WHAT'S NEW IN INTENSIVE CARE

Alveolar recruitment in acute respiratory distress syndrome: should we open the lung (no matter what) or may accept (part of) the lung closed?

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The conceptual model of acute respiratory distress syndrome (ARDS) includes: (a) lung inflammation; (b) severe hypoxemia; (c) edema, hyaline membranes, and alveolar hemorrhage. Common denominator is the loss of aerated lung tissue due to alveolar collapse. To open collapsed alveoli has been, therefore, proposed to restore gas exchange [1] with interventions such as positive endexpiratory pressure (PEEP), recruitment maneuvers, and prone position [2]. Moreover, loss of lung volume may contribute to ventilator-induced lung injury (VILI) [2]. We will review the physiology of alveolar recruitment and address the implications of the "full recruitment" vs "partial recruitment" approaches (Fig. 1).

The physiology of alveolar recruitment

Although the mechanisms and the consequences of alveolar recruitment had been thoroughly studied, it is only after the late 80s [3] and early 90s [4] that alveolar recruitment was quantified. Gattinoni et al. used quantitative analysis of lung CT-scan and showed that PEEP-induced increase in oxygenation was strongly related to a progressive increase in aerated lung tissue, and, thus,

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to the recruitment of perfused and collapsed lung units [3]. Ranieri et al. used quantitative analysis of the volume–pressure curve at different PEEP levels and showed that in some patients, PEEP shifted the curve upward along the volume axis indicating an increase in volume for the same pressure (recruitment). In other patients, PEEP shifted the curve along the pressure axis indicating an increase in pressure for the same volume (hyperinflation) (Fig. 2) [4]. Later, Gattinoni et al. confirmed that the amount of potentially recruited lung was highly variable and, although the study was performed in 68 patients only, it showed that patients with a larger amount of potentially recruitable alveoli tended to have a higher mortality than patients who had smaller amount of alveoli potentially recruitable [5].

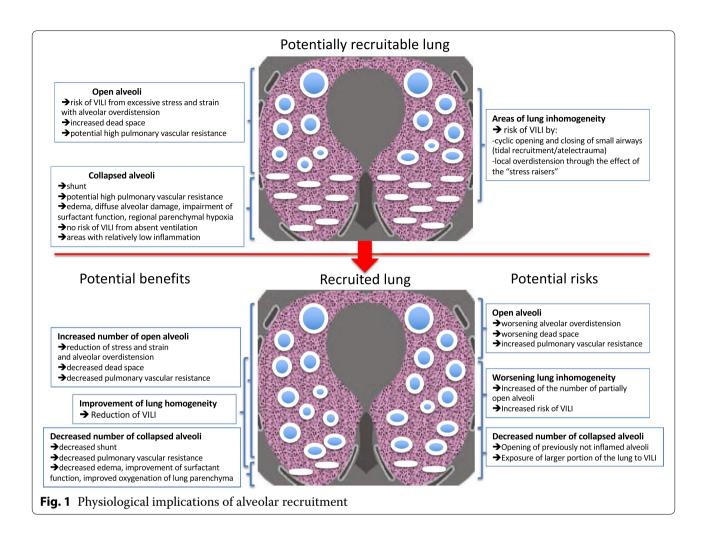
The effects of alveolar recruitment on gas exchange are mainly due to recruitment-induced modifications in venous admixture $(Q_{\rm s}/Q_{\rm t})$ and dead space. An increase in PaO₂ is normally observed after applying PEEP, and this increase is highly correlated with a decrease in $Q_{\rm s}/Q_{\rm t}$ [6]. Interestingly, the increase in PaO₂ is significant also in the non-recruitable patients, in whom the decrease in $Q_{\rm s}/Q_{\rm t}$ cannot be solely explained by re-aeration of alveoli with a low VA/Q. Thus, the effect of PEEP on oxygenation may be—at least in part—explained by a reduction in $Q_{\rm s}/Q_{\rm t}$ due to a PEEP-related decrease in cardiac output [6].

 ${\rm PaCO_2}$ is mainly dependent on dead space $(V_{\rm D}/V_{\rm T}).$ Lung volume has an ambiguous, "U" shaped, effect on $V_{\rm D}/V_{\rm T},$ depending mostly on the recruitment response to the applied PEEP levels [7]. Indeed, opening of previously closed lung units may reduce $V_{\rm D}/V_{\rm T}$ and improve ${\rm CO_2}$



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elimination. However, if the increase in aerated units is accompanied by an even higher increase in overinflated units, this leads invariably to an increase in $V_{\rm D}/V_{\rm T}$ and in PaCO₂ [7].

