ORIGINAL ARTICLE

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Decreased exercise blood lactate concentrations after respiratory endurance training in humans

Abstract For many years, it was believed that ventilation does not limit performance in healthy humans. Recently, however, it has been shown that inspiratory muscles can become fatigued during intense endurance exercise and decrease their exercise performance. Therefore, it is not surprising that respiratory endurance training can prolong intense constant-intensity cycling exercise. To investigate the effects of respiratory endurance training on blood lactate concentration and oxygen consumption ($\dot{V}O_2$) during exercise and their relationship to performance, 20 healthy, active subjects underwent 30 min of voluntary, isocapnic hyperpnoea 5 days a week, for 4 weeks. Respiratory endurance tests, as well as incremental and constant-intensity exercise tests on a cycle ergometer, were performed before and after the 4-week period. Respiratory endurance increased from 4.6 (SD 2.5) to 29.1 (SD 4.0) min ($P < 0.001$) and cycling endurance time was prolonged from 20.9 (SD 5.5) to 26.6 (SD 11.8) min ($P < 0.01$) after respiratory training. The $\dot{V}O_2$ did not change at any exercise intensity whereas blood lactate concentration was lower at the end of the incremental [10.4 (SD 2.1) vs 8.8 (SD 1.9) mmol $\cdot$ l^{-1}, $P < 0.001$] as well as at the end of the endurance exercise [10.4 (SD 3.6) vs 9.6 (SD 2.7) mmol $\cdot$ l^{-1}, $P < 0.01$] test after respiratory training. We speculate that the reduction in blood lactate concentration was most likely caused by an improved lactate uptake by the trained respiratory muscles. However, reduced exercise blood lactate concentrations per se are unlikely to explain the improved cycling performance after respiratory endurance training.

Introduction

It has generally been accepted that ventilation does not limit exercise performance in healthy humans (Leith and Bradley 1976; Dempsey 1986). More recently, Johnson et al. (1993) and Mador et al. (1993) have shown that the diaphragm fatigues during exercise at a constant intensity of at least 80% of maximal oxygen consumption. After an endurance competition, overall inspiratory muscle fatigue (reduced inspiratory function) has been found (Loke et al. 1982; Hill et al. 1991; Chevrolet et al. 1993) and many hours are needed for complete recovery. Fatigued respiratory muscles in turn have been shown to decrease exercise performance (Martin et al. 1982; Mador and Acevedo 1991b). In addition, it has been found that endurance training of respiratory muscles can increase constant-intensity cycling time in sedentary subjects by 50% (Boutellier and Piwko 1992) and in endurance trained (athletic) subjects by 38% (Boutellier et al. 1992).

At present, we do not know the mechanism by which endurance training of the respiratory muscles prolongs the duration of constant-intensity exercise. One possibility is the occurrence of a reduction in blood lactate accumulation during exercise after respiratory training. This hypothesis has been inferred from observations that whole-body endurance training substantially reduces blood lactate concentrations at a given exercise intensity (Casaburi et al. 1987; MacRae et al. 1992). However, in previous studies, we have been unable to demonstrate that respiratory endurance training consistently alters blood lactate concentrations measured at the end of exercise. Although a significant reduction in the increase of blood lactate concentration above the resting level has been seen in sedentary subjects [5.4 (SD 0.3) vs 2.9 (SD 1.0) mmol $\cdot$ l^{-1}; Boutellier and Piwko 1992], in athletic

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subjects, the changes were not significant [2.2 (SD 1.4) vs 2.7 (SD 1.4) mmol · l^{-1}; Boutellier et al. 1992].

With this in mind we decided to examine the accumulation of blood lactate during incremental and constant-intensity exercise before and after endurance training of the respiratory muscles in 20 endurance trained subjects.

Methods

Subjects

A group of 20 healthy, athletic, male subjects [mean age: 26.3 (SD 5.5) years; height: 179.1 (SD 5.9) cm; body mass: 70.3 (SD 7.8) kg] participated in the study. Their weekly endurance training lasted at least 3 h. The subjects were informed in detail about the tests and training involved before they gave their written informed consent (in accordance with the Helsinki Declaration). They kept their habitual physical training constant for 2 weeks prior to the start of the study as well as throughout the entire testing and training period. For the record the subjects kept a diary in which they entered all details of training, including the respiratory training (see below).

