Commentary

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Should We Care about Driving Pressure during Assisted Mechanical Ventilation?

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Introduction

During passive mechanical ventilation, at absence of dynamic hyperinflation, driving pressure of respiratory system (ΔP) is defined as static end-inspiratory plateau pressure (Pplat) minus external positive end-expiratory pressure (PEEP), and equals tidal volume (V_T) to respiratory system compliance (Crs) ratio. ΔP essentially reflects the extent of lung stretch during tidal breathing. Thus, ΔP may reflect better than Pplat or V_T, expressed as ml/kg of ideal body weight, the alveolar distortion during inspiration because it takes into account the available aerated lung volume. Indeed, a large retrospective study in patients with acute respiratory distress syndrome (ARDS) identified ΔP as the main determinant of ventilator-induced lung injury (VILI), and the ventilator parameter most strongly related to mortality, particularly at ΔP values >14 cm H_2O [1].

The association of ΔP with mortality in ARDS patients was confirmed in another large observational study [2]. Other studies also showed that high ΔP may detect lung overstress [3] and be associated with high morbidity [4]. However, we should notice that ΔP is dissipated to counterbalance both the change in transpulmonary pressure (ΔP_{lung}) and that of chest wall (ΔP_{cw}), the first being the key variable for lung damage. Since ΔP_{lung} calculation requires an esophageal catheter insertion for recording esophageal pressure, a procedure not easily applicable for every day practice, ΔP is used as surrogate of ΔP_{lung} ($\Delta P=\Delta P_{lung}+\Delta P_{cw}$).

Although ΔP, as marker of VILI, has been exclusively studied in patients under controlled mechanical ventilation, there is no reason to believe that the potential harmful effects of high ΔP (due to high ΔP_{lung}) are present only during passive ventilation. During assisted mechanical ventilation the ventilator and the respiratory muscles may be considered as pressure generators arranged in series, and thus during inspiration the total pressure applied to respiratory system at any time t ($P_{TOT(t)}$) is the sum of airway pressure (pressure provided by the ventilator, $Paw_{(t)}$) and pressure developed by inspiratory muscles ($Pmus_{(t)}$). This total pressure is dissipated to offset elastic and resistive pressures according to the equation of motion:

$$Paw_{(t)}+Pmus_{(t)}=V_{(t)}/Crs+V'_{(t)} \times Rrs+PEEP \quad \text{(Equation 1)}$$

Where V and V' are volume above end-expiratory lung volume and flow, respectively. Notice that Equation 1 as it stands, is valid if intrinsic PEEP is zero (no dynamic hyperinflation). Obviously during assisted modes ΔP is the pressure dissipated to offset the increase in elastic recoil pressure of respiratory system (ΔPel) due to V_T ($\Delta Pel=\Delta P=V_T/Crs$). It becomes apparent that, as opposed to controlled mechanical ventilation, during assisted mechanical ventilation ΔP is partially depended and, to some extent, regulated by patient effort (Pmus).

May ΔP reach injurious levels during assisted ventilation? ΔP is high, when V_T is high (i.e., as a result of high $P_{TOT}=Paw+Pmus$) and/or when Crs is low. Regarding Crs, one could hypothesize that patients having more severe lung injury and thus lower Crs, would be ventilated on controlled modes, and assisted modes would be reserved for patients with better lung mechanics, limiting thus the risks of high ΔP. However, although a patient with severe ARDS would be more likely sedated and ventilated in controlled mode, at least initially, no studies or guidelines have indicated switching between controlled and assisted ventilation based on lung mechanics. A second hypothesis could be that the system of control of breathing (non-functioning during passive

mechanical ventilation) would prevent the development of high distending pressures. Indeed, the mechanoreceptors of the respiratory system sense the degree of lung stretch and tend to prevent over-distension through reflex mechanisms. The Hering-Breuer inspiratory-inhibitory reflex inhibits inspiratory activity and associated increase in lung volume, when a threshold lung stretch is reached during inspiration [5]. In addition, if high distending pressure results in high V_T this may decrease $PaCO_2$ which via chemical feedback mechanism lowers Pmus limiting thus ΔP. Nevertheless, the effectiveness of these protective mechanisms is heavily depended on the mode of mechanical ventilation and ventilator settings as Equation 1 dictates (i.e., Paw). In addition, assist volume control or pressure supports modes cancel or limit the ability of the patient effort to modulate V_T. Also the proper function of these protective feedback mechanisms may be overridden by other stimuli that increase the respiratory drive of critically ill patients. Indeed it has been shown in experimental settings that metabolic acidosis induces sufficient hyperventilation to develop lung injury [6]. In critically ill patients, conditions increasing respiratory drive, such as hypercapnia, metabolic acidosis, delirium, fever, on-going sepsis are often present and could result in overriding the protective mechanisms of control of breathing [7]. It is therefore, possible that, in the presence of impaired lung mechanics and high respiratory drive, injurious ΔP may develop during assisted ventilation, promoting ventilator-induced lung injury.

