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Commentary: Venovenous extracorporeal membrane oxygenation: The surgeon's clew

Chase Donaldson, MD, and
Andrew D. Shaw, MB, FRCA, FFICM

In the story of Theseus and the minotaur, Theseus is given a clew, or a ball of string, by the princess Ariadne of Crete when entering the labyrinth as means to escape upon completion of his mission. Without the string, even though he may have accomplished his aim of slaying the minotaur, he would have been lost in the labyrinth and perished. Likewise, in cardiothoracic surgery, although technical success in the operating theater of the procedure may be successfully accomplished, hemodynamic instability and specifically refractory hypoxia may occur that limit the ability to liberate the patient from cardiopulmonary bypass or achieve the necessary stability for the patient to recover in the intensive care unit. Venovenous (VV) extracorporeal membrane oxygenation (ECMO) can function as the clew to bridge the patient across the acute process that resulted in refractory hypoxemia and allow the patient to safely leave the operating theater. In this issue, Copeland and colleagues¹ provides a concise review of mechanisms for hypoxia in cardiothoracic surgery patients as well as the indications, contraindications, procedure, and management of VV ECMO in this patient population.

As the risk profile of patients undergoing cardiothoracic surgery continues to increase,² the need for more sophisticated and typically invasive modalities of postoperative respiratory care will likely follow, including postoperative ECMO support. However, a significant complicating factor in the evaluation and treatment of refractory hypoxia in the

Andrew D. Shaw, MB, FRCA, FFICM

CENTRAL MESSAGE

Postoperative hypoxia is due to a variety of causes other than ARDS and VV ECMO remains an important therapeutic option to bridge the cardiothoracic surgery patient to lung recovery.

cardiothoracic surgery patient is the heterogeneity of etiologies for the hypoxia. In addition to the typical causes of acute respiratory distress syndrome (ARDS) described by the authors, a significant burden of postcardiothoracic surgery refractory hypoxia may be due to pulmonary edema from a cardiogenic source as a result of mitral regurgitation, left ventricular systolic and diastolic dysfunction, volume overload, and acute right ventricular failure causing left ventricular failure. These etiologies are definitionally distinct from ARDS, and management may vary on account of differing mechanisms of alveolar injury, specifically damage to epithelial and endothelial barriers as well as the relative sensitivity to driving pressures in the traditional pathophysiology of ARDS.³

Extrapolating management strategies and outcomes from ARDS trials⁴ or even the use of ECMO in ARDS may also be problematic because the populations studied differ significantly from those in cardiothoracic surgery. For example, sepsis and pneumonia cause up to 80% of cases of ARDS and are thus rarely seen in the immediate postoperative course. Instead, typical etiologies of intraoperative or postoperative ARDS described by Copeland and colleagues¹ include inflammation from cardiopulmonary bypass, aspiration of gastric contents, transfusion reactions, ischemia-reperfusion injury, and atelectasis, all of which typically follow a shorter clinical course and often demonstrate more rapid improvement than is seen with sepsis and pneumonia. The short-lived hypoxic effects of many of these etiologies

From the Department of Intensive Care and Resuscitation, Cleveland Clinic Foundation, Cleveland, Ohio.

Disclosures: The authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

Received for publication Oct 26, 2021; revisions received Oct 26, 2021; accepted for publication Oct 26, 2021; available ahead of print Nov 10, 2021.

Address for reprints: Andrew D. Shaw, MB, FRCA, FFICM, Department of Intensive Care and Resuscitation, Cleveland Clinic Foundation, 9500 Euclid Ave, Cleveland, OH 44195 (E-mail: shawad8@ccf.org).

JTCVS Open 2021;8:104-5

2666-2736

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<https://doi.org/10.1016/j.jtcn.2021.10.053>

makes postoperative VV ECMO an ideal strategy to bridge the postoperative cardiothoracic surgery patient to lung recovery. At our institution, intraoperative venous or arterial sheaths are commonly placed in patients at very high risk of postoperative cardiac or pulmonary failure in anticipation of the potential need for veno-arterial or VV ECMO.

Outcomes of VV ECMO in postcardiothoracic surgery are poorly characterized in the literature outside of the lung transplantation population, but its use is commonly accepted and regardless of etiology of the refractory hypoxia, it can be Ariadne's gift for the patient's treatment team in the operating theater.

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