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Respiratory muscle endurance training in humans increases cycling endurance without affecting blood gas concentrations

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Abstract Isolated respiratory muscle endurance training (RMT) can prolong constant-intensity cycling performance. We tested whether RMT affects O₂ supply during exercise, i.e. whether the partial pressure of oxygen in arterial blood (P_aO_2) and/or its oxygen saturation (S_aO_2) are higher during exercise after RMT than before. A group of 28 sedentary subjects were randomly assigned to either an RMT ($n=13$) or a control group ($n=15$). The RMT consisted of 40×30 min sessions of normocapnic hyperpnoea. The control group did not perform any training. Breathing and cycling endurance time as well as P_aO_2 and S_aO_2 during cycling at a constant intensity of 70% maximum power output were measured before and after the RMT or the control period. Mean breathing endurance increased significantly after RMT compared to control [RMT 5.2 (SD 2.9) vs 38.1 (SD 6.8) min, control 6.5 (SD 5.7) vs 6.4 (SD 7.6) min; $P<0.01$], as did mean cycling endurance [RMT 35.6 (SD 11.9) vs 44.0 (SD 17.2) min, control 32.8 (SD 11.6) vs 31.4 (SD 14.4) min; $P<0.05$]. The RMT did not affect P_aO_2 which ranged from 11.6 to 12.3 kPa (87–92 mmHg), and S_aO_2 which ranged from 96% to 98% throughout all tests. In conclusion, RMT substantially increased breathing and cycling endurance in sedentary subjects. These changes, however, cannot be attributed to increased O₂ supply, as neither P_aO_2 nor S_aO_2 were increased during exercise after RMT.

Keywords Arterial oxygen concentration · Arterial oxygen saturation · Endurance performance · Constant intensity exercise · Sedentary subjects

Introduction

Isolated respiratory muscle endurance training (RMT) in the form of normocapnic hyperpnoea can prolong constant-intensity cycling performance (Spengler et al. 1999; Spengler and Boutellier 2000). The mechanisms leading to this improvement of cycling endurance after RMT still need to be determined. Among the potential mechanisms several have been excluded. Markov et al. (1996) have shown that the hypoxic ventilatory response remains unchanged after RMT. Kohl et al. (1997) found no reduction in airway resistance which would have decreased respiratory work and possibly delayed fatigue of the respiratory muscles after RMT. Spengler et al. (1999) reported reduced blood lactate concentrations after RMT, but found no shift in the balance of aerobic as opposed to anaerobic metabolism and thus could not explain the increased cycling endurance performance by reduced blood lactate concentrations. Spengler et al. (1998) found no increase in stroke volume and no decrease in heart rate (HR) after RMT. Thus, the increased cycling endurance performance after RMT was not a result of cardiovascular training induced by increased venous return while breathing very hard during RMT.

A further mechanism that could be responsible for the increased exercise performance after RMT is an increase in the arterial partial pressure of oxygen (P_aO_2) after RMT. Endurance is, for example, increased when subjects are breathing an hyperoxic gas mixture. This increase might either be directly related to an increase in O₂ supply to working muscles by an increased P_aO_2 or it might result from indirect effects of altered O₂ supply, for example changes in blood lactate, H⁺ or HCO₃⁻ concentrations or a decrease in minute ventilation (\dot{V}_E). A decrease in \dot{V}_E and thus a reduction of respiratory work could cause a blood redistribution towards working leg muscles and thereby leave more O₂ to these muscles (Harms et al. 1997). In addition, an increase in P_aO_2 could possibly partially prevent the increase in the

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alveolar-arterial difference in the partial pressure of O_2 [$P_{(A-a)}O_2$] that can be observed during exercise at 65% of maximal O_2 consumption, for example, in trained subjects. The $P_{(A-a)}O_2$ proportionally widens with increasing O_2 consumption ($\dot{V}O_2$) due to both a decrease in P_aO_2 and an increase in the partial pressure of oxygen in the alveoli (P_AO_2) (Cerretelli and Di Prampero 1987). However, there is little data available in sedentary subjects performing constant-intensity exercise where such effects may be more pronounced with increasing exercise time.

Therefore, the aim of the present study was to investigate whether the increased endurance performance in constant-intensity cycling after RMT might be explained by a change in oxygen supply, that is a higher P_aO_2 , a higher oxygen saturation of arterial blood (S_aO_2) or a smaller $P_{(A-a)}O_2$ during exercise compared to the controls.