Although ΔP is an important variable during assisted modes, its calculation requires either Pmus or valid Crs measurements. Pmus measurement can be obtained by recording Pes, in which case, however, it is easier to measure ΔP_{lung}. On the other hand, valid Crs is difficult to be obtained during assisted modes of support such as volume-assist, pressure support or neutrally adjusted ventilator assist (NAVA), because it necessitates passive conditions during the measurement of Pplat. Currently the only assisted mode that permits semi-continuously valid measurement of Crs (and thus of ΔP) is proportional assist ventilation with adjustable gain factors (PAV+), a mode that permits the patients to select their own breathing pattern as dictated by the control of breathing mechanisms. With this mode, the ventilator applies a 300-msec end-inspiratory occlusion randomly every 4-7 breaths and measures airway pressure (P_{plat}PAV+) at the end of this brief occlusion. Crs is calculated as the $V_T/(P_{plat}$PAV+-PEEP) ratio. It has been shown that there is no residual post-inspiratory activity at the point of P_{plat} PAV+ measurement and because it is measured very close to the end of inspiratory flow (which is mainly determined by the end of neural inspiration), it is a good approximation of the true elastic recoil pressure at end inspiration in un-occluded breaths [8]. We have recently reported data on ΔP during assisted ventilation [9] by analyzing 108 patients (64 were recovering from ARDS) ventilated on PAV+ for 48h after switching from controlled ventilation [10]. When patients were switched from passive ventilation to PAV+ they controlled ΔP to low levels (<15 cm H_2O in 90% of measurements) without constraining V_T to narrow limits. It seems that in critically ill patients meeting certain criteria, the control of breathing system was adept at protecting the lungs by preventing high ΔP, while not unnecessarily restricting V_T when this had no protective value **(Figure 1)**. However, in two patients having very low values of Crs, constantly high ΔP (≥ 15 cm H_2O) was observed. In addition, unpublished data from our group indicate that approximately 10% of patients during assisted ventilation on PAV+ may at some point, exhibit high ΔP, associated with low CRS. Notwithstanding that ΔP is a surrogate of ΔP_{lung}, these data indicate that in some patients, especially those with impaired Crs, the protective mechanisms of control of breathing may be overridden by high respiratory drive due to a variety of causes. Monitoring ΔP during assisted mechanical ventilation may identify these patients who are at risk of lung injury and influence decision making.

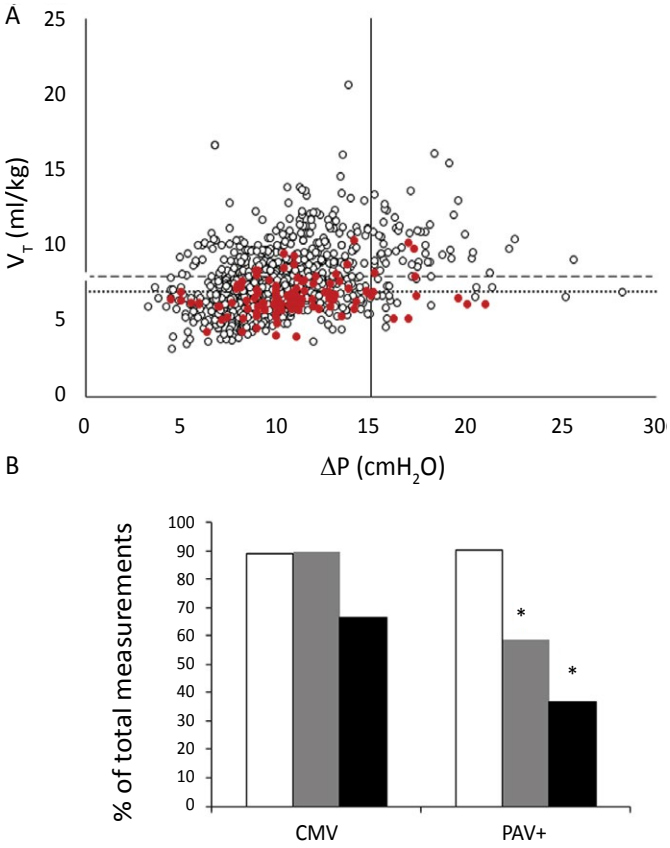

Figure 1 Individual change of ΔP after switching from controlled ventilation (CMV) to assisted ventilation with PAV+ (ΔP_{PAV+}-ΔP_{CMV}), as a function of ΔP during CMV (ΔP_{CMV}) (Data obtained in 108 patients). Each patient is characterized by a number of data points equal to the number of measurements during PAV+. Each dot represents one measurement (total measurements 744). Blue areas highlight measurements with high (≥ 15 cm H_2O) or low (≤ 8 cm H_2O) ΔP during CMV using lung protective strategy. Notice that when the lung protective strategy results in high driving pressure (n=67), the patients' control of breathing system (i.e., feedback mechanisms integrated at brain stem) decreased it in the majority (87%) of measurements. On the contrary when the lung protective strategy results in low driving pressure (n=65), the patients' control of breathing system increased it in 91% of measurements.

Conclusion

The recognition of VILI changed the practice of mechanical ventilation. The pursue of optimal ventilator settings to reduce lung stress and injury is an on-going journey, and the setting of V_T to 6 ml/kg was just the beginning. Targeting ΔP as means to minimize lung injury appears to be reasonable, emphasized by the fact that our brain seems to care more for the ΔP than for V_T. Identifying the appropriate target for driving pressure will be challenging, as the benefits from protection from VILI have to balance against the costs of interventions to reduce ΔP, such as sedation to reduce respiratory drive. To this end, prospective studies evaluating ΔP in both controlled and assisted ventilation are urgently needed.

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