Regional Perfusion During Venoarterial Extracorporeal Membrane Oxygenation: A Case Report and Educational Modules on the Concept of Dual Circulations

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Abstract: A challenging aspect of managing patients on venoarterial extracorporeal membrane oxygenation (V-A ECMO) is a thorough understanding of the relationship between oxygenated blood from the ECMO circuit and blood being pumped from the patient’s native heart. We present an adult V-A ECMO case report, which illustrates a unique encounter with the concept of “dual circulations.” Despite blood gases from the ECMO arterial line showing respiratory acidosis, this patient with cardiogenic shock demonstrated regional respiratory alkalosis when blood was sampled from the right radial arterial line. In response, a sample was obtained from the left radial arterial line, which mimicked the ECMO arterial blood but was dramatically different from the blood sampled from the right radial arterial line. A retrospective analysis of patient data revealed that the mismatch of blood gas values in this patient corresponded to an increased pulse pressure. Having three arterial blood sampling sites and data on the patient’s pulse pressure provided a dynamic view of blood mixing and guided proper management, which contributed to a successful patient outcome that otherwise may not have occurred. As a result of this unique encounter, we created and distributed graphics representing the concept of “dual circulations” to facilitate the education of ECMO specialists at our institution.

Keywords: ECMO, education, cardiopulmonary bypass, cannulation.

Extracorporeal membrane oxygenation (ECMO) is indicated when conventional medical and surgical measures fail to support a patient during cardiac and/or respiratory failure. According to the Extracorporeal Life Support Organization’s (ELSO) International Registry, the number of ECMO cases performed worldwide has increased steadily since 2004 (1). In particular, the number of adult ECMO cases has increased exponentially during that same period for both cardiac and respiratory support (1). A primary factor contributing to the increase in adult ECMO in recent years could be related to the Conventional Ventilatory Support versus Extracorporeal Support for Severe Adult Respiratory Failure (CESAR) trial published in 2009 showing the benefit of ECMO versus conventional ventilator treatment for respiratory failure (2). In addition, the technology used for ECMO support has improved and circuits have become much simpler and safer over time (3), potentially contributing to better outcomes and fewer complications associated with ECMO. In addition to the increase in the clinical use of ECMO, the number of ELSO-recognized centers has also increased. The number of ELSO centers was stable between 110 and 120 from 1993–2004, but has since grown to 170 in 2011 (1). With the increase in ELSO centers, there is an increased demand for ECMO specialists whether perfusionists, respiratory therapists, nurses, or other healthcare
providers. Proper education of ECMO specialists is crucial in ensuring the success of ECMO programs.

One of the more challenging aspects of managing patients on ECMO is a thorough understanding of the relationship between oxygenated blood from the ECMO circuit and blood from the patient’s native systemic circulation with varied cannulation schemes. In safe patient care during venoarterial (V-A) ECMO, it is mandatory for the team to understand and appreciate the distribution and mixing of the patient’s own cardiac output and the arterial flow from the extracorporeal circuit. We present a V-A ECMO case study which illustrates a unique encounter with the concept of “dual,” or “independent,” circulations. After recognizing that this unique encounter may actually be more common than unique, we developed new educational modules to illustrate the concept of blood mixing during ECMO.

CASE STUDY

A 35-year-old man (body surface area = 2.2 m²) underwent orthotopic heart transplant for nonischemic cardiomyopathy after mechanical circulatory support with a Heartware left ventricular assist device at our institution. Seventeen months after transplant, the patient presented to a local emergency department with weakness, epigastric pain, bloating, vomiting, and cough. He was given a “GI cocktail” and initially had relief of symptoms.

