Title:

Inspiratory muscle training reduces blood lactate concentration during volitional hyperpnoea

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Abstract

Intense volitional hyperpnoea can increase blood lactate concentration ($[lac^-]_B$), however, whether this is reduced following pressure-threshold inspiratory muscle training (IMT) is unknown. We hypothesised that volitional hyperpnoea at a breathing pattern specific to intense endurance exercise would increase $[lac^-]_B$ and that specific IMT attenuate such a response. 22 physically active males were matched for 85% maximal exercise minute ventilation ($\dot{V}_E$ max) and divided equally into an IMT or a control group. Prior to and following a 6 week intervention, participants performed 10 min of volitional hyperpnoea at the breathing pattern commensurate with 85% $\dot{V}_E$ max . The IMT group performed 6 weeks of IMT; the control group performed no IMT. Maximal inspiratory mouth pressure increased (mean ± SD) 31 ± 22% following IMT and was unchanged in the control group. Prior to the intervention in the control group, $[lac^-]_B$ increased from 0.76 ± 0.24 mmol·L^{-1} at rest to 1.50 ± 0.60 mmol·L^{-1} and in the IMT group from 0.85 ± 0.40 mmol·L^{-1} at rest to 2.02 ± 0.85 mmol·L^{-1} following 10 min volitional hyperpnoea (P<0.05). Following the intervention the $[lac^-]_B$ response to volitional hyperpnoea was unchanged in the control group. Conversely, following IMT, $[lac^-]_B$ was reduced by 17 ± 37% and 25 ± 34% following 8 and 10 min, respectively (P<0.05). In conclusion, increases in $[lac^-]_B$ during volitional hyperpnoea at 85% $\dot{V}_E$ max were attenuated following IMT. These findings suggest that the inspiratory muscles were the source of at least part of this reduction, and provide a possible explanation for some of the IMT-mediated reductions in $[lac^-]_B$ often observed during whole-body exercise.

Introduction

Specific respiratory muscle training (RMT) can be performed using either voluntary isocapnic hyperpnoea (VIH), flow-resistive loading, or pressure-threshold loading; with the exception of VIH, these are commonly referred to as inspiratory muscle training (IMT). Ventilatory endurance is enhanced with all three techniques, whereas IMT also increases diaphragm thickness (Downey et al. 2007; Enright et al. 2006) and the maximal strength, shortening velocity and power of the inspiratory muscles (for a full review see McConnell and Romer 2004). Furthermore, well controlled studies have shown improvements in endurance exercise performance following both IMT (Gething et al. 2004; Griffiths and McConnell 2007; Johnson et al. 2007; Romer et al. 2002a; Volianitis et al. 2001) and VIH (Leddy et al. 2007).

The mechanisms underlying such performance improvements remain speculative but may include reduced perception of effort (Downey et al. 2007; Gething et al. 2004; Griffiths and McConnell 2007; Romer et al. 2002a; Verges et al. 2007; Volianitis et al. 2001) and possibly reductions in both diaphragm fatigue (Verges et al. 2007) and an associated metaboreflex that attenuates limb blood flow (McConnell and Lomax 2006; Witt et al. 2007). The notion that genuine physiological adaptation explains, in part, RMT-mediated improvements in endurance exercise performance is further supported by the frequently observed reduction in blood lactate concentration ($[lac^-]_B$) during whole-body exercise following both IMT (Griffiths and McConnell 2007; McConnell and Sharpe 2005; Romer et al. 2002b; Volianitis et al. 2001) and VIH (Leddy et al. 2007; Spengler et al. 1999). Furthermore, correlations have been reported between reductions in $[lac^-]_B$ and performance improvements following RMT (Romer et al. 2002b; Spengler et al. 1999), with up to 52% of the variation in performance being attributed to the reduced $[lac^-]_B$ (Romer et al. 2002b).