Equipment

Respiratory training was performed by voluntary hyperpnoea. To avoid dizziness, the subjects used a portable device which allowed normocapnic rebreathing. The training device consisted of a latex balloon connected to a tube equiped with inlet and outlet valves. The valves permitted the addition of fresh air to the air inspired from the rebreathing balloon to add O_2 and to keep the end-tidal CO_2 fraction at a constant level. Tidal volume (V_T) was therefore slightly greater than the volume of the balloon. Breathing frequency (f_b) was paced by a digital metronome, DM-30 (Seiko, Tokyo, Japan).

Respiratory endurance tests (RET) were also performed with the training device. During the endurance run, minute ventilation ($\dot{V}_E$), V_T, and f_b were monitored with an ergo-spirometric apparatus, OxyconBeta (Mijnhardt, Bunnik, Netherlands), a breath-by-breath system which uses fast responding gas analysers (paramagnetic for O_2 and infrared for CO_2) and a turbine for volume measurements and which can be connected to a mouthpiece. Vital capacity (VC), forced expiratory volume in 1 s (FEV$_1$), peak expiratory flow (PEF), and maximal voluntary ventilation (MVV) were also measured with the OxyconBeta.

Incremental and constant-intensity exercise tests were performed on an electronically-braked cycle ergometer, Ergo-metrics 800S (Ergoline, Bitz, Germany). During these tests, $\dot{V}_E$, V_T, f_b, oxygen uptake ($\dot{V}O_2$) and carbon dioxide production were measured. Heart rate (f_c) was recorded in parallel with the respiratory variables using a PE4000 heart rate monitor (Polar Electro, Kempele, Finland).

Blood lactate concentrations were measured with an ESAT 6661 analyser (Eppendorf, Hamburg, Germany) using 20 µl of blood taken from an earlobe. To calibrate the analyser and to check the calibration after the measurements, tubes with a standard solution (10 mmol · l^{-1} lactate concentration) were added before and after a set of blood samples.

Protocol

Firstly, spirometric variables (VC, FEV$_1$, PEF, and MVV) were measured at least three times. The largest of three similar values was used for analysis. While the subjects familiarized themselves with the respiratory training device, they were asked to breathe with a V_T of 60% of their VC and with a f_b of 40–50 breaths · min^{-1}. These preliminary tests allowed us to choose a $\dot{V}_E$ which the subjects could maintain for only 10 min at most during the first respiratory endurance test (RET$_1$).

Peak oxygen consumption ($\dot{V}O_{2peak}$) and maximal work capacity (W_{max}) were measured on the cycle ergometer during an incremental exercise test 1 day later. The subjects started pedalling at 100 W and thereafter, the intensity was increased by 30 W every 2 min. The subjects were allowed to choose their own cycling frequency within a range of 70–90 rpm. After that, they kept the number of revolutions per minute constant to assure a constant muscle efficiency (Heinrich et al. 1968). The cycling frequency was presented to the subjects visually and was supervised by an investigator who told the subjects to adjust the speed if necessary. At the end of each intensity, a blood sample was taken. Ventilatory variables and f_c were recorded continuously. The intensity for the cycling endurance test (W_{CET}) was determined for each subject by averaging the anaerobic threshold values calculated by the following three methods: the modified heart rate deflection method (Conconi et al. 1982), the ventilatory threshold method (Wasserman and McIlroy 1964), and the lactate deflection method (Heck et al. 1985).

At least 3 days later, the first cycling endurance test (CET$_1$) was performed. After 3 min of cycling at 120 W, the intensity was increased to the individual level [W_{CET} = 294 (SD 37) W, representing 85 (SD 3) % W_{max} and 87 (SD 3) % $\dot{V}O_{2peak}$]. Ventilatory variables and f_c were measured continuously. Blood was taken every 5 min to measure lactate concentrations. The subjects were asked to cycle until they were exhausted. When they could no longer hold constant the number of revolutions per minute – even with the encouragement of the investigator – the test was stopped and the duration of the test noted. After 10 min RET$_1$ was performed. Mean $\dot{V}_E$ was 138.5 (SD 18.6) l · min^{-1} corresponding to 71 (SD 10) % of the initial MVV. When the subjects could no longer maintain target V_T or f_b for five consecutive breaths, the test was stopped and the duration of the test recorded.

