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CORRESPONDENCE

Positive or negative pressure: plus ça change, plus c'est la même chose

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We have read with interest the correspondence by Roberts et al. [1] in response to our article [2].

While we agree that negative pressure ventilation (NPV) is generally overlooked in the management of acute respiratory distress syndrome (ARDS), some of the points raised by the authors in their correspondence deserve further discussion. For instance, when they assert that "VILI is not caused by MV itself, but by the supra-atmospheric pressure generated in Positive Pressure Ventilation (PPV)".

A body of evidence indicates that excessive change in lung volume (strain) and the time of exposure to this strain are the primary cause of ventilatory induced lung injury (VILI).

The excessive strain is not caused by the positive airway pressure per se, but by a large difference between the airway and pleural pressure. The same transpulmonary pressure can result in the same risk of VILI regardless of whether this is generated by a positive or negative pleural pressure [3], which may result from either respiratory muscle activity [4–6] or a negative pressure ventilator. The excessive negative pressure can cause pulmonary and hemodynamic consequences, which can be more fully understood if we consider the determinant of pleural pressure as described by equation (1):

$$\Delta P_{pl} = \Delta P_{aw}\,\Delta\frac{E_w}{E_{TOT}} - \Delta P_{musc}\,\Delta\frac{E_L}{E_{TOT}}. \quad (1)$$

This equation describes the changes in pleural pressure when both mechanical ventilation and muscular activity are present. The E_w/E_{TOT} is the ratio between the chest-wall elastance and total respiratory system elastance, while E_L/E_{TOT} is the ratio between the lung elastance and the total respiratory system elastance. The effect of an artificial mechanical negative pressure ventilation is essentially equivalent to the muscular activity during spontaneous or assisted ventilation. In the patients where the negative pressure device is applied, the ΔP_{pl} generated by the negative pressure ventilation ΔP_{NPV} on the pleural pressure ($-\Delta P_{pl}$) depends on the ratio between E_L/E_{TOT}:

$$\Delta P_{pl} = -\Delta P_{NPV}\,\Delta\frac{E_L}{E_{TOT}}. \quad (2)$$

A high E_L/E_{TOT} ratio (high lung elastance) results in an excessively negative pleural pressure with significant effects on hemodynamics and risk of VILI. On the contrary, a normal E_L/E_{TOT} ratio has only modest effects on the pleural pressure, hemodynamics or the risk of VILI. Therefore, we may expect a completely different effect of the same applied negative pressure ventilation in patients with ARDS, obesity, or with neuromuscular disease, in whom the E_L/E_{TOT} ratio varies considerably.

Given these premises, it is understood that if the negative pleural pressure is excessive, the hemodynamic consequences are straightforward, leading to an increase in: (1) the venous return (up to a certain point); (2) pulmonary blood; (3) hydrostatic pressure. Positive or negative pleural pressure that generates the same level of lung inflation will produce equivalent effects on ventricular size and hemodynamic response. Furthermore, in the context of a failing left ventricle, negative pleural pressure can augment transmural pressure and elevate ventricular wall stress, potentially

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exacerbating ventricular failure and pulmonary edema. This can lead to compression atelectasis and worsening gas exchange.

Considering these factors, we stand by the view that 'safe ventilation' is elusive, justifying the use of extracorporeal support. Excessive transpulmonary pressure, whether positive or negative, remains a significant contributor to VILI. Our challenge is to treat lung disease effectively while mitigating injury risks. We remain open minded but skeptical about the effectiveness of negative pressure ventilation.

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Data availability
Not applicable.

Declarations

Conflicts of interest
The authors declare that they have no conflict of interest.

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References

1. Roberts JHM, Vanhoutte J, Howard DJ et al (2024) Negative pressure ventilation, an umbrella against ventilator induced lung injury. Intensive Care Med. https://doi.org/10.1007/s00134-024-07416-5
2. Gattinoni L, Collino F, Camporota L (2024) Ventilator induced lung injury: a case for a larger umbrella? Intensive Care Med 50:275–278. https://doi.org/10.1007/s00134-023-07296-1
3. Sattari S, Mariano CA, Kuschner WG et al (2022) Positive- and negative-pressure ventilation characterized by local and global pulmonary mechanics. Am J Respir Crit Care Med 207:577–586. https://doi.org/10.1164/rccm.202111-2480oc
4. Barach AL, Martin J, Eckman M (1938) Positive pressure respiration and its application to the treatment of acute pulmonary edema. Ann Intern Med 12:754. https://doi.org/10.7326/0003-4819-12-6-754
5. Mascheroni D, Kolobow T, Fumagalli R et al (1988) Acute respiratory failure following pharmacologically induced hyperventilation: an experimental animal study. Intensiv Care Med 15:8–14. https://doi.org/10.1007/bf00255628
6. Brochard L, Slutsky A, Pesenti A (2017) Mechanical ventilation to minimize progression of lung injury in acute respiratory failure. Am J Respir Crit Care Med 195:438–442. https://doi.org/10.1164/rccm.201605-1081cp