Session: L233
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**Anesthetic Management of the Patient on Veno-Venous ECMO Who Requires Emergent Surgery**

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**Stem Case and Key Questions Content**

A 44-year-old female with severe ARDS on veno-venous ECMO through a right internal jugular 31F dual-lumen cannula has been scheduled as an emergent OR case due to a small bowel obstruction. ARDS occurred shortly after an appendectomy at an outside hospital and she has now been on VV ECMO for one week. Over the past 48 hours she has been unable to tolerate tube feeds and has become vasoplegic requiring norepinephrine. There is concern for an intrabdominal abscess causing the obstruction.

Prior to arrival to the operating room the patient had a PaO2 of 72 and a PaCO2 of 46 on with ventilator settings of pressure control Rate 12 IP 12, PEEP 18, 40% FiO2 with TV of 160-200. Her VV ECMO settings were fresh gas blender FiO2 of 100%, flow 7 LPM, 3400 RPMs with flow 3.8 to 4.1L.

The patient is on heparin at 20 units/kg/hr with a PTT of 68 seconds
She had a MAP of 65 on 0.1 mcg/kg/min norepinephrine.

What is the significance of her ECMO settings? How do circuit flow, RPMs, and fresh gas flow impact the patient’s gas exchange and delineate the patient’s severity of illness?

What are the currently used Veno-venous ECMO cannulation strategies? How do these differ from Veno-arterial ECMO?

The patient is currently on heparin with a PTT of 68 seconds. Should anticoagulation be continued during the surgery? How long can anticoagulation be safely stopped? What are the signs of thrombotic complication while on VV ECMO?
Besides me and the patient, which support staff should be present during transport and during the procedure? What monitors would you use during transport?

What is the optimal type of anesthesia? Should TIVA be performed? How does the anesthetic plan differ between VA and VV ECMO patients? Is BIS monitoring appropriate?

Should cardiac output be monitored during the procedure? If so, what are the options?

Is the patient a candidate for laparoscopic surgery? What physiologic changes would be expected with laparoscopic surgery and the requisite positioning? What impact would these changes have on ECMO flows and oxygenation?

A laparoscopic approach is elected. The abdomen is insufflated and the patient is placed into a reverse Trendelenburg position. Shortly after she begins to desaturate to 87%.

What changes can be made to improve the patient’s oxygenation?

What are the optimal ventilator settings for a patient on VV ECMO? How do the ARDSNet lung protective strategies impact ECMO patients? Does this patient need to be on the ventilator at all? With increased ECMO flow, significant line chatter occurs. What can be done to improve blood flow? What would be criteria for transitioning to an open procedure?

The patient is converted to an open procedure. What are the hemodynamic and gas exchange goals for this patient?

The patient has begun to bleed and the surgeon believes it is coagulopathic. Which labs should be assessed? Is there value in collecting a ROTEM? If so, which specific channels should be assessed? What transfusion triggers are present in the ECMO population? Is it safe to give platelets? Cryoprecipitate? FFP? PCC?

The bleeding has resolved and the surgeon reports adequate hemostasis and successful drainage of an abscess. When is it safe to restart anticoagulation? What is the patient’s risk of thrombosis if anticoagulation is not restarted?

Should ECMO be used in patients with sepsis? Is it appropriate to use VV, VA, or both? How should pump flow considerations vary between septic and non-septic patients?
What types of surgical procedures can ECMO patients be expected to undergo?

**Model Discussion Content**

Veno-venous ECMO is being increasingly used for the management of severe respiratory failure and ARDS. Currently there are 490 centers registered with the Extracorporeal Life Support Organization worldwide. As the prevalence of VV ECMO increases, so too will the requirement of these patients to go to the operating room for procedures such as repair of vascular injuries, drainage of hematomas, laparoscopy/laparotomy, tracheostomy, incision and debridement, and lung transplant. An understanding of the cannulation strategies and pump settings is essential to the management of such patients undergoing operative procedures.

Typically 100% oxygen is delivered to the ECMO circuits’ oxygenator. The delivered gas effectively oxygenates the blood that traverses the pump, but the patient’s systemic oxygenation will be based primarily on the degree of oxygen rich post-ECMO blood mixing with oxygen-variable blood that has passed directly into the pulmonary circulation – analogous to shunting from a physiologic standpoint. In order to increase the oxygen delivery in a patient on VV ECMO, the practitioner must either increase the ECMO flow, increase the FiO2 or PEEP on the patient’s ventilator (if the patient has parenchymal gas exchange), or transfuse as blood – as blood leavening the oxygenator is fully saturated and has a PaO2 of approximately 500. CO2 exchange is determined by the rate of fresh gas flow into the ECMO oxygenator, and typically ranges from 1-10 LPM. Higher flows of fresh gas will have only minimal impact on improving oxygenation. Once the upper limit of the rate of fresh gas flow is met, PaCO2 can be decreased by either increasing the patients minute ventilation, again assuming there is native parenchymal gas exchange or decreasing carbon dioxide production, which can be achieved with the utilization of sedation, paralysis, or cooling. Comparison of the patients’ arterial blood gas with ECMO settings and ventilator settings will give insight into the severity of pulmonary dysfunction. For patients with negligible lung function, systemic oxygen saturation will often be below 90%, even with adequate ECMO support.