The mechanism(s) by which RMT reduces $[lac^-]_B$ remains equivocal. An RMT-mediated change in minute ventilation ($\dot{V}_E$), which may conceivably alter both the work of breathing and

acid base balance, is an unlikely mechanism since reductions in $[lac^-]_B$ following RMT have been observed irrespective of whether $\dot{V}_E$ is lower (Leddy et al. 2007), unchanged (McConnell and Sharpe 2005; Spengler et al. 1999; Volianitis et al. 2001), or increased (Kohl et al. 1997). It thus appears that the specific, targeted nature of RMT elicits respiratory muscle adaptations that result in the respiratory muscles being the source of at least part of the reductions observed in $[lac^-]_B$.

Modest increases in $[lac^-]_B$ are observed under resting conditions when $\dot{V}_E$ is increased for 5 min at 72 % maximal voluntary ventilation (MVV) (Martin et al. 1984), or sustained to volitional tolerance at ~70 %MVV (Verges et al. 2007). This increase is reduced during an exhaustive breathing endurance test following VIH training although the reductions observed following RMT failed to exceed a control and the authors neglect to explain their findings (Verges et al. 2007). Notwithstanding these findings, previous studies that have employed a breathing challenge at a given %MVV have little ecological validity with respect to intense endurance exercise since the breathing pattern adopted during volitional hyperpnoea can significantly influence the work of breathing (Coast et al. 1993). Thus for volitional hyperpnoea to reflect the demands of exercise hyperpnoea, $\dot{V}_E$, respiratory frequency (f_R), tidal volume (V_T) and duty cycle (T_I/T_{TOT}) must be rigourously controlledto that of exercise which has not been achieved in previous studies. Furthermore, despite VIH reducing $[lac^-]_B$ during an intense respiratory endurance test to volitional tolerance, it is unknown whether strength based inspiratory muscle training may also reduce systemic $[lac^-]_B$ given the discrete differences in training mode.

Therefore, to investigate this issue further the present study examined two hypothesese: firstly that mimicking at rest the breathing pattern observed during high-intensity endurance exercise would significantly increase $[lac^-]_B$, and secondly that 6 weeks of IMT would attenuate such a response.

Methods

Subjects

Following approval from Nottingham Trent University's ethics committee, 22 non-smoking, recreationally active males provided written informed consent to participate in the study. Throughout the study subjects were instructed to adhere to their usual training regimen and not to engage in strenuous exercise the day before test days, during which subjects refrained from ingesting caffeine and arrived at the laboratory 2 h post-prandial. Descriptive characteristics of the subjects are presented in Table 1.

Experimental procedure

Baseline pulmonary function and maximum inspiratory mouth pressure (MIP) were measured during the first laboratory visit. On a separate occasion, subjects then performed a maximal incremental cycling test, and two 10 min isocapnic volitional hyperpnoea tests (the first being a familiarisation test); all of these tests were separated by a minimum of 48 hours. The volitional hyperpnoea tests were performed at the $\dot{V}_E$, tidal volume (V_T), breathing frequency (f_R) and duty cycle (TI/T_{TOT}) associated with 85% maximal exercise $\dot{V}_E$ ($\dot{V}_E$max) since pilot work showed that this was the maximal exercise breathing pattern that could be maintained for 10 min. During the experimental volitional hyperpnoea test expired respiratory and pulmonary variables were measured breath by breath from min 0 to 10 inclusive and arterialised venous blood gases, pH and $[lac^-]_B$ was measured at rest and every 2 min thereafter. Subjects were subsequently matched for 85% $\dot{V}_E$max and divided into an IMT group (n=11) or a control group (no IMT; n=11). No more than 1 week following a 6 week intervention MIP was measured and at least 48 hours following this, subjects repeated the volitional hyperpnoea test. Each subject completed a 24 h diet record prior to the criterion pre-intervention volitional hyperpnoea test and this was then replicated during the 24 h prior to the post-intervention volitional hyperpnoea test.

