Commentary

Positive end-expiratory pressure in acute respiratory distress syndrome – an old yet mysterious tool

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Related to *Research* by Bruhn *et al*., see page 395

Abstract

A recent study by Bruhn and colleagues, discussed here, confirms that even high levels of positive end-expiratory pressure (PEEP) – up to 20 cmH_2O – may be applied in conditions of moderate acute respiratory distress syndrome. Such levels of PEEP were found to be safe in terms of their impact on cardiac output and adequacy of gastric mucosal perfusion once systemic haemodynamics were stabilized by adequate fluid replacement and catecholamine therapy. However, we strongly recommend that the reader does not oversimplify the conclusions of that study. PEEP therapy is not inherently safe with respect to haemodynamics and regional organ perfusion, but it may be used safely, even at high levels of up to 20 cmH_2O, if haemodynamic therapy is appropriate.

Keywords acute lung injury, acute respiratory distress syndrome, mechanical ventilation, P_{CO_2} gap, positive end-expiratory pressure, regional organ perfusion

The introduction of positive end-expiratory pressure (PEEP) into the practice of mechanical ventilation in patients with acute respiratory distress syndrome (ARDS) [1] was among the most important milestones in critical care therapy. However, despite the clear benefits of this therapeutic tool in improving severely compromised gas exchange, potential dangers related to the use of PEEP – mainly suppression of cardiovascular and other organ function – became evident from experimental data even before that time [2]. During the past few decades a huge amount of research on this topic has been conducted, providing us with important insights into the interaction between mechanical ventilation with PEEP and physiological function in critically ill patients [3–5]. Nevertheless, it is not yet possible to determine clearly the balance between benefit and deleterious effects of PEEP, and the controversy on how to set PEEP at the bedside persists [6]. Current strategies of mechanical ventilation for patients with acute lung injury or ARDS [7] increasingly recommend fairly high levels of PEEP. In view of this, any data that may improve our understanding of the benefits and dangers of PEEP are particularly important.

In this issue of *Critical Care*, Bruhn and colleagues [8] report the effects of PEEP on adequacy of gastric mucosal perfusion in ARDS patients. Their clinical data are interesting and encouraging. This is because, at first glance, fairly high levels of PEEP – up to 20 cmH_2O – apparently did not compromise haemodynamics or significantly affect gastric mucosal–arterial partial carbon dioxide tension gap (ΔP_{CO_2}) in these patients. However, proper interpretation of these data requires consideration of important limitations imposed by the study design. In fact, one should not conclude that PEEP up to 20 cmH_2O is generally safe in patients with ARDS in terms of haemodynamics or even regional organ perfusion. This is clearly not the case, as was shown by various investigators [9,10]. Rather, as demonstrated by previous studies [11,12], adequate replacement of intravascular fluid volumes is required to maintain cardiac

ARDS = acute respiratory distress syndrome; ΔP_{CO_2} = partial carbon dioxide tension gap; PEEP = positive end-expiratory pressure.

output and regional perfusion during mechanical ventilation with high PEEP levels. In their study, which nicely corroborates previous findings, Bruhn and colleagues [8] paid meticulous attention to maintenance of haemodynamics with fluid replacement and intravenous administration of catecholamines at each PEEP stage, and they were successful in this in all but one patient. (Unfortunately, that patient did not complete the study, and hence we do not know whether the failure to maintain haemodynamics may also have impaired gastric mucosal–arterial ΔP_{CO_2}.) Therefore, the important message from the study is that, even at high levels of PEEP (20 cmH_2O), it is possible to maintain haemodynamics and adequate gastric mucosal perfusion with fluid and catecholamine therapy. Such an approach, however, should be considered a prerequisite for safe PEEP ventilation.

