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PHYSIOLOGICAL NOTE

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Work of breathing

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Introduction

The main goal of mechanical ventilation is to help restore gas exchange and reduce the work of breathing (WOB) by assisting respiratory muscle activity. Knowing the determinants of WOB is essential for the effective use of mechanical ventilation and also to assess patient readiness for weaning. The active contraction of the respiratory muscles causes the thoracic compartment to expand, inducing pleural pressure to decrease. This negative pressure generated by the respiratory pump normally produces lung expansion and a decrease in alveolar pressure, causing air to flow into the lung. This driving pressure can be generated in three ways: entirely by the ventilator, as positive airway pressure during passive inflation and controlled mechanical ventilation; entirely by the patient's respiratory muscles during spontaneous unassisted breathing; or as a combination of the two, as in assisted mechanical ventilation. For positive-pressure ventilation to reduce WOB, there needs to be synchronous and smooth interaction between the ventilator and the respiratory muscles [1, 2, 3]. This note will concentrate on how to calculate the part of WOB generated by the patient's respiratory muscles, especially during assisted ventilation.

Esophageal pressure and the Campbell diagram

Measuring WOB is a useful approach to calculate the total expenditure of energy developed by the respiratory muscles [4]. In general, the work performed during each respiratory cycle is mathematically expressed as $\mathrm{WOB} = \int \mathrm{Pressure} \times \mathrm{Volume}$, i.e. the area on a pressure–volume diagram. Esophageal pressure, which is easily measured, is usually taken as a surrogate for intrathoracic (pleural) pressure. The dynamic relation between pleural pressure and lung volume during breathing is referred to as the Campbell diagram [5] (Fig. 1). Esophageal pressure swings during inspiration are needed to overcome two forces: the elastic forces of the lung parenchyma and chest wall, and the resistive forces generated by the movement of gas through the airways. One can calculate these two components (elastic and resistive) by comparing the difference between esophageal pressure during the patient's effort during the breath and the pressure value in passive conditions, represented by the static volume–pressure curve of the relaxed chest wall. This passive volume–pressure curve is a crucial component of the Campbell diagram. It is calculated from the values of esophageal pressure obtained over lung volume when the airways are closed and the muscles are completely relaxed. Unfortunately, as this is difficult to do (because it requires passive inflation and often muscle paralysis), a theoretical value for the slope of this curve is frequently used. However, if a patient is passively ventilated and an esophageal balloon is placed, a true value for the volume–pressure relationship of the chest wall during passive tidal breathing can be obtained [6]. This passive pressure–volume relationship can be used as a reference value for subsequent calculations when the patient develops spontaneous inspiratory efforts.

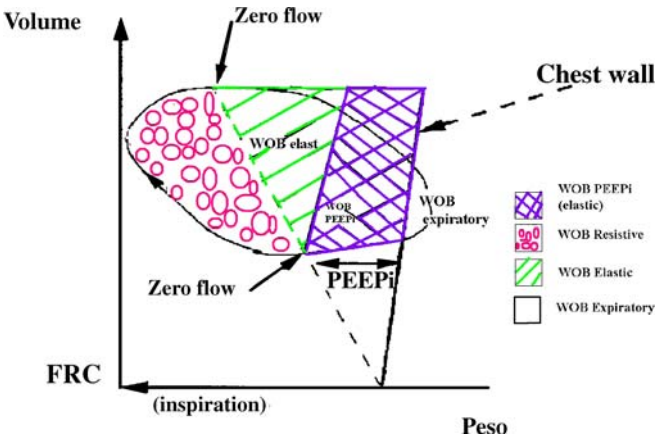

Fig. 1 Campbell's diagram. Work of breathing measured by the esophageal pressure: resistive WOB (W_{resist}), elastic WOB (W_{elast}), WOB related to active expiration (*WOB expiratory*) and WOB related to intrinsic PEEP (W_{PEEPi}). *Chest wall*: this *thick line* (the chest wall compliance) represents the pleural (esophageal) pressure obtained when muscles are totally relaxed and lung volume increases above functional residual capacity, measured in static conditions

The WOB is normally expressed in joules. One joule is the energy needed to move 1 l of gas through a 10-cmH_2O pressure gradient. The work per liter of ventilation (J/l) is the work per cycle divided by the tidal volume (expressed in liters). In a healthy subject the normal value is around 0.35 J/l [7]. Lastly, WOB can be expressed in work per unit of time, multiplying joules per cycle by the respiratory rate (expressed in breaths per minute) to obtain the power of breathing (joules/minute). In a healthy subject the normal value is around 2.4 J/min [7]. As illustrated by the Campbell diagram, two other phenomena affect the WOB: intrinsic PEEP (positive end-expiratory pressure, or PEEPi) and active expiration.