Pulmonary function, maximal inspiratory pressure, and respiratory measurements

Pulmonary function was assessed using a pneumotachograph (ZAN 600USB, Nspire Health, Oberthulba, Germany) calibrated using a 3 L syringe. Each measurement was repeated 3 times and the highest recorded value was used for subsequent analysis (Quanjer et al. 1993). A hand-held mouth pressure meter (Ferraris Respiratory Europe, Hertford, UK) measured MIP as an index of global inspiratory muscle strength. The mouthpiece assembly incorporated a 1 mm orifice to prevent glottic closure during inspiratory efforts. Manoeuvres were performed in an upright standing posture, were initiated from residual volume, and sustained for at least 1 s. Repeat measurements separated by 30 s were taken until 3 values within 5 cmH_2O of each other were produced (McConnell 2007). The highest recorded value was used for subsequent analysis. Throughout hyperpnoea trials and the $\dot{V}O_2$ max test, respiratory variables were measured breath by breath (ZAN 600USB, Nspire Health, Oberthulba, Germany). Subjects wore a facemask (model 7940, Hans Rudolph, Kansas City, Missouri) connected to a pneumotachograph, and during volitional hyperpnoea tests, a two-way non-rebreathing valve (model 2730, Hans Rudolph, Kansas City, Missouri) was attached distally to the pneumotachograph allowing additional CO_2 to be added to the inspirate.

Blood sampling and analysis

Arterialised venous blood was sampled from a dorsal hand vein via an indwelling cannula (Forster et al. 1972; McLoughlin et al. 1992). Arterialisation was ensured by immersing the hand in water at ~40˚C for 10 min prior to cannulation and by warming the hand during volitional hyperpnoea tests using an infrared lamp. Blood samples were drawn into a 2 ml pre-heparinsed syringe (PICO 50, Radiometer, Copenhagen, Denmark) and analysed immediately for blood gases (ABL520, Radiometer, Copenhagen, Denmark), including the partial pressure of carbon dioxide (PCO_2) and pH, and $[lac^-]_B$ (Biosen C_line Sport, EKF Diagnostics, Barleben,

Germany). Plasma bicarbonate concentration ($[HCO_3^-]$) was calculated from PCO_2 and pH values using the Henderson Hasselbalch equation:

$$pH = pK + \log \frac{[HCO_3^-]}{0.03 \times PCO_2}$$

$[HCO_3^-]$ was then subsequently incorporated into the Siggaard-Anderson equation to calculate base excess of the extracellular fluid (BE_{ECF}) (Siggaard-Anderson and Fogh-Anderson, 1995):

$$BE_{ECF} = 0.93 \times \left([HCO_3^-] - 24.4 + 14.83 \times (pH - 7.40)\right)$$

Maximal exercise test

Subjects performed a maximal incremental cycling test on an electromagnetically-braked cycle ergometer (Excalibur Sport, Lode, Groningen, The Netherlands). At the onset of the exercise test, cycling power was 0 W and subsequently increased by 10 W every 15 s in order to result in exercise intolerance within approximately 10 min. This rapid incremental protocol was selected to maximise $\dot{V}_E$ at the cessation of exercise and reflect intense endurance exercise. The power at which exercise intolerance ensued defined maximal power output ($\dot{W}$ max), and the highest oxygen uptake ($\dot{V}O_2$) and $\dot{V}_E$ recorded in any 30 s period defined $\dot{V}O_2$ max and $\dot{V}_E$ max, respectively.

Volitional hyperpnoea

Volitional hyperpnoea was performed whilst seated on the cycle ergometer in an identical body position to that adopted during the maximal exercise test. Subjects were instructed to increase $\dot{V}_E$ and f_R in a square wave manner to a level commensurate with 85 % $\dot{V}_E$ max. An

audio metronome paced f_R and real-time visual feedback of $\dot{V}_E$ was provided throughout the test. In order to provide a breathing challenge representative of the work of breathing of intense exercise hyperpnoea, the volitional hyperpnoea tests was performed at the $\dot{V}_E$, V_T, f_R and T_I/T_{TOT} associated with 85% $\dot{V}_E$max since pilot work showed that this was the maximum square wave response that could be maintained for 10 min. This methodology is deemed superior to an arbitrary %MVV as it reflects the work of breathing of intense endurance exercise as for a given $\dot{V}_E$ greater than approximately 60 L·min^{-1} the work of breathing of exercise hyperpnoea can overestimated by as much as 25 % when a spontaneous breathing pattern is adopted during volitional hyperpnoea (Coast et al. 1993). Isocapnia was maintained during volitional hyperpnoea by adding CO_2 into the inspiratory circuit in order to maintain resting PCO_2. Blood was sampled at rest and at 2 min intervals.

