

Respiratory muscle fatigue limiting physical exercise?

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ABSTRACT: 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 (MVV), 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 (COPD) patients. However, a loss of respiratory muscle strength has rarely been documented following strenuous physical exercise with techniques independent of the subjects' collaboration. Prior inspiratory muscle fatigue decreases exercise tolerance in normal subjects but its effect is largely unknown in COPD patients. Respiratory muscle rest by negative pressure ventilation was reported to improve exercise tolerance in COPD, but this beneficial effect was not confirmed by controlled studies. The effect of inspiratory muscle training on exercise tolerance is still undefined by existing data, in part because of differences in methods and selection criteria between studies. Although respiratory muscle fatigue may occur during exercise, it is not clearly established whether interventions directed at respiratory muscles may improve exercise tolerance in COPD.

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For more than a decade, the phenomenon of respiratory muscle fatigue has been extensively studied in normal humans submitted to inspiratory loads. In these experimental circumstances, the two main determinants of respiratory muscle fatigue appear to be the ratio of pressure developed by inspiratory muscles to their maximal capacity (P/P_{MAX}) and the duty cycle of inspiratory muscles (T_i/T_{TOT}): fatigue occurs above a certain threshold of intensity and duration of contraction in the various inspiratory muscles [1-3]. The ratio of velocity of muscle shortening to maximal velocity probably represents another determinant of fatigue, particularly during unimpeded breathing.

End-expiratory lung volume is another important factor for two reasons. Firstly, the force generating capacity of the diaphragm decreases at higher lung volumes [4]. Secondly, end-expiratory lung volume may be actively increased by persistent contraction of inspiratory muscles, as in asthma [5]. Thus, fatigue occurs with lower inspiratory pressures when end-expiratory lung volume is actively increased [6].

With respect to energetics, respiratory muscle fatigue is determined by efficiency, which is the ratio of external power produced to the energy consumption rate, and by the balance between the energy demand and the energy supplied by blood flow.

In patients, respiratory muscle fatigue has mainly been demonstrated during weaning trials from mechanical ventilation and has been shown to contribute frequently to weaning failures [7-10]. Although less documented in other circumstances, respiratory muscle fatigue is currently deemed to play a major role in the development of acute ventilatory failure [11].

Patients with chronic respiratory insufficiency, in particular those with chronic obstructive pulmonary disease (COPD), are handicapped by a markedly reduced exercise capacity. The question of whether respiratory muscle fatigue represents a limiting factor for them is important. Indeed, depending on the answer to this question, various interventions on respiratory muscles may, or may not, appear justified to improve exercise tolerance in these patients.

Indirect evidence that respiratory muscle fatigue could limit exercise

Maximal exercise capacity is usually considered not to be limited by ventilation in normal subjects. This contention is supported by the observation that maximal exercise ventilation attains approximately 70% of maximal voluntary ventilation (MVV) [12, 13]. Thus,

normal subjects are said to have some "breathing reserve", even if dyspnoeic when stopping exercise [13]. However, MVV is usually measured over 15 s and cannot be maintained much longer. According to different studies, the maximal sustainable ventilation decreases with time and the level that can be sustained for more than 15 min corresponds to 55–80% of MVV [14–16]. The inability to sustain MVV for more than 15–30 s is probably explained by respiratory muscle fatigue. Indeed, fatiguing contractions of the diaphragm have been reported during high level hyperpnoea in normal subjects, as documented by a shift in the electromyographic (EMG) power spectrum [17]. Hyperpnoea also resulted in a loss of maximal transdiaphragmatic pressure, as measured by voluntary efforts or phrenic stimulation [17]. These manifestations of fatigue always occurred at levels of ventilation exceeding 70% of MVV. Therefore, the maximal exercise ventilation should more appropriately be compared to the maximal sustainable ventilation expected at the corresponding time. Computed in this way, the breathing reserve is likely to be thin even in normal subjects.

