Intensive Care Med
https://doi.org/10.1007/s00134-026-08530-2

UNDERSTANDING THE DISEASE

Balancing lung recruitment and venous congestion in ARDS: rethinking the systemic effects of PEEP

Martín H. Benites[1,2,3], Laurent Papazian[4,5] and Jaime Retamal[1*]

Positive end-expiratory pressure (PEEP) is a cornerstone of mechanical ventilation in acute respiratory distress syndrome (ARDS), improving oxygenation and promoting alveolar stability. However, randomized trials comparing higher versus lower PEEP levels have not demonstrated that these physiological benefits translate into a survival advantage [1, 2]. This apparent dissociation suggests that improvements in respiratory physiology may be counterbalanced by adverse hemodynamic and systemic consequences, particularly when PEEP is not appropriately tailored to lung recruitability and the underlying mechanical phenotype.

Despite its central role in critical care, PEEP titration remains largely blind to its systemic effects. Clinically, these effects may manifest as elevated central venous pressure, reduced urine output, increased intracranial pressure, gastrointestinal dysfunction, and biochemical signs of organ dysfunction despite improved oxygenation [3, 4]. In this context, the respiratory benefits of mechanical ventilation may coexist with inflammatory activation, contributing to both local lung injury and systemic effects [5]. At the same time, mechanical ventilation imposes hemodynamic costs that are not captured by conventional monitoring. These effects are traditionally interpreted as reductions in cardiac output and left ventricular preload, yet this framework does not fully explain the patterns of organ dysfunction observed in ventilated patients. In this setting, venous congestion may represent an important hemodynamic mechanism in ARDS, linking ventilatory-induced circulatory alterations to systemic organ dysfunction [3]. Despite its potential physiological relevance, this pathway remains insufficiently characterized, and its systematic assessment and therapeutic implications across different mechanical phenotypes are not yet well defined.

Venous congestion in ARDS

According to Guyton's framework, venous return is determined by the gradient between the mean systemic filling pressure and right atrial pressure and is modulated by venous resistance [6]. PEEP can simultaneously affect both components. On the one hand, it increases pulmonary vascular resistance, thereby increasing right ventricular afterload and right atrial pressure [7]. On the other hand, transmission of airway pressure to the pleural space, together with diaphragmatic displacement, promotes vena cava compression and impairs venous return [8, 9]. The net result is impaired venous drainage from the abdominal organs and a reduction in the effective perfusion gradient, even as systemic oxygenation improves [8, 9].

In this context, venous congestion can be understood as a distinct and underappreciated pathophysiological pathway linking positive pressure ventilation to organ perfusion. Increased intrathoracic pressure impairs venous outflow, elevates central venous pressure, and reduces the effective perfusion gradient, even when arterial pressure and oxygenation appear to be preserved [6, 8]. The kidneys are often among the first organs to reflect these changes, as increased renal venous pressure compromises the filtration gradient [3].

The liver is similarly vulnerable, as even modest elevations in hepatic venous pressure can impair sinusoidal

*Correspondence: jaimeretamal@gmail.com
[1] Departamento de Medicina Intensiva, Facultad de Medicina, Pontificia Universidad Católica de Chile, Santiago, Chile
Full author information is available at the end of the article

flow and oxygen diffusion [10]. In the gastrointestinal tract, elevated venous pressure compromises mucosal perfusion, increases intestinal permeability, and limits lymphatic drainage [11]. At the cerebral level, the transmission of intrathoracic pressure to the jugular system may reduce cerebral perfusion pressure, particularly in patients with reduced intracranial compliance [4].

Together, these observations converge into a consistent physiological pattern: impaired venous drainage, leading to systemic venous congestion.

Mechanical profiles in ARDS

Critically, these circulatory effects are determined not only by the applied PEEP level, but also by its interaction with lung recruitability, respiratory system compliance, and pleural pressure transmission, a distinction with direct therapeutic implications in ARDS. Three representative mechanical phenotypes illustrate this concept.