After these control measurements, respiratory training was started. The subjects trained for 30 min continuously each day, 5 days a week, for 4 weeks. They performed the daily training at home and recorded all training in their diaries. To make sure that the training was being performed as prescribed, the subjects came to the laboratory every week, where the training device was attached to the OxyconBeta system and, under the supervision of an investigator, they performed their respiratory training. This allowed us to judge the progress of training and to determine the increase in $\dot{V}_E$ (5–10 l) either by raising target V_T (up to maximal 60% of VC) or f_b. The choice of breathing pattern during respiratory training has been shown to have no effect on the outcome of a cycling endurance test (Spengler et al. 1996). The subjects began with a $\dot{V}_E$ of 123 (SD 17) l · min^{-1} [corresponding to 63 (SD 11) % of initial MVV] in the 1st week, which increased to 162 (SD 21) l · min^{-1} [83 (SD 15) % of initial or 70 (SD 16) % of trained MVV] by the end of the 4th training week.

After the end of the respiratory training period, at least 5 days passed without respiratory training before the second cycling endurance test (CET$_2$) was performed followed by the second respiratory endurance test (RET$_2$) after a 10-min break. The RET$_2$ was discontinued after 30 min whether or not the subjects were tired. The exercise intensities for CET$_1$ and CET$_2$ were identical [W_{CET} = 294 (SD 37) W] as were V_T and f_b of RET$_1$ and RET$_2$. After a further 3 to 4 days, spirometric variables were measured and the post-training incremental cycling test was performed.

Statistics

For comparison of cycling endurance tests, values are presented at two different times:

1. The steady-state values of the cycling endurance tests were calculated by averaging, for each subject, the data from the 10th to 14th min (Table 2). As one subject stopped cycling after 11 min before respiratory training, we used the data of the

7th–11th min instead of the 10th–14th min for this subject. Steady-state blood lactate concentrations were measured in the 13th min (8th min for 1 subject).

2. To compare results at the end of exercise (Tables 1, 2), breath-by-breath values of the last completed minute were averaged.

Of the many tests performed during the study, four tests failed due to technical problems. Therefore, the number of subjects was not always 20.

To detect significant differences between tests before and after respiratory training, paired Student's t-tests were performed. For comparison of blood lactate concentrations during exercise before and after respiratory training, an analysis of variance was performed with a Student-Newman-Keuls post-hoc analysis. The level of significance was set to $P < 0.05$. Fisher's r to z P-values were used to test the correlation coefficients for significance.

Results

Spirometry and respiratory endurance

The subjects ($n = 19$) had normal lung functions which did not change with respiratory training: mean VC 5.7 (SD 0.7) compared to 6.0 (SD 0.8) l, mean FEV_1 90.1 (SD 8.1) compared to 88.1 (SD 8.4)% of VC, and mean PEF 10.9 (SD 1.8) compared to 11.2 (SD 1.2) $l \cdot s^{-1}$. The mean MVV increased significantly from 195.5 (SD 24.8) to 232.7 (SD 28.4) $l \cdot min^{-1}$ after respiratory training ($P < 0.001$).

Respiratory endurance time increased significantly from an average of 4.6 (SD 2.5) before to 29.1 (SD 4.0) min after training ($P < 0.001$). It should be noted that after training 19 out of 20 subjects reached 30 min without showing any signs of fatigue.