After finding elevated cardiac enzymes, the patient was transferred to our facility for further evaluation and management. Previous echocardiogram had shown an ejection fraction of 71% for the transplanted heart. On presentation to our facility, the patient underwent right heart catheterization, which revealed elevated right atrial pressures and compromised cardiac output (cardiac index = 1.33 L/m²/min) with concerns of possible rejection. An echocardiogram revealed a left ventricular ejection fraction of 25–30% with generalized left ventricular hypokinesis and severe right heart dysfunction. An endocardial biopsy revealed that the patient had level 2R rejection and the patient was immediately treated with aggressive steroids and other antirejection medications. The patient showed steady improvement with treatment until 4 days after admission when he went into atrial fibrillation with rapid ventricular response resulting in compromised circulation. The patient also showed signs of acute renal failure. At this point, an intra-aortic blood pump (IABP) was placed through the right femoral artery to improve perfusion and the right internal jugular vein was also accessed for a hemodialysis catheter. Despite aggressive use of inotropes and the IABP, a day later, the patient continued to decline showing severe metabolic acidosis, and ECMO was placed in the operating room using left femoral access. The femoral artery was cannulated with a 19-Fr. Biomedicus arterial cannula (Medtronic, Minneapolis, MN) using the Seldinger technique and a Fem-Flex 10-Fr. cannula (Edwards Lifesciences, Irvine, CA) was placed in the superficial femoral artery for distal leg perfusion. A 28-Fr. VFEM venous cannula (Edwards Lifesciences) was placed in the right femoral artery and forwarded to the right atrium with transesophageal echocardiographic guidance. The main components of the ECMO circuit were an X-coated custom tubing pack (Terumo Cardiovascular Systems, Ann Arbor, MI), a Quadrox-D oxygenator (Maquet, Bridgewater, NJ), and a Centrimag blood pump (Thoratec Corporation, Pleasanton, CA). The patient was stabilized on initiation of ECMO and showed no signs of significant bleeding as a result of the surgical placement.

To adequately monitor the patient and the ECMO circuit, initial blood samples were being drawn from three sites: 1) the patient’s right radial artery, 2) the ECMO arterial line, and 3) the ECMO venous line. During the initial hours of ECMO support, the blood gases at times presented uniquely (Table 1). At Hour 14, the blood gases drawn from both the ECMO arterial line and the patient’s right radial line were within parameters that we consider normal for V-A ECMO. However, at Hour 24, we recognized that despite the ECMO arterial blood gases becoming slightly hypercapnic, this patient in cardiogenic shock demonstrated regional mild respiratory alkalosis when blood was sampled from the right radial arterial line. Also, the PaO₂ measured at the right radial arterial line was less than half of the PaO₂ as measured at the ECMO arterial line (101 versus 236 mmHg). In an attempt to compensate for the patient’s respiratory alkalosis (at the right radial line), the ECMO specialist decreased the “sweep” (CO₂ control) from 1 LPM to 0.5 LPM. A set of samples drawn 7 hours later (Hour 31) showed normalization of the patient’s right radial gases, but at the cost of respiratory acidosis as measured at the ECMO arterial line. Also, although the FiO₂ setting on the ECMO circuit (80%) was appropriate, the extremely low sweep setting of .5 LPM also resulted in a decreased ECMO PaO₂ (69 mmHg). At this point, it was suggested that the patient may have a unique blood mixing pattern and that the left ventricle may be contributing significantly to regional blood flow. At this point (Hour 31), an arterial line was placed in the left radial artery and blood was sampled from each of the three arterial sites. As we hypothesized, the left radial line correlated more closely with the ECMO arterial line but was markedly different than the blood sampled from the right radial line (Table 1).

Based on blood gases sampled early during ECMO support, we hypothesized that blood sampled from the right radial line largely represented blood that was being ejected from the left ventricle and that blood sampled at the left radial line represented blood being pumped from
the ECMO circuit. We also hypothesized that the mismatch of gases would correlate with higher pulse pressures because it has been shown that pulse pressure directly correlates with left ventricular stroke volume (4,5). To test our hypothesis, we analyzed the effects of left ventricular ejection (i.e., pulse pressure) on blood gases in this patient.

