Intensive Care Med (2008) 34:1336–1339
DOI 10.1007/s00134-008-1095-7

Theodoros Vassilakopoulos

Understanding wasted/ineffective efforts in mechanically ventilated COPD patients using the Campbell diagram

Received: 15 January 2008
Accepted: 29 February 2008
Published online: 4 April 2008

Electronic supplementary material
The online version of this article (doi:10.1007/s00134-008-1095-7) contains supplementary material, which is available to authorized users.

This physiological note is accompanied by Electronic Supplementary Material containing a Powerpoint presentation. This is an independent Powerpoint presentation that has a structured animation of ventilator waveforms exhibiting wasted efforts and the use of the Campbell diagram to understand wasted efforts in COPD. It is thus accompanied by notes beneath each slide so that the reader can go through the presentation without referring to the text.

T. Vassilakopoulos (✉)
University of Athens Medical School, Evangelismos Hospital, Department of Critical Care and Pulmonary Services, 45–47 Ipsilandou Street, 10675 Athens, Greece
e-mail: tvassil@med.uoa.gr
Tel.: +30-210-7253915
Fax: +30-210-7216503

Introduction

Wasted or ineffective efforts are inspiratory efforts that fail to trigger the ventilator [1]. Nearly 25% of mechanically ventilated patients exhibit ineffective efforts which are even more frequent in COPD patients [2]. The pathophysiology of wasted efforts can be illustratively presented using the Campbell diagram.

Campbell diagram

The Campbell diagram is constructed by plotting the dynamic relation between pleural pressure (measured with an esophageal balloon) and lung volume during breathing in relation to the passive pressure-volume curves of the lung Pel(L) and the chest wall Pel(cw) [3]. The Pel(cw) is constructed by connecting the values taken by the esophageal pressure during passive inflation (i. e., with no respiratory muscle activity) at different lung volumes; thus, any change in esophageal pressure is referred to this line in the Campbell diagram in order to calculate the true muscular pressure developed by the patient.

In normal subjects inspiration starts from the relaxation volume of the respiratory system (Vr), where the Pel(L) and Pel(cw) intersect (i. e., where the tendency of the lung to recoil inward is equal to the tendency of the chest wall to expand; Fig. 1a). Inspiratory muscle action results in pressure development (Pinsp) on the left of the Pel(cw). Inspiratory flow, and thus increases in volume (V_L), take place on the left of the Pel(L) and coincide with the beginning of inspiratory muscle action. At any volume, the horizontal distance between the Pel(cw) and Pel(L) represents the portion of inspiratory muscle action devoted to expanding the lung at this volume with open airways and the portion on the left of the Pel(L) represents the pressure dissipated to generate airflow. Inspiration ends on the Pel(L) (point of zero flow) and the inspiratory muscles relax [so that pressure returns on the Pel(cw)]. Expiration is usually passive, and the respiratory system returns to its relaxation volume on the Pel(cw); however, in patients with respiratory distress, such as mechanically ventilated COPD patients, expiration is frequently active. In the case of active expiration, pressure develops on the right of the Pel(cw) due to activity of expiratory muscles (Pexp). This returns volume back to the relaxation volume of the respiratory system.

Why are COPD patients prone to develop wasted/ineffective efforts?

In COPD patients with dynamic hyperinflation, inspiration starts from an increased end-expiratory lung volume (Fig. 1b). Inspiratory muscle action has to overcome the intrinsic positive end expiratory pressure [PEEPi, horizontal distance between the Pel(L) and Pel(cw)] before it results in inspiratory flow and thus increases in volume (V_L). In mechanically ventilated patients, inspiratory muscle action has to additionally overcome the trigger sensitivity (Ptr) of the ventilator [horizontal distance between the $Pel_{(L)}$ and Ptr] before it results in inspiratory flow and thus increases in volume (V_L); thus, in mechanically ventilated COPD patients, inspiratory muscle action has to overcome PEEPi plus the trigger sensitivity (Ptr).

When the magnitude of inspiratory muscle action is less than the sum of PEEPi plus Ptr this inspiratory effort (Fig. 1b, orange line) cannot trigger the ventilator, and consequently does not result in inspiratory flow and thus increases in volume (V_L). This inspiratory effort is called ineffective or wasted.

