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The respiratory system as an exercise limiting factor in normal sedentary subjects

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Summary. The present study was undertaken to investigate the respiratory system as an exercise limiting factor. Breathing and cycle endurance (i.e. the time until exhaustion at a given performance level) as well as physical working capacity 170 (i.e. the exercise intensity corresponding to a heart rate of 170 beats·min^{-1} on a cycle ergometer) were determined in four healthy sedentary subjects. Subsequently, the subjects trained their respiratory system for 4 weeks by breathing daily about 90 l·min^{-1} for 30 min. Otherwise they continued their sedentary lifestyle. Immediately after the respiratory training and 18 months later, all performance tests carried out at the beginning of the study were repeated. The respiratory training increased breathing endurance from 4.2 (SD 1.9) min to 15.3 (SD 3.8) min. Cycle endurance was improved from 26.8 (SD 5.9) min to 40.2 (SD 9.2) min whereas physical working capacity 170 remained essentially the same. During the endurance cycling test in the respiratory untrained state, the subjects continuously increased their ventilation up to hyperventilation [ventilation at exhaustion = 96.9 (SD 23.6) l·min^{-1}] while after the respiratory training they reached a respiratory steady-state without hyperventilation [ventilation at exhaustion = 63.3 (SD 14.5) l·min^{-1}]. The absence of this marked hyperventilation was the cause of the impressive increase of cycle endurance in normal sedentary subjects after respiratory training. The effects gained by the respiratory training were completely lost after 18 months. Our results indicated that the respiratory system was an exercise limiting factor during an endurance test in normal sedentary subjects.

Key words: Exercise limiting factor - Respiratory muscle training - Breathing endurance - Cycle endurance - Physical working capacity 170

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Introduction

There are many forms of exercise and therefore different factors can limit exercise. Among the exercise limiting factors, the availability and the turnover of oxygen plays a predominant role. The oxygen transport from the atmosphere to the mitochondria of skeletal muscle involves respiration, circulation, erythrocytes, the heart, and skeletal muscles. Among these, respiration is generally assumed to be the least important exercise limiting factor because ventilation seems to have enough reserve. The literature is almost unanimous in reporting that respiration does not limit maximal oxygen consumption ($\dot{V}O_{2max}$) in normal subjects (Ouellet et al. 1969; Dempsey 1986; di Prampero and Ferretti 1990) except for highly trained endurance athletes, whose blood oxygen saturation drops during severe exercise (Dempsey et al. 1984; Williams et al. 1986; Wagner 1987). However, Martin et al. (1982) have shown that ventilatory work designed to decrease ventilatory muscle endurance prior to short-term maximal running reduced performance and O_2 uptake compared to the same exercise test without prior ventilatory work.

There are two arguments in favour of the suggestion that respiration can never be pushed to its limits by exercise in healthy subjects:

1. Maximal voluntary ventilation always exceeds the maximal ventilation reached even at exhausting exercise intensities.
2. All subjects - from the sedentary to the endurance athletes - hyperventilate above the anaerobic threshold in incremental exercise tests although this yields probably only a small benefit (Ouellet et al. 1969).

The first argument, however, does not consider the longer duration of exhausting exercise compared with the 12-20 s of a common maximal voluntary ventilation manoeuvre. As soon as this manoeuvre lasts 60 s and more, maximal voluntary ventilation is reduced (Tenney and Reese 1968). Therefore, respiratory muscles may fatigue during long lasting exercise (Leith and Bradley 1976; Bye et al. 1983; Grassino and Macklem 1984; Belman and Gaesser 1988). Respiratory muscle

fatigue could reduce ventilation which becomes critical when arterial oxygen saturation starts to decrease. At first sight, this point of view is contradicted by the second argument given above because even untrained subjects are able to increase ventilation overproportionately during an incremental exercise test at high intensities. This surplus ventilation supports strongly the view that respiration is not an exercise limiting factor (Ouellet et al. 1969; Dempsey 1986). Nevertheless, this hyperventilation lasts only a few minutes if $\dot{V}O_{2max}$ is also measured. Therefore, it is still a controversial issue as to whether respiratory muscles become fatigued while maintaining a high ventilation during intense steady-state exercise (Anholm et al. 1989).

