

## Commentary

# Low tidal volume, high respiratory rate and auto-PEEP: the importance of the basics

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## Abstract

Recent studies have shown that application of the ARDSNet low tidal volume strategy (i.e. allowing an increase in respiratory rate in order to minimize hypercapnia in those with low tidal volume) may generate consistent auto-PEEP (positive end-expiratory pressure), and this is not efficient in improving clearance of carbon dioxide. The present commentary deals with some of the recent controversies related to use of a low tidal volume strategy, as implemented in the ARDSNet trial, which has proved successful in reducing mortality rates in patients with acute respiratory distress syndrome. We emphasize the importance of basic physiological knowledge and sound respiratory monitoring.

**Keywords** acute respiratory distress syndrome, auto-PEEP, high respiratory rate, hypercapnia, low-tidal volume strategy

"... the wisdom of old men. They do not grow wise. They grow careful."

*A Farewell to Arms*, Ernest Hemingway

The ARDSNet trial [1], which compared a low versus a high tidal volume (Vt) ventilation strategy, appears to have said the final word in the controversy surrounding the clinical relevance of ventilator-induced lung injury. That study indeed showed that patients with acute respiratory distress syndrome (ARDS) who were ventilated at 6 ml/kg benefited from a 22% reduction in mortality, as compared with the group ventilated with a Vt of 12 ml/kg. As always, and in spite of these important results, some aspects of the study design and speculation regarding the mechanisms involved generated some controversial interpretations [2–5].

Of those, a major interpretation of the ARDSNet findings pertains to the possible role of auto-PEEP (positive end-expiratory pressure) in causing the observed difference in mortality between low and high Vt strategies. The protocol

allowed investigators to increase the respiratory rate in the low Vt group to 35 breaths/min, in order to minimize hypercapnia and respiratory acidosis – the major side effects of low Vt ventilation. A recent study conducted by de Durante and coworkers [4] demonstrated that the ventilatory settings employed in the ARDSNet low Vt group may generate an auto-PEEP of  $5.8 \pm 3$  cmH<sub>2</sub>O. Based upon these data, those authors suggested that in the ARDSNet study, in spite of comparable external PEEP settings, total PEEP was substantially higher in the low Vt group because the high respiratory rate generated a substantial auto-PEEP. It is possible to speculate that the observed difference in survival was more related to the difference in total PEEP than to the difference in Vt.

Vieillard Baron and coworkers [5] recently investigated the effects of increasing respiratory rate from 15 to 30 breaths/min, while maintaining a constant plateau pressure ( $\leq 25$  cmH<sub>2</sub>O). Those authors reported an auto-PEEP of  $6.4 \pm 2.7$  cmH<sub>2</sub>O at 30 breaths/min, which was associated with an increased right ventricular outflow

ALI = acute lung injury; ARDS = acute respiratory distress syndrome; Pco<sub>2</sub> = partial carbon dioxide tension; PEEP = positive end-expiratory pressure; Vt = tidal volume.

impedance and a decreased cardiac index. Moreover, despite the higher respiratory rate, partial carbon dioxide tension ( $PCO_2$ ) was not significantly different, whereas the ratio between (alveolar) dead space and  $V_t$  increased significantly. Those authors concluded that, in acute lung injury (ALI)/ARDS patients, the use of higher respiratory rate at constant plateau pressure in order to increase minute ventilation is unable to improve elimination of carbon dioxide, while it generates auto-PEEP.

What lesson can we derive from those studies? What should clinicians be careful about? Clearly, there should be no doubt that a ventilatory strategy based on high  $V_t$  and high plateau pressure should be avoided; years of experimental data indicate a need for a gentler ventilatory approach. However, we do not have, and probably will never have, a simple 'cookbook' for ventilatory management of patients with ALI/ARDS. Above all, clinicians should rely on scientific knowledge, clinical expertise, monitoring and a degree of wisdom. As stated by Tobin [6], "... there is no substitute for the clinician's standing by the ventilator, making necessary adjustments, and monitoring the effects of such adjustments. The treatment of patients with the acute respiratory distress syndrome involves trade-offs in which improvement in one type of measure ... can lead to worsening of another." If a single lesson should be picked out from the many controversies in ventilatory management, then it is that respiratory mechanics should be monitored.

