EDITORIAL

"Intrinsic" PEEP (PEEPi): role of expiratory muscles

V. Ninane

In normal subjects breathing at rest, lung volume at end-expiration corresponds with the elastic equilibrium volume, or relaxation volume (Vr), of the respiratory system. If expiratory duration is not long enough, then inspiration may begin before the system has returned to Vr, such that end-expiratory lung volume is higher than Vr. This condition is named dynamic pulmonary hyperinflation and "intrinsic" positive end-expiratory pressure (PEEPi), also called auto PEEP, corresponds to the elastic recoil of the lung at end-expiration [1, 2].

In the spontaneously breathing subject, this dynamic hyperinflation occurs mainly when the rate of lung emptying is decreased owing to high respiratory resistance or flow limitation. The highest degrees of dynamic hyperinflation are, thus, encountered in patients with airway diseases, particularly chronic obstructive pulmonary disease (COPD) patients. In the ventilated patient, external determinants may also induce dynamic pulmonary hyperinflation, or increase the magnitude of dynamic hyperinflation that was already present before the onset of mechanical ventilation. These additional external determinants include a preceding inflation volume that is too large, a time available for expiration that is reduced or a decreased rate of lung emptying owing to high resistance associated with the tube, the ventilator tubing or devices [3].

Dynamic pulmonary hyperinflation has important pathophysiological and therapeutic implications mainly in critical care medicine [3]. In particular, it implies that as the inspiratory muscles start to contract they must first offset the elastic recoil pressure of the system before inspiratory flow can begin and lung volume can increase. PEEPi associated with dynamic pulmonary hyperinflation thus corresponds to an inspiratory threshold load and is associated with an increased inspiratory work of breathing. This has led several investigators to give external PEEP or continuous positive airway pressure (CPAP) in the belief that this would unload the inspiratory muscles and, therefore, reduce the inspiratory work of breathing [4–6].

It must be stressed, however, that PEEPi and dynamic pulmonary hyperinflation are not always linked. In fact, alveolar pressure can also remain positive throughout expiration when the expiratory muscles contract [1, 5]. With respect to this, recent studies have shown that in many patients with stable but severe COPD [7] as well as in many mechanically ventilated patients [8, 9], expiration is a mechanically active process. These observations, thus, provide strong evidence that the active nature of expiration is an important determinant of PEEPi in

such patients and, therefore, that the importance of dynamic pulmonary hyperinflation has been overestimated. Since a positive end-expiratory alveolar pressure resulting from an expiratory muscle contraction does not represent an inspiratory threshold load, the level of external PEEP that is needed in such patients must be significantly less than conventionally thought. The contribution of expiratory muscle contraction to the increase in end-expiratory alveolar pressure has to be taken into account when applying external PEEP or CPAP to offset PEEPi or these procedures might, in fact, result in enhanced pulmonary dynamic hyperinflation with further deleterious consequences [4, 10].

In this issue of the European Respiratory Journal, ZAKYNTHINOS and co-workers [11] report on a method for detecting expiratory muscle use and quantifying its contribution to PEEPi. The two points at issue in this and other studies [7–9] are:

1) How to detect expiratory muscle use? The four different muscles of the anterolateral wall of the abdomen (*i.e.* the external and internal oblique, the rectus abdominis and the transversus abdominis muscles) are conventionally thought to be the main expiratory muscles. It must be stressed, however, that they do not operate as a unit during breathing [12]. This is well illustrated by the observation that the contraction of the abdominal muscles that occurs during expiration in many stable patients with severe chronic airflow obstruction is usually confined to the deepest abdominal muscle layer, namely the transversus abdominis muscle [13]. In contrast, during resting breathing, these patients generally do not contract the muscles of the superficial abdominal layer (namely, the external oblique and rectus abdominis) which are clearly not the most prominent abdominal expiratory muscles [12]. Therefore, when assessing abdominal muscle use with electromyography (EMG), the activity of the transversus rather than the external oblique or the rectus abdominis should be recorded. Surface electrodes record essentially from the superficial abdominal layers, and these technical reasons probably explain why previous studies [14–18] using surface electrodes have reported that COPD patients in general do not show any abdominal muscle EMG activity. Therefore, to avoid underestimation of abdominal muscle use with EMG studies, concentric needle electrodes should be used instead, and implanted selectively in the transversus abdominis with the aid of a high-resolution ultrasound scan [12, 13]. For the same reasons, abdominal wall palpation that explores mainly the superficial abdominal layers may also underestimate active contraction of the abdominal muscles. Indeed, palpation failed to reveal expiratory use of abdominal muscles during resting breathing in 14 out

Correspondence: V. Ninane, Chest Service, Saint-Pierre University Hospital, Rue Haute 322, Brussels 1000, Belgium.

of 17 stable patients with severe COPD who showed EMG evidence of continuous phasic expiratory contraction of the deeply located transversus abdominis [12].

