

ORIGINAL ARTICLE

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Effect of exercise-induced hyperventilation on airway resistance and cycling endurance

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Abstract The purpose of the present study was to investigate the effect of exercise induced hyperventilation and hypocapnia on airway resistance (R_{aw}), and to try to answer the question whether a reduction of R_{aw} is a mechanism contributing to the increase of endurance time associated with a reduction of exercise induced hyperventilation as for example has been observed after respiratory training. Eight healthy volunteers of both sexes participated in the study. Cycling endurance tests (CET) at 223 (SD 47) W, i.e. at 74 (SD 5)% of the subject's peak exercise intensity, breathing endurance tests and body plethysmograph measurements of pre- and postexercise R_{aw} were carried out before and after a 4-week period of respiratory training. In one of the two CET before the respiratory training CO_2 was added to the inspired air to keep its end-tidal concentration at 5.4% to avoid hyperventilatory hypocapnia (CO_2 -test); the other test was the control. The pre-exercise values of specific expiratory R_{aw} were 8.1 (SD 2.8), 6.8 (SD 2.6) and 8.0 (SD 2.1) cm $H_2O \cdot s$ and the postexercise values were 8.5 (SD 2.6), 7.4 (SD 1.9) and 8.0 (SD 2.7) cm $H_2O \cdot s$ for control CET, CO_2 -CET and CET after respiratory training, respectively, all differences between these tests being nonsignificant. The respiratory training significantly increased the respiratory endurance time during breathing of 70% of maximal voluntary ventilation from 5.8 (SD 2.9) min to 26.7 (SD 12.5) min. Mean values of the cycling endurance time (t_{cend}) were 22.7 (SD 6.5) min in the control, 19.4 (SD 5.4) min in the CO_2 -test and 18.4 (SD 6.0) min after respiratory training. Mean values of ventilation (\dot{V}_E) during the last 3 min of CET were 123 (SD 35.8) $l \cdot min^{-1}$ in the control, 133.5 (SD 35.1) $l \cdot min^{-1}$ in the CO_2 -test and 130.9 (SD 29.1)

$l \cdot min^{-1}$ after respiratory training. In fact, six subjects ventilated more and cycled for a shorter time, whereas two subjects ventilated less and cycled for a longer time after the respiratory training than in the control CET. In general, the subjects cycled longer the lower the \dot{V}_E , if all three CET are compared. It is concluded that R_{aw} measured immediately after exercise is independent of exercise-induced hyperventilation and hypocapnia and is probably not involved in limiting t_{cend} , and that t_{cend} at a given exercise intensity is shorter when \dot{V}_E is higher, no matter whether the higher \dot{V}_E occurs before or after respiratory training or after CO_2 inhalation.

Key words Exercise · Respiratory training · Hyperventilation · Airway resistance

Introduction

Two recent studies dealing with exercise limiting factors have reported that an untrained respiratory system might limit endurance time during intense cycling exercise. After 4 weeks of respiratory training, the endurance time during constant intensity exercise, corresponding to 64 (SD 8)% of peak oxygen uptake ($\dot{V}O_{2peak}$) in sedentary subjects and to 77 (SD 6)% of $\dot{V}O_{2peak}$ in athletes has been shown to be prolonged by 50% and by 38%, respectively (Boutellier and Piwko 1992; Boutellier et al. 1992). Simultaneously, minute ventilation (\dot{V}_E) at the same exercise intensity was lower and thus end tidal partial pressure of carbon dioxide ($P_{ET}CO_2$) was higher after respiratory training than before.

Alveolar hypocapnia has been shown to induce bronchoconstriction (Newhouse et al. 1964; Coon and Kampine 1975; Twort et al. 1985). Therefore, the question arises whether the lower CO_2 concentration during the higher exercise \dot{V}_E would affect airway resistance (R_{aw}). Although exercise per se has been found to have rather a bronchodilating effect in normal as well as in asthmatic subjects (Kagawa and Kerr 1970; Stirling et al.

