise. Significant improvements in 20- and 40-km time trial performance were seen (3.8 ± 1.7% and 4.6 ± 1.9%, respectively; $P < 0.05$) and postexercise reductions in muscle function were attenuated with IMT. **Conclusion:** These data support existing evidence that there is significant global inspiratory muscle fatigue after sustained heavy endurance exercise. Furthermore, the present study provides new evidence that performance enhancements observed after IMT are accompanied by a decrease in inspiratory muscle fatigue. **Key Words:** CYCLING, ERGOGENIC AID, RESPIRATORY MUSCLE

Inspiratory muscle fatigue occurs after prolonged submaximal exercise (22) and short-term maximal exercise (18,24). Evidence from a number of sources suggests that such fatigue may influence exercise tolerance in healthy young adults. For example, prior fatigue of the inspiratory muscles using either sustained maximal isocapnic hyperpnea (25) or resistive loading (23) decreases time-to-exhaustion during subsequent short-term, high-intensity exercise. In addition, several authors have noted improvements in exercise capacity in response to partial unloading of the inspiratory muscles using either reduced viscosity gas mixtures (1,36) or proportional assist ventilation (15); presumably because inspiratory muscle fatigue was prevented (4). Collectively, these data suggest that the respiratory muscles may reach the limit of their capacity during certain types of activity.

More recently, there has been considerable interest in the influence of specific inspiratory muscle training (IMT) upon exercise performance. Specific IMT improves respiratory muscle function in patients (19) and healthy individuals (21), and is associated with improvements in whole-body endurance capacity (7,8,11,33) and short-duration, high-intensity, time-trial performance (34). Recent evidence suggests that pressure threshold IMT attenuates inspiratory muscle fatigue after 6 min of all-out rowing in trained women (34). However, some have argued that trained individuals may be resistant to inspiratory muscle fatigue (12), by virtue of the adaptations induced in the inspiratory muscles by whole-body training (26,31).

We have described the performance benefits associated with IMT in trained male cyclists (32). The present paper presents data from the same group of subjects but reports the influence of 20- and 40-km time trials upon inspiratory muscle function pre- and post-IMT. We hypothesize that even trained endurance athletes will experience inspiratory muscle fatigue and that IMT will attenuate this.

METHODS

Participants

After local ethics committee approval and written informed consent, 16 male nonsmoking competitive road cyclists (5 triathletes) with normal resting pulmonary function volunteered to participate in the study. Descriptive characteristics of the participant group are presented in Table 1.

General Design

Participants were assigned randomly to either an experimental inspiratory muscle training (IMT) or sham-training

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TABLE 1. Descriptive characteristics of the participants (mean ± SEM).

	IMT[a]	Placebo[a]
Anthrompometry		
Age (yr)	29.5 ± 3.3	30.3 ± 2.6
Training history (yr)	8.9 ± 2.0	9.3 ± 1.9
Stature (m)	1.78 ± 0.02	1.80 ± 0.02
Body mass (kg)	70.1 ± 2.3	74.5 ± 2.3
Sum of 4 skinfolds (mm)	24.8 ± 1.6	31.0 ± 3.4
Estimated body fat (%)	12.3 ± 1.1	15.0 ± 1.5
Resting pulmonary function		
FVC (L)	5.53 ± 0.26 (108 ± 3)	6.13 ± 0.3 (117 ± 5)
FEV_1 (L)	4.71 ± 0.29 (109 ± 5)	5.17 ± 0.32 (118 ± 7)
FEV_1/FVC (%)	84.8 ± 1.8 (101 ± 2)	84.0 ± 2.1 (101 ± 2)
PEF ($L \cdot s^{-1}$)	10.1 ± 0.8 (110 ± 9)	10.6 ± 0.5 (107 ± 6)
MVV ($L \cdot min^{-1}$)	195.4 ± 16.3 (142 ± 12)	197.1 ± 11.3 (130 ± 7)
Maximal incremental exercise		
$\dot{W}_{max}$ (W)	355 ± 10	380 ± 17
$\dot{V}O_{2max}$ ($L \cdot min^{-1}$)	4.58 ± 0.17	4.70 ± 0.08
Time trial performance		
20 km (min)	29.62 ± 0.47	30.52 ± 0.35
40 km (min)	59.00 ± 1.53	60.03 ± 1.02

[a] $N = 8$. FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 s; PEF, peak expiratory flow; MVV, 15 s maximum voluntary ventilation; $\dot{W}_{max}$, maximum external power; $\dot{V}O_{2max}$, maximal oxygen uptake. Numbers in parentheses represent % of predicted value based on age, stature, and gender (30).

control (placebo) group using a double-blind, placebo-controlled design. Participation required seven visits to the laboratory. Participants were thoroughly familiarized with the test procedures on visit 1. Visit 2 required the assessment of pulmonary and inspiratory muscle function, and the physiological response to maximal incremental cycling. Visits 3 and 4 required participants to complete a simulated 20- and 40-km time trial, the order of which was counterbalanced. Pre-intervention trials (visits 2–4) were separated by at least 48 h, completed within 2 wk and repeated (visits 5–7) after a 6-wk period of IMT. Thus, the overall duration of the study was ~10 wk.