Apart from invasive EMG studies using needle electrodes, expiratory use of abdominal muscles may also be inferred from measurements of abdominal pressure and chest wall motion [19–21]. Indeed, when the abdominal muscles contract, they pull the abdominal wall inward and increase abdominal (gastric) pressure. The presence of this characteristic volume-pressure relationship of the abdomen during expiration has been shown to be a good indicator of the presence or absence of significant expiratory activity in the transversus abdominis [7]. This approach, however, requires the positioning of a gastric balloon as well as assessment of the respiratory changes in abdominal dimensions by means of magnetometry [7] or inductive plethysmography [8].

2) How to correct PEEPi for expiratory muscle contraction? During controlled mechanical ventilation, respiratory muscles, including expiratory muscles, are presumably at rest and PEEPi then corresponds mainly to dynamic pulmonary hyperinflation. In addition, there is no indication for external PEEP in this condition since there is no need for inspiratory muscle unloading. In contrast, expiratory muscle activity may occur and contribute to PEEPi in conditions of spontaneous breathing efforts, such as during spontaneous breathing in COPD patients [7] or assisted ventilation [8, 9]. Firstly, it is important to stress that even COPD patients in a stable condition are generally unable to voluntarily diminish the expiratory recruitment of the abdominal muscles when this recruitment is present [7]. This implies that the only way to suppress this activity is to administer sedative or paralyzing medication. For obvious reasons, alternative approaches have been developed to assess the relative contributions of expiratory muscle contraction and dynamic pulmonary hyperinflation to the positive end-expiratory alveolar pressure.

The increase in abdominal pressure associated with abdominal muscle contraction causes the diaphragm to move cranially into the thoracic cavity, and this displacement, in turn, results in a rise in pleural pressure (P_{pl}). On this basis, it seems logical to correct PEEPi for expiratory muscle contraction by subtracting from the initial measurement the gastric pressure (P_{ga}) rise associated with abdominal muscle contraction (*i.e.* associated with a decrease in abdominal dimensions). This reasoning is however based on the assumption that the diaphragm is relaxed and the increase in P_{ga} is transmitted into an equal increase in P_{pl}. If this assumption is correct, the increase in P_{ga} is probably also a good estimate of the overall mechanical effect of expiratory muscles. Indeed, it is likely that patients with abdominal expiratory activity contract additional expiratory muscles, in particular rib cage expiratory muscles such as the triangularis sterni and the internal interosseous intercostals [22]. These muscles may then accentuate the rise in alveolar pressure caused by the contraction of the abdominal muscles. Provided this rise in pressure is well transmitted to the abdominal compartment, one may assume that the rise in P_{ga} is a good estimate of overall expiratory muscle effect.

ZAKYNTHINOS and co-workers [11], in this issue of the Journal, have used the increase in P_{ga} observed between the end-inspiratory and peak end-expiratory values, or the total decrease in P_{ga} at end-expiration, to correct the initial PEEPi, measured in static conditions (PEEPi,stat) [1], and during end-expiratory airway occlusion. It must be stressed, however, that these measurements do not always reflect expiratory muscle effect with accuracy because P_{ga} tracing is also affected by the contraction of the inspiratory muscles. Indeed, the descent of the dome of the diaphragm during inspiration causes an increase in abdominal (gastric) pressure. At the end of inspiration, P_{ga} drops rapidly as the diaphragm relaxes and starts to rise again while the abdominal muscles become active. If the time available at end-inspiration for P_{ga} to return to its relaxation value is insufficient, then the mechanical effect of the abdominal muscles might be underestimated. The same phenomenon can occur at the onset of inspiration if the descent of the diaphragm causes P_{ga} to increase before this pressure has returned to its relaxation value after the derecruitment of the abdominal muscles. Figure 1, in the paper from ZAKYNTHINOS and co-workers [11] in this issue of the Journal illustrates one example in which such an obvious underestimation of the mechanical effect of the expiratory muscles occurred.