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1983; Freedman 1992), it could be expected that bronchodilation would be smaller, i.e. R_{aw} higher, the lower the alveolar CO_2 concentration owing to higher \dot{V}_E . This relatively higher R_{aw} might in turn increase the work of breathing or give rise to a subjective feeling of short-windedness or breathlessness and thus compel the subject to stop exercise. In the present study, R_{aw} was measured using whole body plethysmography before and immediately after exercise. We assumed that R_{aw} values immediately after exercise would reflect the degree of hyperventilation and hypocapnia during exercise, as has been found to be the case after voluntary hyperventilation at rest (Twort et al. 1985; our preliminary experiments), and as an exercise-induced reduction of forced expiratory volume in the 1st s has been shown to persist in the 5th and 10th min after exercise (O'Kroy et al. 1992). On the other hand, if alveolar hypocapnia were to limit exercise endurance by increasing R_{aw} , then maintaining normocapnic conditions by addition of CO_2 to the inspired air might have a beneficial effect on cycling endurance time.

The purpose of the present study therefore was to investigate the effect of exercise induced hyperventilation and hypocapnia as well as of normocapnia on R_{aw} , and to try to answer the question whether a reduction of R_{aw} is a mechanism contributing to the increase of endurance time associated with a reduction of hyperventilation. The working hypothesis tested in this study was that the prolongation of endurance is, at least in part, due to the effects which the reduced hyperventilation via reduction of hypocapnia might have on R_{aw} : Decreased \dot{V}_E would lead to less severe hypocapnia and hence to a smaller R_{aw} , which in turn would delay the onset of respiratory muscle fatigue and therefore increase the exercise endurance time.

Methods

Subjects

Eight healthy, moderately trained medical students, two women and six men, participated as volunteers in this study. The subjects' mean age was 27.5 (SD 8.9) years, body mass 76.9 (SD 19.0) kg and body height 180.0 (SD 11.1) cm. The subjects were well informed about all the methods and procedures to be used and gave their written consent to participate in the study. The experimental protocol was approved by the local Ethics Committee.

Apparatus

An electrically braked cycling ergometer, Ergometrics 900 (Ergoline, Bitz, Germany), was used for the incremental cycling test and for the cycling endurance tests (CET). Respiratory variables, e.g. tidal volume (V_T), breathing frequency (f_b), concentrations of respiratory gases and oxygen uptake ($\dot{V}O_2$), were continuously measured and calculated by Oxycon Gamma (Mijnhardt B. V., Bunnik, Netherlands). Heart rate (f_c) was continuously measured by Sport Tester PE4000 (Polar Electro, Kempele, Finland) and monitored by Oxycon. The R_{aw} was measured by a constant-volume whole body plethysmograph Bodystar FG 90 (Dr. Fenyves

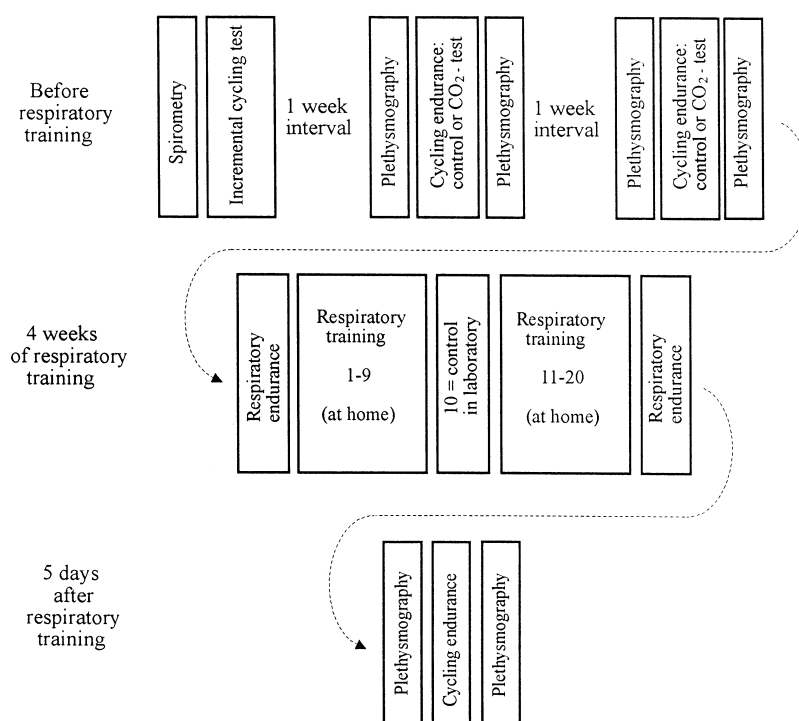
and Gut, Basel, Switzerland); the specific expiratory R_{aw} (equivalent to expiratory R_{aw} related to thoracic gas volume, see below) was used for comparisons between tests. A gas mixing unit, developed in the institute workshop according to Boutellier and Fahri (1986), was used in one of the two CET before respiratory training to keep end-tidal CO_2 concentration at 5.4%. The respiratory training was carried out using a portable semi-open system device which was a variant of the model of Isaak (1992) consisting of a rubber bag for partial rebreathing (to avoid hypocapnia) and two unidirectional valves: one valve permitted room air to enter when the rebreathing bag was collapsed, and through the other one excess expiratory air streamed out when the bag was full. Both the volume of the rebreathing bag and the duty cycle of the breaths were individually adjusted to provide a degree of rebreathing necessary to keep normocapnic conditions during the voluntary hyperpnoea. Lactate concentrations of blood samples withdrawn from the ear lobes were measured by an enzymatic lactate analyser ESAT 6661 (Eppendorf, Hamburg, Germany).