To elucidate this problem further, we decided to investigate the respiratory system as a limiting factor to endurance at a given submaximal intensity by measuring the cycling time until exhaustion in normal subjects who were subsequently to undergo special respiratory training without any other muscle conditioning. After this respiratory training, cycle endurance was measured again. If cycle endurance remained the same after the respiratory training then respiration would not have been an exercise limiting factor, provided the respiratory training was effective. If cycle endurance had improved after training the respiratory system exclusively then ventilation would have limited exercise. This last conclusion would only be valid if the possibility were ruled out that the respiratory training also conditioned other systems in addition to the respiratory system. As physical training also conditions respiratory muscles (Martin and Stager 1981; Anholm et al. 1989), we chose sedentary subjects for our study.

Methods

Subjects. Four healthy sedentary subjects participated in the study. Only persons performing less than 1 h of sport activities a week were considered to be untrained. The subjects' characteristics are listed in Table 1. They were informed about the study in detail and agreed without hesitation to continue their sedentary lifestyle throughout the investigation.

Equipment. A body plethysmograph (Bodystar FG90, Dr. Fenyves and Gut, Basel, Switzerland) was used to measure vital capacity, peak flow, forced expiratory volume in 1 s and specific airway conductance. Maximal voluntary ventilation was determined with a flowmeter (Calculair M403, Sandoz, Basel, Switzerland). A special device was developed to train respiration and to measure breathing endurance. It was similar to the device described in detail elsewhere (Boutellier and Farhi 1986). The device consisted of a gas mixing unit and indicators for respiratory frequency and tidal volume. Because the subjects were asked to hyperventilate, it was necessary to add CO_2 to the inspired air to maintain isocapnia in the subjects. Air was continuously withdrawn from the mouthpiece and CO_2 was measured by an infrared method (medical gas analyser LB-2, Beckman Instruments Inc., Fullerton, CA, USA). The inspired CO_2 concentrations were adjusted manually with the gas mixing unit to maintain end-tidal CO_2 concentrations at 5.4%. Air flow was measured by a pneumotachograph (Fleisch, no. 3, Metabo SA, Epalinges, Switzerland) and transduced into an electrical signal. This signal was integrated and displayed to the subject using a horizontal series of 35 green light-emitting diodes (LED). Before every respiratory training session or endurance breathing test, the system was set with the help of a syringe so that each LED represented 1/35th of the desired tidal volume. Three red LEDs at the end of the scale warned the subjects that the breath exceeded the requested one. Two additional lights, which were used by the subjects to keep to the set frequency, were triggered by a metronome.

Table 1. Subjects' characteristics

Subject	Symbol	Sex	Age (years)	Height (cm)	Body mass (kg)
1	●	Female	21	170	51
2	○	Male	24	179	76
3	□	Male	25	170	52
4	△	Male	23	172	62

A cycle ergometer (Ergocont, Monark-Crescent AB, Yarburg, Sweden) was used to measure physical working capacity 170 (PWC_{170}), cycle endurance and peak oxygen uptake ($\dot{V}O_{2,peak}$). The PWC_{170} test was introduced by Sjöstrand (1960) and determines the mechanical power developed in cycling at a heart rate of $170 \cdot min^{-1}$. Because heart rate increases linearly with increasing exercise intensities, PWC_{170} can be extrapolated from submaximal levels of exercise intensity. Respiratory variables were automatically measured breath-by-breath with a method originally suggested by Beaver et al. (1973) and subsequently, improved (Boutellier et al. 1982, 1987). Heart rate was recorded in parallel with respiratory variables by the same computer. We used a commercially available heart rate transmitter (PE3000, Polar Electro OY, Kempele, Finland) and a receiver developed by us which could be set to calculate heart rate beat-to-beat or to average up to 5 beats. The computer averaged heart rate over a respiratory cycle and stored the values breath-by-breath.

Blood lactate concentrations were measured with an automatic analyser (lactate analyser 23L, Yellow Springs Instrument Co. Inc., Yellow Springs, OH, USA) from blood taken from the fingertip. In this study only the increase in blood lactate concentrations is given, i.e. the concentration at the end of a test minus the resting value measured before the test. Arterial oxygen saturation was determined noninvasively by oximetry (oximeter NC N-200, Nellcor Inc., Hayward, CA, USA) with a durasensor placed on an index finger. The measurement was triggered by the simultaneously recorded electrocardiogram to avoid misreadings provoked by motion artefacts.