Incremental exercise

Respiratory training did not affect $\dot{V}O_{2peak}$, W_{max}, maximal f_c (Table 1), or the intensity averaged from the calculations of the three anaerobic threshold [294 (SD 37) compared to 291 (SD 38) W]. From 220 W onward, blood lactate concentrations were significantly reduced after respiratory training (Fig. 1). Blood lactate concentrations at the end of exercise decreased by

Table 1 Respiratory variables, heart rate (f_c), and exercise intensity of 20 subjects during the last minute of incremental exercise (peak) before and after respiratory training. $\dot{V}_E$ Minute ventilation; V_T tidal volume, f_b breathing frequency, $P_{ET}CO_2$ end-tidal CO_2 partial pressure, $\dot{V}O_2$ oxygen consumption, $\dot{V}CO_2$ carbon dioxide production
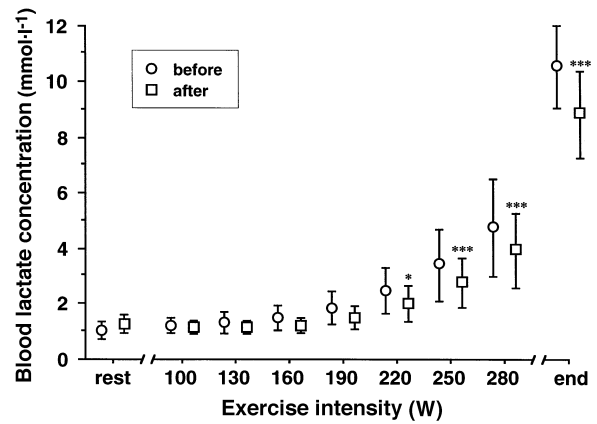
	Before mean	SD	After mean	SD
$\dot{V}_E$ ($l \cdot min^{-1}$)	141.7	26.1	147.3	21.1
V_T ($ml \cdot breath^{-1}$)	3074	418	3259	514 **
f_b ($breaths \cdot min^{-1}$)	46.6	8.2	46.3	9.0
$P_{ET}CO_2$ (mmHg)	36.0	4.4	35.2	4.2
$\dot{V}O_2$ ($ml \cdot min^{-1}$)	4310	591	4452	522
$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	61.5	6.7	63.8	8.0
$\dot{V}CO_2$ ($ml \cdot min^{-1}$)	4624	786	4926	710 ***
f_c ($beats \cdot min^{-1}$)	181	8	179	6
Exercise intensity (W)	343	41	345	40

$**P < 0.01$, $***P < 0.001$

Fig. 1 Blood lactate concentration (mean and SD) plotted at rest and as a function of the intensity of incremental exercise before and after respiratory training. $n = 19$; $*P < 0.05$, $***P < 0.001$

Table 2 Respiratory variables (18 subjects) and heart rate (f_c; 20 subjects) during the 10th to 14th min (steady state) and during the last minute (end) of cycling endurance exercise test before (CET_1) and afte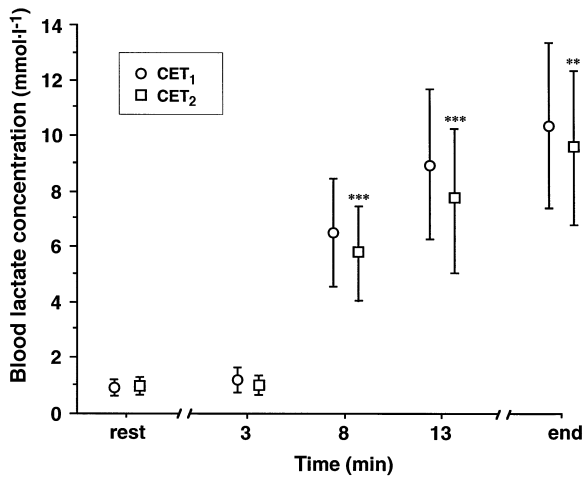r respiratory training (CET_2). $\dot{V}_E$ Minute ventilation, V_T tidal volume, f_b breathing frequency, $P_{ET}CO_2$ end-tidal CO_2 partial pressure, $\dot{V}O_2$ oxygen consumption, $\dot{V}CO_2$ carbon dioxide production