Experiment protocol

Figure 1 gives a summary of the design of the experiment. The whole experimental period for one subject lasted about 8 weeks. In the first experiment session, spirometer measurements of vital capacity, forced expiratory volume in the 1st s and maximal voluntary ventilation (MVV) were carried out. Afterwards the submaximal exercise intensity for CET was established on a cycling ergometer by an incremental procedure starting with an intensity of 50 W. Every minute the intensity was increased by 15 W until the subject was exhausted. Blood samples for the determination of lactate concentration were taken every 2 min. The exercise intensity for CET was then determined from the changes of f_c , \dot{V}_E and lactate concentration with increasing intensity. Each of these three variables was plotted against increasing exercise intensity and the intensity at which the slope of the f_c curves became flatter and the slope of \dot{V}_E and lactate curves became steeper was determined. The mean value of these three intensities was then used as the submaximal intensity for all three CET. After 1 week the first CET was carried out and after a second week a further CET. One of these two CET before respiratory training served as control, and in the other CO_2 was added to the inspired air to prevent hypocapnia as soon as end tidal CO_2 concentration dropped below 5.4%. The sequence of the control and CO_2 -test was randomized. Before and within about 2 min after each CET, R_{aw} was measured by body plethysmography. The 2-min delay between the end of CET and the postexercise plethysmography was necessary to achieve the appropriate temperature and humidity steady state in the prewarmed plethysmograph. This procedure was tested in preliminary experiments, and it was found that an increase of R_{aw} following voluntary hyperventilation at rest was still measurable 2 min after the end of hyperventilation.

At the beginning and at the end of the respiratory training period, breathing endurance tests were carried out. In these tests the subjects had to breathe with the respiratory training device at 70% of their MVV (mean value amounting to $130 \text{ l} \cdot \text{min}^{-1}$) for as long as they were able. The 4-week period of respiratory training consisted of 19 sessions (one 0.5-h session a day, 5 days a week) at home. The 10th session was carried out in the laboratory to monitor the training procedure. In the first training session, the subjects had to breathe at a \dot{V}_E equal to 50% of their MVV. As soon as the subjects were able to maintain such \dot{V}_E for 30 min without difficulty, both f_b (metronome-paced) and V_T were increased for the next session. And as soon as this increased \dot{V}_E could be maintained, a further increase was introduced in the next session, and so on. During the 4 weeks of respiratory training the subjects were asked to continue with their moderate physical activities as practised before respiratory training. After the end of respiratory training, with a time-lag of 5–10 days, CET and plethysmography were carried out in the same way as before the training.

Fig. 1 Diagram of experimental protocol



Evaluation and statistics

The R_{aw} values are given as specific expiratory resistance in centimetres of H₂O and second ($\text{cm} \cdot \text{s}$) which is the reciprocal of specific conductance (per centimetre of H₂O per second; $\text{cm}^{-1} \cdot \text{s}^{-1}$), i.e. conductance (per centimetre of H₂O per second and litre; $\text{cm}^{-1} \cdot \text{s}^{-1} \cdot \text{p}$), divided thoracic gas volume. The effect of CO₂ in the CO₂-CET was quantified as the difference between the slope of breath-by-breath curves of \dot{V}_E (litres per minute per minute) before and during the CO₂ application. For comparisons of cardiorespiratory parameters between the CET, all values were averaged over the period from 5th to 10th min of exercise and over the last 3 min of exercise. Statistical analysis was carried out by means of software Stat View 4.01a (Abacus Concepts Inc., Berkeley, Calif. USA) using the non parametric Wilcoxon test for pair differences and the Spearman rank test for correlations. The minimal accepted level of significance for all tests was P equal to 0.05.