Is the Campbell diagram useful for estimating the work of breathing during wasted efforts?

The Campbell diagram is useless to estimate inspiratory work of breathing when inspiratory triggering does not happen (i. e., during wasted efforts): work is physically defined as the area subtended in a pressure/volume loop. Since there is no inspiratory volume, the work is zero (albeit muscles, indeed, consume energy). The energy expenditure during non-triggered wasted inspiratory efforts can be estimated by the pressure/time product: the product of the pressure developed by the inspiratory muscles [difference between the measured esophageal pressure and the Pel(cw)] multiplied by the time of muscle contraction (i. e., neural Ti).

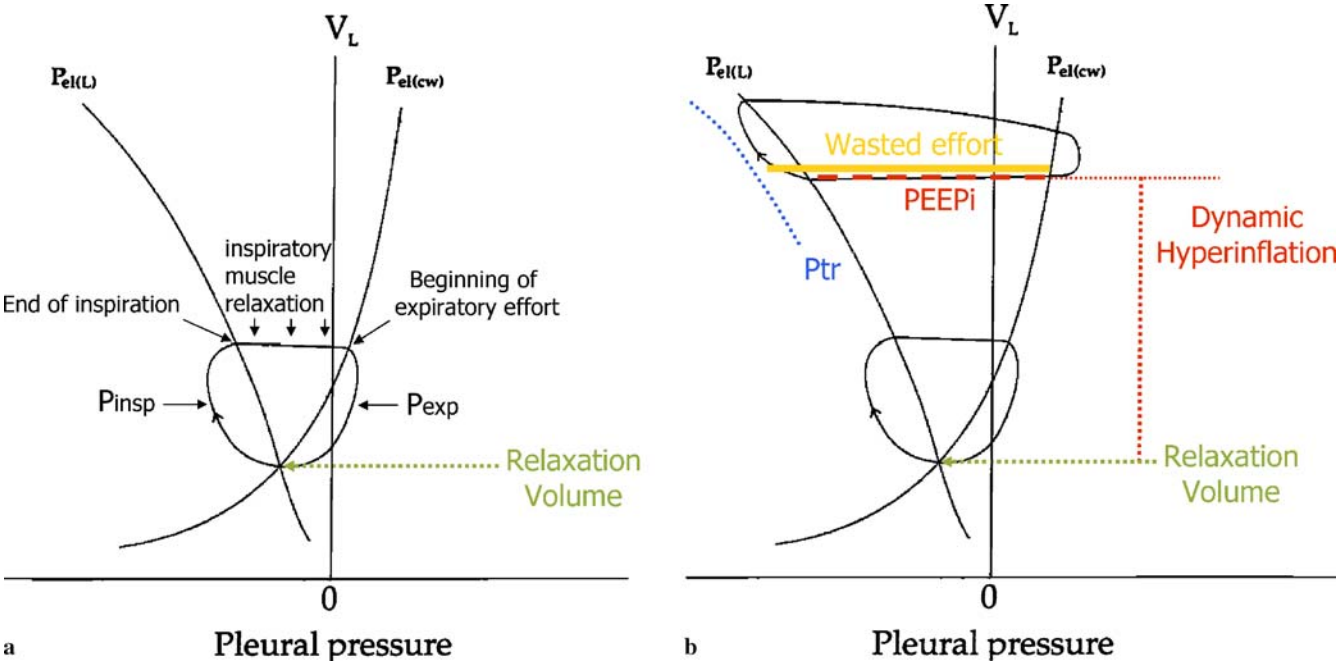

Fig. 1 **a** In normal subjects inspiration starts from the relaxation volume of the respiratory system, where the passive pressure-volume curves of the lung [*Pel(L)*] and chest wall [*Pel(cw)*] intersect. Inspiratory muscle action results in pressure development (*Pinsp*) on the left of the pressure-volume curve of the chest wall [*Pel(cw)*]. Inspiratory flow, and thus increases in volume (V_L) take place on the left of the pressure-volume curve of lung and coincide with the beginning of inspiratory muscle action. Inspiration ends on the pressure-volume curve of the lung and the inspiratory muscles relax (so that pressure returns on the pressure-volume curve of the chest wall). In the case shown, expiration is active so that pressure develops on the right of the pressure-volume curve of the chest wall due to activity of expiratory muscles (*Pexp*). This returns volume back to the relaxation volume of the respiratory system. **b** In COPD patients with dynamic hyperinflation, inspiration starts from an increased end-expiratory lung volume. Inspiratory muscle action has to overcome the intrinsic positive end expiratory pressure [*PEEPi*, *red dashed line*, horizontal distance between the *Pel(L)* and *Pel(cw)*] before it results in inspiratory flow and thus increases in volume. In mechanically ventilated patients, inspiratory muscle action has to overcome PEEPi plus the trigger sensitivity (*Ptr*) before it results in inspiratory flow and thus increases in volume (V_L). When the magnitude of inspiratory muscle action is less than the sum of PEEPi + Ptr this inspiratory effort (*orange line*) cannot trigger the ventilator and consequently does not result in inspiratory flow and thus increases in volume (V_L). This inspiratory effort is called ineffective or wasted