Protocol. Before respiratory training started several determinations were carried out. Vital capacity, peak flow, forced expiratory volume in 1 s, specific airway conductance and maximal voluntary ventilation were measured. Breathing endurance was determined by voluntarily breathing with a frequency of $45 \cdot min^{-1}$ and a tidal volume of 2.5 or 3.0 l (60%–66% of vital capacity). The clock was stopped when the subjects could no longer follow the preset respiratory frequency or tidal volume. This test was repeated 1–3 days later to account for any training or task learning effects. For the determination of PWC_{170}, at least three different exercise intensities were used. The measured result was compared with the value derived from a nomogram (Bühlmann 1965) for a normal, untrained population (see Fig. 2, "norm"). Cycle endurance was determined with an exercise intensity of about 80% PWC_{170} (Table 2) with the prerequisite that the subjects should be exhausted within 20 to 30 min. The test was repeated to adjust the exercise intensity and to account for training effects. The pedal revolutions were kept constant between 60 and $80 \cdot min^{-1}$. Before and after this test, blood lactate concentrations were measured. During the test, respiratory variables and heart rate were recorded continuously.

After the completion of these measurements, a period of respiratory training followed. The subjects trained their respiration over a 4-week period by breathing voluntarily 76–102.5 $l \cdot min^{-1}$

Table 2. Absolute and relative exercise intensities used for the measurements of cycle endurance as well as peak oxygen consumption ($\dot{V}O_{2,peak}$) and the corresponding increase in blood lactate concentration above the resting value

Subject	Exercise intensity (W)	$\%PWC_{170}$	$\%\dot{V}O_{2,peak}$	$\dot{V}O_{2,peak}$ ($l \cdot min^{-1}$)	ΔLactate ($mmol \cdot l^{-1}$)
1	70	75	63	1.8	8.7
2	185	82	66	3.7	9.1
3	105	88	53	2.9	10.6
4	155	86	72	3.1	9.3
Mean	129	83	64	2.9	9.4
SD	51	6	8	0.8	0.8

$\%PWC_{170}$, percentage physical working capacity 170

for 0.5 h five times a week. Tidal volume was set to 2.0 or 2.5 l (50%–53% of vital capacity) and breathing frequency started with $38 \cdot min^{-1}$. Each week breathing frequency was increased by $1 \cdot min^{-1}$. From time to time, the lactate concentration was determined before and after the respiratory training session.

Immediately after the end of the respiratory training period, the whole set of measurements carried out at the beginning of the study was repeated. The subjects continued their sedentary lifestyle during the entire study and abstained from any additional physical activities beside the respiratory training.

Finally, the subjects were asked to repeat some of the measurements 18 months after the completion of the initial study. We wanted to find out if the respiratory training effects were of limited duration or not. Breathing and cycle endurance as well as PWC_{170} were determined once more. Furthermore, we decided to measure $\dot{V}O_{2,peak}$ despite the subjects' untrained state. During the determination of $\dot{V}O_{2,peak}$ and cycle endurance arterial oxygen saturation was continuously recorded.

The results are presented as individual values or as means and standard deviations.

Results

All experiments were performed without major difficulties. The subjects complained sometimes about side stitches and about soreness of muscles involved in heavy breathing. Also the inhalation of dry air for 0.5 h was sometimes unpleasant, especially at the beginning of the respiratory training period.

The effectiveness of the respiratory training was demonstrated by the increase of breathing endurance from 4.2 (SD 1.9) min before to 15.3 (SD 3.8) min after the respiratory training (Fig. 1). Breathing endurance had returned to pre-training values after 18 months [6.0 (SD 3.0) min]. Vital capacity, peak flow, forced expiratory volume in 1 s, specific airway conductance and maximal voluntary ventilation were not influenced by the respiratory training (Table 3).

