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# Breathless Legs? Consider Training Your Respiration

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***The condition of the respiratory system is more important for endurance exercise performance of healthy subjects than hitherto assumed. Not only do respiratory muscles fatigue during intensive endurance exercise, but prefatigued respiratory muscles can also impair performance. In turn, respiratory endurance training can improve endurance exercise performance.***

It is well accepted that the respiratory system may limit exercise performance in disease, e.g., pulmonary or cardiovascular disease, but to what extent the respiratory system may play a significant role in limiting exercise performance of healthy subjects is still controversial. Different studies indicate that exercise does induce respiratory muscle fatigue (6, 9), that res-

piratory muscle fatigue can limit exercise performance (8, 10), that endurance of respiratory muscles can be trained (1–3, 11–13), and that respiratory endurance training can enhance endurance (but not peak) exercise performance (1, 2, 12, 13), even in healthy subjects. Changes in breathing pattern that occur with increasing exercise duration, possibly as a consequence of respiratory muscle fatigue, i.e., increased respiratory frequency, hyperventilation, and/or increased sense of respiratory effort, are reversed after respiratory endurance training, i.e., these changes are delayed or absent.

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## Exercise induces respiratory muscle fatigue

Several studies have brought forward evidence that respiratory muscles may fatigue during exercise; i.e., after long distance races such as a marathon, ultramarathon, or triathlon, respiratory muscle function is impaired. This impairment can last for more than three days after exercise. Also, respiratory muscle fatigue can develop during shorter but more intensive tasks. Diaphragmatic fatigue was shown to occur in subjects who were cycling to exhaustion at a constant workload of ~80% of their maximal workload ( $W_{\max}$ ). At the point of exhaustion, maximal transdiaphragmatic pressure ( $P_{\text{di}}$ , the difference between esophageal and gastric pressure), a measure of maximal diaphragmatic force, was reduced during a maximal inspiratory maneuver and electromyographic recordings of the diaphragm indicated diaphragmatic fatigue. Possible mechanisms accounting for these findings are peripheral fatigue (i.e., muscular fatigue) or central fatigue. As recent as 1993, Johnson et al. (6) and Mador et al. (9) were able to show that the force-generating capacity of the diaphragm is reduced after short (10–30 min) intensive [80–95% of maximal oxygen consumption ( $\dot{V}O_{2\max}$ )] exercise. Both groups used bilateral supramaximal electrical phrenic nerve stimulation to elicit twitches of the diaphragm, and they measured the transdiaphragmatic pressure achieved during the twitch ( $P_{\text{di,tw}}$ ).  $P_{\text{di,tw}}$  is considered an objective measure of diaphragm contractility independent of the subject's effort. These findings (25% decrements in  $P_{\text{di,tw}}$ ) have been confirmed several times since then.

Although all of these studies concentrate on fatigue of the diaphragm, the main inspiratory muscle, we also need to consider potential fatigue of extradiaphragmatic inspiratory muscles (i.e., sternocleidomastoids, parasternals, and scalenes) as well as expiratory muscles, since these muscle groups also come into play during heavy exercise. For instance, Johnson et al. (6) demonstrated that the relative contribution of the diaphragm to total respiratory motor output is progressively reduced with exercise duration, indicating an increasing activity of extradiaphragmatic muscles. Regarding expiratory muscle fatigue, Fuller et al. (4) recently demonstrated that the ability to voluntarily maximally activate abdominal expiratory muscles and to generate maximum expiratory pressures is impaired after exhaustive exercise. Moreover, after only 2 min of maximal isocapnic ventilation, the force-generating capacity of abdominal muscles—tested by magnetic stimulation—is reduced for >90 min (7), which indicates that the reduced ability to maximally activate expiratory muscles after exhaustive exercise is likely to be caused, at least in part, by muscular fatigue. Since respiratory muscles other than the diaphragm become increasingly active during exercise, it is also likely that fatigue of those muscles can contribute to exercise limitation.