Intervention

IMT was performed using an inspiratory pressure-threshold device (POWERbreathe®, Gaiam, UK). The IMT group performed 30 dynamic inspiratory efforts twice daily for 6 weeks against a pressure-threshold load of ~50% MIP. Thereafter, subjects periodically increased the load to a level that would permit them to only just complete 30 manoeuvres. Each inspiratory manoeuvre was initiated from residual volume and subjects strove to maximise V_T. This protocol is known to be effective in eliciting an adaptive response (Johnson et al. 2007; McConnell and Lomax 2006; McConnell and Sharpe 2005; Romer et al. 2002a,b; Volianitis et al. 2001). Subjects completed a training diary to record IMT adherence and habitual training, which the control group also recorded. The control group performed no IMT during the 6 week intervention since the duration and breathing pattern of the volitional hyperpnoea test was fixed pre and post-intervention (i.e. no measures of performance) and therefore the responses between groups were not influenced by either motivation or expectation.

Statistical analyses

Statistical analyses were performed using SPSS for Windows (SPSS, Chicago, Illinois, USA). Pre- and post-intervention results, differences over time during volitional hyperpnoea and group interactions were compared using one-way or two-way ANOVA for repeated measures and Tukey's HSD post-hoc analysis. Pearson product-moment correlation coefficients were calculated to assess the relationship between selected variables. Statistical significance was set at $P \leq 0.05$. Results are presented as mean ± SD.

Results

Pulmonary function and maximal inspiratory pressure

Baseline pulmonary function and MIP were all within normal limits (Table 1). The IMT group demonstrated excellent training compliance (91% adherence) and subjects' habitual training remained unchanged in both IMT and control groups. MIP increased from 147 ± 27 to 189 ± 27 cmH_2O (+31 ± 22%) following IMT ($P<0.01$). No change was observed in the control group (pre- vs. post-: 163 ± 19 vs. 166 ± 20 cmH_2O).

Responses to volitional hyperpnoea

Ventilatory and acid base responses to volitional hyperpnoea pre- and post-intervention for the control and IMT groups are shown in Table 2. Throughout hyperpnoea pre- and post-intervention (min 0 to min 10) there were no differences in breathing pattern and acid base balance between groups (Table 2). $\dot{V}_E$ during volitional hyperpnoea represented 72 ± 8% and 81 ± 19% of MVV_{10} in control and IMT groups, respectively. PCO_2 was maintained at resting levels throughout hyperpnoea and was not different between groups (Figure 1).

Prior to the intervention in the control group, $[lac^-]_B$ increased from 0.76 ± 0.24 mmol·L^{-1} at rest to 1.50 ± 0.60 mmol·L^{-1} and in the IMT group from 0.85 ± 0.40 mmol·L^{-1} at rest to 2.02 ± 0.85 mmol·L^{-1} following 10 min volitional hyperpnoea (P<0.05) (Figure 2). The non-significant difference in the absolute increase in $[lac^-]_B$ between groups is likely due to the different relative loads of the imposed hyperpnoea (control: 72 %MVV; IMT: 81 %MVV). The $[lac^-]_B$ response to volitional hyperpnoea was unchanged in the control group following the intervention. Conversely, $[lac^-]_B$ during volitional hyperpnoea was reduced following IMT, with significant 17 ± 37% and 25 ± 34% reductions being observed at 8 and 10 min, respectively. These changes were different between groups (significant group × time × trial interaction effect, P<0.05).

Correlations amongst variables

Prior to the intervention, increases in $[lac^-]_B$ during volitional hyperpnoea were not correlated with any measure of pulmonary function, MIP, endurance training status ($\dot{V}O_2max$, $\dot{W}max$), or ventilatory responses to volitional hyperpnoea. However, baseline MIP was negatively correlated with relative IMT-induced increases in MIP (r=-0.70, P<0.05).