Results

Specific expiratory R_{aw}

Figure 2 shows the mean values of specific expiratory R_{aw} before and immediately after each of the three CET. There were no significant differences in specific expiratory R_{aw} among the three tests – either before or after the CET; nor were there differences among the pre- and postexercise measurements in all three tests. There was no significant correlation either between R_{aw} and \dot{V}_E or between R_{aw} and $P_{ET}\text{CO}_2$.

Respiratory endurance time

The endurance time during breathing of 70% of MVV under normocapnic conditions was in all the subjects

significantly prolonged after respiratory training ($P < 0.01$), mean values increasing from 5.8 (SD 2.9) min to 26.7 (SD 12.5) min. The effective \dot{V}_E during the respiratory endurance test before the respiratory training was 129.0 (SD 25.3) $\text{l} \cdot \text{min}^{-1}$ and after respiratory training 131.6 (SD 31.6) $\text{l} \cdot \text{min}^{-1}$. The difference between these two values was not significant.

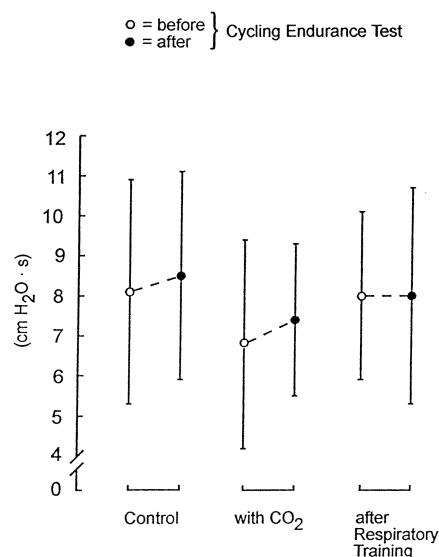


Fig. 2 Mean values and standard deviations of specific expiratory airway resistance before (open circles) and immediately after (closed circles) each of the three CET (control, CO₂-test, test after respiratory training). No differences were statistically significant

Cycling endurance time

Mean values and standard deviations of the cycling endurance time (t_{cend}) are given in Table 1. The t_{cend} was significantly longer in the control CET than in the CO₂-test ($P < 0.01$) and than in CET after respiratory training ($P < 0.05$). There was no significant difference in t_{cend} between the CO₂-test and CET after respiratory training.

 $\dot{V}O_2$ and f_c during CET

The subjects exercised in all three CET at the same submaximal exercise intensity, the mean value being 223 (SD 47) W. This intensity represented 74 (SD 5)% of their peak intensity. To characterize this intensity further $\dot{V}O_2$ and f_c values averaged from the 5th to the 10th min of exercise and during the last 3 min of exercise are given in Table 1. No differences in either $\dot{V}O_2$ or f_c among all CET were statistically significant.

Lactate concentration after CET

Lactate concentrations were compared in blood samples withdrawn immediately after the end of each CET. The mean values and standard deviations are given in Table 1. There was no significant difference between the two CET carried out before respiratory training. However, after respiratory training, lactate concentration at the end of CET was significantly lower ($P < 0.05$) than at the end of the control CET at an identical exercise intensity.

Table 1 Values for cycling endurance time (t_{cend}), oxygen uptake ($\dot{V}O_2$), heart rate (f_c), and of lactate concentration after the end of the test in the three cycling endurance tests. 5–10 Average from 5th to 10th min of exercise, last 3 average from the last 3 min of exercise, RT respiratory training