The demonstration of diaphragmatic fatigue during hyperpnoea may seem surprising because the pressures developed under these circumstances are relatively low. During hyperpnoea or exercise, the peak pleural pressure (Ppl) attains only -30 cmH₂O approximately and is, therefore, well below maximal static inspiratory pressure. However, the performance of inspiratory muscles must always be considered in terms of demand and capacity [18]. As lung volume increases during inspiration, the capacity to generate inspiratory pressure progressively declines. Data obtained in supine animals indicate that increasing lung volume mainly affects the diaphragm, with regard to the force-length relationship. Indeed, diaphragm length is close to optimal at functional residual capacity (FRC) [19] and decreases by 30% at total lung capacity (TLC) [20]. In contrast, the length of parasternal intercostal muscles decreases less and becomes near optimal at TLC [21, 22]. With increasing airflow, a further loss results from the force-velocity relationship, the velocity of shortening of the diaphragm being reflected by inspiratory flow [23]. Thus, in normal subjects at maximal exercise, LEBLANC *et al.* [18] found peak Ppl to be equal to 30% of maximal static inspiratory pressure, but to 42% of the maximal inspiratory capacity at the corresponding lung volume and airflow. Had their subjects not decreased end-expiratory volume during exercise, this ratio would have increased to 55–70% [18]. Therefore, the force reserve of inspiratory muscles is considerably reduced during maximal exercise. Moreover, the blood flow and energy supplied to the respiratory muscles may be less during intense whole body exercise than it is when only inspiratory muscles are exercising as during experimental inspiratory loading.

In this respect, patients with COPD are definitely in a more unfavourable condition. Firstly, the increased ventilatory load results in a greater demand on their inspiratory muscles. Secondly, their diaphragm is

characterized by an abnormal geometry and often a reduced muscle mass, all factors contributing to curtail their capacity to generate inspiratory pressure. Data obtained from models of emphysematous hamsters indicate that the diaphragm adapts by dropping out sarcomeres, so that its normal force-length curve is displaced to the new operating length [24–27]. However, the question is not settled since ARORA and ROCHESTER [28] found no evidence of permanent shortening of the diaphragm in autopsies performed in COPD patients.

Because of their flow limitation during expiration, COPD patients have only two ways to increase ventilation during exercise. One of these is to breathe at higher lung volume where maximal expiratory flow is greater. However, the elastic work increases and the inspiratory muscles operate at a further mechanical disadvantage at high lung volume. The other is to prolong expiration to enhance lung emptying. However, a longer expiration means a shorter inspiration and, therefore, a higher velocity of shortening of inspiratory muscles to increase inspiratory flow [29]. For these theoretical reasons, COPD patients appear at particular risk to develop inspiratory muscle fatigue during exercise.

Direct evidence of respiratory muscle fatigue during exercise

The fact that prolonged strenuous exercise can induce respiratory muscle fatigue is suggested by the measurements performed by LOKE *et al.* [30] in four runners before and after completion of a marathon. At the end of the race, maximal inspiratory pressure (P_{imax}) fell significantly by 16%, maximal expiratory pressure (P_{emax}) by 28%, and MVV by 9% [30]. In a larger number of subjects, CHEVROLET *et al.* [31] found a loss of P_{imax} after both a half-marathon and a full marathon race. Moreover, BYE *et al.* [32] reported a loss of diaphragm strength in normal subjects exercising until exhaustion at 80% of maximal power output on a cycle ergometer, with mean minute ventilation attaining 67% of MVV. Maximal voluntary transdiaphragmatic pressure (P_{di,max}) fell by 12% post-exercise. This loss of strength was possibly due to fatigue as it was associated with a decline in the high/low ratio of diaphragmatic EMG during the runs [32].

Respiratory muscle fatigue during exercise has also been shown to occur in patients with COPD. During exercise at 80% of maximal power output on a cycle ergometer, a fatiguing pattern of the diaphragm developed in five out of eight patients, as shown by a fall in the high/low ratio of the EMG [33]. Electromyographic signs of fatigue could also be observed in the scalenes and intercostal muscles of COPD patients during exercise [34, 35]. Finally, using electrical stimulation to establish force-frequency curves, WILSON *et al.* [36] demonstrated low-frequency fatigue of the sternomastoid in a group of COPD patients after a 12 min walking test.