1. In lungs with low respiratory system compliance and poor recruitability, increased airway pressure predominantly raises the transpulmonary pressure, promoting alveolar overdistension and pulmonary capillary compression. This increases pulmonary vascular resistance and right ventricular afterload, elevates right atrial pressure, and promotes retrograde systemic venous congestion [7].
2. In highly compliant lungs with limited recruitability, airway pressure is preferentially transmitted to the pleural space. Diaphragmatic displacement and thoracic expansion compress the inferior vena cava, limiting venous return and promoting splanchnic congestion through extrapulmonary pressure transmission and impaired venous return rather than pulmonary vascular mechanisms [8, 9].
3. In patients with low lung compliance and preserved recruitability, effective alveolar recruitment with PEEP may increase the end-expiratory lung volume and improve lung homogeneity. Under these conditions, PEEP may attenuate hypoxic pulmonary vasoconstriction and reduce pulmonary vascular resistance, thereby facilitating right ventricular unloading while preserving venous return. When the left ventricle operates on the ascending limb of the Frank–Starling curve, as in relative hypovolemia, increased preload may translate into improved cardiac output and systemic perfusion [12] (Fig. 1).

Across these mechanical profiles, the circulatory response to PEEP reflects the interaction among lung recruitability, pressure transmission, and preload conditions rather than the applied pressure alone. Similar phenotype-dependent responses have been reported in acute brain injury, where the effects of PEEP on intracranial dynamics appear influenced not only by respiratory mechanics and alveolar recruitment, but also by the physiological characteristics of the target organ [13].

These physiological interactions may differ during spontaneous breathing or non-invasive ventilation. Unlike passive positive-pressure ventilation, spontaneous inspiratory effort generates negative pleural pressure swings that may preserve venous return and facilitate transdiaphragmatic venous and lymphatic drainage [11]. During non-invasive ventilation, positive airway pressure may partially counteract these effects, although improved lung recruitment and reduced inspiratory effort may simultaneously attenuate hemodynamic stress and patient self-inflicted lung injury [14]. Thus, the hemodynamic effects of non-invasive respiratory support likely depend on the balance between pressure transmission, inspiratory effort, and lung recruitment.

Clinical implications

These considerations reframe PEEP titration as a systemic decision rather than as a purely respiratory adjustment. At the bedside, interpreting the physiological impact of a PEEP change requires answering three key questions beyond oxygenation: Did respiratory system compliance improve (recruitment vs. overdistension)? How did biventricular function and cardiac output respond to this change? Are there signs of evolving venous congestion?

Importantly, improvements in respiratory mechanics do not necessarily imply improved global physiology. Strategies focused primarily on oxygenation or driving pressure may overlook the cardiovascular consequences of positive pressure ventilation, potentially contributing to the dissociation between pulmonary optimization and clinical outcomes [15].

Declining urine output, rising central venous pressure, abnormal venous Doppler patterns consistent with venous congestion (e.g., Venous Excess Ultrasound (VExUS)), and echocardiographic evidence of right ventricular dysfunction may indicate adverse circulatory effects despite improved gas exchange and should prompt reassessment rather than further escalation of airway pressure.

Conclusion

PEEP reshapes venous return and systemic venous pressure beyond the lung. Across different mechanical phenotypes, these effects may converge into systemic venous congestion, despite improved oxygenation. Current approaches to PEEP titration remain largely insensitive to these consequences, creating a disconnect between pulmonary optimization and global hemodynamic status. Therefore, optimizing the