Procedure

Pretest preparation. Testing took place during a maintenance phase of normal training so that confounding influences were minimized. Participants performed only a light recovery training session (less than 50 km at less than 75% maximal heart rate) 24 h before testing and maintained their regular diet in the days preceding physiological assessment. On a test day, participants were instructed not to eat 2 h before testing, and to avoid drinking alcohol or caffeinated beverages and taking any other substances that are known to affect, or may be suspected to affect, human physiological functions. Participants kept detailed records of all exercise and food intakes for 48 h before the first test and used these records to replicate their activities before subsequent tests. Tests were conducted at the same temperature (18–22°C) and relative humidity (<70%). For each subject, testing was scheduled at a similar time of day (± 1 h) so that diurnal fluctuation effects were minimized.

Pulmonary function. Forced vital capacity (FVC), forced expiratory volume in 1 s (FEV_1), peak expiratory flow rate (PEF), and 15-s maximum voluntary ventilation (MVV) were determined using an on-line turbine spirometer (Mijnhardt Oxycon Alpha, Bunnik, The Netherlands) according to European Respiratory Society recommendations (30).

Maximum dynamic inspiratory muscle function. Pressure-flow data were obtained from maximal inspiratory efforts performed against a pressure-threshold valve arrangement (9) immediately before and after (<2, 10, and 30 min) 20- and 40-km time trials. Inspiratory pressure was measured at the mouth with a pressure transducer (Mercury M14, Glasgow, UK). Inspiratory airflow was measured with an ultrasonic phase-shift flow meter (Birmingham Flowmetrics Ltd., Birmingham, UK) located distal to a pressure-threshold valve. Mean pressure at zero flow (P_0) was measured with complete closure of the threshold valve. A 1-mm orifice was exposed to prevent the subject from producing artificially high inspiratory pressures with the muscles of the buccal cavity (6). To ensure that inspiratory efforts were consistently performed from the same lung volume (residual volume), changes in vital capacity were measured with a pneumotachograph spirometer (Vitalograph 2120, Buckingham, UK) connected in series to the expiratory port of the pressure-threshold device. After the determination of P_0, individuals performed maximal inspiratory maneuvers against a load setting of 30% P_0 for the determination of inspiratory flow rate ($\dot{V}_{30}$). Three technically correct trials were performed at each of the loading intensities. Participants received visual feedback of pressure and flow to maximize respiratory efforts, and were consistently instructed to contract maximally and as rapidly as possible.

The maximal relaxation rate (MRR) of the initial linear decline of the pressure decay curve and the time constant (τ) of the later curved portion of the pressure decay curve were determined for the zero flow condition. The MRR was calculated as being the slope of a tangent drawn to the steepest portion of the curve. Because the MRR is pressure-dependent (14), it was normalized by dividing MRR by the peak contraction pressure. The later curved portion of the pressure decay curve was analyzed by replotting the pressure signal on a logarithmic scale. This yielded a straight line over the lower 50–70% of the curve, indicating that the pressure decay was mono-exponential. The τ of the exponential portion was calculated as the reciprocal of the slope of the line (from the equation for an exponential function: $y = e^{-t/\tau}$).

Incremental exercise. Participants performed an incremental exercise test to volitional exhaustion on a calibrated electromagnetically braked cycle ergometer (Lode Excalibur, Groningen, The Netherlands) set in the "hyperbolic" mode (i.e., independent of pedal rate). Power output was increased by 35 W every 3 min starting from 95 W. The test was terminated when pedal cadence fell below 60 rpm and maximal power output ($\dot{W}_{max}$) was calculated by interpolation from the total time completed during the final stage of the test (20). Ventilatory and pulmonary gas exchange indices were measured breath-by-breath throughout incremental exercise using an on-line system (Mijnhardt Oxycon Alpha).

Time trial performance. Exercise endurance performance was evaluated as the time taken to complete simu-

lated 20- and 40-km time trials, the order of which was counter balanced. Full details of the procedure have been documented previously (16) but, briefly, the cycle ergometer was set in the "linear" mode (i.e., pedal rate dependent) according to the formula: $W = L \cdot (rpm)^2$. The value for L was chosen such that it would elicit a pedalling rate of 95 rpm at 82% and 75% $\dot{W}_{max}$ for the 20- and 40-km time trials, respectively. In other words, the linear factor was dependent on a subject's $\dot{W}_{max}$. Thus, 82% and 75% $\dot{W}_{max}$ could be achieved at about 95 rpm, which appears to be the preferred pedalling rate of most cyclists. After a warm-up (5 min, 40% $\dot{W}_{max}$), subjects performed a predetermined amount of work (equal to ~30 or ~60 min cycling) in the fastest time possible. The measure of performance was the time to complete the target amount of work. This target was based on the maximal power achieved during incremental exercise ($\dot{W}_{max}$), and was calculated according to the formula: target work (J) = 0.82 (or 0.75)·$\dot{W}_{max}$·1800 (or 3600) for the 20-km (or 40-km) time trials.