Methods

Subjects

A group of 28 healthy, sedentary subjects were randomly assigned to one of two experimental groups: 13 subjects (5 women, 8 men), mean age 43 (SD 7) years, mean height 170 (SD 9) cm, mean body mass 67 (SD 12) kg, mean haemoglobin concentration 144 (SD 15) $g\cdot l^{-1}$, mean haematocrit 41 (SD 4)% were included in the RMT group and 15 subjects (7 women, 8 men), mean age 37 (SD 9) years, mean height 170 (SD 7) cm, mean body mass 68 (SD 13) kg, mean haemoglobin concentration 141 (SD 19) $g\cdot l^{-1}$, haematocrit 40 (SD 4)% in the control group. There were no significant differences in age, height, body mass, haemoglobin concentration or haematocrit between groups. The protocol was approved by the Ethics Committee of the Institutes of Physiology and Pharmacology of the University of Zurich and the subjects gave their written informed consent. All the experiments complied with the current laws of Switzerland.

Equipment

Vital capacity (VC), forced expiratory volume in 1 s ($FEV_{1.0}$), peak expiratory flow (PEF), and maximum voluntary ventilation (MVV), as well as \dot{V}_E and gas exchange during exercise were measured using an Oxycon Gamma ergospirometric device (Jaeger, Wuertzburg, Germany). Cycling tests were performed on an electromagnetically braked cycle (Ergometrics 900, Ergoline, Bitz, Germany). The HR was recorded using a PE4000 heart rate monitor (Polar Electro, Kempele, Finland). For RMT and for the respiratory endurance tests, a special device was developed in-house. It consisted of a rebreathing bag connected to two T-shaped tubes which were then connected to a mouthpiece. The volume of the bag was adjusted to be 50%–60% of the subjects' VC. Subjects were instructed to fill and empty the bag completely with each breath. To avoid an increase in the partial pressure of CO_2 in the arterial blood (P_aCO_2) and a fall in S_aO_2 , a small hole in the tube permitted additional inspiratory and expiratory flow to and from the room. During the constant-intensity cycling tests, arterial blood samples of 1.5 ml were withdrawn anaerobically every 3 min from a catheter positioned in a radial artery. Within 30 min of sampling, P_aO_2 , P_aCO_2 , pH, and S_aO_2 were determined automatically at 37°C by a blood gas analyser (BGElectrolytes) connected to an IL482 Co-Oxymeter (Instrumentation Laboratory, Lexington, Mass., USA).

Tests before and after the training or control periods

Each test period consisted of three sessions separated by a minimum of 2 days. In the first session, VC, $FEV_{1.0}$, PEF, and MVV were measured first. After a break of at least 15 min, an incremental cycling test was performed to determine maximum power output (\dot{W}_{max}) and peak oxygen consumption ($\dot{V}O_{2,peak}$). Subjects were free to choose their own pedalling frequencies within the range of 60–100 min^{-1} . Once established, the frequency had to be maintained throughout all cycling tests. In the second session, subjects were further familiarized with the RMT device until they were capable of breathing at a \dot{V}_E corresponding to 70% MVV (respiratory frequency, f_R , was paced by a metronome) for more than 2 min, or, if this time was not achieved, the target \dot{V}_E was reduced to 65% MVV and another trial was performed. In the third session, a catheter was placed in a radial artery and the subjects performed a breathing endurance test to exhaustion (determined by \dot{V}_E having dropped by more than 10% below target or by the subject stopping exercise) at the \dot{V}_E established in the second session. There were 4 subjects who were not exhausted after 15 min, they were stopped, the target \dot{V}_E was increased by 5% MVV and the test was repeated after a break of at least 15 min. Mean target \dot{V}_E was similar for the RMT [69 (SD 6)% MVV] and control group [71 (SD 10)% MVV]. After the breathing endurance test, a recovery of 20 min was allowed to ensure that no carry-over effect on the subsequent cycling test would ensue (Spengler et al. 2000). Subsequently, subjects performed a 5 min warm up on the cycle ergometer at 35% of the individual \dot{W}_{max} immediately followed by the cycling endurance test at 70% \dot{W}_{max} to exhaustion (determined by pedalling frequency dropping by more than 10% below target or by the subject having stopped). Subjects were not given any additional information or encouragement. The HR and \dot{V}_E were measured continuously and arterial blood samples were withdrawn every 3 min.