| | Control | | CO ₂ -Test | | After RT | |
|---|---------|------|-----------------------|------|----------|------|
| | mean | SD | mean | SD | mean | SD |
| t_{cend} (min) | 22.7 | 6.5 | 19.4* | 5.4 | 18.4** | 6.0 |
| $\dot{V}O_2$ 5–10 ($l \cdot \text{min}^{-1}$) | 3.48 | 0.91 | 3.33 | 0.96 | 3.38 | 0.77 |
| $\dot{V}O_2$ last 3 ($l \cdot \text{min}^{-1}$) | 3.48 | 0.93 | 3.41 | 0.83 | 3.34 | 0.95 |
| f_c 5–10 (beats $\cdot \text{min}^{-1}$) | 173.8 | 16.3 | 167.9 | 15.2 | 175.5 | 16.3 |
| f_c last 3 (beats $\cdot \text{min}^{-1}$) | 184.5 | 11.1 | 182.6 | 10.1 | 183.5 | 9.9 |
| Lactate concentration ($\text{mmol} \cdot \text{l}^{-1}$) | 10.5 | 3.2 | 9.7 | 2.7 | 8.1* | 2.9 |

Significance of differences from control * $P < 0.05$, ** $P < 0.01$

Table 2 Values for minute ventilation (\dot{V}_E), tidal volume (V_T) and breathing frequency (f_b) in three cycling endurance tests. 5–10 Average from 5th to 10th min of exercise, last 3 average from the last 3 min of exercise, RT respiratory training

| | Control | | CO ₂ -Test | | After RT | |
|--|---------|------|-----------------------|------|----------|------|
| | mean | SD | mean | SD | mean | SD |
| \dot{V}_E 5–10 ($l \cdot \text{min}^{-1}$) | 106.0 | 25.8 | 107.8 | 21.7 | 117.9* | 24.3 |
| \dot{V}_E last 3 ($l \cdot \text{min}^{-1}$) | 123.0 | 35.8 | 133.5* | 35.1 | 130.9* | 29.1 |
| V_T 5–10 (l) | 2.84 | 0.94 | 2.89 | 0.88 | 2.89 | 0.90 |
| V_T last 3 (l) | 2.54 | 0.75 | 2.63 | 0.67 | 2.59 | 0.74 |
| f_b 5–10 (breaths $\cdot \text{min}^{-1}$) | 39.5 | 8.3 | 38.8 | 8.6 | 42.9* | 9.6 |
| f_b last 3 (breaths $\cdot \text{min}^{-1}$) | 50.9 | 4.9 | 50.9 | 6.2 | 52.3 | 8.3 |

Significance of differences from control * $P < 0.05$

End-tidal CO₂ concentrations and P_{ETCO_2} during CET

The mean values of end-tidal CO₂ concentrations, averaged over the last 3 min of each CET, were 4.90 (SD 0.40)%, 5.44 (SD 0.10)% and 4.73 (SD 0.33)% for the control test, the CO₂-test and the CET after respiratory training, respectively. Expressed as partial pressures, the P_{ETCO_2} reached 33.0 (SD 2.7) mmHg in the control test, 36.4 (SD 0.7) mmHg in CO₂-test, and 31.8 (SD 2.2) mmHg in CET after respiratory training. The P_{ETCO_2} was, as intended, significantly higher ($P < 0.01$) in the CO₂-test than in both other CET. After respiratory training, P_{ETCO_2} was significantly lower ($P < 0.05$) than in the control test, corresponding to the significantly higher \dot{V}_E after respiratory training (see below). However, it should be pointed out that in the two subjects ventilating less after respiratory training P_{ETCO_2} was higher than in the control test, i.e. all the subjects reached lower P_{ETCO_2} in CET with higher \dot{V}_E .

 \dot{V}_E during CET

Mean values and standard deviations of \dot{V}_E averaged during the 5th to 10th min and during the last 3 min of exercise in each CET together with the respective values of V_T and f_b are summarized in Table 2. Between the 5th to the 10th min \dot{V}_E was significantly higher ($P < 0.05$) after respiratory training than in both CET before respiratory training, whereas there was no significant difference between the control and the CO₂-test. In the last 3 min \dot{V}_E was significantly lower in the control than in the CO₂-test ($P < 0.05$) and than in CET after

respiratory training ($P < 0.05$), whereas the difference between the CO_2 -test and CET after respiratory training was not significant.