It should be noted that the latter study is the only one unequivocally demonstrating force failure of respiratory muscles after exercise. Because of their dependence on subject collaboration and their intrasubject variability, the measurements of maximal voluntary pressures are often difficult to interpret, in particular those showing small changes. Thus, if fatiguing contractions of the respiratory muscles have been documented by EMG during exercise, overt muscle failure has only rarely been demonstrated.

Is respiratory muscle fatigue actually limiting exercise?

There is now evidence from several studies that a process of fatigue can develop in respiratory muscles during high level exercise, both in normal subjects and in patients with COPD. The important question is whether or not this fatigue actually limits exercise.

The problem can be approached by assessing the effect of prior fatigue of the respiratory muscles on exercise capacity. One study recently reported the effect of intense, short-term ventilatory work on exercise capacity in normal subjects. It was found that high level isocapnic hyperpnoea for 10 min had no effect on endurance time during a constant load cycle exercise at 85% of maximal power output [37]. In contrast, another study concluded that maximal hyperpnoea for 150 min, corresponding on average to 66% of MVV, decreased exercise capacity in normal subjects. During maximal treadmill running following hyperpnoea, there was a decrease in endurance time, maximal oxygen consumption, maximal ventilation, and maximal heart rate [38]. Finally, another group reported that respiratory muscle fatigue induced in normal subjects by an inspiratory load decreased subsequent endurance time during a constant load cycle exercise at 90% of maximal power output [39]. Thus, maximal exercise capacity can be curtailed by prolonged hyperpnoea or by loaded breathing in normal subjects.

This experimental approach has rarely been applied to patients. It has been reported in four COPD patients that prior fatigue of the sternomastoid, as induced by a 12 min walking test, had no effect on the distance walked during a second test performed immediately afterwards [36]. Thus, from the data available it cannot yet be concluded that prior fatigue of the respiratory muscles affects exercise capacity in patients.

Assessing interventions directed at improving respiratory muscle performance represents another approach to the problem. Because fatigue may be defined as a loss of muscle performance induced by effort and reversible by rest, the latter represents such an intervention. Resting of respiratory muscles can be achieved by mechanical ventilation, via endotracheal intubation or noninvasive means. In an uncontrolled study, GUTIERREZ *et al.* [40] assessed the effect of negative pressure ventilation for eight hours per day, once a week, in five hypercapnic COPD patients. After four months, the mean P_{max} had increased from 45 to

62 cmH₂O and the 12 min walking distance from 573 to 700 m. However, these findings were not supported by three other studies. PLUTO *et al.* [41] reported that eight weeks of negative pressure ventilation, four to seven hours per day, failed to improve P_{max} and exercise performance on a cycle ergometer in five patients with severe COPD. In a randomized, cross-over study, ZIBRAK *et al.* [42] compared negative pressure ventilation, two to six hours per day for six months, to standard care in nine severe COPD patients. No significant change occurred in P_{max} and walking endurance time during either the ventilation or the control periods. Finally, the preliminary results of a large, double-blind, controlled trial in Montreal also indicate that intermittent negative pressure ventilation failed to improve exercise capacity in COPD patients [43]. A key question is whether the respiratory muscles were adequately rested in these studies. If actual rest was achieved, these negative results cast doubt on the existence of chronic respiratory muscle fatigue, or at least on its relevance to exercise capacity.

Training the respiratory muscles through resistive breathing or hyperpnoea can improve their strength and endurance. Therefore, the result of this intervention is susceptible to reveal the actual role of respiratory muscle fatigue as a limiting factor of exercise. In normal elderly subjects, training by the hyperpnoea method has been found to increase respiratory muscle endurance but not physical exercise performance [44]. Numerous studies have assessed respiratory muscle training in COPD and several of them included the parameter of exercise performance, as determined from the 12 min walking distance or from endurance during an incremental or constant load cycle exercise. However, they yielded contradictory results which may in part be explained by important methodological differences. Firstly, most studies did not include selection criteria such as documentation of ventilatory limitation or respiratory muscle fatigue during exercise. Secondly, many studies did not control the training stimulus. This point is of particular relevance to resistive breathing where the patients may use low flow and pressure breathing patterns leading to an insufficient training stimulus.