At least 5 days after the last respiratory training session, breathing and cycling endurance tests were repeated. Procedures and exercise intensities were the same as in the tests before the training or control period except that the breathing endurance test was discontinued after 40 min whether or not the subjects were exhausted.

Training and control period

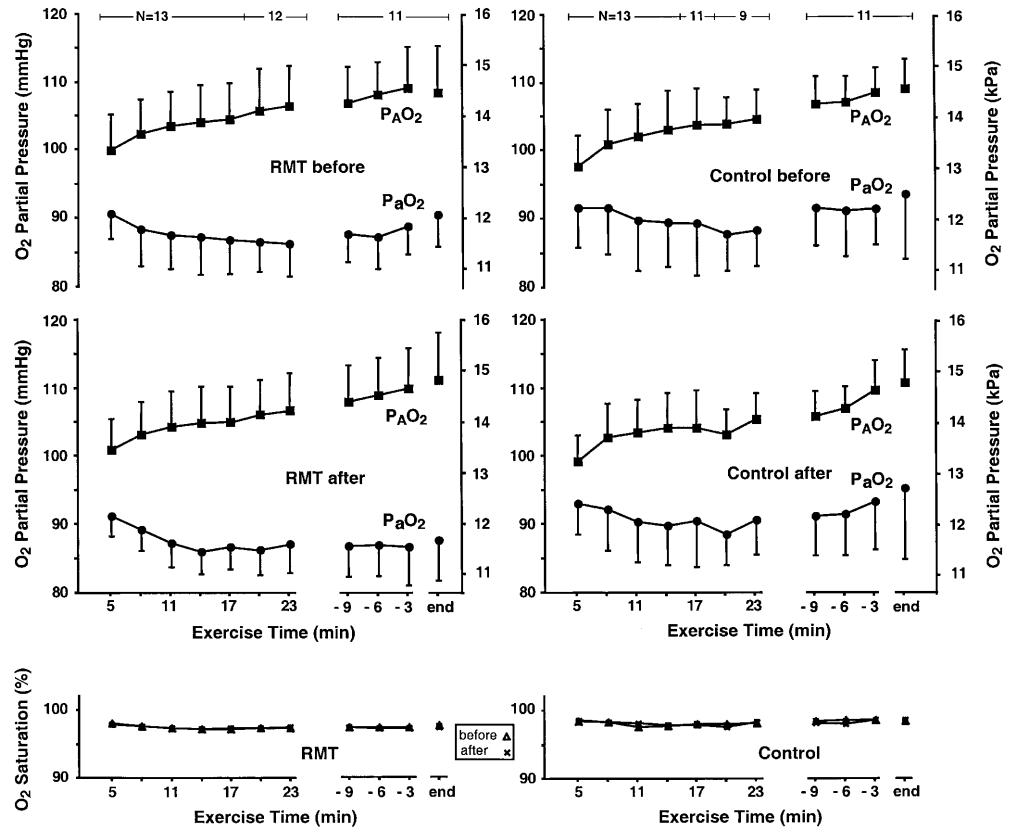
All subjects were asked to maintain their normal sedentary lifestyle. The RMT group performed 40 respiratory training sessions of 30 min duration over a period of 15 (SD 2) weeks. Compliance with the training regimen was excellent in the RMT group, as assured by HR recorded continuously during each RMT session. Mean HR during the training sessions was 104 (SD 11) min^{-1} corresponding to 57 (SD 6)% of the maximal HR in the RMT group.

Analysis and statistics

All statistical comparisons were performed between groups using factorial analysis of variance (ANOVA). This applied to the following variables:

1. Age, height, body mass, haemoglobin concentration, and haematocrit
2. Differences (Δ) in lung function variables, breathing endurance time, and cycling endurance time before compared to after training
3. $\Delta P_{(A-a)}O_2$, ΔP_aO_2 , and ΔS_aO_2 in the cycling endurance tests were obtained every 3 min. Values of corresponding times were compared during two separate periods: 5–23 min after the beginning of cycling and 9–3 min before exhaustion as well as at the end of the cycling endurance tests. Due to inter-subject variation in the length of the cycling endurance tests and values missing for technical reasons, the numbers of subjects included in each analysis is not identical. Exact numbers are given in Fig. 1.