To illustrate the entire time course of \dot{V}_E changes during the three CET, Fig. 3 shows the mean values of \dot{V}_E from all eight subjects breath by breath. The end of each line gives the mean end-point in the CET. The \dot{V}_E in the two tests before respiratory training (control test and CO_2 -test) was almost identical until the moment when the addition of CO_2 started. In response to CO_2 , the slope of \dot{V}_E became steeper, the mean value increasing from 1.97 (SD 1.02) $\text{l} \cdot \text{min}^{-1}$ per min before CO_2 inhalation to 5.03 (SD 1.92) $\text{l} \cdot \text{min}^{-1}$ per min after CO_2 inhalation. After respiratory training, mean \dot{V}_E was from the beginning higher than in the two CET before training, and simultaneously the mean t_{cend} was reduced. Actually, this was the case in six subjects – all of them being tested for cycling endurance within 5 to 7 days after the end of respiratory training. However, two subjects tested on the 9th and 10th day after respiratory training cycled longer and breathed less than before training. A higher \dot{V}_E was found in seven out of eight subjects caused by higher f_b . Thus, the f_b averaged from the 5th to 10th min of cycling was significantly higher ($P < 0.05$) in CET after respiratory training than in the control and in the CO_2 -test, whereas no differences in V_T were significant (Table 2).

Relationship between \dot{V}_E and t_{cend}

Figure 4 shows the relationship between the differences in \dot{V}_E and the differences in t_{cend} among the three CET at identical submaximal exercise intensities as individual values for all eight subjects. Differences in \dot{V}_E during the last 3 min of CET between the control CET and the CET after respiratory training (closed circles) as well as between control CET and CET with CO_2 (open circles) are plotted against the respective differences in t_{cend} . All

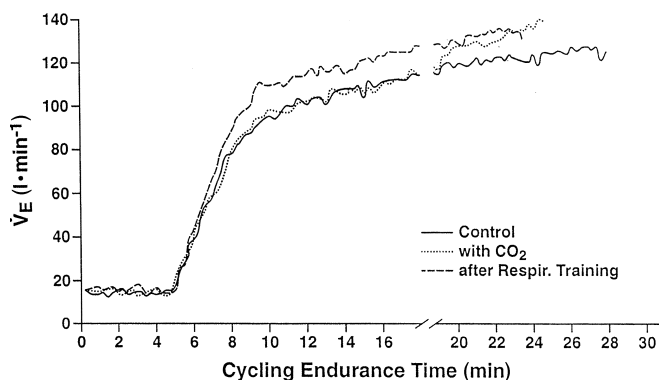


Fig. 3 Breath-by-breath mean values of minute ventilation (\dot{V}_E) and of endurance time from eight subjects in the three CET. Standard deviations of \dot{V}_E ranged between 20% and 30% of mean value. Standard deviations of endurance time were 6.5 min, 5.4 min and 6.0 min for the control test, CO_2 -test and test after respiratory training, respectively

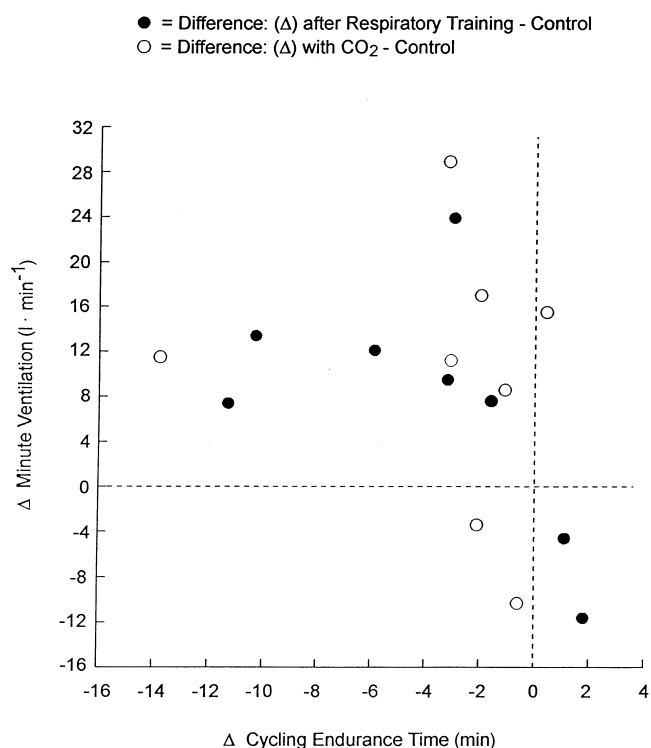


Fig. 4 Correlation between the differences in cycling endurance time and the differences in minute ventilation. *closed circles* Comparison between control test and test after respiratory training in eight subjects, *open circles* comparison between control test and CO_2 -test in eight subjects