The PWC_{170} corresponded to the value from the nomogram in one subject and was below this value in two subjects (Fig. 2). One subject's PWC_{170} was somewhat higher than expected. After the respiratory training, PWC_{170} was increased by 13% and 23%, respectively, in two subjects, whereas it did not change in the two others. Some 18 months later, three subjects had the same

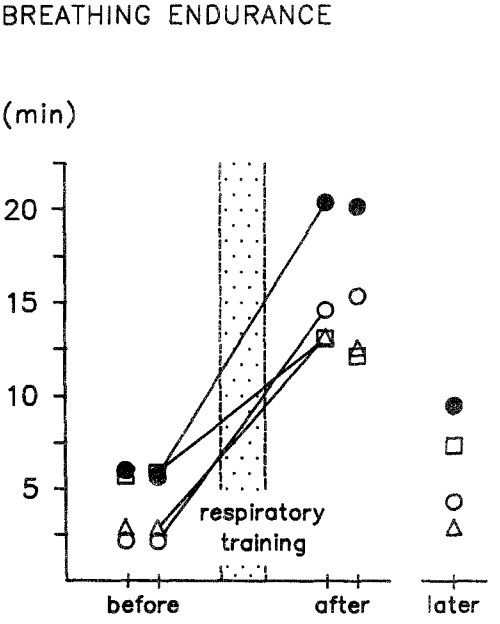

Fig. 1. Breathing endurance of four subjects before, immediately after and 18 months after a respiratory training period of 4 weeks. For definition of symbols see Table 1

values as at the beginning of the study; in one subject PWC_{170} was 21% lower than before (Fig. 2). The $\dot{V}O_{2,peak}$ determined 18 months after the end of the training session and the corresponding increases in blood lactate concentration are given in Table 2.

Cycle endurance was increased by 50% after respiratory training (Fig. 3). While the subjects exercised at 129 (SD 51) W before the respiratory training for 26.8 (SD 5.9) min, they tolerated the same intensity for 40.2 (SD 9.2) min after the respiratory training. However, 18 months later, mean cycle endurance amounted to only 21.9 (SD 2.5) min because that of one subject was substantially reduced by 10 min (Fig. 3). Figure 4 shows the increase in blood lactate concentrations above the resting values at the end of the endurance cycling test. Although the subjects cycled for only about 27 min before the respiratory training, blood lactate concentration increased by 5.4 (SD 0.3) $mmol \cdot l^{-1}$. After the respiratory training the increase in blood lactate concentration was reduced to 2.9 (SD 1.0) $mmol \cdot l^{-1}$ despite a much longer cycling time (about 40 min). After 18 months, lactate rose by 5.6 (SD 1.7) $mmol \cdot l^{-1}$. From time to time, blood lactate concentrations were also measured before and after a respiratory training session. In these random checks we never detected a lactate concentration which exceeded resting values.

Arterial oxygen saturation determined by oximetry showed no significant changes while cycling either at a constant intensity or with an increasing one in the respiratory untrained state. The values remained between 94% and 99% throughout the two exercise tests in all four subjects without clear increasing or decreasing trends.

A finding of some interest was the fact that the subjects increased their ventilation continuously during the endurance cycling test in the respiratory untrained

Table 3. Vital capacity (VC), peak flow, forced expiratory volume in 1 s (FEV_1), specific airway conductance (SAC), and maximal voluntary ventilation (MVV) before and after respiratory training

Variable	Subject	Before	After
VC	1	3.8	4.0
(l)	2	5.0	4.9
	3	3.9	4.0
	4	4.7	4.7
	mean	4.4	4.4
	SD	0.6	0.5
Peak flow	1	5.9	6.3
($l \cdot s^{-1}$)	2	9.5	9.1
	3	5.3	6.6
	4	8.0	8.3
	mean	7.2	7.6
	SD	1.9	1.3
FEV_1	1	2.8	2.7
(l)	2	4.0	3.8
	3	3.4	3.6
	4	4.0	3.9
	mean	3.6	3.5
	SD	0.6	0.5
SAC	1	0.13	0.13
($cm\ H_2O^{-1} \cdot s^{-1}$)	2	0.15	0.18
	3	0.20	0.20
	4	0.29	0.27
	mean	0.19	0.20
	SD	0.07	0.06
MVV	1	112	108
($l \cdot min^{-1}$)	2	174	184
	3	156	164
	4	152	150
	mean	149	152
	SD	26	32

state, whereas they reached a respiratory steady-state after the respiratory training (Table 4).

Discussion

Our prerequisite - to work with physically inactive subjects - turned out to be rather a hindrance in recruiting volunteers. It was difficult to motivate lazy subjects to enrol in a time and energy consuming training study. Finally, four sedentary subjects were willing to participate and their results were so uniform and convincing that it seems justifiable to present the interesting findings despite the small number of subjects.

