
Exercises in COPD

Exercises in COPD: damned if you do and damned if you don’t

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Recruitment of the expiratory muscles by COPD patients during exercise

In this issue of Thorax Aliverti et al present the most complete description of the effect of bronchodilators in chronic obstructive pulmonary disease (COPD) to date. Surprisingly, they found that the patient population segregated cleanly into two groups: those whose exercise endurance was improved and those whose exercise endurance decreased. This unexpected adverse effect was not trivial: endurance time at constant workload decreased on average by 34%, while endurance time in those who improved increased by 86%. They called this group the “improvers”, and those whose exercise performance became worse the “non-improvers”: a more accurate moniker for the non-improvers would be “worseners”. Both groups dynamically hyperinflated to the same extent during exercise after placebo, whereas following bronchodilation the improvers hyperinflated more than the worseners so that, at the limit of endurance, inspiratory reserve volume was considerably larger in the latter.

In a previous paper Aliverti et al studied patients with stable COPD during incremental exercise and found that 60% dynamically hyperinflated while 40% did not. The latter group (which they called “euvolumics”) decreased end expiratory volume during exercise, just as healthy subjects do. The euvolumics had significantly better resting lung function but, paradoxically, significantly worse exercise performance.

The implication of these two papers is that, in COPD, it is better to hyperinflate dynamically because it improves exercise performance than to use a breathing pattern which mimics that used by healthy subjects. What on earth is going on here? According to many experts, dynamic hyperinflation is supposed to be the most important factor limiting exercise in COPD.

In a study of 105 patients with COPD, O’Donnell et al found dynamic hyperinflation occurred in 80% of patients during incremental exercise. These researchers measured inspiratory capacity by integrating flow at the mouth as an index of hyperinflation and found that it decreased by a mean (SD) of 0.37 (0.39) l. On the other hand, Aliverti et al measured chest wall volumes by optoelectronic plethysmography (OEP). Could the difference between the methods of measurement have led to an underestimation of dynamic hyperinflation by OEP and/or an overestimation by spirometry?

Spirometry measures the volume of gas entering or leaving the lungs at the mouth. OEP measures the volume of the trunk. This includes volume changes at the mouth, but also two other variables—gas compression and decompression in the lungs, and blood shifts between the trunk and extremities. In normal subjects exercising with a Starling resistor in the expiratory line to limit expiratory flow to approximately 1 l/s, gas compression due to expiratory muscle recruitment and blood shifts can be substantial.4 On average, normal subjects shift 7.2 ml blood from the trunk to the extremities for every 1 cm H2O increase in alveolar pressure. The euvolumics in Aliverti’s first study4 generated peak expiratory pleural pressures of about 22 cm H2O at maximal exercise workload. Peak alveolar pressures would be higher by an amount equal to the elastic recoil pressure of the lung. Assuming that (1) peak alveolar pressures were 25 cm H2O, (2) blood shifts were 7.2 ml/cm H2O, and (3) the operating lung volumes were 6 litres, OEP would measure a minimal expiratory chest wall volume that would be 330 ml less than the minimal lung volume measured by spirometry. Thus, OEP would not detect 89% of the reduction in inspiratory capacity measured by O’Donnell and colleagues.

However, if this were the case, minimal chest wall volume would occur during expiration and not at end expiration. This has been shown in normal subjects exercising with expiratory flow limitation.4 During control exercise,
volume changes measured by spirometry and OEP went along the line of identity whereas, with flow limitation, expiratory chest wall volume led the spirometric volume in time and magnitude and reached a minimum before expiratory flow at the mouth ceased. Dynamic hyperinflation as assessed by OEP must be done at zero flow points, not at minimal chest wall volume. It is unclear which volume was chosen by Aliverti et al. It would be better to make the comparison between OEP and spirometry at end inspiration. Certainly there would be no gas compression at this point, although it is doubtful whether—