Fig. 1 Alveolar ($P_{A}O_2$) and arterial (P_aO_2) partial pressure of O_2 during constant-intensity cycling at 70% maximum power output before and after 40 sessions of respiratory muscle endurance training (RMT, left) or the control period (right). Arterial O_2 saturation does not differ between RMT (bottom left) and control group (bottom right). For details of calculations and comparisons see Analysis and statistics (3.) in the Methods section



4. Δ of all other variables in the cycling endurance tests were averaged during the steady state phase, i.e. from 7.5 min after the beginning of the test (2.5 min after the beginning of 70% \dot{W}_{max}) to 2.5 min before the end of the shorter test and during the equivalent time period of the longer test. Significance was accepted if $P < 0.05$.

Results

After RMT, significant increases of breathing endurance at 70% MVV [mean RMT, 5.2 (SD 2.9) before compared to 38.1 (SD 6.8) min after; control 6.5 (SD 5.7) before compared to 6.4 (SD 7.6) min after; $P < 0.01$], as well as of cycling endurance at 70% \dot{W}_{max} [mean RMT 35.6 (SD 11.9) compared to 44.0 (SD 17.2) min; control 32.8 (SD 11.6) compared to 31.4 (SD 14.4) min; $P < 0.05$] were observed compared to control. During the cycling endurance tests, no significant changes were observed in mean steady state \dot{V}_E , tidal volume (V_T), f_R [RMT 29 (SD 5) compared to 29 (SD 5) min^{-1} ; control 28 (SD 4) compared to 28 (SD 4) min^{-1}], the inspiratory time (t_I), the expiratory time (t_E), $\dot{V}O_2$, and HR in the RMT group compared to the control group (Table 1).

Also, no significant changes of mean VC [RMT 4.72 (SD 1.00) compared to 4.77 (SD 0.98) l; control 4.82 (SD 0.98) compared to 4.80 (SD 0.97) l], $FEV_{1.0}$ [RMT 3.71 (SD 0.86) compared to 3.78 (SD 0.84) l; control 3.81 (SD 0.73) compared to 3.76 (SD 0.64) l], PEF [RMT 8.4 (SD 1.8) compared to 9.1 (SD 2.2) $\text{l}\cdot\text{s}^{-1}$;

control 8.2 (SD 1.4) compared to 8.5 (SD 1.4) $\text{l}\cdot\text{s}^{-1}$, MVV [RMT 170 (SD 49) compared to 181 (SD 40) $\text{l}\cdot\text{min}^{-1}$; control 151 (SD 37) compared to 162 (SD 37) $\text{l}\cdot\text{min}^{-1}$], $\dot{V}O_{2,peak}$ [RMT 2.39 (SD 0.76) compared to 2.38 (SD 0.77) $\text{l}\cdot\text{min}^{-1}$; control 2.35 (SD 0.60) compared to 2.23 (SD 0.55) $\text{l}\cdot\text{min}^{-1}$], and \dot{W}_{max} [RMT 175 (SD 57) compared to 175 (SD 56) W; control 171 (SD 43) compared to 163 (SD 35) W] were observed after RMT in the training group compared to the control group.

After RMT, neither P_aO_2 , $P_{(A-a)}O_2$, nor S_aO_2 were significantly altered during the cycling endurance tests. However, at the time of exhaustion, $\Delta P_{(A-a)}O_2$ in the RMT group tended to exceed the value in the control group ($P = 0.06$; Fig. 1). Mean pH, HCO_3^- , partial pressure of CO_2 in the alveoli and $P_a\text{CO}_2$ did not change significantly after RMT compared to control (Table 1); moreover, $P_{(A-a)}O_2$ was never more than 0.1 kPa (0.75 mmHg) and did not change significantly in the training group compared to the control group.