The subjects handled all performance tests and the respiratory training without problems despite their reluctance to engage in any sport activities in their daily life. The poor training state of the subjects is obvious from the comparison of the measured PWC_{170} values

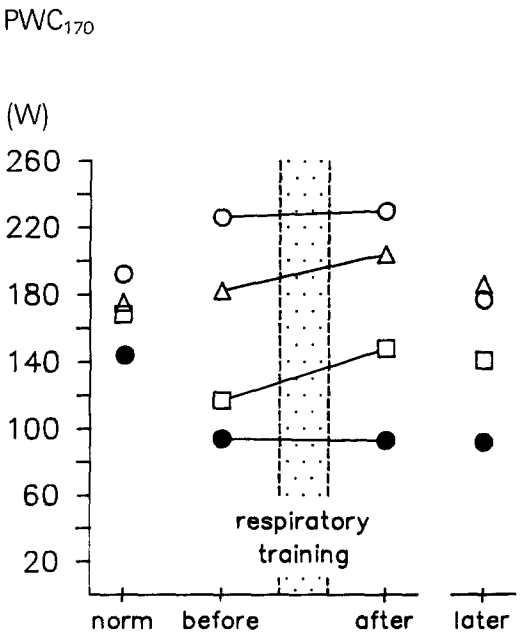

Fig. 2. Physical working capacity 170 (PWC_{170}) of four subjects before, immediately after and 18 months after a respiratory training period of 4 weeks. For definition of symbols see Table 1

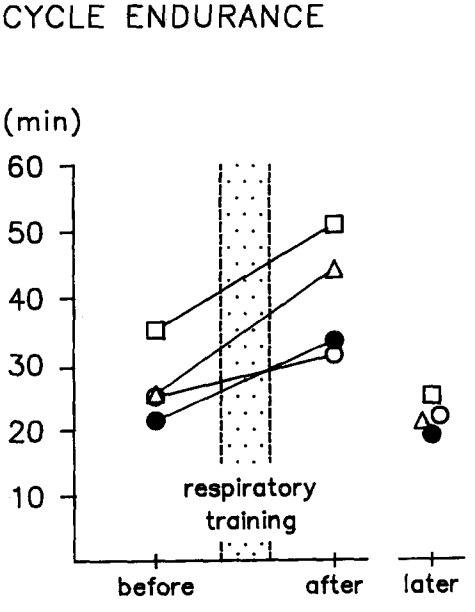

Fig. 3. Cycle endurance of four subjects before, immediately after and 18 months after a respiratory training period of 4 weeks. For definition of symbols see Table 1

with the nomogram of normal values (Fig. 2) and from the $\dot{V}O_{2,peak}$ results (Table 2): three subjects had a PWC_{170} as is typical for an untrained population and a $\dot{V}O_{2,peak}$ between 1.8 and 3.1 $l \cdot min^{-1}$ which indicated a modest aerobic capacity. Subject no. 2 was somewhat better conditioned, although he invested at most 1 h each week in physical activities.

The respiratory training increased breathing endurance by 268%. This improvement was not due to short range training or task learning effects because breath-

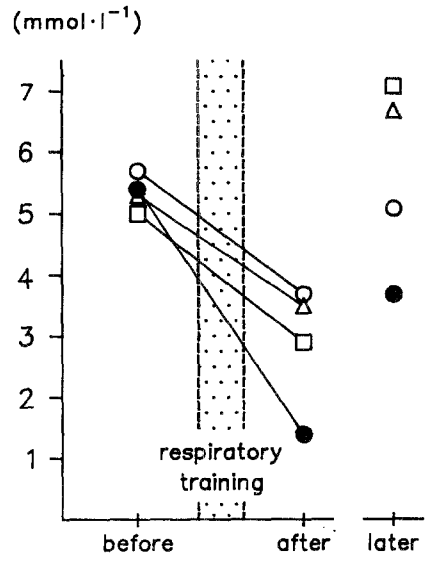

Fig. 4. Increase in blood lactate concentration above the resting value at the end of the endurance cycling test of four subjects before, immediately after and 18 months after a respiratory training period of 4 weeks. For definition of symbols see Table 1

