Issues in the Intensive Care Unit for Patients with Extracorporeal Membrane Oxygenation

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KEYWORDS

• Intensive care • ECMO • Circulation • Ventilator management • End-organ failure

KEY POINTS

- Extracorporeal corporeal oxygenation (ECMO) flow should be maintained to perfuse and recover the end organs.
- Ventilator management involves lung protective ventilator strategies.
- Anticoagulation during ECMO should be appropriately monitored.
- Near-infrared tissue oximetry is an important tool to assess cerebral and limb perfusion.

INTRODUCTION

Critically ill patients require very thorough physical and laboratory assessments to address medical and surgical issues. Patients on extracorporeal corporeal oxygenation (ECMO) support require an additional level of attention. Much of the focus for these patients revolves around cardiovascular support or ventilator needs. The effects of multiorgan dysfunction syndrome can ravage ECMO patients and require multiple levels of support beyond the traditional cardiovascular support. Special consideration must be given to heart–vasopressor–lung–ventilator–ECMO interactions, but that is not all. Attention must be given to patients requiring renal support to correct metabolic derangements. Hepatic support may be necessary to mitigate the effects of hepatic failure. Clinicians must understand that ECMO is a support system to allow organs

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to recover, and patients often get worse before they get better. This article addresses select topics of intensive care unit rounds that arise on ECMO patients. The topics covered in this article should be considered in routine rounding to be sure that the patient is maximally supported once a patient is committed to ECMO support.

CARDIAC CIRCULATION

End-organ perfusion can be defined by blood flow and pressure. To maintain end-organ perfusion, the blood pressure goal of the patients on venoarterial (VA) or venovenous (VV) ECMO should be maintained at a mean arterial pressure of 65 to 70 mm Hg or higher, like other critically ill patients.[1,2] The mean arterial pressure of patients on VA ECMO should be controlled at less than 100 mm Hg owing to competition of systemic vascular resistance, left ventricular (LV) afterload and distention, and ECMO flow. Optimum ECMO flow should be determined by unloading of the ventricles, not solely based on a body surface area (BSA) times 2.2.[3] Our previous review suggested that a higher mean arterial pressure improved survival, but liberal use of vasopressors to achieve a mean arterial pressure goal is yet to be proven. Tissue perfusion is the goal of flow and pressure, with confirmation by both physical (eg, urine output) and laboratory (eg, lactate) parameters.

Cardiac function should be monitored by transthoracic echocardiography or continuous hemodynamic transesophageal echocardiography. In case of cardiac standstill, cardiac decompression needs to occur within 4 to 6 hours before worsening pulmonary edema and LV distention occur. Options for the medical management of cardiac arrest or pulseless electrical activity on ECMO should include full ECMO flow, decreased systemic resistance, correction of temperature, electrolytes, acidosis, and hypoxia, and/or inotropes. Failure of return of pulsatility will mandate the use of surgical vents to decompress the LV cavity and prevent pulmonary edema. Cardiac standstill increases the incidence of blood stasis in the ventricle, which increases the risk of the stroke.[4] All inotropes should be discontinued as soon as the VA ECMO is started, to decrease the myocardial work and allow the VA ECMO to maintain the circulation. An intraaortic balloon pump may interfere with the ECMO flow and not decrease the LV afterload, but will increase diastole filling; it is recommended to remove if the coagulation profile is reasonable.[5] The cardiologists' routine use of the Impella 2.5 to unload the LV before ECMO may be excessive and costly, with minimal benefits and significant risks of hemolysis and displacement or perforation. Swan Ganz monitoring on patients on ECMO is not necessary. The pulmonary artery and central venous pressure are not accurate and it may depend on the ECMO flow.

Maximum ECMO flow is usually determined by cannula. Each cannula has a specific flow–pressure curve. The larger cannula is able deliver a larger flow. For example, a 17-F arterial cannula is only able to provide 4 to 5 L/min, whereas a 21-F arterial cannula can provide 6 L/min without adding extreme pressure on the cannula.

Besides mechanical issue of the cannula size, low ECMO flow can occur anytime during ECMO. Low ECMO flow often results in low perfusion pressure, low end-organ perfusion, hypoxemia, hypercapnia, and worsening of shock. Thus, a low ECMO flow should be addressed immediately. The low flow is usually related patient volume status and frequently observed within 24 hours after ECMO initiation, or observed during aggressive diuresis. Before low ECMO flow, "line chattering" is often observed. Line chattering is the phenomenon that venous cannula hit against venous wall owing to negative pressure created by the ECMO circuit because of low intravascular volume status.[6] Continuous chatter may cause the ECMO to alarm or "suck