with inspiration taking less than 2 seconds—there would be sufficient time for blood to shift back from the extremities to the trunk. The difference in end inspiratory volumes between improvers and worseners just before stopping exercise was about 400 ml, a little large to be accounted for by blood shifts alone. Nevertheless, euvaluomics measured by OEP might well be called “hyperinflators” if measured by spirometry. But it seems clear that there are euvaluomics measured by spirometry. O’Donnell’s data revealed a decrease in mean (SD) inspiratory capacity of 370 (390) ml. Assuming these data to be normally distributed, one would expect to see changes in end expiratory lung volume ranging from an increase of +1150 ml (mean + 2SD) to a decrease of −410 ml (mean − 2SD). The decrease is virtually identical to the mean value measured by Aliverti et al in the euvaluomics. What remains unclear is the percentage of patients with COPD who are euvaluomics or hyperinflators.

Is it relevant to know this percentage? I doubt it. The essential difference between improvers and worseners’ and between euvaluomics and hyperinflators’ was not so much about what happened to their lung volumes, but the degree of expiratory muscle recruitment. The kinematic difference between improvers and worseners was in the behaviour of the abdomen. After bronchodilators there was inward abdominal displacement at end expiration to a volume considerably less than that during quiet breathing in the worseners, but no change from quiet breathing in the improvers. The worseners were using the abdomen to pump the lungs much more than the improvers. This requires coordinated activity of the abdominal muscles and the diaphragm so that, as the diaphragm contracts during inspiration the abdominal muscles relax, and vice versa during expiration. This is the normal response to exercise and it requires a simple respiratory muscle neural control system by which the rib cage muscles and the abdominal muscles are activated 180° out of phase with each other. This neural control mechanism is not used at rest but is brought into play as soon as exercise starts, even at minimal workloads. It is as if a switch occurred in the central neural drive to breathe to recruit expiratory muscles immediately at the onset of exercise. This is what happened in the euvaluomics and worseners so the neural drive to their respiratory muscles was “normal”. This is hardly surprising. The problem is that, in COPD, the expiratory flow generated by the abdominal pump becomes limited by dynamic airway compression. Thus, the power output of the abdominal muscles (the product of their velocity of shortening and the force they develop) is expressed more as pressure and less as flow. This was first recognised by Potter et al 34 years ago. They observed that, during exercise, some patients with COPD were euvaluomics and that excessive expiratory muscle recruitment occurred. They questioned whether or not the high expiratory pressures had adverse circulatory effects. It is now known that expiratory muscle recruitment during flow limited exercise acts as a Valsava manoeuvre, decreasing cardiac output and producing the blood shifts from trunk to extremities. In addition, the oxygen cost of breathing is so high in COPD that it can become a very large percentage of total body oxygen uptake. This can establish competition between respiratory and locomotor muscles for available oxygen supply at low exercise workloads. It seems that the combination of high ventilatory oxygen demands and limitation of cardiac output—both caused by excessive expiratory pressures—can be a potent factor limiting exercise performance in COPD.

This raises the question whether exercise might be improved if appropriate physiotherapy programmes could train patients to relax their expiratory muscles. The benefits could be substantial: there would be a reduction in the oxygen cost of breathing and the cardiac output should increase, and the competition between respiratory and locomotor muscles would be alleviated. This would require an “abnormal” response to exercise so that the abdominal pump would be activated to a much lesser degree. Although this may be an interesting question, I think we already have the answer. This is what the improvers and the hyperinflators do. Their expiratory muscle recruitment, work of breathing, and competition between respiratory and locomotor muscles are all considerably less than in euvaluomics and worseners. They have learned to bypass the normal control of the respiratory muscles and, hence, their expiratory pressures remain low. Even if expiratory pressures are substantial, dynamic hyperinflation appears to minimise its effect on cardiac output. Are they improved as a result? Not much. Without expiratory muscle recruitment, dynamic hyperinflation is inevitable if patients are sufficiently flow limited. There can be no doubt that dynamic hyperinflation is a common and potent mechanism limiting exercise in COPD. A great deal of research has shown that patients are crippled as a result. Recruit your expiratory muscles or not, patients with COPD are damned if they do and damned if they don’t. Nevertheless, dynamic hyperinflation does seem to be a better strategy for exercise in COPD than the one adopted by healthy subjects and worseners. But it does not confer much benefit.


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