How are ventilator settings affecting the incidence of wasted/ineffective efforts?

Excessive ventilator support predisposes to ineffective efforts irrespective of the mode used [2, 4–7]. This is because, in the case of COPD, excessive pressure or volume delivered by the ventilator combined with the long time constant of the respiratory system (which retards lung emptying) and/or a short imposed expiratory time (in case of volume or pressure control) results in further increased end-expiratory lung volume before the next inspiratory effort begins (Fig. 2a, green curve). At the same time, excessive ventilator assistance reduces inspiratory muscle effort, via either a phenomenon called neuromechanical inhibition (the main mechanism most likely being the Hering–Breuer reflex) [8], and/or by producing alkalemia via excessive CO_2 reduction in patients with chronic bicarbonate elevation, thus reducing the drive to breathe [2]. The ensuing inspiratory effort is inadequate to overcome PEEPi plus Ptr and thus, this inspiratory effort fails to trigger the ventilator (Fig. 2a, red line). This is of course exaggerated in the presence of respiratory muscle weakness [7]. The following expiratory effort (Fig. 2a, blue curve) decreases the end expiratory lung volume. When the end expiratory lung volume decreases to a level where the ensuing inspiratory effort exceeds PEEPi plus Ptr, the ventilator is triggered again to deliver a machine breath (Fig. 2a, mauve curve). The breath-to-breath variability in breathing pattern contributes to the variability in the end-expiratory lung volume and thus to the frequency of ineffective efforts [7].

Alternatively, during assist control mechanical ventilation, prolonged imposed inspiratory time (machine Ti) greater than the patient's neural Ti results in a situation where the ventilator is inflating the patient long after the inspiratory muscles have stopped their contraction, i. e., during the neural expiration [6, 9]. During pressure support ventilation, the expiratory trigger threshold (percentage of peak inspiratory flow at which the ventilator cycles to expiration) might be quite low, leading to pressure support being delivered well beyond the patient's neural Ti [10]. In either case, the next inspiratory effort (controlled by the patient's respiratory controller) begins during the early

