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INTRODUCTION

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Pulmonary hyperinflation is commonly divided into static and dynamic in patients with chronic obstructive pulmonary disease (COPD). The former can be directly attributed to the emphysema-related reduction in lung elasticity, leading to a larger volume at which lung and chest wall recoil pressures are balanced. As a consequence, both total lung capacity and functional residual capacity increase above their normal levels. Conversely, dynamic hyperinflation (DH) is caused by expiratory airflow limitation, air trapping and ''auto-positive end-expiratory pressure''. This further increases functional residual capacity.

Extensive literature has accumulated on the objective and subjective manifestations and sequelae of pulmonary hyperinflation in COPD, including effects on respiratory muscle function, ventilation, work of breathing, exercise tolerance, cardiovascular function, dyspnoea and health status. The purpose of the articles in this issue of the *European Respiratory Review* is to summarise some of this information by addressing the physiology, clinical consequences and several established and novel treatment modalities targeting pulmonary hyperinflation in patients with COPD.

Inflammation is now recognised as a key element in the pathogenesis of COPD. AUGUSTI and SORIANO [1] therefore seek to point out interactive processes that may exist between hyperinflation and inflammation.

Soon after the re-introduction of lung volume reduction surgery to treat COPD, it became clear that a major benefit of the procedure was related to a reduction in static and DH, and an increase in inspiratory capacity. Since then, other surgical and nonsurgical therapies have been tested and found to reduce hyperinflation and its clinical consequences. In some of these studies, hyperinflation as an end-point rather than the intervention itself was novel. Other studies have highlighted new experimental approaches to reversing hyperinflation. Most of these therapeutic modalities are covered in this issue of the Review.

O'DONNELL and LAVENEZIANA [2] discuss how expiratory flow limitation leads to DH, with an emphasis on the mechanisms of air trapping. They have contrasted breathing at rest with breathing during exercise and stress the important role of respiratory rate in the process of DH. They also point to the clinical consequences of DH in COPD, especially dyspnoea and exercise intolerance.

AGUSTI and SORIANO [1] propose that DH could have a pro-inflammatory action in patients with COPD and that the resulting inflammation could lead to structural and mechanical changes in the lung, which further promote DH. Given these interrelationships, AGUSTI and SORIANO [1] speculate that bronchodilators should have anti-inflammatory effects by reducing DH, and that anti-inflammatory agents should have a beneficial effect on DH in patients with COPD.

CALVERLEY [3] addresses the relationship between exercise and dyspnoea, and the critical role of DH in the process. By using a new noninvasive method to continuously assess chest wall dimensions, CALVERLEY [3] has shown that there is a wide variation in the magnitude and timing of DH during and after exercise among patients with COPD, and that the inflation response to bronchodilators is also variable.

MACKLEM [4] focuses on the cardiovascular effects of expiratory flow limitation in exercising healthy subjects breathing through an expiratory flow resistor and in patients with COPD. In both groups, exercise was limited because of dyspnoea and was associated with excessive respiratory muscle recruitment, peripheral blood distribution, hypoxaemia and hypercarbia. DH was not consistently observed. This indicates that DH is just one of several factors that limit exercise in subjects with expiratory airflow limitation.

O'DONNELL and LAVENEZIANA [5] also summarise recent studies, suggesting that bronchodilator-induced symptom relief in COPD may be more related to a decrease in end-expiratory lung volume than improvements in spirometric parameters. In this sense, bronchodilator therapy could be likened to surgical lung volume reduction.

WOUTERS [6] stresses the point that bronchodilators are not the only therapeutic intervention that can attenuate DH in COPD. Any intervention that reduces ventilatory demand, thereby prolonging expiratory time, is expected to decrease end-expiratory lung volume. These interventions include oxygen and pulmonary rehabilitation.

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Finally, POLKEY [7] and, in another paper, POLKEY and HOPKINSON [8] review surgical and bronchoscopic procedures, respectively, that are designed to reduce pulmonary hyperinflation, including DH in patients with COPD. Of special interest are recent data from their institution on the role of endoscopic procedures that collapse diseased areas of the lung and the creation of extrapulmonary pathways, whereby larger bronchi are made to anastomose directly with the lung parenchyma in order to reduce resistance to airflow.

The following articles in this issue of the *European Respiratory Review* reflect presentations made at a GlaxoSmithKline-sponsored COPD Expert Panel discussion, which took place in Dublin (Ireland) on March 11 and 12, 2005. Given the growing awareness of pulmonary hyperinflation as a prognostic factor and therapeutic target in chronic obstructive pulmonary disease, the meeting participants felt that combining the presentations in a symposium format would be of interest to the readers of the *European Respiratory Review*.

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