## Fatigued respiratory muscles impair endurance exercise performance

A few authors have demonstrated impairment of exercise performance after subjects have voluntarily fatigued their respiratory muscles. Martin et al. (10), for example, had their subjects breathe at 60% of maximal voluntary ventilation for

150 min. After this enormous ventilatory work, the subjects' running time at high speed was significantly reduced, from 7.6 to 6.5 min. Also, respiratory muscle fatigue induced by breathing with a threshold inspiratory load of ~80% of the maximal inspiratory pressure ( $P_{\text{imax}}$ ) compromised endurance time of a subsequent cycling test, i.e., exercise time was reduced from 5.2 to 4 min (8). At the same time, minute ventilation and breathing frequency were increased during exercise. It is indeed astonishing that the output of a fatigued system is larger than normal, a fact that is not yet fully explained. A similar type of hyperventilation, i.e., increased breathing frequency with or without a reduction in tidal volume, frequently occurs toward the end of an endurance exercise test. It is thus possible that this hyperventilation is a result of developing respiratory muscle fatigue. Since in the two studies mentioned above respiratory muscles were likely fatigued to a much larger extent than occurs during exercise, it is still unresolved whether the diaphragmatic fatigue measured after exhaustive cycling tests (6, 9) is large enough to impair endurance performance.

## Unloading respiratory muscles may improve endurance exercise performance

Several authors have used unloading of respiratory muscles during exercise to investigate whether a reduction of respiratory work, and thus less development of fatigue, would allow subjects to improve endurance performance (Table 1). Two different approaches were used to unload respiratory muscles: subjects either breathed a helium/oxygen mixture or their breathing was assisted by a ventilator. The authors found either no effect or a small and insignificant increase of exercise time if subjects were studied at workloads of <80%  $W_{\max}$  and significant increases in exercise time at intensities >90%  $\dot{V}O_{2\max}$ , independent of the method used. These results suggest that respiratory muscle fatigue and/or the respiratory load plays a significant role in limiting human performance at intensities exceeding 90%  $\dot{V}O_{2\max}$ . This improvement was likely the result of the concomitant increase in blood flow to working leg muscles (5). The results also seem consistent with the fact that diaphragmatic fatigue was detected most commonly during exercise at loads >85%  $\dot{V}O_{2\max}$  (6). On the other hand, we need to be aware that helium breathing, as well as assisted ventilation, may have effects other than simply unloading the respiratory muscles, e.g., they may alter respiratory sensations and normal breathing mechanics, affect ventilation distribution (helium), change distribution of blood flow (ventilator), and so forth. Thus other, more "natural" approaches to testing for respiratory limitation may give further insight into the question of respiratory limitation, e.g., respiratory endurance training. If respiratory muscle training can increase cycling endurance time, then it is more than likely that exercise was limited by the respiratory system before the specific training.

## Respiratory muscle training may improve endurance exercise performance

It is known that respiratory muscles of healthy humans can be trained, as is true for any other skeletal muscles, to specifically

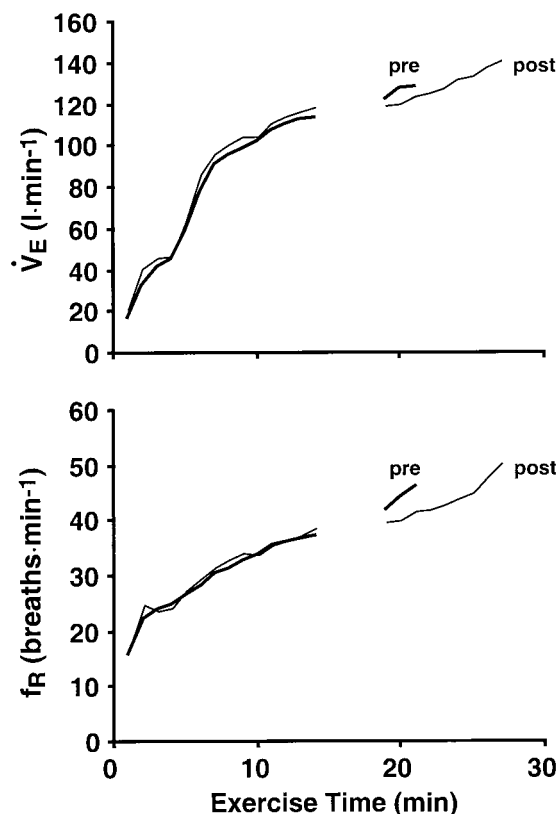
**TABLE 1. Overview of reported changes in endurance performance after respiratory endurance training and with respiratory muscle unloading**