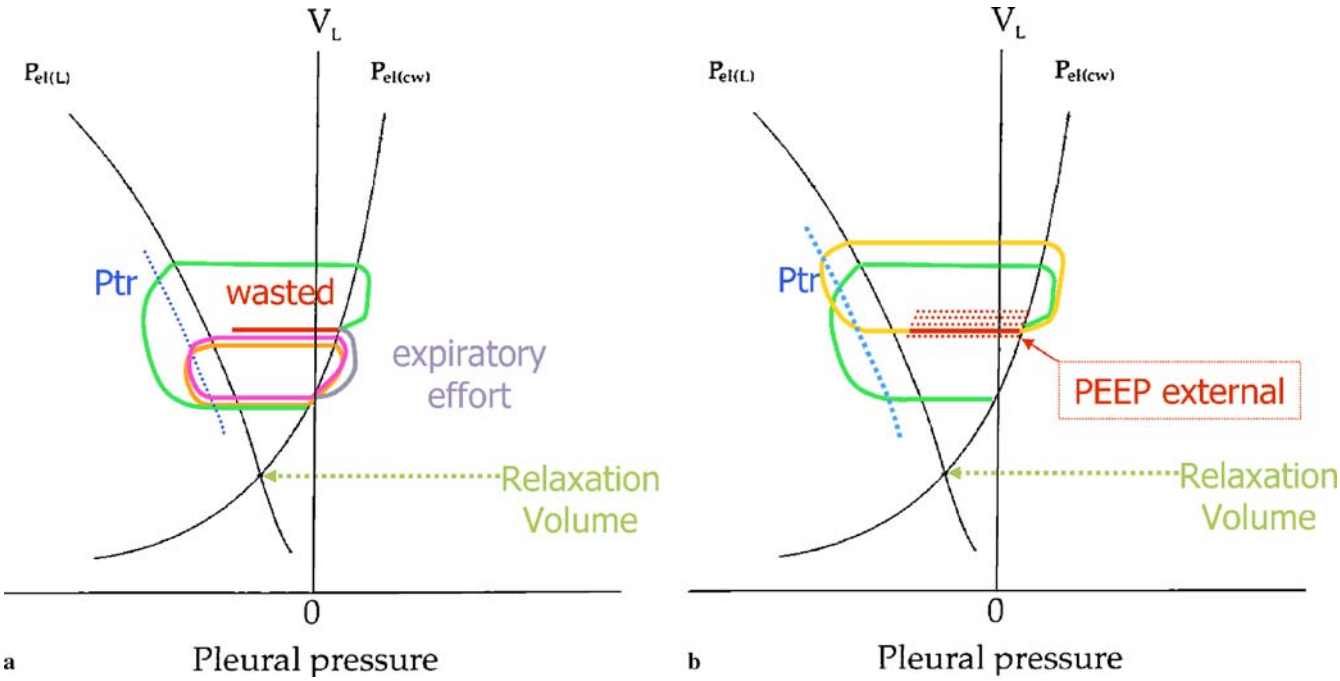

Fig. 2 **a** In the case of COPD presented, the first inspiratory effort (*orange curve*) triggers the ventilator. In the next breath that triggered the ventilator, excessive ventilator assistance (either pressure or volume) resulted in large tidal volume (*green curve*), which combined with the long time constant of the respiratory system (which retards lung emptying) and/or a short imposed expiratory time (in case of volume or pressure control) led to further increased end-expiratory lung volume before the next inspiratory effort begins (*green curve*). The ensuing inspiratory effort is inadequate to overcome PEEPi + Ptr due to the increased end expiratory lung volume; thus, this inspiratory effort fails to trigger the ventilator (wasted or ineffective effort, *red line*). The following expiratory effort (*blue curve*) decreases the end-expiratory lung volume. When the end-expiratory lung volume decreases to a level where the ensuing inspiratory effort exceeds PEEPi + Ptr, the ventilator is triggered again to deliver a machine breath (*mauve curve*). **b** In the presence of increased end-expiratory lung volume (*green curve*), addition of an amount of external PEEP lower than the intrinsic PEEP (*red dotted lines*) offers part of the pressure required to overcome PEEPi + Ptr. The inspiratory effort starts closer to the passive pressure-volume curve of the lung [*Pel(L)*]. The horizontal distance between this point and the passive pressure-volume curve of the chest wall [*Pel(cw)*] is the applied PEEP (*PEEP external*). The inspiratory effort is now adequate to trigger the ventilator (*orange curve*)

phase of ventilator expiration, i. e., at an increased lung volume [5]. This effort might not be sufficient to overcome PEEPi plus Ptr, and thus this inspiratory effort also fails to trigger the ventilator.

How can wasted efforts be detected at the bedside?

Wasted efforts can be clinically detected when the breaths delivered by the ventilator (measured rate on the ventilator display) are less than the number of inspiratory efforts of the patient (on clinical examination) at the same time interval (see ESM, slides 1 and 2). On modern ventilator screens, ineffective efforts can be detected as abrupt airway pressure drop simultaneous to an abrupt decrease in expiratory flow (from the flow trajectory established earlier during expiration) and not followed by a machine breath (see ESM, slide 2). Monitors that can automatically detect wasted efforts are under clinical testing and will be become available in the future [11, 12].