	Steady state				End			
	CET_1		CET_2		CET_1		CET_2	
	mean	SD	mean	SD	mean	SD	mean	SD
$\dot{V}_E$ ($l \cdot min^{-1}$)	109.7	17.1	113.9	15.4	130.1	19.7	141.6	24.1***
V_T ($ml \cdot breath^{-1}$)	3088	552	3161	611	2830	402	2829	410
f_b ($breaths \cdot min^{-1}$)	36.5	6.3	36.8	6.7	46.4	7.2	50.6	9.0**
$P_{ET}CO_2$ (mmHg)	39.7	4.7	38.8	3.4	32.9	3.5	30.8	3.4***
$\dot{V}O_2$ ($ml \cdot min^{-1}$)	3941	631	3986	605	3956	691	4035	660
$\dot{V}CO_2$ ($ml \cdot min^{-1}$)	3926	625	4049	742	3922	614	4071	727*
f_c ($beats \cdot min^{-1}$)	171	9	169	8	182	9	181	8

$* P < 0.05$, $** P < 0.01$, $*** P < 0.001$

Fig. 2 Blood lactate concentration (mean and SD) measured at rest and during cycling endurance exercise test before (*CET₁*) and after (*CET₂*) respiratory training. $n = 20$; $**P < 0.01$, $***P < 0.001$
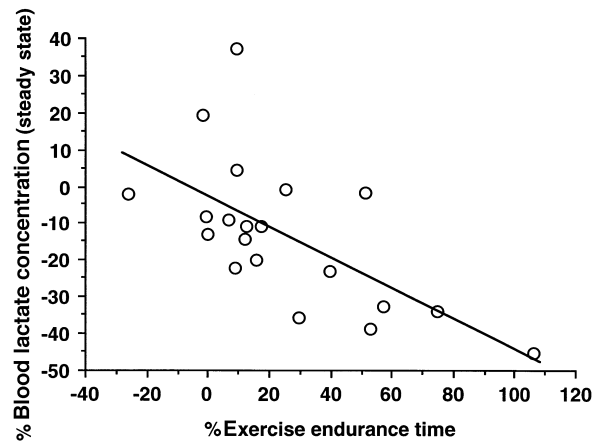

Fig. 3 Decrease in blood lactate concentration during steady-state exercise (13th min) plotted as a function of the increase in exercise time. Values are calculated as the percentage change in both parameters measured from before to after respiratory endurance training. $n = 20$; $y = -0.42\,x - 2.49$

Fig. 4 Change of oxygen uptake ($\dot{V}O_2$ mean of 10th to 14th min) plotted as a function of the change in blood lactate concentration during steady-state exercise (13th min). Values are calculated as the difference in both parameters measured from before to after respiratory endurance training. $n = 18$

1.53 mmol · l^{-1} on average (Fig. 1, $P < 0.001$) after respiratory training.
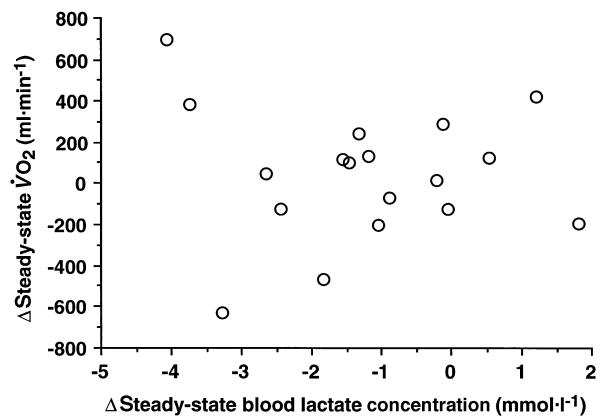
Endurance exercise

Mean cycling time during endurance exercise significantly increased from 20.9 (SD 5.5) min before, to 26.6 (SD 11.8) min after respiratory training ($P < 0.01$). This represents an average 27% elevation above CET$_1$. After respiratory training, $\dot{V}O_2$ and f_c, whether measured during the 10th to 14th min or at the end of CET$_2$ were unchanged (Table 2). Blood lactate concentrations were significantly reduced from 5 min of exercise onward (Fig. 2). Steady-state $\dot{V}_E$ of CET$_2$ was not influenced by respiratory training (Table 2). Also, V_T and f_b as well as end tidal partial pressure of carbon dioxide ($P_{ET}CO_2$) did not change significantly. During the last minute of CET$_2$, $\dot{V}_E$ was higher compared to CET$_1$ (Table 2). This increase was caused by a higher f_b whereas V_T remained constant. The higher $\dot{V}_E$ caused a significant reduction of $P_{ET}CO_2$.