Inspiratory muscle training. Participants were ranked according to maximal inspiratory mouth pressure (P_0) and subsequently divided into matched pairs. One individual of each pair was randomly assigned to the IMT group by an independent observer and the other to the placebo group. The principle investigators were therefore blinded to the training condition. The IMT group performed 30 dynamic inspiratory efforts twice daily for 6 wk against a pressure-threshold load equivalent to ~50% P_0, a protocol known to be effective in eliciting an adaptive response (10). The placebo group trained using 60 slow protracted breaths once daily for 6 wk at ~15% P_0 a protocol known to elicit negligible changes in inspiratory muscle function (11). Subjects were instructed to initiate each breath from residual volume and to continue the inspiratory effort up to the lung volume where the inspiratory muscle force output for the given load limited further excursion of the thorax. Because of the increased tidal volume, a decreased breathing frequency was adopted to avoid hyperventilation and the consequent hypocapnia. Subjects were instructed to cease training 48 h before post-IMT trials. Loading characteristics of the inspiratory muscle-training device (POWERbreathe®, IMT Technologies Ltd., Birmingham, UK) have been documented (9). After the initial setting of training loads, subjects in the IMT group were instructed by an independent observer to increase the load periodically to a level that would permit them to only just complete 30 maneuvers; the placebo group was not given these instructions. Subjects were told they were participating in a study to compare the influence of strength (IMT group) versus endurance (placebo group) protocols and, as a consequence, were blinded to the true purpose of the study and the expected outcomes. Subjects were instructed to cease training 48 h before post-IMT trials.

Training adherence. The number of inspiratory efforts completed by subjects throughout the intervention period was monitored using a thermistor suspended within the main body of the training device that sensed acute drops in air temperature associated with changes in airflow. Detailed physical activity diaries were used to monitor training volume and intensity. Training impulse (TRIMP), which is a measure of the quantity of training in any given session, was calculated week-by-week as the product of training volume (duration of training in minutes) and intensity, indicated by the delta heart rate ratio ([exercise f_c − resting f_c]/[maximum f_c − resting f_c]) (5).

Data Analyses

Mixed factorial ANOVAs were used to test for between group effects due to "treatment" (IMT or placebo) and within group effects due to "intervention" (pre- and post-treatment), "distance" (20- and 40-km), and "time" (pre-, 2, 10, and 30 min post-time trial) on each of the dependent variables. Mauchly's sphericity test was used to check homogeneity of covariance. Violations of the assumption of sphericity were corrected using the Greenhouse-Geisser adjustment. The residuals from the ANOVA were checked for normality. Planned pairwise comparisons were made with repeated measures *t*-tests and the Bonferroni adjustment was used to modify the per family type I error rate per comparison.

Pearson product moment correlation coefficients were computed to assess the degree of relationship between the relative changes in selected physiological variables after IMT. Stepwise multiple regression analysis was used to determine which variables accounted for the largest amount of variation in the time trial performance expressed relative to pre-IMT values. Results are expressed as mean ± standard error of the mean (SEM) unless otherwise stated. An alpha level of 0.05 was chosen *a priori* to represent statistical significance. All statistical analyses were performed using the 8.0 release version of SPSS for Windows (SPSS Inc., Chicago IL).

RESULTS

Routine Physical Exercise and IMT Compliance

Participants' physical activity did not vary between (IMT vs placebo) or within groups (1–6 wk) as evidenced by nonsignificant frequency, duration, intensity, and training impulse (TRIMP) score differences. Furthermore, total TRIMP score was not correlated with changes in $\dot{W}_{I\ max}$ or 20- and 40-km time trial performance ($r = 0.13$, 0.18, and 0.20, respectively). Excellent compliance with the prescribed IMT regimen was found for both groups. The IMT group completed 81 ± 2 of the 84 training sessions (96% adherence), whereas the placebo group completed 40 ± 2 of the 42 sessions (95% adherence).

Preexercise Pulmonary and Respiratory Muscle Function

All baseline pulmonary function values were within normal limits (Table 1). For the placebo group, none of the pulmonary or respiratory muscle function measures were different after the 6 wk of sham training. In contrast,

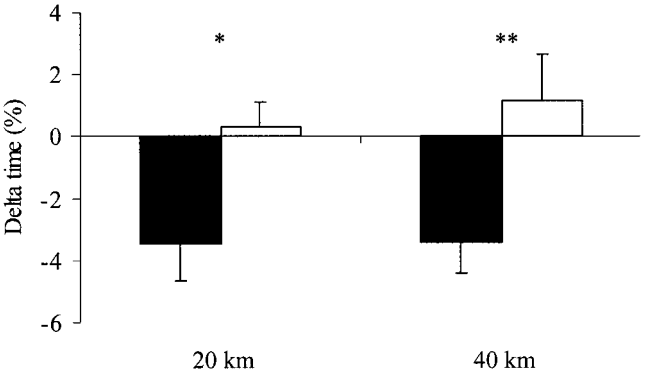

FIGURE 1—Relative changes in 20- and 40-km time trial performance for IMT (*closed bars*) and placebo (*open bars*) groups (mean ± SEM); * significant interaction effect ($P \leq 0.05$); ** significant interaction effect ($P \leq 0.01$).

significant improvements in P_0 (28 ± 7%) and $\dot{V}_{30}$ (22 ± 8%) were observed in the IMT group.