PEEPi and active expiration

The distending pressure of the lungs is called the transpulmonary pressure and it can be estimated as the difference between airway and esophageal (pleural) pressure. At the end of a normal expiration, alveolar and airway pressures are zero relative to atmosphere, and esophageal pressure is negative, reflecting the resting transpulmonary pressure (around 5 cmH_2O in normal conditions). However, in the presence of PEEPi, the alveolar pressure remains positive throughout expiration, because of either dynamic airway collapse or inadequate time to exhale [8]. This implies that some degree of dynamic hyperinflation does exist (lung volume at end-expiration is higher than passive functional residual capacity). Importantly, for lung volume to further increase in a patient with PEEPi, the inspiratory muscles contract to an amount equal to PEEPi before any volume is displaced.

PEEPi can be quite high in patients with chronic obstructive pulmonary disease (COPD) and may represent a high proportion of the total WOB [9]. For example, a patient who displaces 0.5 l of tidal volume through a 7-cmH_2O pressure gradient will perform an amount of work of 0.35 J/cycle. If nothing else changes except that this patient develops 5 cmH_2O of PEEPi, 0.25 J will be required to counterbalance this, meaning that the total WOB will be 0.60 J (0.35 + 0.25), which represents around 40% of the total work required for the inspiration. The PEEPi value is measured as the drop in esophageal pressure occurring during expiration when the inspiratory muscles start contraction, until the flow reaches the point of zero (see Fig. 1).

In the case of ineffective respiratory efforts, that is, muscle contraction without volume displacement, WOB cannot be measured from the Campbell diagram, since this calculation is based on volume displacement. In this situation, measurement of the pressure–time product (PTP) may more accurately reflect the energy expenditure of these muscles. The PTP is the product of the pressure developed by the respiratory muscles multiplied by the time of muscle contraction, expressed in cmH_2O per second. The relevant pressure is again the difference between the measured esophageal pressure and the static relaxation curve of the chest wall.

Expiration normally occurs passively. However, the coexistence of PEEPi and active expiration is common, especially in COPD patients [10]. Positive expiratory swings in gastric pressure are observed during active expiration as a consequence of abdominal muscle recruitment. When the patient starts contracting the inspiratory muscles, the expiratory muscles also start to relax. The drop in esophageal pressure used to estimate PEEPi is therefore also due to the relaxation of the expiratory muscles. To avoid overestimating the value of PEEPi, the abdominal pressure swing resulting from the active expiration must thus be subtracted from the initial drop in esophageal pressure [10].

Technical aspects of WOB calculation

Two other calculations can be obtained from pressure and volume measurements: airway pressure WOB and transpulmonary pressure WOB. The airway pressure WOB displays the energy dissipated by the ventilator to inflate the respiratory system. The transpulmonary pressure WOB shows the energy needed to inflate the lung parenchyma and reflects the mechanical characteristics of the pulmonary tissue. The limitation of these two measurements is that the amount of WOB performed by the patient's respiratory muscles is ignored.

The main tools used to measure the WOB are a double-lumen polyethylene gastro-esophageal catheter–balloon system and a pneumotachygraph. The catheter has an esophageal and a gastric balloon, usually filled with

0.5 and 1 ml of air to measure the esophageal and gastric pressures, respectively. Correct positioning of the esophageal balloon is assessed by an occlusion test: when the airways are closed at the end of expiration and an active inspiration occurs, a drop in esophageal pressure occurs. In this scenario, there are no changes in lung volume and the decrease in esophageal pressure equals the decrease in airway pressure (because in the absence of volume displacement, the transpulmonary pressure has to be nil) [11]. The catheter–balloon system should be placed to obtain a ratio between airway pressure and esophageal pressure changes as close as possible to 1. Also, the correct positioning of the gastric balloon needs to be checked [12].

Limitations

The calculation of WOB has several limitations. The first is that it requires insertion of a double-balloon gastro-esophageal catheter system. The second is the validity of the esophageal pressure value. Since pleural pressure is influenced by gravity, it can be modified by the weight of the thoracic content and by the posture. In the supine position, end-expiratory esophageal pressure is usually positive because of the weight of the heart and mediastinum on the esophagus. However, the amplitude of the changes in esophageal pressure is not usually affected. The third limitation is that the theoretical value for chest wall compliance is often used rather than a true measured value. Furthermore, chest wall deformation can occur if levels of ventilation are high [13]. Lastly, it is difficult to determine what the optimal WOB level should be for each patient on clinical grounds.

Conclusion

From the standpoint of clinical research, the measurement of WOB is extremely useful in the field of mechanical ventilation, having contributed to important progress in the management of patients for optimizing and understanding the effects of ventilator settings such as trigger, external PEEP, peak inspiratory flow, etc. WOB has also been used to evaluate the physiological effects of a number of agents such as helium and bronchodilators [9, 14, 15, 16, 17, 18, 19]. Studies on WOB have given us greater insight into the pathophysiology of weaning failure [3] and have also contributed to the progress made in the field of non-invasive mechanical ventilation [20, 21]. Bedside measurements of WOB in clinical practice, however, should be reserved for individuals in whom assessment of this parameter can provide further insight into the patient ability to breath and the patient–ventilator interactions.

