Still no room to breathe: insights on supine lung mechanics from oscillometry in COPD

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Shareable abstract (@ERSpublications)
A study in *ERJ Open Research* documents the effects of supine posture on lung mechanics in patients with COPD. Specifically, it finds that hyperinflation is related to expiratory flow limitation and increased dynamic elastance. https://bit.ly/4eQG7G7

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Although COPD is characterised by the presence of airflow obstruction during forced expiration, it is the downstream consequences of airflow obstruction that result in many of the symptoms of COPD. In particular, airflow obstruction leads to pulmonary hyperinflation, which in turn reduces inspiratory capacity (IC), increases work of breathing and produces dyspnoea [1]. These problems are compounded when the mechanical load on the respiratory system of the patient with COPD increases as it does during exercise [2] or when patients lie down [3]. In healthy people, lying down results in a slight drop in end-expiratory lung volume (EELV) [4], which is associated with a higher end-expiratory position of the diaphragm [5]. The lower EELV in the face of preserved total lung capacity (TLC) results in an increase in IC. In people with COPD and hyperinflation, lying down results in less of a fall in EELV [4] and the diaphragm remains in a more flattened position at the end of expiration [5], resulting in a reduced ability to increase IC. These events are accompanied by the onset of expiratory flow limitation which can be detected during tidal breathing by the negative expiratory pressure manoeuvre [6] or the within-breath reactance change on oscillometry [7]. In addition, the neural drive to breathe is increased in patients with COPD when supine, but this signalling is relatively ineffective and results in neuromechanical uncoupling, further increasing the sensation of dyspnoea [8]. However, the link between these neuromechanical events and overall respiratory system dynamic elastance, which reflects lung stiffness, has not been previously demonstrated.

The paper by Srinivasan *et al.* [9] in this issue of *ERJ Open Research* addresses this link directly. In an elegantly simple study combining spirometry and oscillometry, the authors demonstrate that persistent hyperinflation occurs in patients with COPD when supine, and this is associated with increased dynamic elastance and tidal expiratory flow limitation. The authors studied 42 participants with moderately severe, stable COPD and 14 healthy controls. Baseline spirometry, body plethysmography and oscillometry were measured while upright, with the latter focusing on respiratory system resistance (R_{rs_5}) and reactance (X_{rs_5}) at 5 Hz during both inspiration and expiration. The patients with COPD had moderate airflow obstruction, with hyperinflation and gas trapping when compared to the healthy control subjects while, as expected, oscillometry demonstrated higher R_{rs_5} and more negative X_{rs_5} values averaged across each breath in those with COPD. The IC and oscillometry measurements were repeated when supine. While IC increased in healthy controls, it did not change significantly in participants with COPD. Meanwhile change in X_{rs_5} was much greater in participants with COPD than in healthy controls, with the mean change when supine exceeding the accepted threshold for defining expiratory flow limitation, which was not the case in the healthy controls. Similarly, total X_{rs_5} became much more negative when supine in the participants with COPD, indicating an increased dynamic elastance or apparent stiffening of the respiratory system. Both expiratory flow limitation and dynamic elastance were independent predictors of persistent hyperinflation when supine, as was body mass index (BMI) (with higher BMI associated with a larger IC/TLC ratio,

indicating less hyperinflation). While the authors rightfully acknowledged various limitations, one major shortcoming of this study is that the authors did not study the effects of longer periods of time supine to mimic what might happen to lung mechanics over the course of a night. However, the findings were straightforward and convincing enough to validate the concept that the inability to increase IC was related to expiratory flow limitation and worsening dynamic elastance.

This study illustrates several important points. First, the availability of oscillometric measurement in a standardised format [10] offers new insights into common clinical problems [11]. Secondly, postural change is one of the commonest mechanical loads that the respiratory system faces in daily life and has markedly different consequences depending on baseline lung mechanics. Although this study shows that the effects on lung mechanics are qualitatively similar in both healthy people and those with COPD, the absolute changes are very different when the system is already stiffened by the presence of static pulmonary hyperinflation. Finally, the findings from this study demonstrate that the inability to increase IC relates not just to expiratory flow limitation, which was to be expected, but also to increased dynamic elastance. Dynamic elastance is the component of respiratory system impedance that reflects energy storage during breathing. As such, it is related to the stiffness of the respiratory system and hence to the size of the lung in communication with the oscillatory pressure signal. The marked drop in X_{rs_5} during expiration indicates that less lung volume is being "seen" by the oscillatory signal, which may be due to either actual or functional airway closure, or due to increased ventilation heterogeneity [12, 13]. The supine posture may result in either process, the former by a drop in functional residual capacity exceeding the closing volume, and the latter by uneven airway closure or narrowing from intraluminal mucus pooling, airway wall oedema, or blood volume redistribution. In all cases, the lung will be functionally stiffer to inflate and the work of breathing will increase, resulting in increased dyspnoea.