Time Trial Performance

After intervention, the IMT group completed the simulated 20- and 40-km time trials faster than the placebo group (65 ± 30 and 114 ± 38 s faster, respectively; $P = 0.025$ and 0.009; 3.8 ± 1.7 and 4.6 ± 1.9%, respectively). The relative changes in time trial performance are shown in Figure 1.

Postexercise Inspiratory Muscle Function

Pre-IMT. Absolute values for inspiratory muscle function before and after the time trials in the two groups before training began are presented in Tables 2 and 3. Relative changes from preexercise values after 20- and 40-km time trials for IMT and placebo groups are shown in Figures 2 and 3, respectively. Measurements made within 2 min of the end of the 20- and 40-km time trials revealed significant declines in P_0 from baseline values, averaging −17 ± 4 and −13 ± 3% for the IMT group versus −18 ± 2 and −14 ± 2% for the placebo group, respectively, before IMT. Values for P_0 after 20- and 40-km time trials had not returned to near preexercise levels within 30 min postexercise. After 20- and 40-km time trials, group mean values for pressure normalized maximum relaxation rate (MRR/P_0) were significantly slower (−14 ± 2 and −13 ± 2% for IMT vs −15 ± 2 and −14 ± 2% for placebo, respectively) and recovered almost completely by 30 min postexercise. A similar trend was observed for τ after the 20- and 40-km time trials (44 ± 2 and 23 ± 2% for IMT vs 40 ± 3 and 25 ± 2% for placebo, respectively). The 20- and 40-km time trials induced a reduction in $\dot{V}_{30}$ of −14 ± 6 and −6 ± 4% in the IMT group versus −13 ± 5 and −7 ± 5% for the placebo group, respectively. Values for $\dot{V}_{30}$ after 20- and 40-km time trials had not returned to near preexercise levels within 30 min post exercise. Although the postexercise changes in inspiratory muscle function appeared to be greater for the 20-km versus the 40-km time trial, only the relative change in τ reached statistical significance ($P \leq 0.01$).

Post-IMT. Relative changes in inspiratory muscle function from preintervention values after 20- and 40-km time trials for IMT and placebo groups are shown in Figures 2 and 3, respectively. The relative changes in P_0, MRR, $\dot{V}_{30}$, and τ induced by the time trials were significantly smaller after training for the IMT group ($P \leq 0.001$). Furthermore, most parameters were not significantly different from preexercise values within 30 min postexercise. The magnitude of these changes for the placebo group remained the same postintervention.

Correlations among Variables with Inspiratory Muscle Training

Interindividual differences with IMT for several variables were significantly interrelated (see Table 4). The relative changes in postexercise inspiratory muscle function compared to pre-IMT values were significantly correlated with the relative changes in performance time. In particular, changes in P_0 and MRR/P_0 accounted for 38 and 36% of the variance in performance time expressed relative to pre-IMT values. On the basis of stepwise multiple regression analysis, changes in P_0 and τ accounted for 58% of the variance in %Δ performance time (see Equation 1).

$$\%\Delta \text{ performance time} = -0.56(\%\Delta P_0) - 0.48(\%\Delta\tau) - 2.10(\text{SEE} = 2.1\%) \quad (1)$$

DISCUSSION

Main Findings

The aim of the present study was to determine the influence of 20- and 40-km cycling time trials and specific IMT upon postexercise inspiratory muscle function in trained cyclists. The main findings were that both time trial distances induced inspiratory muscle fatigue and that the extent of this fatigue was diminished after IMT.

Exercise-Induced Alterations in Inspiratory Muscle Function

Inspiratory muscle fatigue was defined as a loss in the capacity for developing inspiratory pressure and/or flow, resulting from whole-body exercise and which was reversible by rest (29). Using this definition, it is clear that significant global inspiratory muscle fatigue occurred in response to simulated 20- and 40-km time trial performance in these trained cyclists. Indeed, maximum inspiratory pressure (P_0) measured within 2 min of completing the 20- and 40-km time trial rides was reduced by 18 and 13%, respectively, and remained below preexercise values at 30 min. Furthermore, a significant increase in the time constant of pressure decay (τ) and a significant decrease in the maximum relaxation rate (MRR) was observed immediately postexercise; these trends were almost completely reversed by 30 min postexercise. Our findings are in agreement with those of previous studies that have shown that the diaphragm muscle of healthy individuals is susceptible to fa-

TABLE 2. Changes in inspiratory muscle function from preexercise values following 20- and 40-km time trials for IMT group (mean ± SEM).