Discussion

Main findings

The main findings of this study were that 10 min of volitional hyperpnoea approximately doubled resting $[lac^-]_B$, and that 6 weeks of pressure threshold IMT attenuated this increase by 25%. These findings strongly support the notion that the respiratory muscles are capable of increasing $[lac^-]_B$ and are the first to show that this can be attenuated through specific IMT. This observation may help to explain some of the RMT-mediated reductions in $[lac^-]_B$ previously observed during whole-body exercise.

Volitional hyperpnoea and blood lactate concentration

We report an increased $[lac^-]_B$ from rest of 0.96 ± 0.58 mmol·L^{-1} (n=22; range: 0.20 – 2.50 mmol·L^{-1}) during 10 min of intense volitional hyperpnoea at 85% $\dot{V}_E$ max (131 ± 4.36 L·min^{-1}; n=22). These findings contrast those of Spengler et al. (2000) who reported unchanged $[lac^-]_B$ during volitional hyperpnoea at a lower relative $\dot{V}_E$ (~62 %MVV; 122.4 L·min^{-1}), however, are similar to others with a similar relative breathing challenge (72 %MVV, Martin et al. 1984; 70 %MVV, Verges et al. 2007). These data confirm that increases in $[lac^-]_B$ during volitional hyperpnoea are positively related to the ratio of $\dot{V}_E$ to MVV (Martin et al. 1984; Johnson et al. 2006) and may, in part, explain the different $[lac^-]_B$ responses observed in previous studies in response to volitional hyperpnoea and between groups in this study. This study provides novel data that the work of breathing of volitional hyperpnoea when rigorously matched to high-intensity exercise hyperpnoea is sufficient to result in net lactate release from the respiratory muscles.

The potential for respiratory alkalosis to elevate $[lac^-]_B$ is well documented (Davies et al. 1986; LeBlanc et al. 2002). Consequently we were careful to maintain, with considerable accuracy, resting PCO_2 throughout the 10 min of volitional hyperpnoea (see Figure 1). Other measures of acid base status also remained unchanged from rest during volitional hyperpnoea in both groups pre- and post-intervention. We are thus confident that the increase in $[lac^-]_B$ during volitional hyperpnoea was not a consequence of respiratory alkalosis and we attribute the increase in $[lac^-]_B$ to lactate efflux from the respiratory muscles

Inspiratory muscle training and blood lactate concentration

The attenuated increase in $[lac^-]_B$ during volitional hyperpnoea following IMT is similar to that observed in healthy subjects performing an exhaustive respiratory endurance test at ~70 %MVV following VIH training, although, this reduction did not exceed that of a control (Verges

et al. 2007). However, the authors fail to report their attempts to maintain end tidal CO_2 and / or PCO_2 during the respiratory endurance test, furthermore, subjects were prescribed a pre-determined arbitrary breathing pattern, of which has been criticised previously for failing to accurately represent the work of breathing of exercise hyperpnoea (Coast et al. 1993). The IMT-mediated reduction in $[lac^-]_B$ observed in the present study is also similar to the reduction often observed during submaximal, whole-body exercise following both IMT (Griffiths and McConnell 2007; McConnell and Sharpe 2005; Romer et al. 2002b; Volianitis et al. 2001) and VIH (Leddy et al. 2007; Spengler et al. 1999), however, whether these observations during volitional hyperpnoea and exercise share a common mechanistic explanation is unclear.

RMT-mediated reductions in $[lac^-]_B$ at submaximal exercise intensities occur (Leddy et al. 2007; McConnell and Sharpe 2005) when lactate production and release from the respiratory muscles is probably negligible given the relative ventilatory demand and the reduced activation of less efficient accessory muscles (Martin et al. 1984; Johnson et al. 2006). Hence, under such conditions it seems more likely that reductions in $[lac^-]_B$ result from increased metabolism of lactate by the trained respiratory muscles (Spengler et al. 1999) rather than a decrease in net lactate release. Conversely, during high-intensity exercise where $\dot{V}_E$ is increased above that of sub-maximal exercise similar to the $\dot{V}_E$ of volitional hyperpnoea in this study (Kohl et al. 1997: 130.9 $L \cdot min^{-1}$; Spengler et al. 1999; 147.3 $L \cdot min^{-1}$), it is possible that IMT-mediated inspiratory muscle adaptation contributed to lowering $[lac^-]_B$ through affecting both lactate clearance by and efflux from the trained inspiratory muscles.