So how can we use this information to help our patients with COPD feel less short of breath when they lie down? Any answer to this problem needs to focus on relieving expiratory flow limitation and promoting more even ventilation. Bronchodilators are the mainstay of improving lung emptying and can abolish airflow limitation in some patients with COPD, which should also improve inhomogeneity of ventilation [14]. Positive airway pressure at night has been shown to mitigate expiratory flow limitation in patients with COPD [7]. Perhaps a clue to another possible therapy offered by this study is the association of BMI with persistent hyperinflation. Patients with COPD who are obese have preserved IC and exercise tolerance compared to their lean counterparts [15]. This appears to be due to enhanced lung elastic recoil and a more advantageous operating length of the diaphragm due to a lower EELV. How might we mimic these effects of obesity in nonobese individuals? Abdominal binders have been shown to enhance neuromuscular efficiency in patients with COPD during exercise [16]. Perhaps an abdominal binder might help people with COPD get a good night's rest when they lie down. Certainly, the tools now exist to show if this is the case in a simple and noninvasive fashion.

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References

1 Thomas M, Decramer M, O'Donnell DE. No room to breathe: the importance of lung hyperinflation in COPD. *Prim Care Respir J* 2013; 22: 101–111.
2 O'Donnell DE, Elbehairy AF, Webb KA, *et al.* The link between reduced inspiratory capacity and exercise intolerance in chronic obstructive pulmonary disease. *Ann Am Thorac Soc* 2017; 14: S30–S39.
3 Eltayara L, Ghezzo H, Milic-Emili J. Orthopnea and tidal expiratory flow limitation in patients with stable COPD. *Chest* 2001; 119: 99–104.
4 Elbehairy AF, Faisal A, McIsaac H, *et al.* Mechanisms of orthopnoea in patients with advanced COPD. *Eur Respir J* 2021; 57: 2000754.
5 Hellebrandová L, Chlumský J, Vostatek P, *et al.* Airflow limitation is accompanied by diaphragm dysfunction. *Physiol Res* 2016; 65: 469–479.
6 Uccelli S, Pini L, Bottone D, *et al.* Dyspnea during night-time and at early morning in patients with stable COPD is associated with supine tidal expiratory flow limitation. *Int J Chron Obstruct Pulmon Dis* 2020; 15: 2549–2558.
7 McKenzie J, Nisha P, Cannon-Bailey S, *et al.* Overnight variation in tidal expiratory flow limitation in COPD patients and its correction: an observational study. *Respir Res* 2021; 22: 319.

8 Domnik NJ, Phillips DB, James MD, *et al.* Compensatory responses to increased mechanical abnormalities in COPD during sleep. *Eur J Appl Physiol* 2022; 122: 663–676.
9 Srinivasan M, Pollard H, Chapman DG, *et al.* The effect of expiratory flow limitation on supine persistent hyperinflation in COPD: a prospective observational study. *ERJ Open Res* 2024; 10: 00255-2024.
10 King GG, Bates J, Berger KI, *et al.* Technical standards for respiratory oscillometry. *Eur Respir J* 2020; 55: 1900753.
11 Kaminsky DA, Simpson SJ, Berger KI, *et al.* Clinical significance and applications of oscillometry. *Eur Respir Rev* 2022; 31: 210208.
12 Milne S, Jetmalani K, Chapman DG, *et al.* Respiratory system reactance reflects communicating lung volume in chronic obstructive pulmonary disease. *J Appl Physiol* 2019; 126: 1223–1231.
13 Young HM, Guo F, Eddy RL, *et al.* Oscillometry and pulmonary MRI measurements of ventilation heterogeneity in obstructive lung disease: relationship to quality of life and disease control. *J Appl Physiol (1985)* 2018; 125: 73–85.
14 Dellacà RL, Pompilio PP, Walker PP, *et al.* Effect of bronchodilation on expiratory flow limitation and resting lung mechanics in COPD. *Eur Respir J* 2009; 33: 1329–1337.
15 Ora J, Laveneziana P, Wadell K, *et al.* Effect of obesity on respiratory mechanics during rest and exercise in COPD. *J Appl Physiol* 2011; 111: 10–19.
16 Abdallah SJ, Smith BM, Wilkinson-Maitland C, *et al.* Effect of abdominal binding on diaphragmatic neuromuscular efficiency, exertional breathlessness, and exercise endurance in chronic obstructive pulmonary disease. *Front Physiol* 2018; 9: 1618.