A different situation may arise in some patients with acute diaphragm dysfunction and excessive recruitment of the rib cage inspiratory muscles. In this condition, P_{ga} may in fact decrease rather than increase during inspiration [23] with the consequence that the measurement of the overall P_{ga} rise during expiration may, in fact, lead to overestimation of the mechanical effects of expiratory muscle contraction. Such paradoxical movements of the abdomen during breathing have been described during weaning from mechanical ventilation [24]. One major limitation to the measurement of expiratory muscle effect by the amount of P_{ga} rise during expiration is, thus, related to the fact that the relaxation value of P_{ga} may be required and this measurement can only be obtained if expiratory muscles are put at rest.

In addition, interpretation of the corrected PEEPi has to be even more cautious if the initial PEEPi is determined dynamically (PEEPi,dyn) as the fall in pleural pressure before the inspiratory flow starts [6, 7, 25, 26]. In fact, PEEPi,dyn is not only influenced by the (static) elastic recoil of the respiratory system but also by varying (dynamic) factors, including inequality of the time constant of different lung units, such that PEEPi,dyn measurement shows values that are different from the values of PEEPi,stat [27, 28]. It has been suggested to correct PEEPi,dyn for expiratory muscle contraction by subtracting from its value either the increase in P_{ga} during expiration [8], with the limitations discussed earlier, or the fall in P_{ga} that occurs during the time interval (from the onset of inspiratory effort to the onset of inspiratory flow) in which PEEPi,dyn was measured [9]. These two approaches clearly differ. Due to the fact that P_{ga}, at end-expiration, may decrease before the beginning of the inspiratory effort and may go on decreasing after the onset of the inspiratory flow, the P_{ga} values obtained by the latter method are lower than the results obtained with the former approach. Further studies are, therefore, needed to validate and compare these methods in order to correct PEEPi,dyn accurately for expiratory muscle use.

In summary, assessment of expiratory muscle use and of its mechanical effect in order to correct PEEPi and titrate external PEEP with accuracy requires the use of respiratory inductive plethysmography (or magnetometry) and the positioning of a gastric balloon. This approach is invasive and still has potential limitations. These factors will undoubtedly limit its widespread clinical application and, in the absence of a simple measurement of the degree of dynamic hyperinflation in the actively breathing patient, some controversy will remain as to the best level of PEEP or CPAP that should be added in conditions such as assisted ventilation or weaning [3].

Whatever the best strategy may be, a good understanding of the underlying mechanisms and pathophysiological consequences of pulmonary dynamic hyperinflation is needed. With regard to this, it is important to recall that, although external PEEP or CPAP may help to reduce the work of breathing associated with PEEPi, this is true only in patients with airflow limitation. When pulmonary dynamic hyperinflation is linked to other determinants [3], application of an external positive pressure may cause a further increase in hyperinflation and in its deleterious consequences [4, 10]. Finally, it is also important to stress that application of external PEEP does nothing to reduce the degree of pulmonary dynamic hyperinflation since it does not relieve its underlying mechanisms. We must then also pay special attention to therapeutic measures which may effectively diminish dynamic hyperinflation, including use of bronchodilator therapy in patients with airflow diseases, changes in the ventilator setting and the use of a large bore endotracheal tube [3].