The three most common cannulation strategies for VV ECMO are through a double lumen right internal jugular cannula, a combination of Right internal jugular and femoral cannulas, or dual femoral cannulas. These cannulas offer differing flow rates, which are limited by cannula diameter and necessitate different surgical position and venous access strategies. It is important to differentiate veno-venous ECMO from veno-arterial ECMO. In VV ECMO, the patient’s heart is responsible for all systemic circulation and some, if not the majority of the patient’s cardiac output will enter pulmonary and systemic circulation without having gone through the oxygenator. Generally, maintain an ECMO flow greater than 60% of cardiac output will be needed to prevent systemic hypoxemia. In VA ECMO, the heart and lungs are almost completely bypassed and the patient’s “cardiac output”
is almost entirely dictated by ECMO flows. On VA ECMO, our patient would tolerate extubation, however on VV ECMO, our patient, like most others would become progressively hypoxemic and hypercapnic without concurrent mechanical ventilation.

Dual lumen cannulas are available in 23F, 27F, and 31F sizes, which have maximum flows of 4-6 liters per minute, respectively. They are a proprietary product, currently only manufactured by the Maquet Corporation (Rastatt, Germany) and marketed under the trade name Avalon ®. Femoral-femoral cannulas usually are 23-29F, with the premise for both types of cannulation strategies being the usage of the largest cannula possible to maximize flow or to match cannula size to desired flow. A 23F femoral cannula can flow up to 5 LPM with a 29F flowing up to 8LPM. The flow is almost always limited by the drainage or ECMO inflow cannula as ECMO outflow cannulas are pressurized. The main indication for VV ECMO is for patients with severe ARDS, or similar severe acute lung injury patterns, in which the patient cannot be effectively oxygenated or ventilated using lung protective ventilation strategies. A proposed benefit of ECMO is that is can unload the lungs of the potential volutrauma and barotrauma associated with mechanical ventilation in the patient with injured lungs/ARDS. While on ECMO, lung protective ventilation should still be maintained, especially when it is still needed for effective gas exchange. Consideration of tidal volumes of even less than the 6 cc/kg proposed by ARDSNet should be considered in patients who have very poor pulmonary compliance. Managing the risks of thrombosis with bleeding is easier in patients on VV ECMO than those who are on VA ECMO. All circuits are heparin bounded which greatly decreases thrombogenesis from contact activation. In VV ECMO, the primary risks are thrombosis around the ECMO cannula, which is similar in structure to a central venous catheter and progressive clot formation within the oxygenator. Oxygenator thrombosis is generally a slow process happening over days to weeks, but may occur acutely in patients with hypercoagulable states. Generally, patients are managed with an ACT goal of 1.5 baseline or aPTT goal of 1.2-1.8x baseline, or 60-80 seconds, but institutional practices vary greatly. A case report has been published for a patient on VV ECMO who was managed for 20 consecutive days off of anticoagulation, which demonstrates the increased importance of considering bleeding risk in the anticoagulation of VV ECMO patients when compared to VA ECMO patients. In VV ECMO, the oxygenator serves as the major site of clot formation, and can be exchanged, compared to VA ECMO where there is risk of intra-cardiac clot formation due to stagnant flow in the left atrium and ventricle, which is potentially catastrophic.

Use of ECMO in septic patients is controversial and often avoided as the high cardiac output and impaired oxygen extraction creates a relatively low ratio of ECMO blood flow to systemic blood flow, which will lead to systemic hypoxemia. Any critically ill or ARDS patient is at risk of developing sepsis, so even if cannulation of septic patients is avoided, they are at risk of developing the condition. While broad spectrum antibiotic prophylaxis is commonplace, and routine blood culturing may be considered (ECMO patients will not typically develop fever due to heat loss from the circuit, and those treated with steroids for ARDS will not have reliable white blood cell counts), bacteremia still may occur, especially
in patients without source control of an infection. Probably the most important consideration in septic ECMO patients is cardiac output. As sepsis creates a hyperdynamic cardiac output state - the effect of this on ECMO flows must be considered. For VV ECMO it means that a greater proportion of blood will enter the pulmonary circulation without having passed through the oxygenator. For VA ECMO it means that an "adequate" pump flow will likely not match the physiologic needs of a septic patient who would otherwise have a highly elevated cardiac output.

