With interest we have read the paper by Carvalho and colleagues, in which the authors report the results of their elegant study on the role of positive end-expiratory pressure (PEEP) in acute lung injury [1]. Carvalho and colleagues demonstrated in an acute respiratory distress syndrome (ARDS) model that alveolar hyperinflation and nonaerated areas may coincide during a stepwise reduction of PEEP. Their finding is in line with our findings in a mathematical ARDS model and with other studies [2,3], and emphasises that the PEEP at which the respiratory system compliance is maximal coincides with a compromise between hyperinflation and poor aeration [3].

The accompanying commentary by Rouby and colleagues, however, raises questions about the contribution by Carvalho and colleagues on which we would like to comment [4]. First, Rouby and colleagues state that PEEP may produce end-expiratory hyperinflation of aerated areas. This statement is clear, but the authors also state that PEEP produces end-expiratory reaeration of nonaerated parts of the lung (recruitment). This last statement is puzzling, since PEEP is an end-expiratory phenomenon and as such cannot in itself recruit collapsed lung units. PEEP can maintain volume that has been recruited by higher pressures.

Second, we fully agree that a lung-protective ventilator strategy should not only include a reduced tidal volume, but also should combine a PEEP level that corresponds to an optimal compromise between alveolar recruitment and hyperinflation. Rouby and colleagues conclude that this compromise yields equal numbers of hyperinflated and non-aerated lung regions. Evidently, both regional consolidation and regional hyperinflation may lead to ventilator-induced lung injury [5]. To our knowledge, however, whether both are equally harmful has yet to be demonstrated. Furthermore (at least during high-frequency oscillatory ventilation), airway pressures at which oxygenation is maximal do not equal pressures at which the respiratory system compliance is maximal [6]. We therefore propose that for the survival of the individual patient it is unclear whether to aim at optimal oxygenation or at optimal compliance.

**Authors’ response**

Jean-Jacques Rouby, Fabio Ferrari, Bélaïd Bouhemad and Qin Lu

We thank Dr Markhorst and colleagues for their comment. We fully agree that PEEP prevents end-expiratory derecruitment and does not provide lung recruitment per se. When referring to the effects of PEEP, it is true that it would have been more appropriate to replace recruitment with prevention of derecruitment as we did in a recent paper measuring the effect of PEEP on lung aeration [7].

The second comment raises important questions. There is clear evidence that regional lung inflammation is not only caused by cyclic opening and closing of distal lung units, but also by hyperinflation of aerated lung units: this has been shown in patients with ARDS [8] and in patients with healthy lungs undergoing mechanical ventilation during general anaesthesia [9]. As shown in Figure 1, in ARDS survivors the distribution of pseudo cysts and bronchiectasis characterizing late ventilator-induced lung injury [10] is exactly the same as the distribution of lung hyperinflation measured in the early phase [11]. Such a result clearly suggests that initial pulmonary hyperinflation is the prelude to secondary lung distortion and bronchial damage, and that early ventilator-induced hyperinflation has a significant secondary harmful impact.

**ARDS** = acute respiratory distress syndrome; **PEEP** = positive end-expiratory pressure.
It is true that the best oxygenation does not coincide with the best compliance. The relevant issue remains, however, whether high oxygenation levels are of any significant benefit compared with oxygenation levels providing arterial oxygen saturation >90% and oxygen transportation sufficient to meet tissue oxygen demand. A strategy aimed at obtaining the highest arterial oxygenation cannot be considered safe if the price to pay is early hyperinflation with impaired cardiac output and oxygen transportation followed by late bronchial and alveolar damage.

Competing interests
The authors declare that they have no competing interests.

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