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References

1. Nava S, Bruschi C, Rubini F, Palo A, Iotti G, Braschi A (1995) Respiratory response and inspiratory effort during pressure support ventilation in COPD patients. Intensive Care Med 21:871–879
2. Leung P, Jubran A, Tobin M (1997) Comparison of assisted ventilator modes on triggering, patient effort and dyspnea. Am J Respir Crit Care Med 155:1940–1948
3. Jubran A, Tobin MJ (1997) Pathophysiologic basis of acute respiratory distress in patients who fail a trial of weaning from mechanical ventilation. Am J Respir Crit Care Med 155:906–915
4. Roussos C (1985) Structure and function of the thorax: energetics. In: Roussos C, Macklem PT (eds) The thorax. Dekker, New York, pp 437–492
5. Mead J, Loring SJ (1985) Volume displacements of the chest wall and their mechanical significance. In: Roussos C, Macklem PT (eds) The thorax: part A. Dekker, New York, pp 369–392
6. Jubran A, Van de Graaff WB, Tobin MJ (1995) Variability of patient-ventilator interaction with pressure support ventilation in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 152:129–136
7. Mancebo J, Isabey D, Lorino H, Lofaso F, Lemaire F, Brochard L (1995) Comparative effects of pressure support ventilation and intermittent positive pressure breathing (IPPB) in non intubated healthy subjects. Eur Respir J 8:1901–1909
8. Brochard L (2002) Intrinsic (or auto-) PEEP during controlled mechanical ventilation. Intensive Care Med 28:1376–1378
9. Brochard L, Harf A, Lorino H, Lemaire F (1989) Inspiratory pressure support prevents diaphragmatic fatigue during weaning from mechanical ventilation. Am Rev Respir Dis 139:513–521
10. Lessard MR, Lofaso F, Brochard L (1995) Expiratory muscle activity increases intrinsic positive end-expiratory pressure independently of dynamic hyperinflation in mechanically ventilated patients. Am J Respir Crit Care Med 151:562–569
11. Baydur A, Behrakis PK, Zin WA, Jaeger MJ, Milic-Emili J (1982) A simple method for assessing the validity of the esophageal balloon technique. Am Rev Respir Dis 126:788–791
12. Diehl JL, Lofaso F, Deleuze P, Similowski T, Lemaire F, Brochard L (1994) Clinically relevant diaphragmatic dysfunction after cardiac operations. J Thoracic Cardiovasc Surg 107:487–498
13. Fleury B, Murciano D, Talamo C, Aubier M, Pariente R, Milic Emili J (1985) Work of breathing in patients with chronic obstructive pulmonary disease in acute respiratory failure. Am Rev Respir Dis 131:822–827

14. Jaber S, Fodil R, Carlucci A, Boussarsar M, Pigeot J, Lemaire F, Harf A, Lofaso F, Isabey D, Brochard L (2000) Noninvasive ventilation with helium–oxygen in acute exacerbations of chronic obstructive pulmonary disease. Am J Respir Crit Care Med 161:1191–1200
15. Tassaux D, Gainnier M, Battisti A, Jolliet P (2005) Helium–oxygen decreases inspiratory effort and work of breathing during pressure support in intubated patients with chronic obstructive pulmonary disease. Intensive Care Med 31:1501–1507
16. Mancebo J, Amaro P, Lorino H, Lemaire F, Harf A, Brochard L (1991) Effects of albuterol inhalation on the work of breathing during weaning from mechanical ventilation. Am Rev Respir Dis 144:95–100
17. Mancebo J, Albaladejo P, Touchard D, Bak E, Subirana M, Lemaire F, Harf A, Brochard L (2000) Airway occlusion pressure to titrate positive end-expiratory pressure in patients with dynamic hyperinflation. Anesthesiology 93:81–90
18. Cinnella G, Conti G, Lofaso F, Lorino H, Harf A, Lemaire F, Brochard L (1996) Effects of assisted ventilation on the work of breathing: volume-controlled versus pressure-controlled ventilation. Am J Respir Crit Care Med 153:1025–1033
19. Aslanian P, El Atrous S, Isabey D, Valente E, Corsi D, Harf A, Lemaire F, Brochard L (1998) Effects of flow triggering on breathing effort during partial ventilatory support. Am J Respir Crit Care Med 157:135–143
20. Lellouche F, Maggiore SM, Deye N, Taille S, Pigeot J, Harf A, Brochard L (2002) Effect of the humidification device on the work of breathing during noninvasive ventilation. Intensive Care Med 28:1582–1589
21. L'Her E, Deye N, Lellouche F, Taille S, Demoule A, Fraticelli A, Mancebo J, Brochard L (2005) Physiologic effects of noninvasive ventilation during acute lung injury. Am J Respir Crit Care Med 172:1112–1118