VV ECMO patients, such as the one described, who have ongoing blood loss due to a combination of both surgical and coagulopathic etiologies are at further risk for dilutional coagulopathies. Patients who have ARDS may also be at risk of DIC. Regardless of etiology, factor and platelet deficiency should be treated with targeted transfusion strategies and assessed with either a combination of traditional labs or using thromboelastography. Use of ACT may also be used to monitor anticoagulation in ECMO patients.

Transportation of a patient on ECMO represents a critical time and a period in which catastrophe can occur. A minimal amount of staff would include practitioners dedicated to the physical transport of the patient/bed, ventilator (bag-mask ventilation should not be used in the transport of ARDS patients), infusion pumps, and ECMO circuit plus the anesthesiologist who can assess monitors and administer drugs, and a perfusionist or respiratory therapist that can adjust ECMO settings if needed. In the OR, having a dedicated practitioner (either a respiratory therapist or perfusionist) to manage the ECMO circuit is highly beneficial as the machine will divert attention away from the anesthesiologist and often is located at the side of the bed, with the settings outside of reach and or view. Changes to ECMO settings can then be discussed between the anesthesiologist and the practitioner managing the ECMO circuit.

Consideration should be made for the provision of TIVA versus volatile anesthesia as severe parenchymal dysfunction can significantly slow the uptake of inhaled anesthetics. Disconnection of the patient from the ICU ventilator and connection to the OR ventilator can result in de-recruitment of alveoli, especially in patients on high degrees of PEEP. Depending on the degree of sedation in the ICU (some ECMO patients with dual lumen cannulation are managed to a RASS of 0 to +1, and some are even ambulatory), continuation of existing infusions can also be considered. In patients with severe ARDS, tidal volumes, and especially gas exchange can be severely limited, which would in turn limit the delivery of volatile anesthetics. During VA ECMO, there will be very minimal blood flow through the lungs, so TIVA should always be selected.

Regardless of anesthetic selection, BIS or EEG monitoring should be highly considered, as changes in vital signs will be neither sensitive nor specific for determining anesthetic plane in this critically ill
population and IV anesthetics, especially lipophilic drugs may absorb to the ECMO circuit. BIS (Covidien, Boulder, CO) will also provide a confirmation that the anesthetic is being effectively delivered, and can uncover high degrees of ICU sedation, which can significantly impact the need for additional surgical anesthetics. Regardless of the choice of anesthetic, a specific plan should be developed for the delivery and monitoring of the depth of surgical anesthetic that reflects the specific physiologic characteristics of the VV ECMO population.

The decision to monitor cardiac output in a VV ECMO patient is highly dependent on the patient and provider. No single technique is without limitations. Arterial waveform based monitoring can be considered, but is not validated in this population. The use of area-under-the-curve based techniques to calculate cardiac output should have no physiologic limitations in ECMO patients. Pulse pressure variation will not be accurate due to the high intrathroacic pressures and small tidal volumes in patients with ARDS. A CCO Pulmonary artery catheter could be used to estimate cardiac output, specifically by following the trend. These devices can either under or overestimate cardiac output as temperature loss or heating may occur in the ECMO circuit, limiting the utility of thermodilution to calculate cardiac output. Utilization of TEE or TTE to calculate cardiac output would remain accurate, but is not a continuous technique and requires a specific skill set. The use of one, or a combination of these techniques can help direct management of ECMO patients. Cardiac output may also be estimated based on comparison of pre-ECMO saturations to systemic oxygen saturations.

Laparoscopic surgery introduces multiple physiologic challenges to the patient on VV ECMO. First, insufflation with carbon dioxide will need to be compensated by increased ECMO “sweep.” If the patient is already requiring a high level of sweep gas, there is a risk for the development of progressive hypercapnea and acidemia if CO2 production overwhelms the ability of ECMO to remove carbon dioxide. A second concern is that insufflation can decrease venous return. This can cause a decrease in the ECMO flows and lead to an unpredictable change in proportion of blood that bypasses the ECMO circuit – with the ultimate concern being systemic hypoxemia, or more importantly decreased oxygen delivery due to decreased ECMO flow.

In the patient from the scenario, there was significant ECMO cannula “chatter” or vibration of the cannulas – which represents decreased venous return and caval collapse which forced a decrease in pump RPMs, and therefore flows. This lead to both a decrease in systemic oxygen concentration and decreased CO2 elimination causing an increase in end-tidal CO2. Reverse trendelenburg positioning, used for upper abdominal procedures, can exacerbate this decrease in venous return and ECMO function, but positioning changes in ECMO patients may have unpredictable effects on pump function. Patients’ ECMO settings should be closely monitored during all changes in positioning.

