Least damaging invasive ventilation during extracorporeal respiratory support after lung transplantation

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We were interested to read of the surgical experience at Turin over the last decade with 25 veno-venous (VV) extracorporeal membrane oxygenation (ECMO) runs in support of cadaveric sequential lung transplantation (1). This is a great piece of work indeed in an expanding application of artificial organs.

There are certain points of interest to be discussed from the cardiothoracic intensivist’s and perioperative physician’s corner:

(I) ‘Graft protective’ invasive ventilation: Boffini et al. did not include perioperative or VV-ECMO ventilatory strategy variables such as tidal volume, plateau pressure ($P_{\text{plat}}$), positive-end expiratory pressure (PEEP) or driving pressure ($\Delta P = P_{\text{plat}} - \text{total PEEP}$) in the univariate or multivariate analyses of risk factors for postoperative need for VV-ECMO, liberation from ECMO or in-hospital mortality (1). Any positive pressure ventilation could in theory lead to ventilator-induced lung injury (VILI) and it has been shown that healthy (‘non-injured’) lungs are at risk of subclinical VILI (2). There is lack of rigorous prospective data relating to the optimal invasive ventilatory strategy for the deceased organ donor and lung transplant recipient. One might argue that even if a ‘low tidal volume’ ventilation was utilized in the study cohort, primary graft dysfunction due to VILI is still a possibility. It stands to reason that during VV-ECMO support in the context of primary graft dysfunction (‘injured lungs’) a ‘least damaging’ or ‘ultra-protective’ ventilatory strategy is adopted. ‘Ultra-low’ tidal volumes (3–4 mL/kg predicted body weight) to limit total lung and dynamic strain as reflected by $P_{\text{plat}}$ ($P_{\text{plat}} < 25 \text{ cmH}_2\text{O}$), combined with low respiratory rate (5–10 breaths per minute), and low fraction of inspired oxygen ($\text{FiO}_2 < 0.4$) seems a reasonable ‘graft-protective’ strategy that could potentially improve outcomes in lung transplant patients on VV-ECMO support (3). Driving pressure in patients with severe acute respiratory distress syndrome (ARDS) requiring VV-ECMO has been shown to be independently associated with in-hospital mortality (3,4). The aforementioned parameters of pulmonary mechanics during VV-ECMO for primary graft dysfunction, their role in mitigating VILI and the concept of elimination of invasive ventilation during VV-ECMO support should be tested in large scale prospective trials.

(II) Right ventricle (RV)-protective ventilation: VV ECMO reverses hypoxemia and hypercapnia and also facilitates low volume/low pressure mechanical ventilation leading to unloading of the RV which may be impaired in acute severe respiratory failure due to increased RV afterload (5). Perioperative RV injury in the setting of primary...
graft dysfunction could potentially worsen outcomes and echocardiography-guided ventilatory management during VVECMO support may confer hemodynamic benefit.

Minimizing VILI and RV injury in the high risk lung transplant patient population using least injurious ventilation settings (low tidal volume, low $P_{\text{plat}}$, low $\Delta P$) is paramount. The causative link, if any, between VILI, RV dysfunction and primary graft dysfunction in lung transplantation and the protective role of perioperative extracorporeal respiratory support is yet to be determined.

The effect of extracorporeal technologies and non-injurious ventilation strategies on patient-centred outcomes in recipients with primary graft dysfunction needs to be tested in large randomized controlled trials.

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Footnote

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References


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