Some additional limitations were addressed in part by the authors themselves, and should be kept in mind when considering the findings of the study. First, the patients included fulfilled the criteria for ARDS when they entered the study, but their respiratory status apparently improved between entrance into the study and the start of evaluation. In fact, arterial oxygen tension/fractional inspired oxygen ratio was greater than 200 in five out of eight patients, and respiratory system compliance was only moderately decreased in most patients. Second, ARDS may represent widely differing pathophysiological conditions depending on its primary cause, which may derive from pulmonary or extrapulmonary disorders [13]. In the case of abdominal diseases leading to ARDS (which was the case in three out of eight patients), intra-abdominal pressure may be increased, thus compromising both systemic haemodynamics and perfusion of intra-abdominal organs independent from PEEP derived cardiocirculatory effects at thoracic levels. Therefore, the impact of PEEP on regional perfusion may depend to a significant degree on the primary cause of ARDS. Finally, in the study the arterial carbon dioxide tension significantly increased between baseline and application of 20 cmH_2O PEEP. This might have interfered with regional perfusion, thus potentially influencing the findings of the study [14].

The limitations mentioned thus far should not be interpreted as a critique of the study, which is clear in terms of the study design and conclusions drawn from the findings. Indeed, the authors elegantly demonstrate how PEEP may safely be employed even at high levels, at least in patients with moderate ARDS. However, the limitations mentioned above are indicators of the huge complexity of the interactions between mechanical ventilation and organ perfusion, which continue to limit our understanding of the impact of mechanical ventilation in critical care.

Competing interests

The authors declare that they have no competing interests.

References

1. Ashbaugh DG, Bigelow DB, Petty TL, Levine BE: **Acute respiratory distress in adults.** *Lancet* 1967, **2:**319-323.
2. Cournand A, Motley HL, Werko L, Richards DW: **Physiological studies of the effects of intermittent positive pressure breathing on cardiac output in man.** *Am J Physiol* 1948, **152:**163-174.
3. Miro AM, Pinsky MR: **Heart-lung interactions.** In: *Principles and Practice of Mechanical Ventilation.* Edited by Tobin MJ. New York: McGraw-Hill Inc.; 1994:647-671.
4. Pannu N, Mehta RL. **Mechanical ventilation and renal function: an area for concern?** *Am J Kidney Dis* 2002, **39:**616-624.
5. Beyer J, Beckenlechner P, Messmer K: **The influence of PEEP ventilation on organ blood flow and peripheral oxygen delivery.** *Intensive Care Med* 1982, **8:**75-80.
6. Saura P, Blanch L: **How to set positive end-expiratory pressure.** *Respir Care* 2002, **47:**279-295.
7. Amato MB, Barabas CS, Medeiros DM, Magaldi RB, Schettino GP, Lorenzi-Filho G, Kairalla RA, Deheinzelin D, Munoz C, Oliveira R, Takagaki TY, Carvalho CR: **Effect of a protective-ventilation strategy on mortality in the acute respiratory distress syndrome.** *N Engl J Med* 1998, **338:**347-354.
8. Bruhn A, Hernandez G, Bugedo G, Castillo L: **Effects of positive end-expiratory pressure on gastric mucosal perfusion in acute respiratory distress syndrome.** *Crit Care* 2004, **8:**R306-R311.
9. Bonnet F, Richard C, Glaser P, Lafay M, Guesde R: **Changes in hepatic flow induced by continuous positive pressure ventilation in critically ill patients.** *Crit Care Med* 1982, **10:**703-705.
10. Winsö O, Biber B, Gustavsson B, Holm C, Milsom I, Niemand D: **Portal blood flow in man during graded positive end-expiratory pressure ventilation.** *Intensive Care Med* 1986, **12:**80-85.
11. Brienza N, Revelly JP, Ayuse T, Robotham JL: **Effects of PEEP on liver arterial and venous blood flows.** *Am J Respir Crit Care Med* 1995, **152:**504-510.
12. Matuschak GM, Pinsky MR, Rogers RM: **Effects of positive end-expiratory pressure on hepatic blood flow and performance.** *J Appl Physiol* 1977, **62:**1377-1383.
13. Pelosi P, Gattinoni L: **Acute respiratory distress syndrome of pulmonary and extra-pulmonary origin: fancy or reality?** *Intensive Care Med* 2001, **27:**457-460.
14. Blobner M, Bogdanski R, Kochs E, Henke J, Findeis A, Jelen-Esselborn S: **Effects of intraabdominally insufflated carbon dioxide and elevated intraabdominal pressure on splanchnic circulation: an experimental study in pigs.** *Anesthesiology* 1998, **89:**475-482.