



SHORT REPORT

Does training of respiratory muscles affect exercise performance in healthy subjects?

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Is there a physiologic rationale for respiratory muscle training effects on exercise performance?

Ventilatory demand during exercise is met by increased volume and airflow. They require higher negative pleural pressure (Pes) which peak ranges from 49% to 90% of maximal pressure generating capacity (Pcap) of the respiratory muscles during both incremental maximal^{1,2} and endurance exercise.^{3,4} In these conditions the diaphragm progressively reduces over time its relative pressure contribution (Pdi) to total inspiratory muscle output (Pdi/Pes).³⁻⁵ Studies employing non-volitional methods have demonstrated that high-intensity whole body exercise endurance (>85% VO₂max) induces global respiratory muscle fatigue. In this context, the functional significance of improved respiratory muscle function with respiratory muscle training (RMT) would presumably be to prevent or delay the diaphragm fatigue that is known to occur during sustained high-intensity exercise.³ Competition for

blood flow distribution and/or acidification of the diaphragm via its uptake of circulating lactate in conjunction with a contracting diaphragm accounts for most of the exercise-induced diaphragm fatigue in healthy subjects.⁶ To define the contribution of the work of breathing to diaphragm fatigue, Babcock et al.⁷ were able to show that endurance exercise at 80–85% VO₂max (55 ml/kg/min) resulted in both high- and low-frequency diaphragm fatigue. Partial unloading of the diaphragm via pressure assist ventilation (PAV) reduced the work of breathing and total VO₂ and prevented diaphragm fatigue at all stimulation frequencies and after exercise. These findings are consistent with the contribution of work load per se to diaphragm fatigue.

Development of diaphragm fatigue during exercise is a function of the relationship between the magnitude of diaphragm work and adequacy of its blood supply: the less the blood flow available, the less the diaphragm work required to produce fatigue. Muscle force output vs blood flow imbalance of the diaphragm (which favors fatigue) may occur during endurance exercise only when the intensity of exercise exceeds 85% VO₂max or arterial hypoxemia is present.⁸

Recent evidence shows that inspiratory muscle fatigue causes a sympathetically mediated vasoconstriction of lower limbs. St Croix et al.⁹ fatigued

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the diaphragm by imposing high-intensity contraction and high duty cycle on healthy subjects. Fatigue caused a time-dependent increase in muscle sympathetic nerve activity (MSNA) in resting legs. MSNA is likely due to type IV nerve ending stimulation of the diaphragm. In a later study, MSNA was found to be accompanied by significant decreases in limb vascular conductance and limb blood flow along with increased mean arterial pressure and heart rate.¹⁰ A high MSNA also results as a metabo-reflex during high-intensity and high-frequency contractions of the expiratory muscles.¹¹ In turn, Romer and Dempsey¹² postulated that reflex mechanisms of sympathoexcitation are triggered by metaboreceptors in the diaphragm as the muscle begins to accumulate metabolic end products during heavy exercise when cardiac output is insufficient to adequately meet the high metabolic requirements of both respiratory and limb musculature.

It appears therefore that respiratory muscle fatigue must occur for unloading to improve performances.^{13,14} This might explain why other studies have not found a significant effect of respiratory muscle unloading on exercise capacity.¹⁵ An important observation is that respiratory muscle fatigue during heavy exercise^{6,7} could negatively impact limb work capacity due to blood flow redistribution even when cardiac output is not near maximal level.

Based on the above results it has been postulated that RMT might improve exercise capacity as follows: (i) by preventing or delaying respiratory muscle fatigue and its effect on blood flow distribution, (ii) by causing less recruitment of accessory respiratory muscles, and therefore (iii) by increasing efficiency and decreasing blood flow requirements of respiratory muscles during exercise,¹² (iv) by reducing the amount of sympathetically induced reflex vasoconstriction in response to isometric exercise.^{16,17}

RMT effects on exercise response

Several studies in recent years have examined the effects of specific respiratory muscle endurance training on the performance of both respiratory muscles and exercise performance itself. However, the effects of RMT on exercise performance in healthy subjects are controversial. As shown in Table 1, 25–50% improvement in endurance exercise performance has been reported by some,^{18–27} but not by others.^{28–34} A reduction in exercise ventilation^{18,19} and blood lactate^{20,21,27}

Table 1 Effects of RMT on whole body endurance capacity in healthy subjects.