	Pre-IMT[a]				Post-IMT[a]			
	Preexercise	<2 min	10 min	30 min	Preexercise	<2 min	10 min	30 min
20 km								
P_0 (−cm H_2O)	104.9 ± 8.3	87.7 ± 9.1**	91.5 ± 9.4**	94.3 ± 8.0**	125.3 ± 7.6	113.7 ± 8.7	116.7 ± 8.9	119.0 ± 8.3
(%Δ)		(−17 ± 4)**	(−14 ± 3)**	(−10 ± 3)**		(−10 ± 3)††	(−7 ± 2)††	(−5 ± 3)††
MRR/P_0 (ms^{-1}·10^{-3})	6.8 ± 0.4	5.9 ± 0.4**	6.4 ± 0.4*	6.6 ± 0.4	7.0 ± 0.3	6.4 ± 0.3	6.8 ± 0.4	7.0 ± 0.0
(%Δ)		(−14 ± 2)**	(−7 ± 1)*	(−3 ± 1)		(−9 ± 1)†	(−4 ± 1)	(0 ± 1)
τ (ms)	57.4 ± 3.2	82.4 ± 3.8**	71.2 ± 3.4**	61.6 ± 3.1*	60.8 ± 3.1	80.4 ± 3.6	68.7 ± 3.1	61.8 ± 3.0
(%Δ)		(44 ± 2)**	(25 ± 2)**	(8 ± 1)*		(33 ± 2)††	(13 ± 1)††	(2 ± 1)†
$\dot{V}_{30}$ ($L·s^{-1}$)	6.63 ± 0.70	5.89 ± 0.70**	6.24 ± 0.83	6.19 ± 0.79*	7.78 ± 0.30	7.21 ± 0.26	7.55 ± 0.45	7.70 ± 0.40
(%Δ)		(−14 ± 6)**	(−7 ± 4)*	(−7 ± 5)*		(−7 ± 3)††	(−3 ± 5)†	(−1 ± 5)††
40 km								
P_0 (−cm H_2O)	99.8 ± 7.9	87.1 ± 6.9**	89.8 ± 8.9**	91.5 ± 8.4**	127.6 ± 7.5	118.7 ± 7.2	120.4 ± 8.6	123.4 ± 8.2
(%Δ)		(−13 ± 3)**	(−11 ± 3)**	(−9 ± 3)**		(−7 ± 2)††	(−6 ± 2)†	(−4 ± 2)†
MRR/P_0 (ms^{-1}·10^{-3})	6.5 ± 0.3	5.7 ± 0.4**	6.3 ± 0.4	6.5 ± 0.3	6.8 ± 0.3	6.3 ± 0.3	6.7 ± 0.4	6.8 ± 0.3
(%Δ)		(−13 ± 2)**	(−5 ± 2)*	(−1 ± 1)		(−8 ± 2)†	(−3 ± 1)	(−1 ± 1)
τ (ms)	60.2 ± 2.8	73.9 ± 3.5**	67.4 ± 3.1**	61.4 ± 2.9	61.4 ± 2.5	70.1 ± 3.3	65.2 ± 3.0	61.4 ± 3.0
(%Δ)		(23 ± 2)**	(12 ± 1)**	(2 ± 1)		(14 ± 1)††	(6 ± 1)†	(0 ± 2)
$\dot{V}_{30}$ ($L·s^{-1}$)	6.99 ± 0.56	6.62 ± 0.65*	6.63 ± 0.55	6.76 ± 0.65	8.08 ± 0.40	7.76 ± 0.40	7.86 ± 0.41	7.94 ± 0.31
(%Δ)		(−6 ± 4)*	(−5 ± 5)	(−4 ± 3)		(−4 ± 3)	(−3 ± 2)	(−1 ± 2)

[a] N = 8. P_0, maximum inspiratory mouth pressure at zero flow; MRR, maximum relaxation rate; τ, time constant of relaxation pressure; $\dot{V}_{30}$, inspiratory flow at 30% of P_0. Values in parentheses represent percentage changes from preexercise values.
* Significantly different from pre-exercise baseline values ($P \leq 0.05$); ** significantly different from pre-exercise baseline values ($P \leq 0.01$); † significantly different from pre-IMT values ($P \leq 0.05$); †† significantly different from pre-IMT values ($P \leq 0.01$).

tigue after sustained (>10 min) heavy endurance exercise (>80–85% $\dot{V}O_{2max}$) (18,24). Furthermore, numerous studies have shown that inspiratory muscle fatigue slows the relaxation rate in healthy subjects, as attested by an increase in τ and/or a decrease in MRR (13).

Results from the present study suggest that the intensity/duration of exercise influence the magnitude of inspiratory muscle fatigue. Indeed, the reduction in postexercise inspiratory muscle function appeared to be greater after the 20-km versus the 40-km time trial (although only the change in τ reached statistical significance). The expected increase in inspiratory muscle force output for the 20-km time trial would be a significant determinant of inspiratory muscle fatigue (18). However, differences related to whole-body exercise itself may also explain the observed differences in fatigue. For example, an elevated acid milieu of the locomotor muscles or competition for blood flow between locomotor muscles and the respiratory muscles may have contributed toward the elevated inspiratory muscle fatigue experienced by subjects after the 20-km time trial (17). These events are not necessarily mutually exclusive because the latter might increase the metabolites produced by the respiratory and/or locomotor muscles (17).

The majority of studies examining the effect of exercise upon inspiratory muscle function have primarily tested changes in quasi-static muscle force production (i.e., maximum inspiratory mouth pressure). However, it is becoming increasingly appreciated that other changes may occur in conjunction with the decreased ability to produce force, such as changes in the velocity of shortening or in the ability to shorten under load (2). In the present study, we observed a decrease in maximum inspiratory flow rate at 30% P_0 ($\dot{V}_{30}$) after both 20- and 40-km time trials. Indeed, $\dot{V}_{30}$ measured within 2 min post 20- and 40-km time trial rides was reduced on average by 14 and 7%, respectively, and remained below preexercise values up to 30 min postexercise.