A possible influence of blood lactate concentration on cycling endurance could be inferred from the significant correlation between the percentage decrease in blood lactate concentration, measured either at steady-state ($r = 0.64$, $P < 0.01$; Fig. 3) or end exercise ($r = 0.54$, $P < 0.05$), with the percentage increase of endurance time.

To detect a shift in the balance of aerobic versus anaerobic metabolism, we compared blood lactate concentrations with $\dot{V}O_2$. Not only was $\dot{V}O_2$ unchanged after respiratory training (Table 2) but also there was no correlation between the change in blood lactate concentration and the change in $\dot{V}O_2$ before and after respiratory training whether measured during steady state ($r = 0.06$, $P = 0.80$; Fig. 4) or at the end of exercise ($r = 0.27$, $P = 0.29$).

Discussion

The main findings of the present study were prolonged cycling endurance times but unchanged incremental cycling performance after respiratory training. During both tests after respiratory training, blood lactate concentrations were reduced and $\dot{V}O_2$ remained unchanged. We will discuss first the effectiveness of the respiratory training and then focus on the main findings.

The effectiveness of the respiratory training was evident from the significant increases of MVV and respiratory endurance time. Our observation of increased MVV and respiratory endurance time was in agreement with other studies where healthy subjects have received vigorous respiratory endurance training (Leith and Bradley 1976; Morgan et al. 1987; Fairbarn et al. 1991; Boutellier et al. 1992). The results of the incremental

exercise test and the information in the training diaries indicated that the subjects did not change their level of activity during the study period: $\dot{V}O_{2peak}$, W_{max}, and the intensity at the anaerobic threshold were similar before and after respiratory training. As has been observed in earlier studies (Morgan et al. 1987; Fairbarn et al. 1991; Boutellier et al. 1992), respiratory endurance training did not affect maximal performance in the incremental exercise test. Since the physical status of the subjects did not change with respiratory training but MVV and respiratory endurance time increased, we concluded that respiratory training was effective and that it was the most likely reason for the changes observed during the study.

As a consequence of respiratory training, in the incremental exercise test, blood lactate concentrations were significantly reduced at intensities higher than 220 W. The reduced blood lactate concentration had no influence on either the subjects' maximal performance or on $\dot{V}_E$. During constant-intensity exercise after respiratory training, blood lactate concentrations were also reduced whereas steady-state ventilation (10th–14th min) remained the same. As the decrease of blood lactate concentration was not accompanied by a decrease of $\dot{V}_E$ during incremental and endurance exercise, the two variables are not necessarily linked. This observation confirms earlier studies (Hagberg et al. 1990; Busse et al. 1991) which have also indicated that the link between $\dot{V}_E$ and blood lactate concentration is not as tight as has been shown by Casaburi et al. (1987) after whole-body endurance training. They, however, have concluded that the endurance training resulted in substantially reduced $\dot{V}_E$, an effect probably linked to the reduction in blood lactate concentration. This link does not exist after respiratory training.

To explain the reduced blood lactate concentrations after respiratory training, we would suggest two possible mechanisms:

1. Working muscles produced less lactate because of a reduced overall energy demand due to less respiratory work or
2. Trained respiratory muscles used more lactate as fuel for their own activity.

Previously (Boutellier and Piwko 1992), we have favoured the first explanation because $\dot{V}_E$ was drastically reduced during endurance cycling after respiratory training in sedentary subjects. The present findings clearly disagree with our earlier hypothesis because $\dot{V}_E$ was not reduced during the steady-state phase after respiratory training. Therefore, a reduced lactate production after respiratory training was unlikely in the present study. In addition, increased aerobic relative to anaerobic energy production would have been associated with an increased $\dot{V}O_2$ (Boutellier et al. 1990). We did not observe a higher $\dot{V}O_2$ during CET_2 compared to CET_1 (Table 2) nor did the changes in blood lactate concentration correlate with changes in $\dot{V}O_2$ (Fig. 4). This suggests that the reduced blood lactate accumulation

after respiratory training was not due to a decreased lactate production.