Does high respiratory rate produce intrinsic PEEP in ALI/ARDS patients? There should be no doubt as to the answer to this question. Intrinsic PEEP is a function of minute ventilation (respiratory rate and  $V_t$ ), ventilatory setting (expiratory time) and the mechanical properties of the respiratory system (flow limitation when present [7], along with compliance and resistance, taking into account the ventilator and circuitry). Whenever the expiratory time is too short to exhale the inspired  $V_t$ , given the mechanical properties of the patient-ventilator complex, auto-PEEP will occur. Higher compliance and resistance of the respiratory system, and the possible occurrence of flow limitation will favour generation of auto-PEEP. These simple principles concerning auto-PEEP are well established, and anyone involved in the ventilatory management of any kind of patient (not just ALI/ARDS patients) should be aware of them and measure auto-PEEP. Auto-PEEP may easily be assessed by a simple end-expiratory pause – one of the easiest procedures on a ventilator. In our opinion, measurement of auto-PEEP should be part of any monitoring routine.

Should the clinicians bother about auto-PEEP and associated haemodynamic drawbacks? Of course, the answer to this question is, again, 'yes'. However, ventilatory settings that predispose to auto-PEEP would probably derive from attempts to control some other variable, such as  $PCO_2$ . As always, the final decision is a matter of clinical common

sense and experience in weighing the advantages and the disadvantages of every option. The haemodynamic effects associated with auto-PEEP are well known, and the clinician should be aware of them and monitor them. In the presence of a known haemodynamic impairment, such as a decreased cardiac index (as was observed in the study by Vieillard-Baron and coworkers [5]), the clinician has several choices. If the priority is to limit ventilator-induced lung injury, and we persist on a low  $V_t$  strategy, then the use of a higher respiratory rate appears unavoidable if we are to maintain acceptable  $PCO_2$  and pH levels. To limit auto-PEEP, we can try to increase the expiratory time by increasing the inspiratory flow rate and decreasing the plateau time. However, a reduction in inspiratory time and in the ratio of inspiratory to expiratory time may have a negative impact on oxygenation, which may not be acceptable. On the other hand, we may tolerate auto-PEEP and try to limit the haemodynamic impairment associated with auto-PEEP by directly acting on cardiac function (fluid loads, inotropes), as we often do to compensate for high levels of external PEEP. Once again, basic physiology and appropriate monitoring will lead the clinician to the wisest choice.

Should an increased respiratory rate be used to improve clearance of carbon dioxide when using a low  $V_t$  strategy? Vieillard-Baron and coworkers [5] stated that increasing the respiratory rate is not an efficient strategy for improving carbon dioxide clearance. This is true if a low  $V_t$ /high respiratory rate strategy is compared with a higher  $V_t$ /lower respiratory rate at comparable minute ventilation (physiology tells us that the dead space : minute ventilation ratio will rise, decreasing the efficiency of the system). However, the important issue is not to compare two different strategies of carbon dioxide clearance, but to recognize that, once we have selected a low  $V_t$  strategy, an increased respiratory rate is the simplest way to maintain adequate  $PCO_2$  and pH levels.

In conclusion, in order to survive the numerous discussions and ideas on how patients with ALI/ARDS should be managed, the clinician should always count on basic physiology, which has taught us almost all of the major principles that we need. Every so often some of these principles are rediscovered, and this strengthens their role. After all, it is no bad thing to go back and periodically refresh our understanding of basic physiology. Appropriate monitoring will provide us with the necessary information to appreciate ongoing events and to take the best decision.

## Competing interests

None declared.

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