TABLE 3. Changes in inspiratory muscle function from preexercise values after 20- and 40-km time trials for placebo group (mean ± SEM).

	Pre-IMT[a]				Post-IMT[a]			
	Preexercise	<2 min	10 min	30 min	Preexercise	<2 min	10 min	30 min
20 km								
P_0 (−cm H_2O)	99.0 ± 9.3	81.3 ± 8.1**	86.8 ± 7.2**	87.9 ± 7.2**	101.7 ± 9.6	84.1 ± 7.9	90.4 ± 7.6	90.6 ± 7.4
(%Δ)		(−18 ± 2)**	(−12 ± 2)**	(−10 ± 2)**		(−17 ± 2)	(−10 ± 2)	(−10 ± 2)
MRR/P_0 (ms^{-1}·10^{-3})	6.3 ± 0.5	5.4 ± 0.4**	6.0 ± 0.5	6.2 ± 0.5	6.6 ± 0.4	5.5 ± 0.4	6.3 ± 0.5	6.4 ± 0.4
(%Δ)		(−15 ± 2)**	(−4 ± 1)	(−1 ± 1)		(−18 ± 3)	(−5 ± 1)	(−3 ± 1)
τ (ms)	64.9 ± 4.8	89.7 ± 4.8**	77.6 ± 4.8**	67.2 ± 4.6	62.9 ± 5.0	86.2 ± 4.4	73.3 ± 4.4	64.7 ± 4.7
(%Δ)		(40 ± 3)**	(21 ± 3)**	(4 ± 1)		(40 ± 5)	(18 ± 3)	(3 ± 1)
$\dot{V}_{30}$ ($L·s^{-1}$)	6.69 ± 0.62	5.82 ± 0.68	6.22 ± 0.70	6.30 ± 0.62	6.73 ± 0.58	5.92 ± 0.64	6.33 ± 0.60	6.35 ± 0.63
(%Δ)		(−13 ± 5)	(−7 ± 4)	(−6 ± 4)		(−12 ± 4)	(−6 ± 4)	(−6 ± 5)
40 km								
P_0 (−cm H_2O)	100.7 ± 9.2	87.1 ± 8.5**	93.2 ± 7.7*	94.2 ± 7.8*	99.3 ± 8.8	87.6 ± 7.9	92.9 ± 8.0	94.8 ± 7.5
(%Δ)		(−14 ± 2)**	(−7 ± 2)*	(−6 ± 2)*		(−12 ± 2)	(−6 ± 2)	(−4 ± 2)
MRR/P_0 (ms^{-1}·10^{-3})	6.2 ± 0.6	5.3 ± 0.5**	6.3 ± 0.4	6.4 ± 0.4	6.4 ± 0.5	5.6 ± 0.5	6.1 ± 0.4	6.3 ± 0.4
(%Δ)		(−14 ± 2)**	(3 ± 3)	(1 ± 3)		(−13 ± 2)	(−4 ± 2)	(0 ± 2)
τ (ms)	67.3 ± 4.6	83.5 ± 4.6**	75.3 ± 4.6**	69.5 ± 4.5	64.2 ± 4.2	82.0 ± 4.3	71.9 ± 4.2	65.2 ± 4.5
(%Δ)		(25 ± 2)**	(13 ± 2)**	(4 ± 1)		(28 ± 3)	(12 ± 3)	(1 ± 2)
$\dot{V}_{30}$ ($L·s^{-1}$)	6.58 ± 0.59	6.12 ± 0.66	6.25 ± 0.62	6.26 ± 0.58	6.67 ± 0.53	6.14 ± 0.59	6.34 ± 0.55	6.42 ± 0.54
(%Δ)		(−7 ± 5)	(−5 ± 5)	(−5 ± 4)		(−8 ± 4)	(−5 ± 3)	(−4 ± 2)

[a] N = 8. P_0, maximum inspiratory mouth pressure at zero flow; MRR, maximum relaxation rate; τ, time constant of relaxation pressure; $\dot{V}_{30}$, inspiratory flow at 30% of P_0. Values in parentheses represent percentage changes from preexercise values.
* Significantly different from preexercise baseline values ($P \leq 0.05$); ** significantly different from preexercise baseline values ($P \leq 0.01$).