Alveolar recruitment and VILI

Overall, the above presented data would suggest that recruitment of collapsed alveolar units should be maximized in patients with high potential for recruitment to improve their outcome [1]. Nevertheless, evidences failed to conclusively demonstrate that strategies leading to "full recruitment" improve outcome, and application of these strategies is prudently suggested (and not recommended) by recent guidelines [8]. Two recent randomized clinical trials showed that (a) maximization of lung recruitment through the use of high-frequency oscillatory ventilation was associated with higher mortality [9]; (b) mechanical ventilation with higher PEEP titrated on best lung compliance had higher mortality compared with patients treated with lower PEEP [10]. We must underline that (a)

high-frequency oscillatory ventilation substantially differs from conventional mechanical ventilation; (b) these studies used completely different ventilatory approaches that were applied regardless of individual assessment of patient physiology. Nevertheless, these results seem to point out that maximizing recruitment may not be always the right strategy to minimize alveolar overdistension despite the delivery of low tidal volumes since "high potential for recruitment" [5] may in fact be just a marker of ARDS severity, rather than a pathological feature that need to be reversed. In addition, the change in lung inhomogeneity with mechanical ventilation may be unpredictable [11], and its magnitude may critically modify the relationship between total lung volume and tidal volume.

Lung collapse is not always detrimental. During fetal development, the lung remains collapsed and yet healthy, with gas exchange provided by the placenta. Moreover, the collapsed and infiltrated lung in patients with lobar pneumonia that are not mechanically ventilated can completely heal without any artificial lung recruitment

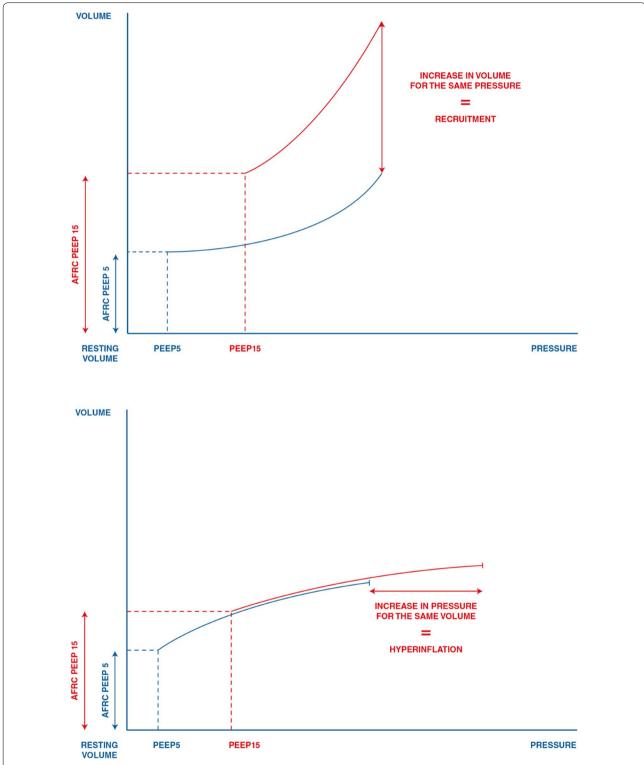


Fig. 2 Quantitative analysis of the volume–pressure curve of the respiratory system to identify and quantify recruitment with PEEP. Top: alveolar recruitment; PEEP shifted upward the curve showing an increase in volume for the same pressure. Bottom: alveolar hyperinflation; PEEP shifted the curve along the pressure axis causing an increase in pressure for almost the same volume. PEEP is positive end-expiratory pressure; resting volume is the volume at the elastic equilibrium point of the respiratory system. ΔFRC is the change in functional residual capacity caused by different levels of PEEP. Modified from Reference 4

maneuver. Furthermore, the first successful treatment provided to patients with pulmonary infection caused by Mycobacterium tuberculosis was the induction of therapeutic pneumothorax with consequent lung collapse [12]. Although fetal development, lobar pneumonia, and M. tuberculosis infection have nothing to do with ARDS, and to date, no clinical trial has irrevocably shown that a "partial recruitment" strategy would result in a better outcome, it should be noted that recent data suggest that the "partial recruitment" approach may better protect the lung from VILI compared to "full recruitment" approaches. Bellani et al. showed in patients with ARDS that aerated pulmonary regions are more inflamed than the collapsed alveolar units [13]. In an ex vivo models, Chu et al. showed that atelectatic lungs had less inflammatory cytokines compared to recruited lungs [14] and Fanelli et al. showed that a "partial recruitment" strategy significantly reduced apoptosis when compared to a "full recruitment" strategy [15].

In conclusion, it remains unknown whether achieving and then maintaining maximal alveolar recruitment are required to minimize lung injury and facilitate lung healing. The clarification of important issues in the pathophysiology of ARDS, such as the interaction between tidal volume and total lung volume (drastically affected by potential for recruitment) and the optimal compromise between alveolar recruitment and overdistension, will guide the design of the future successful clinical trials to improve outcome in patients with ARDS.

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Compliance with ethical standards

Conflicts of interest

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