Ultimately, due to a combination of increase CO2 generation and decreased ECMO flows due to a decrease in venous return - our patient could not tolerate laparoscopy and a conversion to laparotomy occurred within minutes. The patient also had significant “oozing” during the laparoscopy, which made
conversion to an open procedure a practical choice. All ECMO patients will potentially experience challenges with laparoscopic surgery – but the surgical technique does not need to be abandoned in the patient population, especially if the physiologic changes and demands for ECMO patients are well understood.

Common challenges experienced by VV ECMO patients are hypercapnia, hypoxemia, and line chatter with the potential interventions being the increase in pump RPMs (or flow), increasing the amount of sweep gas, and utilizing 100% oxygen infusion. Outside of the ECMO circuit, patient oxygen demand can be decreased by using muscle relaxants or cooling, and oxygen delivery can be increased by transfusing blood and increasing the patient’s ventilator FiO2 or PEEP, if gas exchange is occurring in the native lungs.

Line chatter, or vibration of the ECMO cannula (typically inflow) is a result of excessively high negative force drawing blood into the pump which is a sign of collapse of the vessel from which the ECMO pump is receiving deoxygenated blood from the patient. When this acutely occurs, it is typically caused by hypovolemia, but cannula migration should also be considered following patient transport and re-positioning. A volume challenge should be given, especially if the decrease in flows is impacting gas exchange. To remedy the issue while volume administration is being prepared, the pump RPMs and thus the suction force and flow must be decreased.

The described patient developed a significant coagulopathy due to a combination of prolonged heparinization prior to the procedure, thrombocytopenia due to critical illness, dilution of clotting factors from volume administration, as well as hypofibrinogenemia. ECMO patients have physiologic reasons to be coagulopathic, as mentioned above – but may also form clots due to acute phase reactants such as factor VIII and fibrinogen. PTT, INR, Fibrinogen, and Platelet count are the traditional coagulation labs utilized to guide transfusion. Newer options, such as thromboelastography can provide specific advantages over these labs in that it can identify normal or hypercoagulable clot formation in the presence of abnormal INR and separate heparin from the analysis.

A key decision point is if and when to restart anticoagulation post-surgery. In general, the thrombotic risks on VV ECMO are low, compared to potentially significant bleeding risks from surgery. If thrombus were to occur, it would generally be on the ECMO oxygenator, which can be replaced if needed. This differs from VA ECMO where the greatest concern is for clot formation to occur in stagnant blood in the ventricle or aorta. If coagulation is immediately restarted, and the patient were to continue to bleed, progressive coagulopathy may occur. Withholding heparin for 24 hours or longer is generally considered safe in such patients, but is not without controversy. Post-operative management of anticoagulation should be discussed among the anesthesiologist, intensivist, and operative surgeon, and a clearly defined and explained plan occurring at patient handover, which balances the risks and benefits of systemic anticoagulation.
In summary, the main problems which occur in the operative management of the ECMO patient are:

1) Systemic Hypoxemia/Low systemic oxygen delivery – which can be managed by increasing ECMO flows or giving blood
2) Hypercapnia/Acidemia – which can be managed by increasing sweep gas or decreasing carbon dioxide production
3) Hypovolemia – This can be identified by a venous inflow cannula which is becoming progressively more negative, new line chatter or vibration, decreasing ECMO flows at a stable RPM speed, or decreasing trend in CVP or cardiac output monitoring

![Diagram of ECMO setup and blood oxygen content calculation]

**Calculation of Blood Oxygen Content**

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\text{O}_2\text{ Content}_{\text{ECMO, Flow}_{\text{ECMO}}} = \frac{\text{O}_2\text{ Content}_{\text{systemic}}}{\text{Total Blood Flow}}
\]

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\text{O}_2\text{ Content}_{\text{systemic}} = (\text{Hb} \times 1.36 \times (\text{SpO}_2 / 100)) + (\text{pO}_2 \times 0.0031)
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= (10 \times 1.36)(100/100) + (500 \times 0.0031) = 15.2 \text{ ml}
\]

\[
= 8.3 \text{ ml}
\]

\[
= 11.8 \text{ ml/dl}
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**Patient Characteristics**

ECMO Flow = 4.5 LPM
- Pump Recirculation = 0.5 LPM
- Net ECMO Flow = 4.0 LPM
- Cardiac Output = 8 LPM
- Lung Function: Severe ARDS with negligible gas exchange
  - S\text{O}_2 = 60\% (IVC,SVC blood)
  - Hemoglobin = 10 mg/dl

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References