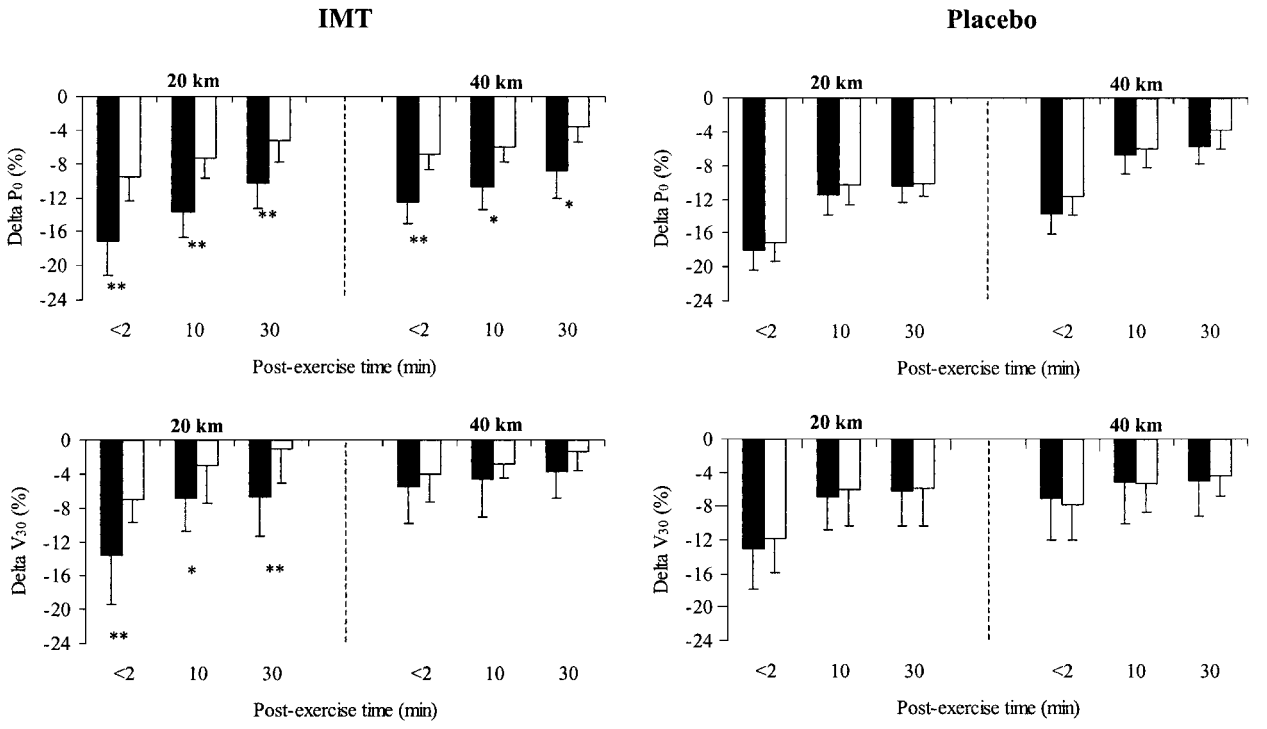

FIGURE 2—Relative changes in postexercise inspiratory muscle function (P_0 and $\dot{V}_{30}$) for IMT (*left panels*) and placebo (*right panels*) groups preintervention (*closed bars*) and postintervention (*open bars*) (mean ± SEM); * significant interaction effect ($P \leq 0.05$); ** significant interaction effect ($P \leq 0.01$).

These findings are in agreement with the observation of McCool et al. (28) that fatigue induced by high-flow tasks preferentially fatigues the diaphragm and reduces maximum flow generating capacity. Thus, our data indicate that even trained subjects are susceptible to inspiratory muscle fatigue and add support to the notion that, during certain types of activity, the inspiratory muscles are working close to the limit of their capacity.