What ventilator adjustments should be done in the presence of wasted/ineffective efforts?

The self-evident solution to reduce the frequency of wasted efforts is to decrease the level of excessive ventilator assistance, thus reducing hyperinflation and the pathophysiology presented above [4, 6, 7]; however, this might not be always clinically feasible, since it might lead to respiratory distress and to derangement of blood gases [6].

Another solution is the use of PEEP [1, 6]. The addition of an amount of external PEEP lower than the intrinsic PEEP (Fig. 2b, red dotted lines) offers part of the pressure required to overcome PEEPi plus Ptr (Fig. 2b). The inspiratory effort starts closer to the Pel(L) [the horizontal distance between this point and the [Pel(cw) being the applied PEEP (PEEP external)]. The inspiratory effort is now adequate to trigger the ventilator (orange curve).

During assist control reducing machine Ti (or equivalently increasing the inspiratory flow) may prevent ventilator delivery beyond the patients' neural Ti and will reduce wasted efforts. Similarly, during pressure support increasing the expiratory trigger threshold will stop the breath earlier and will reduce wasted efforts [13].

Conclusion

Wasted efforts are a major cause of patient ventilator dyssynchrony that increase the energy expenditure of the respiratory muscles and may injure them. Understanding their pathophysiology is essential to properly adjust the ventilator settings to attenuate or eliminate them. Wasted efforts should be searched before any change in ventilator settings is implemented during assisted modes of mechanical ventilation, since any ensuing increase in ventilator rate might be caused by the attenuation of wasted efforts (the ventilator rate now approaching the patient's respiratory controller rate) and not by the development of respiratory distress with the new settings.

References

1. Fernandez R, Benito S, Blanch L, Net A (1988) Intrinsic PEEP: a cause of inspiratory muscle ineffectivity. Intensive Care Med 15:51–52
2. Thille AW, Rodriguez P, Cabello B, Lellouche F, Brochard L (2006) Patient-ventilator asynchrony during assisted mechanical ventilation. Intensive Care Med 32:1515–1522
3. Cabello B, Mancebo J (2006) Work of breathing. Intensive Care Med 32:1311–1314
4. Leung P, Jubran A, Tobin MJ (1997) Comparison of assisted ventilator modes on triggering, patient effort, and dyspnea. Am J Respir Crit Care Med 155:1940–1948
5. Tobin MJ, Jubran A, Laghi F (2001) Patient-ventilator interaction. Am J Respir Crit Care Med 163:1059–1063
6. 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
7. Chao DC, Scheinhorn DJ, Stearn-Hassenpflug M (1997) Patient-ventilator trigger asynchrony in prolonged mechanical ventilation. Chest 112:1592–1599
8. Dempsey JA, Skatrud JB (2001) Apnea following mechanical ventilation may be caused by nonchemical neuromechanical influences. Am J Respir Crit Care Med 163:1297–1298
9. Parthasarathy S, Jubran A, Tobin MJ (1998) Cycling of inspiratory and expiratory muscle groups with the ventilator in airflow limitation. Am J Respir Crit Care Med 158:1471–1478
10. Beck J, Gottfried SB, Navalesi P, Skrobik Y, Comtois N, Rossini M, Sinderby C (2001) Electrical activity of the diaphragm during pressure support ventilation in acute respiratory failure. Am J Respir Crit Care Med 164:419–424
11. Younes M, Brochard L, Grasso S, Kun J, Mancebo J, Ranieri M, Richard JC, Younes H (2007) A method for monitoring and improving patient: ventilator interaction. Intensive Care Med 33:1337–1346
12. Mulqueeny Q, Ceriana P, Carlucci A, Fanfulla F, Delmastro M, Nava S (2007) Automatic detection of ineffective triggering and double triggering during mechanical ventilation. Intensive Care Med 33:2014–2018
13. Tassaux D, Gainnier M, Battisti A, Jolliet P (2005) Impact of expiratory trigger setting on delayed cycling and inspiratory muscle workload. Am J Respir Crit Care Med 172:1283–1289