The plasticity of the inspiratory muscles has been well documented (McConnell and Romer 2004; Powers et al. 1997). It is thus attractive to suggest that changes in inspiratory muscle morphology may explain, in part, the attenuated hyperpnoea-mediated increase in $[lac^-]_B$ following IMT.. An increase in the content of inspiratory muscle monocarboxylate transport (MCT) proteins (McConnell and Sharpe 2005), which facilitate inter- and intra-cellular lactate

shuttling in sarcolemmal and mitochondrial membranes, respectively (Brooks et al. 1999; Dubouchaud et al. 2000) have been reported following endurance (Baker et al. 1998; Burgomaster et al. 2007) and strength (Juel et al. 2004) based training regimens. It is possible that similar adaptations would occur following both IMT (strength-orientated) and VIH (endurance-orientated) training and may explain, in part, the decrease in $[lac^-]_B$ observed during whole-body exercise and volitional hyperpnoea.

Diaphragm hypertrophy has been reported with an approximate 10% increase in diaphragm thickness (Downey et al. 2007; Enright et al. 2006) and 21 % increase in the size of type II muscle fibres (Ramírez-Sarmiento et al. 2002) occurring after 6 and 5 weeks of IMT, respectively. Increasing inspiratory muscle fibre cross-sectional area and subsequently strength decreases the relative intensity for a given absolute work load, which may reduce/delay fast twitch fibre recruitment and thus lactate production (Marcinik et al. 1991). A decrease in relative workload per muscle fibre may also decrease blood flow occlusion, which may influence lactate production and/or clearance (Marcinik et al. 1991).

Finally, the attenuated $[lac^-]_B$ response to volitional hyperpnoea following IMT may also reside in a training-induced increase in the oxidative capacity of the inspiratory muscles. In support of this notion, Ramírez-Sarmiento et al. (2002) reported 38% increases in the number of type I muscle fibres in the external intercostals following 5 weeks IMT. Moderate intensity, high repetitions strength training, similar to the IMT protocol used in the this study can increase oxidative enzyme activity (Costill et al. 1979; Sale et al. 1990) and reduce $[lac^-]_B$ via an increase in mitochondria derived ATP and lactate oxidation (Holloszy and Coyle 1984). Since it is probable that similar oxidative adaptations would also occur following VIH (endurance-orientated) training (Kohl et al. 1997; Leddy et al. 2007; Spengler et al. 1999), this offers an attractive explanation for the decrease in $[lac^-]_B$ observed during whole body exercise (Griffiths and McConnell 2007; Kohl et al. 1997; Leddy et al. 2007; McConnell and Sharpe 2005; Romer

et al. 2002b; Spengler et al. 1999; Volianitis et al. 2001) and volitional hyperpnoea (present study; Verges et al. 2007) following these dissimilar training stimuli.

Inspiratory muscle strength

The 32% increase in MIP following 6 weeks of IMT is consistent with previous studies (Downey et al. 2007; Edwards and Cooke 2004; Gething et al. 2004; Griffiths and McConnell 2007; McConnell and Sharpe 2005; Romer et al. 2002a,b; Williams et al. 2002). The suggestion that IMT-mediated increases in MIP are partly dependent upon baseline MIP (Johnson et al. 2007) was substantiated in the present study by the negative correlation (r=-0.70) observed between these variables. These novel data lend credence to the concept that resistance training-induced increases in strength are partly dependent upon baseline status (Kraemer and Ratamess 2004). However, the significance of our observation is unclear since IMT-mediated increases in MIP were not related to the reduction in $[lac^-]_B$, suggesting that an increase in inspiratory muscle strength *per-se* is not an important determinant of the physiological adaptations following-IMT.

Conclusions

In summary, the present study provides novel evidence that increases in $[lac^-]_B$ during volitional hyperpnoea can be attenuated following IMT. These data thus suggest that the inspiratory muscles were the source of at least part of this reduction, and provide a possible explanation for at least some of the IMT-mediated reductions in $[lac^-]_B$ previously observed during whole-body exercise. The precise mechanisms that underpin these changes remain unknown, but an IMT-mediated increase in the oxidative and/or lactate transport capacity of the inspiratory muscles is an attractive possibility that merits further investigation.

