Respiratory muscle fitness and exercise endurance in healthy humans

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ABSTRACT

BOUTELLIER, U. Respiratory muscle fitness and exercise endurance in healthy humans. *Med. Sci. Sports Exerc.*, Vol. 30, No. 7, pp. 1169–1172, 1998. New evidence exists that the respiratory muscles may limit exercise performance in healthy humans. Four weeks of isolated respiratory training (30 min normocapnic hyperpnea, 5 d·wk^{-1}) significantly increased the endurance time of respiratory muscles and the endurance time of constant-load bicycle tests in sedentary as well as physically active subjects once respiratory muscles had recovered from the training. Minute ventilation and blood lactate concentration were reduced during post-training exercise. Furthermore, respiratory trained subjects had lost the sensation of breathlessness. Maximal oxygen consumption was not affected by respiratory training. The mechanism by which respiratory training improves overall physical performance is as yet unknown. Key Words: RESPIRATORY TRAINING, ENDURANCE PERFORMANCE, EXERCISE LIMITING FACTORS, RESPIRATORY MUSCLE FATIGUE

Ten years ago, it was commonly accepted that "the lung is built for exercise" (10) with just one exception: In some well-trained subjects oxygen saturation drops while exercising at intensities corresponding to oxygen consumptions higher than 3 L·min^{-1} (11). The explanation for the lower oxygen saturation was that blood passed through the pulmonary capillaries too fast, thus causing diffusion limitation at the alveolo-capillary level. Two reasons are used to argue that otherwise respiration is not an exercise limiting factor: 1) Ventilation ($\dot{V}_E$) at a maximal workload never reaches maximum voluntary ventilation; and 2) even untrained subjects are able to hyperventilate while performing exercise at intensities above the anaerobic threshold. The first argument is not convincing because determination of maximum voluntary ventilation lasts only 10–20 s and is therefore not representative for exercise that takes many minutes before leading to exhaustion. In addition, if normocapnic maximum voluntary ventilation is prolonged it declines (15). The second argument is more valid and more difficult to oppose. Nevertheless, Bye et al. (6) discussed several possibilities for how respiratory factors may limit exercise. Limitations may arise in terms of gas exchange, respiratory mechanics, energetics of the respiratory muscles, or because of the development of respiratory muscle fatigue. Concerning respiratory muscle fatigue, Leith and Bradley (24) wrote: "We see three kinds of applications to be explored, in which ventilatory muscle fatigue may limit human performance and in which prior ventilatory muscle training might then improve performance or at least minimize decrements. The first is in sports, if it turned out that exercise capacity in fit individuals were ever limited by ventilatory muscle endurance. This is not generally thought to be the case."

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RESPIRATORY MUSCLE TRAINING AND EXERCISE PERFORMANCE

In the late 1980s, three groups independently challenged the idea that the performance of the respiratory system is always adequate to sustain exercise. They investigated the effects of respiratory muscle training (RMT) on overall physical performance (4,13,31). RMT—performed as isocapnic hyperpnea—significantly improved respiratory muscle endurance in all subjects, thereby confirming earlier studies (2,20,24). While RMT did not have an effect on physical performance in two studies (13,31), we demonstrated an improvement of exercise endurance (3,5). We explain this discrepancy by the different levels of constant workloads used. In the two former studies, intensity was almost maximal, whereas we used lower intensities (64 and 77% $\dot{V}O_{2max}$) in our investigations.

There are two reasons for using submaximal constant workloads instead of high intensity exercise: First, the literature (e.g., (10)) provides ample evidence that respiration does not limit $\dot{V}O_{2max}$. Second, short lasting, maximal exercise hardly applies to daily life situations in contrast to long lasting, more moderate intensities of exercise (endurance exercise). This is also true for most sporting events. RMT did indeed prolong constant-load exercise in physically active subjects, whereas $\dot{V}O_{2max}$ remained unchanged despite lower blood lactate concentrations (34). During constant-load exercise, minute ventilation and blood lactate

concentration were reduced once respiratory muscles had recovered from RMT (5,22,34). Furthermore, respiratory trained subjects lost the sensation of breathlessness (3).

Possible mechanisms leading to the prolongation of exercise endurance will be discussed later. For the moment it is sufficient to remember that RMT significantly increased respiratory muscle endurance, i.e., respiratory muscles become more fatigue resistant.

RESPIRATORY MUSCLE FATIGUE

The definition of respiratory muscle fatigue and how this fatigue can be measured is still controversial (32). Fitting (14) wrote: "Inspiratory muscle fatigue has been documented during loaded breathing or acute respiratory failure, but its role in exercise limitation is still undetermined. Electromyographic (EMG) signs of diaphragmatic fatigue develop in normal subjects hyperventilating above 70% of maximal voluntary ventilation, a ventilatory level commonly attained at peak exercise. EMG signs of diaphragmatic fatigue also occur during high power cycling exercise in normal subjects and chronic obstructive pulmonary disease patients. However, a loss of respiratory muscle strength has rarely been documented following strenuous physical exercise with techniques independent of the subjects' collaboration." Two years later, Johnson et al. (19) and Mador et al. (27) demonstrated diaphragmatic fatigue at the end of constant-load exercise to exhaustion for the first time with techniques independent of the subjects' collaboration. Diaphragmatic fatigue was measured as reduction of the transdiaphragmatic pressure during bilateral transcutaneous supramaximal phrenic nerve stimulation, following intensive exercise corresponding to at least 80% $\dot{V}O_{2max}$.