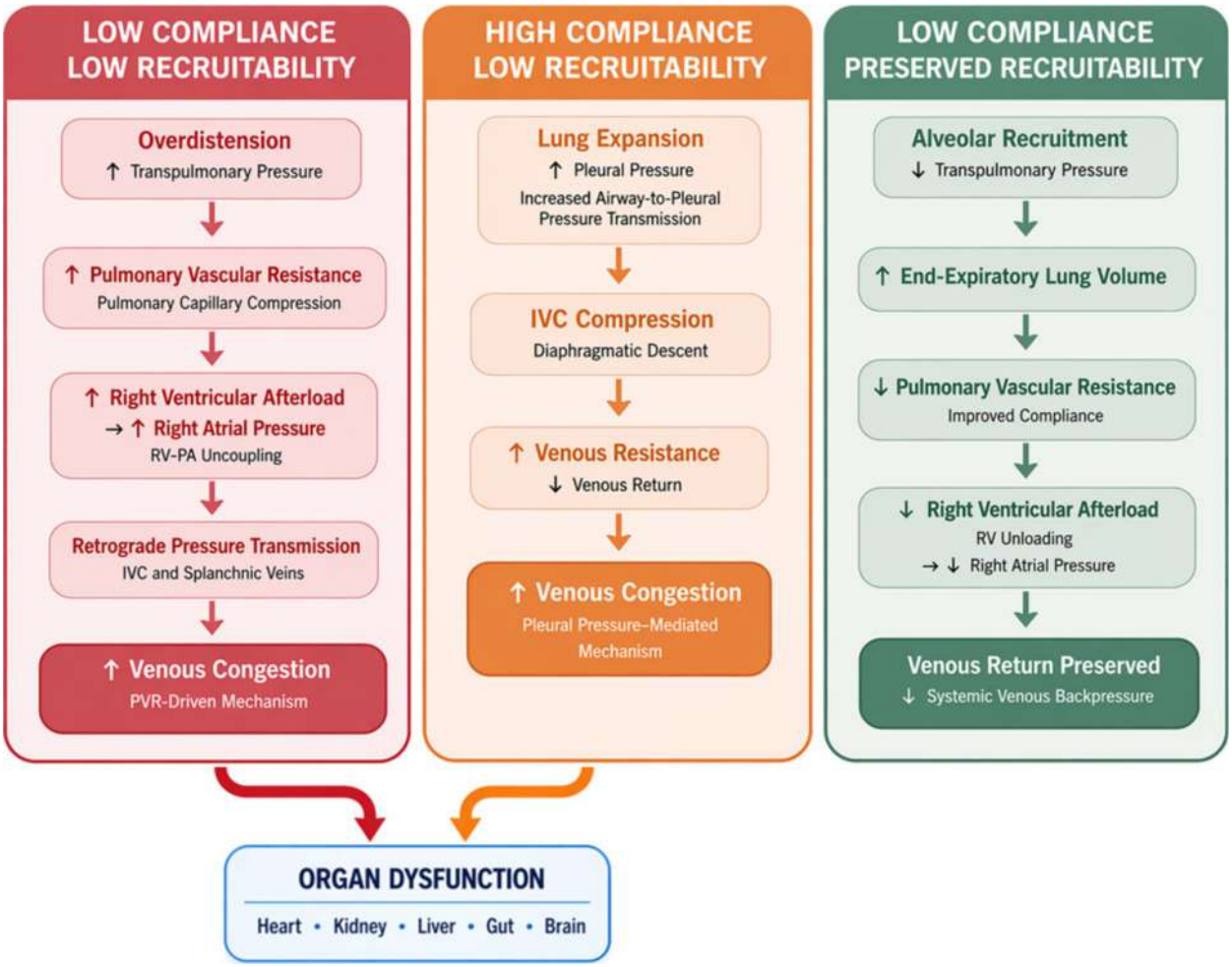

Fig. 1 Hemodynamic effects of PEEP across mechanical phenotypes in ARDS. PEEP induces distinct hemodynamic responses depending on lung recruitability, respiratory system compliance, and pressure transmission. In poorly recruitable phenotypes, increased transpulmonary distending pressure or pleural pressure transmission may promote venous congestion through pulmonary vascular effects or impaired venous return related to pleural pressure transmission. In contrast, effective alveolar recruitment may reduce pulmonary vascular resistance, facilitate right ventricular unloading, and preserve venous return. These phenotype-dependent responses may promote systemic venous congestion and organ dysfunction or preserve circulatory homeostasis despite similar effects on oxygenation. *PEEP* positive end-expiratory pressure, *ARDS* acute respiratory distress syndrome, *RV* right ventricle/right ventricular, *RAP* right atrial pressure, *IVC* inferior vena cava

ventilatory strategy requires balancing lung mechanics with the preservation of venous return and organ perfusion, supporting a physiology-guided, individualized approach in which the lung and circulation are managed as a single integrated system.