the subjects performed longer with the lower \dot{V}_E when the tests after respiratory training were compared with controls. Two subjects ventilated less and cycled longer after respiratory training (closed circles in the lower right quadrant) and six subjects ventilated less and cycled longer before respiratory training (closed circles in the upper left quadrant). When CO_2 -tests were compared with controls then five subjects performed longer and ventilated less in the control test (open circles in the upper left quadrant). Furthermore, when all three types of CET were compared in each subject, then all the subjects, except one, cycled for the longest time in the test with the lowest \dot{V}_E during the last 3 min of the test.

Discussion

The discussion is focused on two main findings of the present study. Firstly, the working hypothesis – that R_{aw} would increase in proportion to the degree of exercise-induced hyperventilation and hypocapnia and thus by increasing the work of breathing play a role in determining the exercise endurance time – was not confirmed. Secondly, the present study clearly showed that the higher the exercise-induced hyperventilation at a given exercise intensity, the shorter the exercise endurance time – independent of the P_{ETCO_2} or lactate

concentration and independent of the cause of the higher \dot{V}_E .

Effect of exercise induced hyperventilation on R_{aw}

The results obtained in the present study do not support the working hypothesis that a higher \dot{V}_E at a given exercise intensity would via hypocapnia increase R_{aw} , which in turn by increasing the work of breathing would accelerate respiratory muscle fatigue and therefore reduce t_{cend} . Our results suggested that R_{aw} is independent both of \dot{V}_E and of CO_2 values. On the one hand, there was no correlation between the level of \dot{V}_E and R_{aw} measured immediately after exercise. On the other hand, the addition of CO_2 to the inspired air to keep $P_{ET}CO_2$ higher did not reduce R_{aw} . The contrary was the case in that the inhalation of CO_2 in an amount allowing its end tidal concentration to be kept at 5.4% caused an increase of \dot{V}_E and a shortening of t_{cend} .

Effect of exercise induced hyperventilation on t_{cend}

The present study indicated that the level of exercise induced hyperventilation per se, independent of the kind of respiratory drive and of $P_{ET}CO_2$ or blood lactate concentrations, is a factor determining the exercise endurance time. In that sense, the results of the present study fully confirmed and extended the finding of Boutellier and Piwko (1992) and Boutellier et al. (1992) that a reduction of \dot{V}_E during a given high exercise intensity is associated with an increase of t_{cend} . In those two studies the subjects achieved the reduction of \dot{V}_E and a simultaneous increase of t_{cend} by respiratory training. The same held true for two subjects in the present study, whereas in six subjects this effect of respiratory training was not only missing but even reversed. These six subjects ventilated more and cycled for a shorter time after respiratory training than at the same exercise intensity before respiratory training, probably because of respiratory muscle fatigue (see below). There is no doubt that all the subjects in the present study trained their respiratory muscles for 4 weeks properly and successfully. The results of the respiratory endurance tests were convincing. All the subjects after the respiratory training were able to maintain \dot{V}_E at 70% of MVV much longer than before training. Furthermore, blood lactate concentrations at the end of CET with the same exercise intensity were significantly lower after respiratory training than in control tests before training, a phenomenon which has previously been reported by Boutellier and Piwko (1992) after successful respiratory training in sedentary subjects. However, the mechanism of this decrease in lactate concentration is unknown.

In spite of the unexpected effect of respiratory training on \dot{V}_E in the present study, it was possible to compare in all eight subjects three CET with three different

levels of \dot{V}_E and the respective t_{cend} . This comparison revealed that whenever \dot{V}_E was higher during a given exercise intensity, the t_{cend} was lower and vice versa. No matter for what reason the \dot{V}_E increased, the effect on the t_{cend} was the same.

The question arises, what other mechanisms, if not $P_{ET}CO_2$ and its eventual effect on R_{aw} , could be involved in linking the higher \dot{V}_E and shorter t_{cend} ? One possibility could be an increased energy consumption of the respiratory muscles during higher \dot{V}_E , thus less oxygen and substrate would be available for exercising muscles. However, considering the values given by Coast et al. (1993) for oxygen costs of breathing during both voluntary and exercise hyperventilation, the differences between our three endurance tests were very small. An increase of exercise \dot{V}_E from 100 to 120 $l \cdot min^{-1}$ would increase $\dot{V}O_2$ by less than 50 $ml \cdot min^{-1}$. The most likely link between the high \dot{V}_E and short t_{cend} would seem to be some kind of fatigue in the respiratory muscles.