Besides direct proof of diaphragmatic fatigue by phrenic nerve stimulation, other studies showed what was thought to be an indirect sign of respiratory muscle fatigue, i.e., a change in breathing pattern (17,25): respiratory frequency (f_{resp}) increases whereas tidal volume (V_T) decreases ("rapid shallow breathing"). Rather than showing this rapid shallow breathing, our subjects, before they had undergone RMT, increased f_{resp} before exhaustion without any significant change in V_T (3,5,34). This breathing pattern caused an increase in $\dot{V}_E$ with a decrease in end-tidal PCO_2. It seemed as if the hyperventilating subjects became short of breath. We attributed the increase in f_{resp} to respiratory muscle fatigue. A study recently performed by Sliwinski et al. (33) supports our assumption. Their subjects fatigued all inspiratory muscles by loaded breathing before they performed constant-load exercise. While the breathing pattern did not change during mild or moderate exercise (30 and 60% maximal workload), f_{resp} and $\dot{V}_E$ increased with minor changes of V_T during heavy exercise (90% maximal workload).

Fatigued respiratory muscles (26,30) or respiratory muscles that did not have a chance to fully recover after RMT (3,22) reduced the duration of constant-load exercise. Fatigued respiratory muscles may need more than 24 h to fully recover (21,23).

Effects of respiratory muscle training. How can we explain the effects of RMT enhancing the tolerance to constant-load exercise? Usually, increased performance is attributed to improvements of the cardiocirculatory system or to improvements of the skeletal muscles. As neither physical working capacity 170 nor heart rate during the cycling endurance test changed with RMT in sedentary subjects (5), cardiocirculatory improvements are very unlikely to account for the increase in endurance performance. Also improvements of leg muscles are unlikely because the sedentary subjects remained inactive during the RMT study. The physically active subjects did not change their activity levels other than RMT which had no influence on the anaerobic threshold or on $\dot{V}O_{2max}$ (3,34). Therefore, we attribute the improved cycling endurance to the improved fitness of respiratory muscles. It might be that the improved exercise endurance was related to delayed respiratory muscle fatigue. Sedentary subjects continuously increased ventilation without reaching a steady state during constant-load exercise until they became short of breath and had to stop the test (5). After RMT the subjects reached a respiratory steady state while cycling at the same exercise level as before RMT. The necessity to increase f_{resp} was absent. Instead of 26.8 ± 5.9 min they were able to cycle for 40.2 ± 9.2 min which represents a prolongation of 50%. After 18 months, all RMT effects had disappeared and the subjects hyperventilated during constant-load exercise ($PetCO_2$ = 28.8 ± 4.4 mm Hg before vs 36.6 ± 3.5 mm Hg after RMT) as they did before RMT. In other words, with unfit respiratory muscles, sedentary subjects breathed more than necessary for a given level of exercise and cycled for a shorter time.

Physically active subjects showed a similar effect (3,34). Most of them first reached a respiratory steady state and increased $\dot{V}_E$ a second time shortly before they stopped exercising because of exhaustion. After RMT if the onset of this final hyperventilation was postponed, the subjects could cycle longer before they became short of breath. It is not clear so far where the additional ventilatory drive originated, forcing the subjects to increase f_{resp}. Also unclear is why a fatigued system would increase its output (expressed as $\dot{V}_E$). When respiratory muscle fatigue leads to rapid shallow breathing, $\dot{V}_E$ remains constant (17,25). In our tests, $\dot{V}_E$ increased and this increase was of no obvious benefit for the subject. After RMT when $\dot{V}_E$ remained low, the subjects could perform longer, suggesting that the $\dot{V}_E$ increase before RMT was counterproductive. A possible explanation for the "physiological value" (if any) of the f_{resp} increase might be that becoming short of breath forces the subject to reduce the exercise intensity. This could protect respiratory muscles from overuse. At the moment, this explanation sounds rather strange.

Besides postponed respiratory muscle fatigue, other factors may explain why many subjects can cycle longer after RMT. One possibility could be the subjects' lower blood lactate concentrations after RMT compared with before (5,22,34). We have good reasons to assume that after RMT respiratory muscles burn more lactate than before RMT (34). Normally, lower blood lactate concentrations are sup-

posed to enhance performance. However, despite the fact that RMT also decreased blood lactate concentrations during incremental exercise, it did not improve performance in this kind of exertion (34). In another study of RMT (22), cycling endurance was decreased and $\dot{V}_E$ was increased despite lower lactate concentrations after RMT. In the same study (22), it was suggested that the respiratory muscles had not yet fully recovered from RMT, thus causing the still fatigued respiratory muscles to induce the $\dot{V}_E$ increase. This is in harmony with the idea that ventilatory load rather than blood lactate concentrations determines endurance.