Author details
[1] Departamento de Medicina Intensiva, Facultad de Medicina, Pontificia Universidad Católica de Chile, Santiago, Chile. [2] Centro de Pacientes Críticos, Clínica las Condes, Santiago, Chile. [3] Facultad de Medicina, Escuela de Medicina, Universidad Finis Terrae, Santiago, Chile. [4] Centre Hospitalier de Bastia, Bastia, France. [5] Unité Des Virus Émergents, UVE Aix-Marseille Univ, Università Di Corsica, IRD 190, Inserm 1207, IRBA, Marseille, France.

Funding
This work was supported by ANID–FONDECYT Grant 1241897 (Chile).

Availability of data and materials
Not applicable.

Declarations

Conflicts of interest
The authors declare no conflict of interest.

Ethics approval
Not applicable.

Consent to participate
Not applicable.

Consent for publication
Not applicable.

Publisher's Note

Received: 20 April 2026 Accepted: 13 June 2026
Published online: 22 June 2026

References

1. Brower RG, Lanken PN, MacIntyre N et al (2004) Higher versus lower positive end-expiratory pressures in patients with the acute respiratory distress syndrome. N Engl J Med 351(4):327–336
2. Mercat A, Richard JCM, Vielle B et al (2008) Positive end-expiratory pressure setting in adults with acute lung injury and acute respiratory distress syndrome: a randomized controlled trial. JAMA 299(6):646–655
3. Benites MH, Suarez-Sipmann F, Kattan E et al (2025) Ventilation-induced acute kidney injury in acute respiratory failure: do PEEP levels matter? Crit Care 29(1):130
4. Iavarone IG, Rocco PRM, Grieco DL et al (2025) Pathophysiology and clinical applications of PEEP in acute brain injury. Intensive Care Med 51(11):2104–2116
5. Tremblay LN, Slutsky AS (2006) Ventilator-induced lung injury: from the bench to the bedside. Intensive Care Med 32(1):24–33
6. Persichini R, Lai C, Teboul JL et al (2022) Venous return and mean systemic filling pressure: physiology and clinical applications. Crit Care 26(1):150
7. CappioBorlino S, Hagry J, Lai C et al (2024) The effect of positive end-expiratory pressure on pulmonary vascular resistance depends on lung recruitability in patients with acute respiratory distress syndrome. Am J Respir Crit Care Med 210(7):900–907
8. Fessler HE, Brower RG, Shapiro EP et al (1993) Effects of positive end-expiratory pressure and body position on pressure in the thoracic great veins. Am Rev Respir Dis 148(6 Pt 1):1657–1664
9. Feihl F, Broccard AF (2009) Interactions between respiration and systemic hemodynamics. Part I: basic concepts. Intensive Care Med 35(1):45–54
10. Brienza N, Ayuse T, O'Donnell CP et al (1995) Regional control of venous return: liver blood flow. Am J Respir Crit Care Med 152(2):511–518
11. Lattuada M, Bergquist M, Maripuu E et al (2013) Mechanical ventilation worsens abdominal edema and inflammation in porcine endotoxemia. Crit Care 17(3):R126
12. Santos A, Lucchetta L, Monge-Garcia MI et al (2017) The open lung approach improves pulmonary vascular mechanics in an experimental model of acute respiratory distress syndrome. Crit Care Med 45(3):e298–e305
13. Barea-Mendoza JA, Llompart-Pou JA, Ballesteros-Sanz MÁ et al (2026) Effects of PEEP on ICP in ABI patients and its relationship with etiology. Intensive Care Med 52:629–630
14. Duan J, Liu X, Shu W et al (2025) Low versus high positive end expiratory pressure in noninvasive ventilation for hypoxemic respiratory failure: a multicenter randomized controlled trial. Intensive Care Med 51:861–869
15. Futier E, De Jong A, Cirenei C et al (2025) Personalized driving pressure-guided positive end-expiratory pressure in patients at risk of postoperative respiratory failure (IMPROVE-2): a multicenter, pragmatic, randomized clinical trial. Intensive Care Med 51:1797–1808