Therefore we speculate that in the present study the subject's respiratory muscles increased their ability to metabolize lactate and, as a result, increased lactate removal from circulating blood. This is in accordance with investigations in animals where respiratory muscles have been shown to consume rather than produce lactate even during intense exercise (Fregosi and Dempsey 1986; Manohar and Hassan 1991). Rochester and Briscoe (1979) have shown that approximately one half of the energy required by the diaphragm is derived from carbohydrate metabolism, primarily in the form of lactate utilisation. Furthermore, after whole-body endurance training in humans, blood lactate concentrations have been found to be reduced due to an increased rate of lactate metabolic clearance during intense exercise (MacRae et al. 1992).

As a possible mechanism for an improved lactate clearance after respiratory training, we would suggest that trained respiratory muscles increase their ability to metabolize lactate. A study performed by Bigard et al. (1992) has supported this suggestion. They have found that 12 weeks of endurance training enhanced the ratio of the heart-specific lactate dehydrogenase isozyme (LDH_1) to total LDH activity in the rat diaphragm by 92%. Since the heart is known to metabolize a significant amount of lactate, an increased lactate metabolization by the diaphragm as the result of a shift of isozyme activity might be the cause for the decrease in blood lactate concentration found during exercise in our subjects. If respiratory muscles use more lactate as fuel, they can spare glycogen. A significant relationship between contractile fatigue of the diaphragm and glycogen depletion has been shown in rabbits (Ferguson et al. 1990) and suggested in humans (Martin et al. 1982; Chevrolet et al. 1993). So, the subjects of the present study might have postponed glycogen depletion and thereby postponed respiratory muscle fatigue.

As well as the mechanism of glycogen sparing, we also should consider a direct effect of reduced blood lactate concentrations on performance. It is well known that increased blood lactate concentrations impair performance. Therefore, one can speculate that reduced blood lactate concentrations might improve performance. The significant correlation between the percentage decrease in blood lactate concentration, measured either at steady-state or end exercise, with the percentage increase of endurance time, points in this direction. But incremental exercise was not improved despite lower blood lactate concentrations after respiratory training. Also, to our knowledge, a direct, beneficial effect of reduced blood lactate concentrations on performance has never been shown. Thus, we do not believe that the reduced blood lactate concentration per se had a beneficial effect on performance.

Therefore we would suggest that delayed respiratory muscle fatigue could possibly explain improved cycling performance after respiratory training. During CET, $\dot{V}_E$

of active subjects (Boutellier et al. 1992; this study) reached a plateau with a small incline with time during exercise. Before exhaustion, a further ventilatory increase occurred caused by an augmented f_b. This final respiratory increase has often been observed before subjects finally stop intense constant-intensity exercise (Kearon et al. 1991; Boutellier et al. 1992; Johnson et al. 1993). The increase in f_b suggests that fatigue of respiratory muscles might be a possible explanation for the final increase of $\dot{V}_E$. It has been shown that rapid shallow breathing occurs when respiratory muscles are fatigued (Gallagher et al. 1985; Mador 1991; Mador and Acevedo 1991a). However, in these studies the increase in f_b was accompanied by a reduced V_T which we have not observed (Boutellier and Piwko 1992; Boutellier et al. 1992; present study). Sliwinski et al. (1996) have just recently confirmed our results by showing that overall inspiratory muscle fatigue increased f_b and $\dot{V}_E$ with minor changes in V_T during subsequent intense exercise whereas mild or moderate exercise did not affect ventilatory variables. As, in the present study, constant-intensity performance was prolonged despite an unchanged steady-state $\dot{V}_E$, one can assume that steady-state $\dot{V}_E$ is less important for the duration of exercise than the start of final hyperventilation.

Conclusions

Endurance training of respiratory muscles significantly prolonged intense constant-intensity exercise and reduced blood lactate concentrations during exercise. The reduced lactate concentrations were most likely caused by an improved lactate uptake by trained respiratory muscles. However, reduced exercise blood lactate concentrations per se were probably not the reason for better cycling performance after respiratory endurance training.

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