ing endurance was determined twice on different days with similar results before and after the respiratory training (Fig. 1). Vital capacity, peak flow, forced expiratory volume in 1 s, specific airway conductance and maximal voluntary ventilation were not influenced by the respiratory training. These results differ somewhat from other studies (Leith and Bradley 1976; Morgan et al. 1987; Belman and Gaesser 1988) where the respiratory training increased in some subjects their vital capacity or maximal voluntary ventilation. The reason for these differences is probably a different training regimen. Leith and Bradley (1976) have reported that respiratory endurance training increased maximal voluntary ventilation which was not the case when respiratory strength training was applied. As none of the pulmonary function tests showed an improvement after respiratory training, we assumed that the forced voluntary breathing had increased the endurance of respiratory muscles (diaphragm and auxiliary muscles). This assumption was justified because the basic physiological behaviour of respiratory muscles has been found to be similar to that of other skeletal muscles (Grassino and Macklem 1984). The fibres of diaphragm muscle have been shown not to be different from limb muscle fibres, either in structure or in function (Lieberman et al. 1973). The assumption, that respiratory muscles were conditioned by the respiratory training, was supported by the subjects sporadic complaints about side stitches and soreness of muscles involved in heavy breathing. After 18 months, the respiratory training effects were lost and the endurance breathing tests yielded more or less the same results as before the respiratory training. Others have observed detraining effects within 4 to 8 weeks after the cessation of respiratory training (Leith and Bradley 1976; Keens et al. 1977; Belman and Gaesser 1988).

Table 4. Ventilation ($\dot{V}_E$), alveolar partial pressure of oxygen (P_AO_2), alveolar partial pressure of carbon dioxide (P_ACO_2), oxygen consumption ($\dot{V}O_2$), ventilatory equivalent ($\dot{V}_E:\dot{V}O_2$) and heart rate during and after the endurance cycling test

Variable	Subject	Untrained	Trained	
		end time	end time untrained	end time trained
$\dot{V}_E$ ($l \cdot min^{-1}$)	1	64.1	54.7	53.1
	2	114.6	86.6	84.7
	3	113.6	56.0	56.1
	4	95.2	59.4	59.2
	mean	96.9	64.2	63.3
	SD	23.6	15.1	14.5
P_AO_2 (mmHg)	1	114.8	106.4	109.0
	2	115.4	107.8	111.5
	3	124.5	108.8	107.7
	4	112.3	109.3	110.8
	mean	116.8	108.1	109.8
	SD	5.3	1.3	1.7
P_ACO_2 (mmHg)	1	31.2	39.4	36.6
	2	31.0	36.2	32.8
	3	22.3	36.1	35.8
	4	30.8	42.1	41.2
	mean	28.8	38.4	36.6
	SD	4.4	2.9	3.5
$\dot{V}O_2$ ($l \cdot min^{-1}$)	1	1.17	1.14	1.11
	2	2.41	2.60	2.35
	3	1.51	1.50	1.47
	4	2.22	2.32	2.32
	mean	1.83	1.89	1.81
	SD	0.58	0.68	0.62
$\dot{V}_E:\dot{V}O_2$	1	54.8	48.0	47.8
	2	47.6	33.3	36.0
	3	75.2	37.3	38.2
	4	42.9	25.6	25.5
	mean	55.1	36.0	36.9
	SD	14.3	9.3	9.2
Heart rate (min^{-1})	1	183	185	183
	2	195	187	195
	3	198	178	190
	4	183	165	180
	mean	190	179	187
	SD	7	10	7

In the respiratory untrained subjects, the values shown were measured at the end of the test. In the respiratory trained subjects, the values shown were measured during the test at the moment they had stopped in the untrained state and at the end of the test

The training of respiratory muscles, without any intentional conditioning of other skeletal muscles, increased cycle endurance by 50%. This improvement was due neither to cyclo-ergometer training nor to psycho-

logical effects. The endurance cycling test was repeated at least once in all subjects before the respiratory training started to account for training effects. In addition, training effects from a single test would not be expected to last for 4 weeks. Thus, cyclo-ergometer training effects could not explain the increase in cycle endurance. With respect to psychological effects, one could argue that the subjects were more motivated after the respiratory training and continued exercising at the edge of exhaustion for another 14 min. This argument is hardly convincing, especially in untrained subjects. In addition, it would be difficult to offer a psychological explanation for a reduced blood lactate accumulation at the end of the exercise test after the respiratory training. The subjects reported that exercising was easier after the respiratory training despite the identical exercise intensity. Their breathing was much more comfortable. The respiratory training effects on cycle endurance had disappeared after 18 months as was the case with breathing endurance. Three subjects cycled after 18 months as long as they did before the respiratory training. Subject no. 3 reduced his performance on the cycle after 18 months by 10 min, although he really pushed himself to his limits as proved by a 2 $mmol \cdot l^{-1}$ higher blood lactate level in comparison with the test before the respiratory training (Fig. 4). Except that subject no. 3 had given up smoking in the meantime, we found no other difference between the two test conditions and we cannot therefore explain the marked reduction in his cycle endurance time after 18 months.