The following arguments speak in favour of fatigued respiratory muscles. In spite of the successful respiratory training (increase of respiratory endurance time, see above) and in contrast to the previous finding of Boutellier and Piwko (1992) in sedentary subjects and of Boutellier et al. (1992) in athletic subjects, in the present study \dot{V}_E during CET with identical exercise intensities was increased after respiratory training in six out of eight subjects. The most likely explanation for the higher \dot{V}_E is some kind of tiredness of the respiratory muscles after the intense respiratory training. Mador and Acevedo (1991a, b) have found after fatigue of respiratory muscles an increase of \dot{V}_E mainly caused by an increase of f_b . Also in the present study, seven out of eight subjects increased their \dot{V}_E by increasing f_b whereas the differences in V_T were not significant.

Further evidence for the role of respiratory muscle fatigue in increasing \dot{V}_E during exercise has been described by Boutellier et al. (1992). The authors observed in one of the well-trained subjects in a CET, carried out immediately after the end of respiratory training, higher \dot{V}_E and shorter t_{cend} than before training. When CET was repeated 5 days later, \dot{V}_E was significantly reduced and t_{cend} prolonged in comparison with the pretraining values. Possibly 5 days were necessary for the recovery of respiratory muscles from respiratory training therefore in the present study the post-training tests were carried out with a delay of 5–7 days after the end of training. It seems that this recovery time was not sufficient for our subjects, maybe because they trained with a slightly different training device to that used by Boutellier and Piwko (1992) and Boutellier et al. (1992). In the last two subjects the delay was therefore prolonged to 9 and 10 days, and these two subjects were found to breathe less and cycle longer in the endurance test after respiratory training than before it. These findings would suggest that the ventilation-reducing and endurance-lengthening effect of respiratory training only occurs after a sufficiently long recovery period. Tired respiratory muscles obviously increase \dot{V}_E at a given ex-

ercise intensity, mainly due to increased f_b (Mador and Acevedo 1991a), and simultaneously impair subsequent performance of high intensity exercise (Mador and Acevedo 1991b).

It is of interest that this kind of respiratory muscle fatigue does not impair the performance of respiratory muscles itself. Two findings support this suggestion. On the one hand, all the subjects in the present study were able to breathe significantly longer during the respiratory endurance test (70% of MVV) in spite of signs of respiratory muscle fatigue in CET after respiratory training. On the other hand, \dot{V}_E during the same exercise intensity was even higher with fatigued respiratory muscles than before (Mador and Acevedo 1991a, b; present study). A similar finding has been reported just recently by Sliwinski et al. (1996) where overall inspiratory muscle fatigue increased \dot{V}_E and f_b during high intensity exercise.

It should be pointed out that the higher \dot{V}_E during endurance exercise after respiratory training in the present study was associated with lower blood lactate concentrations than before training. This is an important finding suggesting that the increase of \dot{V}_E was not mainly due to lactacidity, and vice versa that the eventual decrease of \dot{V}_E after respiratory training was not due to lower lactate concentrations. This suggestion is in contrast to the conclusion of Casaburi et al. (1987) who have found a reduced ventilatory response to exercise after endurance training and suggested it was linked to the reduction in blood lactate concentration. Probably other mechanisms as for example increased potassium (Busse et al. 1989) or catecholamines concentrations might also be involved in increasing \dot{V}_E during the same exercise intensity.

In conclusion, it is suggested that lower R_{aw} and thus a lower work of breathing is probably not the mechanism underlying the prolonging of the duration of intense exercise observed during lower \dot{V}_E at a given exercise intensity. Some other mechanism, probably a fatigue of respiratory muscles, reduces endurance in hyperventilating subjects. Whenever \dot{V}_E during an endurance test at a given high exercise intensity is lower, t_{cend} is prolonged and vice versa. This holds true no matter whether \dot{V}_E is increased by increasing inspiratory CO_2 or by fatigued respiratory muscles, or whether \dot{V}_E is reduced by respiratory training. The beneficial effect of respiratory training on endurance exercise only occurs after respiratory muscles have recovered from training.

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