ICU
MANAGEMENT & PRACTICE

Effect of PEEP on PVR in Patients With Acute Respiratory Distress Syndrome

In mechanically ventilated patients with acute respiratory distress syndrome (ARDS), the use of positive end-expiratory pressure (PEEP) presents a therapeutic dilemma. PEEP can improve gas exchange and lung mechanics but may impair haemodynamics. Its effects primarily impact right ventricular (RV) preload and afterload, while left ventricular (LV) function is less affected. Increased intrathoracic pressure from PEEP reduces venous return, decreasing RV preload, which in turn reduces cardiac output (CO) if both ventricles are preload-dependent. Elevated transpulmonary pressure (PL) may increase pulmonary vascular resistance (PVR), further decreasing CO, potentially via a leftward septal shift.

The impact of PEEP on PVR is complex and depends on lung volume. Pulmonary vessels are classified into alveolar and extra-alveolar. When lung volume increases from collapse towards functional residual capacity (FRC), PVR decreases due to the expansion of extra-alveolar vessels. However, further lung distension above FRC compresses alveolar vessels, increasing PVR. This creates a U-shaped relationship between PVR and lung volume, with the nadir at FRC. The effect of PEEP on PVR depends on whether it promotes lung recruitment (decreasing PVR) or distension (increasing PVR).

A new study aimed to test the hypothesis that increasing PEEP would raise PVR if it induces lung distension but would minimally change or even decrease PVR if it induces lung recruitment. This hypothesis was tested in ARDS patients using a pulmonary artery catheter (PAC) and echocardiography, with lung recruitment assessed using the recruitment-to-inflation (R/I) ratio.

In patients with ARDS, haemodynamic, echocardiographic, and ventilatory variables were measured at low and high PEEP levels, with an increase of 10 cm H2O. Preload responsiveness was evaluated using the passive leg-raising test at high PEEP.

The study enrolled 23 patients, divided into 10 low recruiters (R/I < 0.5) and 13 high recruiters (R/I > 0.5). When PEEP was raised from 4 cm H2O to 14 cm H2O, pulmonary vascular resistance (PVR) increased in low recruiters (from 160 to 243 dyn·s/cm5), while it remained unchanged in high recruiters (from 224 to 235 dyn·s/cm5). The right-to-left ventricular end-diastolic area ratio increased in low recruiters (from 0.54 to 0.64) but remained stable in high recruiters (from 0.70 to 0.68). Raising PEEP decreased cardiac index only in preload-responsive patients.

This study suggests that, in patients with ARDS, the effect of PEEP on PVR depends on the extent of lung recruitment versus distension. When the recruitment-to-inflation (R/I) ratio was >0.5, indicating significant PEEP-induced recruitment, PVR remained unchanged with higher PEEP. However, when the R/I ratio was <0.5, suggesting PEEP-induced lung distension, PVR significantly increased. This was confirmed by changes in RV dimensions and function, highlighting the complex haemodynamic effects of PEEP.

The study confirms that PEEP decreases cardiac preload, leading to a decrease in CI only in preload-responsive patients. It also demonstrates the influence of lung recruitment on RV afterload, with increasing lung volume raising RV afterload through increased PVR and RV enlargement. The study observes that the relationship between PEEP-induced changes in PVR and lung recruitment is continuous, with RV afterload increasing progressively with alveolar distension during PEEP application. In low recruiters, changes in PVR were due to a change in the mean pulmonary artery pressure (MPAP) and pulmonary artery occlusion pressure (PAOP) gradient rather than cardiac output alone.

The study shows that PEEP-induced changes in CO and PVR are not solely explained by preload responsiveness, as both low and high recruiters had similar proportions of preload responders, and PEEP reduced CO equally in both groups. The study also supports the clinical idea that PEEP levels should be tailored based on the degree of lung recruitability. High PEEP is more beneficial in improving oxygenation and outcomes for patients with significant lung recruitment, and it has a lesser impact on RV afterload in these patients.

Overall, these findings show that in patients with ARDS receiving protective ventilation, PEEP increases PVR and RV afterload only in those with low lung recruitability. In contrast, PEEP has no effect in patients with high lung recruitability. Additionally, high PEEP decreases cardiac preload, which affects CO only in preload-responsive patients. These findings suggest that both lung recruitability (assessed by the recruitment-to-inflation ratio) and the degree of preload responsiveness should be considered when personalising PEEP levels based on the patient's condition.

Source: American Journal of Respiratory and Critical Care Medicine
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Published on : Mon, 21 Oct 2024