The question arises, what contributed to the marked improvement of cycle endurance after respiratory training? Leg muscles were not trained and therefore it is unlikely that they contributed to the improvement. In animals, chronic resistive loading of respiratory muscles has shown training effects on the diaphragm but not on hindlimb muscles (Akabas et al. 1989; Tarasiuk et al. 1991). The heart is another candidate which could provide an explanation of the improvement in endurance. As cardiac output has been seen as the most important limiting factor in oxygen transport (di Prampero and Ferretti 1990), we have to consider cardiac conditioning very carefully. One would have expected cardiac conditioning to be accompanied by a reduced heart rate at a given exercise intensity or an improved PWC_{170}. These effects would have occurred after a short time of endurance training especially in untrained subjects. In our study, PWC_{170} increased slightly in two subjects and was unchanged in the two others (Fig. 2). Therefore, we consider that PWC_{170} was not influenced by the respiratory training. Also heart rate at the end of the endurance cycling test was practically the same before and after the respiratory training (Table 4).

Is there any reason at all to expect cardiac conditioning during respiratory training? An increase in ventilation yields a higher venous return and – following the Frank-Starling law – would have elicited a higher stroke volume leading to an increase in cardiac output. The increase in cardiac output has been found to amount to 35 $ml \cdot l^{-1}$ increase in ventilation in sitting subjects under isocapnic conditions (Boutellier and Farhi 1986). Considering an increase in ventilation of 100 $l \cdot min^{-1}$, cardiac output was probably increased by an additional 3.5 $l \cdot min^{-1}$ which would have led to a total cardiac output of 8.5–9.5 $l \cdot min^{-1}$ during the respiratory training sessions in our subjects. An average exercise intensity of 129 W during the endurance cycling tests has been found to require a cardiac output of about 16 $l \cdot min^{-1}$ (Ferretti et al. 1990). It is unlikely then that a modest additional increase in cardiac output of the order of 3.5 $l \cdot min^{-1}$ elicited a significant cardiac training effect. Therefore, it is not surprising that we did not find any signs of cardiac conditioning.

This leaves the respiratory system itself to account for the marked improvement of cycle endurance after respiratory training. As mentioned before, respiratory muscles have not been found to be fundamentally different from other skeletal muscles (Lieberman et al. 1973; Grassino and Macklem 1984). Therefore, it is conceivable that respiratory muscles might have become tired during an exercise session (Leith and Bradley 1976; Bye et al. 1983; Belman and Gaesser 1988) and provoked a decrease in arterial oxygen saturation which would have reduced the oxygen availability for working muscles. Our findings disproved this hypothesis because arterial oxygen saturation did not decrease either during the incremental or during the steady-state cycling tests in the respiratory untrained state. Furthermore, ventilation never decreased towards the end of an endurance cycling test. On the contrary, in the respiratory untrained state ventilation continued to rise after the rapid increase at the beginning of exercise until the subjects were exhausted. One reason why the subjects stopped cycling was their eventual inability to increase their ventilation any more. In the respiratory trained state, the subjects also increased ventilation very rapidly at the beginning of exercise but afterwards a steady-state was reached without any further increase in breathing and without hyperventilation.

A further reason why the subjects stopped cycling in the respiratory untrained state might have been high blood lactate concentrations. There are two possible explanations for the lower blood lactate concentrations at the end of the endurance cycling test after respiratory training.

1. The trained respiratory muscles used more lactate as fuel for their own activity.
2. Less lactate was produced by active muscles.