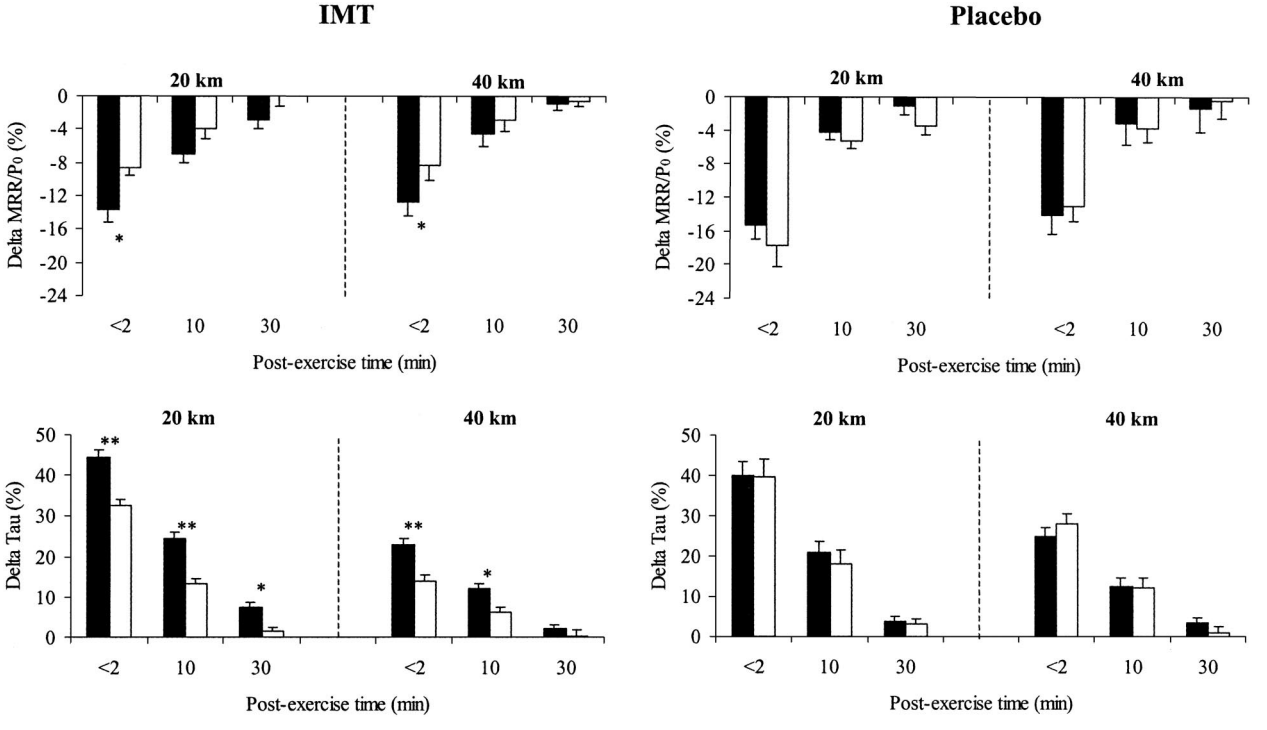

FIGURE 3—Relative changes in postexercise inspiratory muscle function (MRR/P_0 and τ) for IMT (*left panels*) and placebo (*right panels*) groups preintervention (*closed bars*) and postintervention (*open bars*) (mean ± SEM); * significant interaction effect ($P \leq 0.05$); ** significant interaction effect ($P \leq 0.01$).

TABLE 4. Correlation matrix of the relative changes from baseline with IMT for postexercise measurements of inspiratory muscle function.

	%Δ P_0	%Δ MRR/P_0	%Δ τ	%Δ $\dot{V}_{30}$
%Δ Performance time	0.62**	0.60*	0.44	0.45
%Δ P_0		0.41	0.01	0.31
%Δ MRR/P_0			0.68**	0.44
%Δ τ				0.35

$N = 16$ (pooled 20- and 40-km time trial data for IMT group). Δ, change; P_0, pressure at zero flow; MRR maximum relaxation rate; τ, time constant of relaxation; $\dot{V}_{30}$, inspiratory flow at 30% P_0. * $P \leq 0.05$; ** $P \leq 0.01$.

After the intervention, the IMT group exhibited significant reductions in the extent of exercise-induced inspiratory muscle fatigue (see Figs. 2 and 3). This was evident in all of the parameters of inspiratory muscle function assessed (P_0, MRR, $\dot{V}_{30}$, and τ; $P \leq 0.001$). Furthermore, recovery of these parameters was accelerated after IMT. That inspiratory muscle fatigue was diminished in response to IMT is in agreement with recent evidence from our laboratory that pressure threshold IMT attenuates the reduction in maximum inspiratory mouth pressure after 6 min of all-out rowing in well-trained women (34). These findings are perhaps unsurprising considering that the fatigability of these muscles is to some extent governed by their baseline strength (27). These observations suggest that normal endurance training fails to provide an optimal training stimulus to the inspiratory muscles and that, further, favorable adaptation is possible with specific IMT. It was interesting to note that 38% and 36% of the total variance in time trial performance expressed relative to pre-IMT values was accounted for by relative changes in P_0 and MRR, respectively. Collectively, these data support the notion that the inspiratory muscles represent a potential site of limitation to exercise performance.

Although exercise-induced diaphragm fatigue is probably not sufficient to compromise the adequacy of alveolar ventilation (18), theoretically it might curtail exercise performance by reducing the relative contribution of the diaphragm, promoting greater use of accessory respiratory muscles and causing a mechanically inefficient means of producing ventilation (17). The importance of relaxation in the regulation of breathing is crucial, because the inspiratory muscles must return to their optimal muscle lengths between each inspiration and because diaphragmatic perfusion depends in part on rapid and efficient muscle relaxation (13). Although we chose not to collect respiratory data during the time trials (to optimally simulate time trial performance), breathing pattern changes were observed after IMT during separate incremental exercise tests at intensities similar to those experienced by subjects during the time trial rides. The IMT group maintained tidal volume during the later stages of incremental exercise, whereas the placebo group resorted to a more tachypneic breathing pattern, characteristic of respiratory muscle fatigue, for the maintenance of minute ventilation.

The mechanism(s) by which IMT improves whole-body exercise performance is, as yet, unclear. If the effect of IMT was to delay the recruitment of accessory muscles and/or improve accessory muscle function, this might lead to reduced chest wall distortion and improved efficiency of breathing. Such changes might, in turn, translate into a lower work of breathing and a reduced metabolic and blood flow demand by the inspiratory muscles (35). A reduction in inspiratory muscle work would also be expected to attenuate sensory input to the central nervous system and therefore decrease the perception of inspiratory muscle effort (3). We believe that the ergogenic effect of IMT has a multifactorial etiology that may include: 1) the direct effect of IMT upon inspiratory muscle fatigue, 2) IMT's indirect effects upon improving blood flow distribution to limb locomotor muscles in very heavy exercise, and 3) IMT's direct and indirect effect upon the intensity with which both respiratory and peripheral efforts are perceived (35).

CONCLUSIONS

Data from the present study suggest that inspiratory muscle fatigue is present after heavy exercise in trained competitive cyclists but that the extent of this fatigue is attenuated by IMT. Further studies are required to clarify the mechanisms by which improvements in inspiratory muscle function and fatigue resistance improve endurance performance.

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