Intervention	Exercise Load	Endurance Change with Intervention	Subjects' Fitness	References
<b>Respiratory endurance training</b>				
85% MVV : 15 x (2+5+9+12) min in 3 wks	95% $\dot{V}O_{2max}$	┆ -6% n.s.	trained	Morgan et al., <i>Int. J. Sports Med.</i> 8: 88-93, 1987
≥ MSVC : 16 x (8+8+8) min in 4 wks	90% $W_{max}$	┆ + 14% n.s.	trained	Fairbairn et al., <i>Int. J. Sports Med.</i> 12: 66-70, 1991
65–85% MVV : 20 x 30 min in 4 wks	86% $W_{max}$	┆ + 28%	trained	Spengler et al., <i>Eur. J. Appl. Physiol.</i> 79: 299-305, 1999
55–68% MVV : 20 x 30 min in 4 wks	77% $\dot{V}O_{2max}$	┆ + 38%	trained	Boutellier et al., <i>Eur. J. Appl. Physiol.</i> 65: 347-353, 1992
60–80% MVV : 40 x 30 min in 13–17 wks	70% $W_{max}$	┆ + 26%	sedentary	Spengler et al., <i>Am. J. Resp. Crit. Care Med.</i> 157: A782, 1998
58–63% MVV : 20 x 30 min in 4 wks	64% $\dot{V}O_{2max}$	┆ + 50%	sedentary	Boutellier and Piwko, <i>Eur. J. Appl. Physiol.</i> 64:145-152, 1992
<b>Respiratory muscle unloading</b>				
He/O <sub>2</sub>	>95% $\dot{V}O_{2max}$	┆ + 21%	highly trained	Aaron et al., <i>Med. Sci. Sports Exerc.</i> 17: 290, 1985
Ventilator	>90% $\dot{V}O_{2max}$	┆ ... sig.	trained	Dempsey et al., <i>FASEB J.</i> 12: A41, 1998
He/O <sub>2</sub>	80–85% $\dot{V}O_{2max}$	┆ + 13%	highly trained	Aaron et al., <i>Med. Sci. Sports Exerc.</i> 17: 290, 1985
He/O <sub>2</sub>	80% $W_{max}$	┆ + 11% n.s.	trained	Krishnan et al., <i>J. Physiol. (Lond.)</i> 490: 537-550, 1996
Ventilator	72–82% $W_{max}$	┆ + 0.7% n.s.	unknown	Marciniuk et al., <i>J. Appl. Physiol.</i> 76: 236-241, 1994

MVV, maximal voluntary ventilation; MSVC, maximal sustainable ventilatory capacity;  $W_{max}$ , maximal power output; and  $\dot{V}O_{2max}$  and  $\dot{V}O_{2peak}$ , maximal and peak oxygen consumption; n.s., not significant.

improve either strength or endurance. Although some authors used hyperpnea training and showed increases in either maximal voluntary ventilation or maximal sustainable ventilatory capacity, others had their subjects breathe with an inspiratory resistance or

threshold load, and those subjects improved respiratory muscle strength. Because ventilation during exercise is high-flow, low-resistance respiratory work, hyperpnea training is likely to improve not only respiratory capacity but also exercise performance.



**FIGURE 1.** Minute ventilation ( $\dot{V}_E$ ) and respiratory frequency ( $f_R$ ) before (pre) and after (post) 20 respiratory endurance training sessions of 30-min duration. Group average data ( $n = 20$ ) before gap are aligned to beginning of exercise. Data after gap are aligned to end of exercise. Curves end at mean time of exhaustion. After respiratory endurance training, increase of  $\dot{V}_E$  and  $f_R$  shortly before exhaustion is delayed compared with before training.

