COMMENTARY

Covid-19-induced lung recruit ability and positive end-expiratory pressure setting in ARDS

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ABSTRACT

Mechanical ventilation is the gold standard therapy in ARDS for restoring normal gas exchange, but it can exacerbate lung damage by causing ventilator-induced lung injury when improper ventilator settings are used. Existing techniques for preventing ventilatorinduced lung damage include restricting tidal volume, plate--au and driving pressure, and widespread use of the prone position, all of which can improve survival. In contrast, the role Of Positive End Expiratory Pressure (PEEP) is debatable: while using low PEEP in mild-to-moderate appears prudent, there is no conclusive evidence to support higher vs lower PEEP in patients with moderate-to-severe disease.

Key Words: *Hyperinflation.*

INTRODUCTION

Five different randomized studies that compared higher vs lower PEEP, with high PEEP set according to respiratory system mecha--nics, oxygenation impairment, maximizing respiratory system compliance, or achieving various degrees of positive end-expiratory trans pulmonary pressure, failed to detect a significant clinical benefit.

Physiologically, PEEP always causes hyperinflation in the aerated compartment (ie, the baby lung), though it may reduce the risk of ventilator-induced lung injury only when significant alveolar recruitment occurs due to the reopening of collapsed tissue, ultimately increasing the size of the aerated lung available for tidal ventilation. Indeed, the potential for lung recruitment in response to PEEP varies greatly between individuals. PEEP should seek to strike a balance between its potential to recruit new alveoli and the unavoidable over-inflation caused in previously open tissue in early moderate-to-severe ARDS. High PEEP is only advantageous in patients with a higher potential for lung recruitment, in whom PEEP is increased.

Patients with COVID-19-induced acute respiratory failure are treated with relatively high PEEP (14 cm H2O on average), which may be because of the favourable oxygenation response to PEEP that is prevalent in these patients, which may be a deceptively comforting clinical finding. Improved oxygenation does not always imply alveolar recruitment and less harmful breathing, but it might be due to a PEEP-induced decrease in cardiac output, a shift in the distribution of alveolar perfusion, and/or hypoxic vasoconstriction. PEEP-induced substantial recruitment, on the other hand, may be accompanied by worsened or unaltered oxygenation when the intracardiac shunt is enhanced in the presence of a patent foramen ovale.

The effect of PEEP in patients with COVD-19 ARDS and whether the level of recruit ability is comparable to or different from ARDS from other causes are still debatable questions. There is well-conducted physiologic research in this issue of CHEST to investigate the potential for lung recruitment and its link with PEEP-induced alterations in respiratory mechanics in 40 patients with moderate-to-severe ARDS caused by COVID-19 shortly after intubation. The investigation was carried out meticulously, and the data was extensively examined. A CT scan was used to determine the potential for lung recruitment. Similar to other causes of ARDS, the data revealed significant variation in the potential for lung recruitment, supporting the hypothesis that PEEP should be personalized based on individual response in COVID-19 patients as well. A second important finding was that PEEP-induced changes in respiratory mechanics (compliance and driving pressure) provide no information about the potential for lung recruitment and may mislead clinicians; importantly, maximization of compliance (and reduction of driving pressure) is a popular PEEP-setting strategy. In the research, the occurrence of considerable recruiting was not regularly associated with increases in compliance. It is similar to what has previously been found in both COVID-19 and other forms of ARDS and may be explained by infant lung hyperinflation and the possibility of tidal recruitment at low PEEP. Compliance and driving pressure are global metrics that do not account for lung tissue's localised activity. Tidal recruitment is the cyclic opening and closing of alveolar units during tidal ventilation; tidal recruitment causes static respiratory system compliance to be very high at low PEEP, which explains why increases in PEEP may result in worsening compliance even when there is significant recruitment.

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The authors found that variations in PaCO2 caused by PEEP cannot predict the potential for lung recruitment. Changes in PaCO2 reflect changes in dead space with continual minute ventilation. With alveolar recruitment, tidal volume over distension should be reduced, resulting in a decrease in ventilation-perfusion mismatch and easier CO2 clearance. In the event of weakly recruitable lungs, alveolar dead space may rise due to pulmonary vascular compression, and airway dead space may increase due to gas compression in the respiratory circuit and airways; these processes should impede CO2 removal. However, CO2 dynamics are complicated and heavily influenced by hemodynamic equilibrium and the quantity of CO2 produced, potentially masking any PEEP-induced direct impact driven by these processes.

We believe the authors should be applauded for their well-executed study in such a difficult clinical context; the findings have significant clinical implications. First, the inter-individual variation in lung recruitment potential necessitates the development of techniques for individualizing the PEEP setting at the bedside. The most precise technique for measuring recruitment is a CT scan, but it needs expertise, resources, time, and transportation of the patient outsidethe ICU, which may not be accessible for all patients, particularly in the setting of a pandemic. Novel approaches based on electrical impedance tomography or simplified ventilator manoeuvres to estimate the extent of recruitment at the bedside have been proposed with promising results, necessitating further research to determine whether PEEP-setting strategies based on these monitoring are effective.

Second, routinely used metrics thought to be accurate for assessing the effect of PEEP, such as oxygenation, PaCO2, respiratory system compliance, and driving pressure, can mislead doctors. They do not represent the occurrence of alveolar recruitment and are hampered by the involvement of multiple complicated and interdependent physiologic systems, the effects of which cannot be distinguished using traditional bedside monitoring methods. The findings reflect a significant advancement in our knowledge of the complicated physiology that governs the interaction between the particular patient and ventilator. These findings underscore the need of developing techniques for individualizing PEEP settings in ARDS induced by COVID-19.