Although we can only speculate, we favour the second explanation. If the respiratory muscles really consumed so much more lactate after the respiratory training that it did not accumulate in the blood any more, then no hyperventilation would occur. However, hyperventilation may occur without a preceding increase in blood lactate concentration since patients with McArdle's disease have been found to hyperventilate above the anaerobic threshold despite the lack of an increase in blood lactate concentration or H^+ levels (Hagberg et al. 1990). Also other findings have cast considerable doubt on the role of lactic acidosis on the ventilatory response to heavy exercise (Jeyaranjan et al. 1989). Therefore, hyperventilation as the direct or indirect cause of an in-

creased anaerobic glycolysis in our study cannot be ruled out. This hyperventilation increased the overall energy demand which was not followed by an increase in $\dot{V}O_2$ (Table 4). Therefore, the higher energy demand had to be satisfied with a higher anaerobic glycolysis. It seems unlikely that in our experimental conditions the respiratory muscles produced much lactate because we never found an increase in lactate concentrations after a respiratory training session where the subjects breathed during 0.5 h as much as they did at the end of an endurance cycling test in the respiratory untrained state. Hyperventilation per se has been found to increase the blood lactate concentration (Huckabee 1958). It has been shown (Davies et al. 1986) that an additional ventilation of about $30\,l \cdot min^{-1}$ increased lactate concentration by about $1\,mmol \cdot l^{-1}$ at 125 W. We found an additional increase in lactate concentration of at least $2.4\,mmol \cdot l^{-1}$ (the exercise session with the lower lactate concentration lasted 50% longer). Thus, hyperventilation alone is hardly responsible for the increased lactate concentration in the respiratory untrained state.

The additional lactate in the blood probably comes from working limb skeletal muscles. The exercise intensity and thus the energy demand of leg muscles was the same with or without respiratory training, whereas the energy demand of respiratory muscles was higher in the respiratory untrained state due to hyperventilation (Bye et al. 1983). But $\dot{V}O_2$ during the endurance cycling test was as high with hyperventilation as without it. Training has not been found to reduce the oxygen cost of breathing (Bradley and Leith 1978; Martin and Stager 1981) which depends only upon the air ventilated. The unchanged $\dot{V}O_2$ despite an increased respiratory muscle activity has been supported by another study (Demedts and Anthonisen 1973) in which again no increase in total $\dot{V}O_2$ was found when the work of breathing was increased during exercise. Therefore, more oxygen was consumed by the hyperventilating respiratory muscles in the respiratory untrained state. Working skeletal muscles [or perhaps nevertheless respiratory muscles (Jardim et al. 1981; Freedman et al. 1983)] had to gain additional energy by anaerobic glycolysis. Whether this additional demand for oxygen by the respiratory muscle was high enough to account for a $1\text{–}2\,mmol \cdot l^{-1}$ higher blood lactate concentration at the end of exercise is difficult to say because reported oxygen costs of breathing vary widely (Bradley and Leith 1978; Martin and Stager 1981).

We have demonstrated that respiratory training abolished the continuous increase of ventilation otherwise present in four normal sedentary subjects during exercise at a constant intensity of 64% of $\dot{V}O_{2,peak}$. A respiratory steady-state without progressive hyperventilation allowed the subjects to prolong their endurance cycling test by 50%. Therefore, we are convinced that the untrained respiratory system can limit this form of exercise in normal sedentary subjects. At first sight, this conclusion seems to contradict the statement that the respiratory system is not normally an exercise limiting factor (Oullet et al. 1969; Dempsey 1986; di Prampero and Ferretti 1990). In fact, the respiratory system is not an important limiting factor if one is measuring $\dot{V}O_{2max}$ or aerobic performance with an incremental exercise test because neither we nor others (Leith and Bradley 1976; Keens et al. 1977; Morgan et al. 1987; Belman and Gaesser 1988) have found an improvement in these variables after respiratory training. However, Martin et al. (1982) have demonstrated that 150 min of sustained maximal ventilation before a short-term maximal running test can reduce performance and $\dot{V}O_2$ as compared with control.

Conclusions

We have identified the respiratory system as an exercise limiting factor in normal sedentary subjects. Exercising at a constant submaximal intensity (64% $\dot{V}O_{2,peak}$), subjects increased their ventilation continuously until exhaustion in the respiratory untrained state. Respiratory training abolished this hyperventilation and prolonged cycle endurance by 50%. Therefore, the respiratory system should be considered when exercise limiting factors are discussed except in incremental exercise tests of rather short duration as for instance the determination of $\dot{V}O_{2max}$.

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