Training of inspiratory or expiratory muscles was reported to improve exercise tolerance in several studies which, however, did not comprise selection criteria regarding ventilatory limitation on exercise or control of training stimulus [45–47]. Enhanced exercise tolerance was also reported by two studies where the stimulus was controlled, either by sustained hyperventilation [48] or by inspiratory threshold loads [49]. Controlling the training stimulus by the detection of EMG signs of fatigue, PARDY *et al.* [50] also found that inspiratory muscle training improved exercise tolerance in COPD. However, this beneficial effect could not be attained in all patients. They observed that exercise performance improved in only seven out of twelve patients after inspiratory muscle training. These authors pointed out that six of the seven patients manifested electromyographic signs of inspiratory muscle fatigue

during the pretraining cycling exercise tests. In contrast, electromyographic signs of fatigue were not observed in the five patients who showed no improvement in exercise performance with training [35]. DEKHUIZEN [51] studied selected patients showing ventilatory limitation during exercise. Resistive inspiratory muscle training was applied and the training stimulus was controlled with a target-flow system. It was found that maximal work load and maximal oxygen consumption increased equally with standard pulmonary rehabilitation and inspiratory muscle training, whereas the 12 min walking distance increased more with inspiratory muscle training [51].

Another group of studies concluded that respiratory muscle training had no beneficial effect on exercise performance. Selection criteria regarding ventilatory limitation during exercise were not included, with the exception of a single study [52]. Three of these studies did not control the training stimulus either [52-54]. The inspiratory pressures generated by resistive breathing were controlled at the onset of training in one study [55]. Two other studies applied a controlled stimulus via sustained hyperventilation [56] or inspiratory threshold load [57]. It appeared from studies including a control group that exercise performance could improve similarly with sham and with real respiratory muscle training. This placebo effect has been attributed to increased co-ordination, desensitization to the fear of dyspnoea, group therapy and close attention [53, 56].

Conclusion

From the evidence available, it can be stated that fatiguing contractions of the respiratory muscles can occur during maximal exercise in normal subjects and in patients with COPD. It is much less clear if, and how often, respiratory muscle fatigue actually limits exercise. Until now, respiratory muscle rest or training failed to convincingly improve exercise capacity in patients with COPD. It may be erroneous to try to isolate a single limiting factor to exercise capacity in patients. Even if ventilation and respiratory muscles may represent the primary limiting factor in a severe COPD patient, the peripheral muscles can be expected to contribute also to exercise limitation because of generalized muscle weakness. Although respiratory muscle fatigue may occur during exercise, there is at present little basis to recommend an intervention directed solely at the respiratory muscles to improve exercise tolerance in COPD patients.

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La fatigue musculaire respiratoire, facteur limitant l'exercice?
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RÉSUMÉ: La fatigue des muscles inspiratoires a été démontrée en présence de charges ventilatoires et d'insuffisance respiratoire aiguë, mais son rôle comme facteur limitant l'exercice n'est pas connu. Les signes électromyographiques (EMG) de fatigue diaphragmatique apparaissent chez des sujets normaux hyperventilant au-delà de 70% de la ventilation maximale volontaire, niveau ventilatoire communément atteint à l'exercice maximal. Les signes EMG de fatigue diaphragmatique apparaissent également à l'effort intense sur bicyclette ergométrique chez des sujets normaux et des patients BPCO. Une diminution de la force musculaire respiratoire a été documentée après un exercice intense, mais rarement au moyen de techniques indépendantes de la collaboration des sujets. Une

fatigue musculaire inspiratoire préalable réduit la capacité d'effort chez des sujets normaux, mais son effet est largement inconnu dans la BPCO. La mise au repos des muscles respiratoires par ventilation sous pression négative a été considérée comme améliorant la capacité d'effort des patients BPCO, mais cet effet favorable ne fut pas confirmé par des études contrôlées. L'effet de l'entraînement des muscles inspiratoires sur la capacité à l'exercice n'est pas clairement démontré par les données existantes, en partie à cause de différences de méthodes et de critères de sélection entre les études. Bien que la fatigue musculaire respiratoire puisse apparaître à l'exercice, il n'est pas établi que des interventions dirigées sur les muscles respiratoires puissent améliorer la capacité à l'exercice dans la BPCO.

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