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Table 1. Descriptive characteristics of the subjects (mean ± SD).

	Control (n=11)	IMT (n=11)
Age (years)	28.5 ± 4.1	22.4 ± 4.5 *
Body mass (kg)	75.5 ± 5.6	78.6 ± 9.7
Height (cm)	176.9 ± 7.4	181.6 ± 7.6
FVC (L)	5.32 ± 0.55 (104 ± 8)	5.67 ± 0.92 (106 ± 12)
FEV_1 (L)	4.28 ± 0.62 (99 ± 11)	4.93 ± 0.67 (109 ± 11)
FEV_1/FVC (%)	80.3 ± 7.1 (96 ± 9)	87.7 ± 8.3 (103 ± 9) *
MVV_{10} ($L \cdot min^{-1}$)	176.3 ± 15.0 (102.3±10.9)	173.4 ± 53.7 (122.4±30.3))
MIP (cmH_2O)	163 ± 19 (113 ± 4)	147 ± 27 (119 ± 5)
$\dot{V}O_2 max$ ($L \cdot min^{-1}$)	3.75 ± 0.55	3.77 ± 0.75
$\dot{W}max$ (W)	353 ± 44	362 ± 38

FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 s; MVV_{10}, maximum voluntary ventilation in 10 s. Values in parenthesis represent the percent of predicted values (Quanjer et al. 1993; Wilson et al. 1984). *, $P<0.05$.

Table 2. Ventilatory and acid-base responses to volitional hyperpnoea prior to and following the intervention. Data are mean of min 2 to 10 during volitional hyperpnoea (mean ± SD).

	Control (n=11)		IMT (n=11)	
	Pre	Post	Pre	Post
$\dot{V}_E$ ($L \cdot min^{-1}$)	127.1 ± 2.3	128.7 ± 2.4	132.9 ± 9.6	136.8 ± 3.2
V_T (L)	2.62 ± 0.04	2.64 ± 0.07	2.60 ± 0.03	2.66 ± 0.06
f_R ($breaths \cdot min^{-1}$)	50 ± 0	50 ± 0	52 ± 0	52 ± 0
T_I/T_{TOT}	0.44 ± 0.00	0.44 ± 0.00	0.52 ± 0.00	0.49 ± 0.00
pH	7.392 ± 0.031	7.406 ± 0.024	7.397 ± 0.023	7.395 ± 0.014
$[H^+]$ ($nmol \cdot L^{-1}$)	40.6 ± 2.9	39.4 ± 2.2	40.2 ± 2.2	40.3 ± 1.0
$[HCO_3^-]$ ($mmol \cdot L^{-1}$)	26.0 ± 0.9	26.9 ± 2.5	26.5 ± 1.4	27.0 ± 1.3
BE_{ECF} ($mEq \cdot L^{-1}$)	1.38 ± 0.91	1.72 ± 2.04	1.52 ± 1.11	2.35 ± 1.23

$\dot{V}_E$, minute ventilation; V_T, tidal volume; f_R, respiratory frequency; T_I/T_{TOT}, duty cycle; $[H^+]$, hydrogen ion concentration; $[HCO_3^-]$, plasma bicarbonate concentration; BE_{ECF}, base excess of the extracellular fluid.