Several studies investigated the effect of hyperpnea training on exercise performance, most of them interested in improving patients' symptoms. Twelve-minute walking distance, a common measure in hospital settings, was improved after hyperpnea training or target flow training in patients with chronic obstructive pulmonary disease. However, the benefits of increased physical performance at submaximal exercise intensities are not limited to patients (Table 1). Increased cycling endurance times were shown after hyperpnea training in sedentary subjects cycling at 64%  $\dot{V}O_{2peak}$  [+50%, from 26.8 to 40.2 min (2)], as well as in trained subjects exercising at 75–85%  $\dot{V}O_{2peak}$  [+38%, from 22.8 to 31.5 min (1) and +28%, from 17.9 to 23.6 min (13)]. These benefits in endurance may be limited to submaximal exercise levels in the range of 65–85%  $\dot{V}O_{2peak}$  (depending on the subject's physical fitness), since improvements were less evident or even absent when endurance was tested at higher exercise intensities by other investigators. Highly trained cyclists showed an insignificant increase in cycling time (from 5.6 to 6.4 min) at 90%  $W_{max}$  after 16 sessions of hyperpnea training (3), whereas moderately trained cyclists did not increase cycling endurance time at 95%  $\dot{V}O_{2max}$  after 3 weeks of very intense hyperpnea training (11).

Why does respiratory endurance training seem to be mostly effective at and below 85%  $\dot{V}O_{2max}$ , whereas diaphrag-

matic fatigue occurs mainly above this exercise intensity? Different factors, which do not need to be mutually exclusive, could be contributing to this difference. Respiratory endurance training might mainly train those extradiaphragmatic respiratory muscles (inspiratory and expiratory) that are used less than the diaphragm during daily living and that are increasingly recruited as exercise proceeds (6). Thus, at submaximal workloads at which subjects cycle for a longer time (>10 min), the subjects could benefit more from respiratory training. Fatigue of extradiaphragmatic muscles is not tested by measurement of  $P_{di,tw}$ . Also, because hyperpnea training intensity and the regimes used are different among research groups (1–3, 11, 13) and the training status of subjects differs as well, results from different studies cannot be readily compared. More studies are needed, including measurement of diaphragmatic and extradiaphragmatic muscle fatigue before and after respiratory endurance training, to shed light on these seemingly conflicting results.

Interestingly, in those subjects who showed a clear improvement in exercise endurance after hyperpnea training, the increase in breathing frequency was delayed during exhaustive exercise after respiratory training (Fig. 1) and the relative decrease in ventilation (compared at the time of pre-training exhaustion) correlated significantly with the relative increase in exercise time (1). If this change in breathing pattern toward the end of exercise is indeed, in part, a result of increasing respiratory muscle fatigue, this might be indirect evidence for a delayed onset of respiratory muscle fatigue after respiratory training. Further investigations are needed to determine which muscle groups are trained the most with hyperpnea training, i.e., inspiratory vs. expiratory muscles, and whether the onset of respiratory muscle fatigue during exercise is indeed postponed.

### Respiratory muscle training may decrease perception of respiratory exertion and breathlessness during exercise

In addition to muscular changes after respiratory endurance training, changes in perception of breathing, such as decreased sense of respiratory exertion or breathlessness, may also contribute to increased endurance times. Some subjects, in fact, need to stop exercise because of an extreme sensation of breathlessness, whereas others experience some breathlessness, but leg fatigue ("breathless legs"?) makes them stop the test. Indeed, Harms et al. (5) have shown an interaction between ventilatory and leg muscle work. They showed that locomotor muscle perfusion and  $\dot{V}O_2$  are compromised to some extent by the work of breathing.

Preliminary results from our laboratory indicate that perceived respiratory exertion is diminished after hyperpnea training (1, 12). It is possible that breathlessness is reduced as well; at least some subjects spontaneously report that they experience less breathlessness during their sports activities or when climbing stairs after the respiratory training. Mechanisms responsible for the perception of respiratory exertion and breathlessness during exercise are still debated. Although some authors suggest that the degree of reflex ventilatory

activation is the important determinant of the intensity of the sensation of breathlessness irrespective of the exact nature of the ventilatory stimulus, others find a disproportionate increase in the perceived intensity of breathlessness above some threshold level of ventilation. This may suggest an increase in breathlessness with developing respiratory muscle fatigue despite minimal change in ventilation. Because Johnson et al. (6) showed a time-dependent decrease in the relative contribution of the diaphragm to total ventilation during constant-load exercise, i.e., extradiaphragmatic muscles are taking over a larger part of the respiratory work, increasing breathlessness with little change in ventilation might mainly be caused by increased perception of extradiaphragmatic muscle activity and/or development of rib cage and expiratory muscle fatigue. Further studies will show whether this hypothesis holds true.