Casaburi et al. (7) attributed the ventilatory decrease after physical training to reduced blood lactate concentrations. From our results we may speculate that the observed reductions of $\dot{V}_E$ and lactate concentration are linked but in a different way than suggested by Casaburi et al. (7). RMT and, to a lesser extent, physical training increase the fitness of respiratory muscles. People with fully trained respiratory muscles have the lowest $\dot{V}_E$ possible, and their respiratory muscles fatigue later and burn more lactate. The $\dot{V}_E$, as well as blood lactate concentrations, is lowered by physical training or RMT. The two processes happen in parallel, which has led to the suggestion of a cause/effect relationship (7), whereas the correlation may be coincidental.

Therefore, the lower blood lactate concentrations are very likely not the cause of the lower $\dot{V}_E$ or the prolongation of constant-load exercise after RMT. Another possibility explaining the prolongation of constant-load exercise after RMT is the change of respiratory sensations. After RMT, there was no sensation of breathlessness while breathing heavily during cycling or running (3). If one discontinues RMT, the sensation of breathlessness slowly returns while breathing heavily. Against a change in respiratory sensations as a reason for the improved cycling endurance speaks the fact that only sedentary subjects indicated heavy breathing as a cause for exhaustion while cycling. Subjects who were physically trained but not specifically respiratory trained said that tired legs forced them to stop exercise, although they were breathing heavily with high f_{resp}. This was also true for subjects who clearly benefited from RMT.

Since leg fatigue subjectively dominates over respiratory muscle fatigue at the end of exhaustive constant-load exercise in physically fit but not specifically respiratory trained subjects, another explanation for the benefit of RMT must be considered. Toward the end of constant-load exercise with an intensity of at least 80% $\dot{V}O_{2max}$, the diaphragm develops less force (19,27), yet $\dot{V}_E$ increases at the same time. An increased $\dot{V}_E$ most likely will cause an increased energy expenditure in the respiratory muscles which probably will be covered by increasing oxygen consumption (16,28). Surprisingly, a higher $\dot{V}_E$ (3,5,34) or an increased breathing load (9) apparently does not increase total oxygen consumption while one is cycling at a constant workload. Thus, respiratory muscles might cover their higher oxygen demand at the expense of oxygen supply to leg muscles (6,12,18). The lower $\dot{V}_E$ after RMT means that the respiratory muscles consume less oxygen, thus making more oxygen available for the leg muscles.

SIGNIFICANCE OF RESPIRATORY MUSCLE TRAINING IN HEALTHY HUMANS

Even if we cannot yet explain how RMT prolongs constant-load exercise, four studies have demonstrated the phenomenon (3,5,22,34). This new finding weakens to some extent the concept that the lung, or rather the respiratory system, is built for exercise because the fitness of the respiratory muscles is important for healthy humans' ability to sustain constant-load exercise. Although the common reaction to our results still is surprise or disbelief, the importance of the fitness of the respiratory muscles for endurance exercise should have been realized long ago. If respiratory muscle fatigue reduces performance (26,30), then increased resistance to respiratory muscle fatigue should improve performance. In addition, it is widely accepted that physical training reduces $\dot{V}_E$ at a given workload (7). We specifically trained respiratory muscles and achieved a greater effect in many subjects as $\dot{V}_E$ further decreased at a given workload. Actually, it would be more precise to say that $\dot{V}_E$ was no longer unnecessarily increased after RMT as it was before RMT.

Recently respiratory muscle fatigue was discussed as a factor limiting endurance during exercise with large muscle groups at altitude (8). Since RMT did not influence the hypoxic ventilatory drive (29), RMT could significantly improve exercise performance at altitude. RMT could also reduce the fatiguing aspect of anti-G straining maneuver in pilots flying high performance aircraft because respiratory muscle fatigue was identified as possible candidate to explain the inability of subjects to continue a simulated air combat maneuver centrifuge profile (1).

Overall, evidence is growing that the fitness of the respiratory muscles is more important in healthy humans than previously assumed. In addition to very ambitious athletes, RMT might become an useful exercise for persons who tend to suffer from labored breathing and are unable or unwilling to perform "whole-body" exercise training.

CONCLUSIONS

Intensive respiratory training significantly improves the endurance of respiratory muscles and maximal voluntary ventilation in healthy humans. The majority of our subjects benefited with respect to their ability to endure constant-load exercise. Thus, improved fitness of the respiratory muscles enhances the duration of constant-load exercise without having a significant effect on $\dot{V}O_{2max}$. The mechanism by which respiratory training improves overall physical performance is as yet unknown.

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