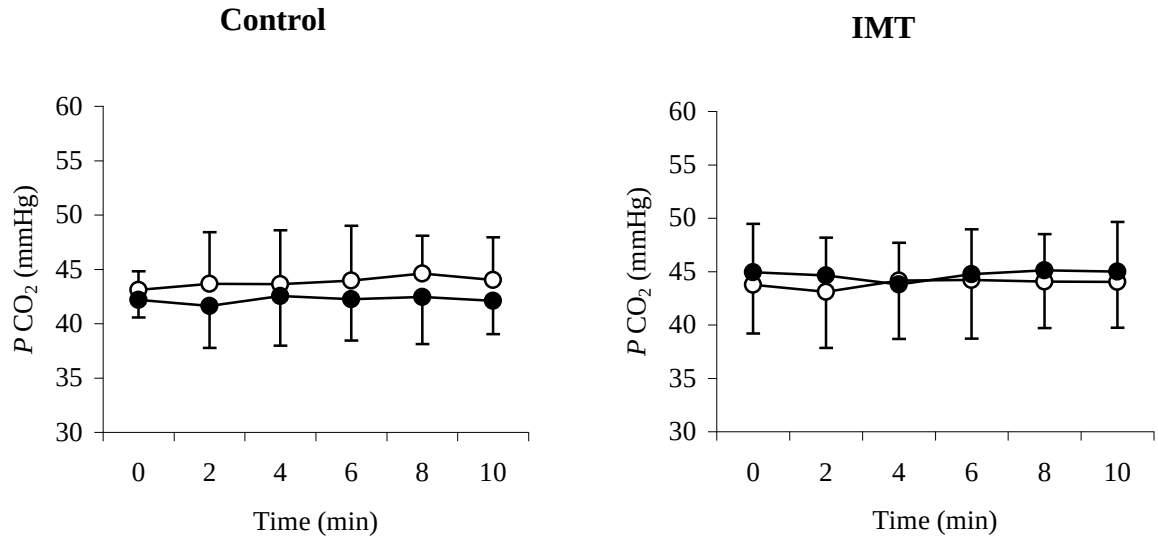

Fig. 1 Partial pressure of carbon dioxide in arterialised venous blood (PCO_2) during volitional hyperpnoea pre- (○) and post- (●) intervention in control and IMT groups.

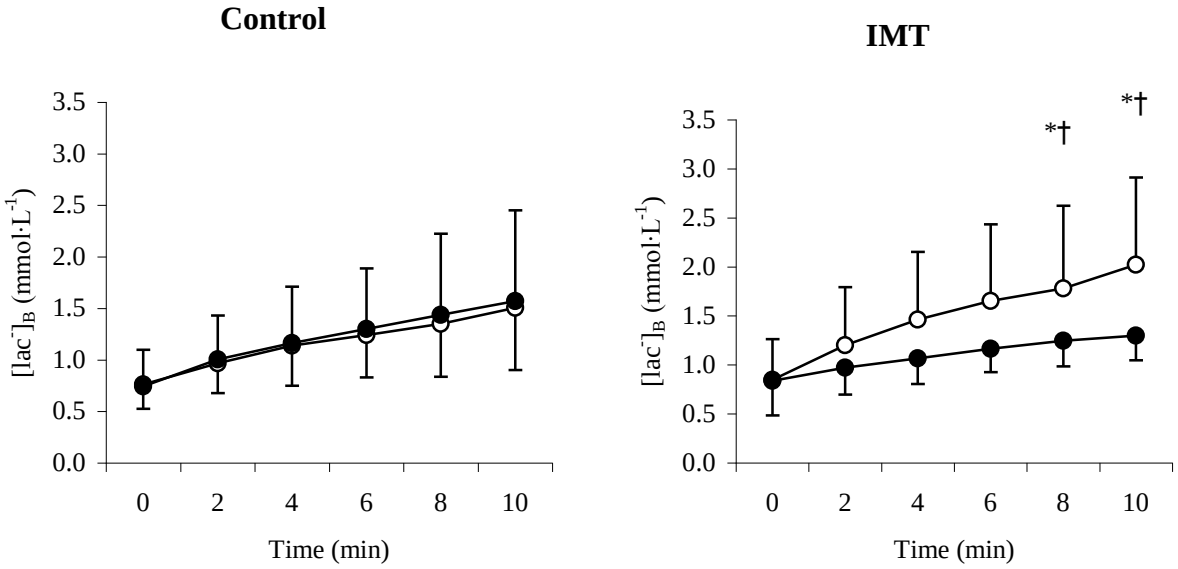

Fig. 2 Blood lactate concentration ($[lac^-]_B$) during volitional hyperpnoea pre- (○) and post- (●) intervention in control and IMT groups. *Significant difference from pre-IMT (P<0.05). †Significant group × time interaction effect (P<0.05).