Discussion

Cycling endurance was significantly increased in a group of 13 sedentary subjects after 40 \times 30 min sessions of RMT, when compared with a control group which did not perform any training. This improvement occurred despite an unchanged $\dot{V}O_{2,peak}$ and \dot{W}_{max} . The fact that RMT does not influence maximum exercise performance has been shown repeatedly (Kohl et al. 1997; Markov

Table 1 Means of averaged steady state pH, arterial standard bicarbonate (HCO_3^-) concentration, alveolar and arterial partial pressure of CO_2 ($P_{\text{A}}\text{CO}_2$, $P_{\text{a}}\text{CO}_2$, respectively), minute ventilation (\dot{V}_{E}), tidal volume (V_{T}), inspiratory time (t_{I}), expiratory time (t_{E}),

oxygen consumption (\dot{V}_{O_2}), and heart rate (HR) during the cycling endurance tests before and after the respiratory muscle endurance training (RMT) or control period

	RMT				Control			
	before		after		before		after	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
pH	7.38	0.02	7.38	0.02	7.39	0.03	7.38	0.03
HCO_3^- (mmol l^{-1})	21.0	2.2	20.6	1.7	21.1	1.8	20.8	2.3
$P_{\text{a}}\text{CO}_2$ (kPa)	4.7	0.4	4.6	0.3	4.6	0.4	4.6	0.5
(mmHg)	35.2	2.7	34.7	2.3	34.6	3.2	34.8	3.3
$P_{\text{A}}\text{CO}_2$ (kPa)	4.7	0.5	4.7	0.5	4.6	0.4	4.7	0.5
(mmHg)	35.3	3.6	35.3	3.3	34.8	3.3	35.3	3.3
\dot{V}_{E} (l min^{-1})	60.8	15.2	59.5	13.0	56.5	11.0	57.1	11.1
V_{T} (l)	2.13	0.55	2.08	0.47	2.03	0.33	2.07	0.42
t_{I} (s)	1.00	0.16	0.98	0.18	1.02	0.20	1.01	0.22
t_{E} (s)	1.15	0.23	1.17	0.26	1.19	0.26	1.22	0.22
\dot{V}_{O_2} (l min^{-1})	1.79	0.52	1.78	0.53	1.75	0.39	1.77	0.37
HR (beats min^{-1})	164	9	163	10	163	15	160	14

et al. 1996; Spengler et al. 1999). Also, breathing endurance increased significantly despite an unchanged lung function. These increases in cycling and breathing endurance are similar to those reported previously (e.g. Spengler and Boutellier 2000).

We wondered whether an increase in $P_{\text{a}}\text{O}_2$ and/or $S_{\text{a}}\text{O}_2$ during endurance exercise after RMT could possibly explain the increased cycling endurance. However, $P_{\text{a}}\text{O}_2$ and $S_{\text{a}}\text{O}_2$, as well as $P_{\text{A}}\text{O}_2$ and the resulting $P_{(\text{A-a})}\text{O}_2$ were not affected by RMT (Fig. 1). In fact, after RMT, a tendency towards a lower $P_{\text{a}}\text{O}_2$ and a larger widening of $P_{(\text{A-a})}\text{O}_2$ at the time of exhaustion was observed. This would tend to cause a decrease rather than an increase in cycling endurance. Moreover, the unchanged \dot{V}_{O_2} , pH, blood lactate and HCO_3^- concentrations during steady-state exercise after RMT compared to control supports the notion that RMT does not shift the balance of aerobic compared to anaerobic metabolism. Also, no changes in \dot{V}_{E} , V_{T} , f_{R} , t_{I} and t_{E} were observed during steady-state exercise after RMT and thus a change in the oxygen cost of breathing (potentially causing a blood redistribution towards leg muscles; Harms et al. 1997) as a consequence of a change in breathing pattern seems unlikely. Considering the present results, the improvements in cycling endurance must be attributed to a mechanism other than an increase in $P_{\text{a}}\text{O}_2$ and/or $S_{\text{a}}\text{O}_2$. Alternative mechanisms to account for the improvement in cycling endurance could be a decrease in perceived exertion, and/or a decrease in the development of respiratory

muscle fatigue that was shown to develop during exhausting exercise, even in sedentary subjects. A reduction of respiratory muscle fatigue could result from increased endurance of inspiratory or expiratory muscles per se or from a more efficient recruitment of the different respiratory muscles. Further studies will have to address these issues.

Conclusion

In sedentary subjects, RMT substantially increased breathing and cycling endurance. These changes could not be attributed to increased O_2 supply, as neither $P_{\text{a}}\text{O}_2$ nor $S_{\text{a}}\text{O}_2$ were increased during exercise after RMT.

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