Endurance improvement	VE			VO ₂ max			Blood lactate reduction		
	Yes	Not	Reduction	No changes	Increase	No changes	Yes	Not	
	18*,19†,20*,21‡,22‡,23†,24†,25‡,26‡,27‡,28‡,40*	29‡,30‡,31*,32‡,33‡,34‡	18,19,40	23,24,26,28,30–34	20–22,25,27,29	26,28–31,40	18,20,21,28	25,26,33	

RMT: respiratory muscle training; VE: minute ventilation; VO₂: oxygen uptake.
 *Trained subjects.
 †Sedentary subjects.
 ‡Athletes.

has been reported as evidence of the effect of training on respiratory muscles. The two studies by Boutelier and Piwko¹⁸ and Boutelier et al.¹⁹ are exceptions in that endurance performance at about 75–80% of VO_2max was increased by 38% in non-athletes and 50% in athletes. These changes were accompanied by substantial but highly variable reductions in ventilatory responses to exercise. No reduction in ventilation^{23,24,26,28,32–34} and even its increase^{20–22,25,29} was reported in other studies whereas aerobic capacity resulted either unchanged^{29–31} or improved.²⁸ The discrepancies among studies may reflect differences in exercise intensities and durations used for testing and the markedly different experimental designs of the studies. Sonetti et al.³³ have recently stressed the probable reasons for discrepancies (subjective nature of the performance tests, variation in the relative intensity and duration of exercise, fitness level of subjects, inclusion and exclusion of control groups), and two major confounding effects: (1) use of a fixed work rate performance test which has little relevance to the subject's competition experience, and does not usually yield reproducible results³⁵; (2) absence of a legitimate placebo group (most of the performance outcomes tested volitional effort and therefore on the subject's expectation and motivation).

After the seminal paper by Harms et al.³⁶ showing that mechanical unloading of the respiratory muscles with PAV during maximal whole body exercise resulted in 5–7% blood flow increase in leg muscles, and 14% increase in endurance capacity, with no changes in circulating lactate, and decrease in VO_2 , cardiac output, dyspnea and leg effort, it seems inconceivable that the effect of RMT could surpass those seen with PAV. Sonetti et al.³³ do not suspect this to be the case since they observed a similarly large performance improvement which was not due to RMT but primarily to a significant placebo plus familiarization effect for fixed work rate exercise. They also maintained that specific RMT in highly fit competitive subjects may influence peak exercise performance to a very limited extent and most of this influence can be explained by a placebo (expectation) effect. Some studies³² used "minimal exercise" placebo that may not be sufficient to activate placebo factors such as expectation.³⁶ These data strengthen the need for applying the criteria suggested by Ojaunen³⁷ for a true placebo: (1) the placebo needs to be inert (no effect on ventilation, and carbon dioxide tension), (2) it should generate expectations, involvement, subjective utility and be meaningful to the subjects.

Is the present method for RMT useless in healthy subjects? Recent data by De Palo et al.³⁸ argue against this possibility. Based on the observation of diaphragm recruitment and Pdi increase with non-respiratory manoeuvres, De Palo et al.³⁸ show that strength training of non-respiratory manoeuvres may strengthen the respiratory muscles in healthy subjects by increasing maximal Pdi, maximal inspiratory (MIP) and expiratory (MEP) muscle pressures, and maximal gastric pressure after 16 weeks of training. Because diaphragm thickness also increased, it was unlikely that the effects were due to training. Whether and to what extent non-respiratory manoeuvres also affects exercise performance remains to be defined.

Conclusions

Recent studies provide evidence that RMT improves exercise performance in well-controlled and rigorously designed studies utilizing exercise tests with the lowest coefficient of variation for repeated testing, appropriate outcome measures, and fitness of the participants.^{28,39,40} Further studies, however, are needed to define the mechanisms by which RMT improves exercise performance.

No amount of experimentation can prove me right; a simple experiment may at any time prove me wrong (A. Einstein).

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