## Perspectives

Respiratory muscle fatigue seems more important than previously assumed in limiting submaximal exercise performance in healthy subjects. Hyperpnea training, shown to improve endurance performance of healthy sedentary as well as trained subjects, may not only be an advantage in the improvement of endurance athletes' performance but also for people who need to compete or work under extreme environmental conditions, such as at high altitude or under water. Also, patients with respiratory disease may benefit from this type of respiratory training, getting additional degrees of freedom in daily living or profiting from a decrease in adverse respiratory sensations during daily exertions.

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## References

1. Boutellier, U., R. Büchel, A. Kundert, and C. Spengler. The respiratory system as an exercise limiting factor in normal trained subjects. *Eur. J. Appl. Physiol.* 65: 347–353, 1992.
2. Boutellier, U., and P. Piwko. The respiratory system as an exercise limiting factor in normal sedentary subjects. *Eur. J. Appl. Physiol.* 64: 145–152, 1992.
3. Fairbairn, M. S., K. C. Coutts, R. L. Pardy, and D. C. McKenzie. Improved respiratory muscle endurance of highly trained cyclists and the effects on maximal exercise performance. *Int. J. Sports Med.* 12: 66–70, 1991.
4. Fuller, D., J. Sullivan, and R. F. Fregosi. Expiratory muscle endurance performance after exhaustive submaximal exercise. *J. Appl. Physiol.* 80: 1495–1502, 1996.
5. Harms, C. A., T. J. Wetter, S. R. McClaran, D. F. Pegelow, G. A. Nickle, W. B. Nelson, P. Hanson, and J. A. Dempsey. Effects of respiratory muscle work on cardiac output and its distribution during maximal exercise. *J. Appl. Physiol.* 85: 609–618, 1998.
6. Johnson, B. D., M. A. Babcock, O. E. Suman, and J. A. Dempsey. Exercise-induced diaphragmatic fatigue in healthy humans. *J. Physiol. (Lond.)* 460: 385–405, 1993.
7. Kyroussis, D., G. H. Mills, M. I. Polkey, C. H. Hamnegard, S. Wragg, J. Road, M. Green, and J. Moxham. Effect of maximum ventilation on abdominal muscle relaxation rate. *Thorax* 51: 510–515, 1996.
8. Mador, M. J., and F. A. Acevedo. Effect of respiratory muscle fatigue on subsequent exercise performance. *J. Appl. Physiol.* 70: 2059–2065, 1991.
9. Mador, M. J., U. J. Magalang, A. Rodis, and T. J. Kufel. Diaphragmatic fatigue after exercise in healthy human subjects. *Am. Rev. Respir. Dis.* 148: 1571–1575, 1993.
10. Martin, B., M. Heintzelman, and H.-I. Chen. Exercise performance after ventilatory work. *J. Appl. Physiol.* 52: 1581–1585, 1982.
11. Morgan, D. W., W. M. Kohrt, B. J. Bates, and J. S. Skinner. Effects of respiratory muscle endurance training on ventilatory and endurance performance of moderately trained cyclists. *Int. J. Sports Med.* 8: 88–93, 1987.
12. Spengler, C. M., C. Lenzin, C. Stüssi, G. Markov, and U. Boutellier. Decreased perceived respiratory exertion during exercise after respiratory endurance training (Abstract). *Am. J. Respir. Crit. Care Med.* 157: A782, 1998.
13. Spengler, C. M., M. Roos, S. M. Laube, and U. Boutellier. Decreased blood lactate concentrations after respiratory endurance training. *Eur. J. Appl. Physiol.* 79: 299–305, 1999.