References

1. Pepe PE, Marini JJ. Occult positive end-expiratory pressure in mechanically ventilated patients with airflow obstruction. *Am Rev Respir Dis* 1982; 126: 166–170.
2. Rossi A, Gottfried SB, Zocchi L, *et al*. Measurement of static compliance of the total respiratory system in patients with acute respiratory failure during mechanical ventilation: the effect of "intrinsic" PEEP. *Am Rev Respir Dis* 1985; 131: 672–677.
3. Rossi A, Polese G, Brandi G, Conti G. Intrinsic positive end-expiratory pressure (PEEPi). *Intensive Care Med* 1995; 21: 522–536.
4. Smith TC, Marini JJ. Impact of PEEP on lung mechanics and work of breathing in severe airflow obstruction. *J Appl Physiol* 1988; 65: 1488–1499.
5. Marini JJ. Should PEEP be used in airflow obstruction? *Am Rev Respir Dis* 1989; 140: 1–3.
6. Petrof BJ, Legaré M, Goldberg P, Milic-Emili J, Gottfried SB. Continuous positive airway pressure reduces work of breathing and dyspnea during weaning from mechanical ventilation in severe chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1990; 141: 281–289.
7. Ninane V, Yernault JC, De Troyer A. Intrinsic PEEP in patients with chronic obstructive pulmonary disease. Role of expiratory muscles. *Am Rev Respir Dis* 1993; 148: 1037–1042.
8. Lessard MR, Lofaso F, Brochard L. Expiratory muscle activity increases intrinsic positive end-expiratory pressure independently of dynamic hyperinflation in mechanically ventilated patients. *Am J Respir Crit Care Med* 1995 151: 562–569.
9. Appendini L, Patessio A, Zanoboni S, *et al*. Physiologic effects of positive end-expiratory pressure and mask pressure support during exacerbations of chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 1994; 149: 1069–1076.
10. Tuxen DV. Detrimental effects of positive end-expiratory pressure during controlled mechanical ventilation of patients with severe airflow obstruction. *Am Rev Respir Dis* 1989; 140: 5–9.
11. Zakynthinos SG, Vassilakopoulos T, Zakynthinos E, Roussos C. Accurate measurement of intrinsic positive end-expiratory pressure: how to detect and correct for expiratory muscle activity. *Eur Respir J* 1997; 10: 522–529.
12. De Troyer A, Estenne M, Ninane V, Van Gansbeke D, Gorini M. Transversus abdominis muscle function in humans. *J Appl Physiol* 1990; 68: 1010–1016.
13. Ninane V, Rypens F, Yernault JC, De Troyer A. Abdominal muscle use during breathing in patients with chronic airflow obstruction. *Am Rev Respir Dis* 1992; 146: 16–21.
14. Campbell EJM, Friend J. Action of breathing exercises in pulmonary emphysema. *Lancet* 1955; 1: 325–329.
15. Gronbaek P, Skouby AP. The activity pattern of the diaphragm and some muscles of the neck and trunk in chronic asthmatics and normal controls. *Acta Med Scand* 1960; 168: 413–425.
16. Skarvan K. The ventilatory function of abdominal muscles in normal subjects and in patients with chronic obstructive lung disease. *Respiration* 1971; 28: 347–359.
17. Sharp JT, Druz WS, Moisan T, Foster J, Machnach W. Postural relief of dyspnea in severe chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1980; 122: 201–211.
18. Morris MJ, Madgwick RG, Frew AJ, Lane DJ. Breathing muscle activity during expiration in patients with chronic airflow obstruction. *Eur Respir J* 1990; 3: 901–909.
19. Dodd DS, Brancatisano T, Engel LA. Chest wall mechanics during exercise in patients with severe chronic airflow obstruction. *Am Rev Respir Dis* 1984; 129: 33–38.
20. Gilmartin JJ, Gibson GJ. Mechanisms of paradoxical rib cage motion in patients with chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1986; 134: 683–687.
21. Martinez FJ, Couser JI, Celli BR. Factors influencing ventilatory muscle recruitment in patients with chronic airflow obstruction. *Am Rev Respir Dis* 1990; 142: 276–282.
22. De Troyer A, Loring SH. Actions of respiratory muscles. *In*: Roussos C, ed. The Thorax. Part A: Physiology. Vol. 85. New York, Marcel Dekker, 1995; pp. 535–563.
23. Sharp JT, Goldberg NB, Druz WS, Fishman HC, Danon J. Thoracoabdominal motion in chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1977; 115: 47–56.
24. Cohen C, Zagelbaum G, Gross D, Roussos C, Macklem PT. Clinical manifestations of inspiratory muscle fatigue. *Am J Med* 1982; 73: 308–316.
25. Dal Vecchio L, Polese G, Poggi R, Rossi A. "Intrinsic" positive end-expiratory pressure in stable patients with chronic obstructive pulmonary disease. *Eur Respir J* 1990; 3: 74–80.
26. Haluszka J, Chartrand DA, Grassino AE, Milic-Emili J. Intrinsic PEEP and arterial P_{CO_2} in stable patients with chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1990; 141: 1194–1197.
27. Maltais F, Reissmann H, Navalesi P, *et al*. Comparison of static and dynamic measurements of intrinsic PEEP in mechanically ventilated patients. A*m J Respir Crit Care Med* 1994; 150: 1318–1324.
28. Yan S, Kayser B, Tobiasz M, Sliwinski P. Comparison of static and dynamic intrinsic positive end-expiratory pressure using the Campbell diagram. *Am J Respir Crit Care Med* 1996; 154: 938–944.