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Recruitment Maneuvers in Acute Respiratory Distress Syndrome: Do They Harm?

Antonia Koutsoukou[1]

1 1st Department of Respiratory Medicine, "Sotiria" Hospital for Diseases of the Chest, University of Athens Medical School, Athens, Greece

Correspondence:
Antonia Koutsoukou, MD

Koutsoukou@yahoo.gr

Associate Professor, ICU, 1st Department of Respiratory Medicine, "Sotiria" Hospital for Diseases of the Chest, University of Athens Medical School, 152 Mesogion Ave, 115 27 Athens Greece

Tel: +30 2107763718

Fax: +30 2107781250

Acute respiratory distress syndrome (ARDS) represents a serious problem in critically ill patients and is associated with an in-hospital mortality between 33 to 52%. It is characterized by severe impairment of oxygenation caused by an inhomogeneous ventilation-perfusion distribution and an increase in shunt fraction [1]. The amount of aerated lung volume is markedly reduced due to alveolar collapse and flooding [1]. Lung protective mechanical ventilation based on low tidal volume (V_T) and positive end-expiratory pressure (PEEP) mechanical ventilation been recommended to improve outcome in ARDS patients [2]. However, low V_T may yield a progressive derecruitment with atelectasis leading to deterioration in respiratory function. Recruitment maneuvers (RMs) have been proposed to open the collapsed lung tissue in these patients. Lung recruitment is defined as the re-aeration of previously collapsed lung alveoli through an intentional transient increase in airway pressure [3]. The rationale for the use of RMs in ARDS is to gain patency of refractory lung units, thus leading to increased end-expiratory lung volume, improvement of gas exchange, and to reduce amount of intra-tidal lung opening and closing, one of the main determinants of ventilator-induced lung injury (VILI). Changes in oxygenation and respiratory system elastance have been used to evaluate RMs response. However, alveolar recruitment is an anatomical phenomenon, whereas arterial oxygenation may be influenced by ventilation-perfusion distribution, or reduction in cardiac output. Chest computed tomography is the gold standard for identifying the 'anatomical' lung recruitment [4], but it is not feasible for bedside routine measurements in ARDS patients.

Several studies have confirmed, though not consistently, a beneficial effect of RMs on oxygenation and/or respiratory mechanics [5]. Inconsistent findings may be due to: a) heterogeneity of patients studied, etiology and phase of ARDS; b) the characteristics of the recruiting technique, i.e., the way a recruiting pressure is applied plus the duration of its application; and c) the magnitude of the effective recruiting pressure, that is the generated transpulmonary pressure (P_{TP}), but not the applied airway pressure [6]. The P_{TP} (applied airway pressure - pleural pressure; P_{AW}-P_{PL}) is a function of both the applied pressure and changes occurring in the pleural pressure [6]. Consequently, conditions that affect P_{PL} may substantially and unpredictably play a role on the effectiveness of RMs [7]. After achieving recruitment, it is important to sustain the previously collapsed areas open by applying sufficient PEEP levels.

In several studies, diverse RMs methods have been described, such as sustained inflation, intermittent or extended sighs, and pressure control ventilation with higher airway pressures, with the sustained inflation being the most popular [5]. Despite the increasing body of literature, few studies have compared the various methods in terms of efficacy or adverse effects and the best RM technique is yet unknown.

Recruitment maneuvers are not without risks. Applying high distending pressures, even for a short period, may compromise lung parenchymal and vascular function via overdistention. The negative impact on pulmonary physiology of high airway pressures during mechanical ventilation has been known for

over 4 decades [8]. The most commonly studied adverse events of RMs are hemodynamic compromise and barotrauma. High airway pressures may impair hemodynamics by impeding venus return, or by increasing pulmonary vascular resistance and right ventricular afterload. Transient hemodynamic changes have been reported by a number of investigators during RMs, but persistent hemodynamic instability post RMs has not been an issue [5]. In this regard, it seems that ensuring patients' volumic status before RMs implementation may attenuate potent circulatory depression. Barotrauma is another common concern, given the high airway pressures applied during aggressive RMs. Fortunately, RMs associated pneumothorax has been reported to be rare (1%) [5].

Another issue that needs consideration, however, is the biological impact that high distending pressures might have on lung tissue. In fact, recent experimental studies have shown that RMs may exacerbate epithelial and endothelial cell damage [9-13], and that this damage differs according to the etiology of acute lung injury and RM technique, being more pronounced if high airway pressures are applied abruptly as compared to stepwise approaches. In an animal ARDS model, Rzezinski et al. compared a single sustained inflation, in which positive airway pressure is abruptly raised, to a progressive RM in which the target end-inspiratory pressure was reached within 12 minutes. They found that lung inflammation, alveolar epithelial cell apoptosis, and alveolar-capillary membrane injury were significantly lower with progressive RM than with the common sustained inflation [9]. Similarly, in rats with paraquat-induced ARDS, Riva et al. compared a common sustained inflation to a RM in which the target pressure was reached after 40 seconds as a ramp. The RM generated as a ramp reduced overdistention, alveolar collapse, and lung expression of mRNA of procollagen III [10]. Santiago et al., were among the first who tested the hypothesis that RMs effects may differ depending on the severity of lung injury [11]. They found that a single sustained inflation induced a significantly greater endothelial and epithelial cells injury and apoptosis to the lungs and kidneys in severe rather than moderate ARDS in rats. Furthermore, it has been suggested that the detrimental effects of RMs may depend on the underlying disease. Pulmonary ARDS has typically a high degree of lung tissue consolidation, whereas extrapulmonary ARDS is associated with alveolar collapse that is potentially reversible. In different lung injury animal models, Riva et al found that three sustained inflations led to more hyperinflation and activation of fibrogenesis in pulmonary than in extrapulmonary lung injury [12]. Silva et al., [13] investigated the impact of various RMs strategies on pulmonary epithelial and endothelial cell injury in a rat model of primary and secondary endotoxin-induced ARDS. They found that all RMs improved respiratory mechanics, but the impact on the molecular and cellular lung components varied with RM strategy in both models. Recruitment maneuvers associated with a sudden increase in airway inspiratory pressure and peak flow yielded a greater lung injury, implying that the rate of inspiration and the flow velocity during inspiration are not of secondary importance. In fact, in normal open-chest rabbits high inflation flows have been shown to induce lung injury [14].

All these experimental data need to be confirmed in clinical studies before any conclusions on the practical significance of such findings can be reached. The problem is that a feasible bedside strategy to identify those patients in whom RMs will achieve and maintain alveolar recruitment without causing structural and biological damage to the lungs is yet to be found. Changes in respiratory mechanics and gas exchange, parameters that are traditionally used for assessing RMs effects, have limited value in evaluating these biological phenomena. Future research needs to be done in order to elucidate if the RMs associated transient injuries found in experimental models represent a risk for VILI in mechanically ventilated ARDS patients.

In conclusion, although the potential of RMs to cause harm remains unanswered, clinicians should not only consider the effects of RMs on hemodynamics and lung recruitment when they do apply RMs, but also on overdistention and biotrauma.

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