Blood gas samples from this single patient were separated into three groups, each of which are represented in Table 2. Group 1 (n = 7) represents those time points during the first 50 hours when the pulse pressure was 16 mmHg or less, whereas Group 2 (n = 8) represents those time points during the first 50 hours when the pulse pressure was 17 mmHg or higher. The data show that when the pulse pressure was low (<16.5 mmHg), there was no statistically significant difference between the ECMO PaCO₂ (43.1 mmHg) and the PaCO₂ measured at the right radial line (41.7 mmHg). There was also no statistically significant difference between the ECMO PaO₂ (198 mmHg) and the PaO₂ measured at the right radial line (189 mmHg). Interestingly, when the pulse pressure was >16.5 mmHg (representing significant left ventricular ejection), we report a dramatic and statistically significant difference in the PaCO₂ in the ECMO arterial line (52.7 mmHg) versus the patient’s right radial line (30.5 mmHg). It should be noted that the right radial PaCO₂ demonstrated a difference when the pulse pressure was >16 mmHg (PaCO₂ = 30.5 mmHg) versus a pulse pressure of ≤16 mmHg (PaCO₂ = 41.7 mmHg). This statistically significant difference, however, was not accompanied by a statistically significant difference in minute ventilation measured by the patient’s mechanical ventilator, indicating that another factor (i.e., left ventricular ejection?) must be the cause of the change in PaCO₂ with increasing pulse pressure. When considering the effect of the ECMO sweep rate on PaCO₂ with changes in pulse pressure, we found that the sweep rate was significantly lower when the pulse pressure was high and that

Table 2. Extracorporeal membrane oxygenation (ECMO) and patient parameter averages during ECMO support.

<table>
<thead>
<tr>
<th>Group 1 (n = 7) Hours 0–50</th>
<th>Group 2 (n = 8) Hours 51–100</th>
<th>p Values</th>
<th>Group 1 vs. 2</th>
<th>Group 2 vs. 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Blood gases</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulse pressure (mmHg)</td>
<td>7.6 ± 4.7</td>
<td>28.6 ± 4.1</td>
<td>29.7 ± 7.2</td>
<td>.000</td>
</tr>
<tr>
<td>ECMO PaCO₂ (mmHg)</td>
<td>43.1 ± 6.4</td>
<td>52.7 ± 13.2*</td>
<td>44.8 ± 6.5*</td>
<td>.121</td>
</tr>
<tr>
<td>Right radial PaCO₂ (mmHg)</td>
<td>41.7 ± 7.1</td>
<td>30.5 ± 5.4</td>
<td>35.2 ± 5.2</td>
<td>.012</td>
</tr>
<tr>
<td>ECMO PaO₂ (mmHg)</td>
<td>197.6 ± 42.8</td>
<td>164.3 ± 58.5</td>
<td>186.4 ± 23.0*</td>
<td>.272</td>
</tr>
<tr>
<td>Right radial PaO₂ (mmHg)</td>
<td>188.9 ± 58.9</td>
<td>161.5 ± 58.7</td>
<td>124.7 ± 20.1</td>
<td>.435</td>
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<td><strong>ECMO settings</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FiO₂ (%)</td>
<td>76.4 ± 9.6</td>
<td>77.5 ± 4.3</td>
<td>70.0 ± 0.0</td>
<td>.818</td>
</tr>
<tr>
<td>Sweep (LPM)</td>
<td>3.0 ± .6</td>
<td>1.5 ± .6</td>
<td>1.8 ± .3</td>
<td>.001</td>
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<tr>
<td>Blood flow (L/min)</td>
<td>5.4 ± .2</td>
<td>5.5 ± .3</td>
<td>5.2 ± 1.1</td>
<td>.422</td>
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<tr>
<td><strong>Ventilator settings</strong></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>FiO₂ (L/min)</td>
<td>40 ± 0.0</td>
<td>40 ± 0.0</td>
<td>40 ± .0</td>
<td>N/A</td>
</tr>
<tr>
<td>Minute ventilation (mL/min)</td>
<td>5236 ± 1816</td>
<td>7600 ± 3670</td>
<td>3230 ± 736</td>
<td>.167</td>
</tr>
</tbody>
</table>

Group 1 = average values during first 50 hours of ECMO when pulse pressure was <16.5 mmHg; Group 2 = average values during first 50 hours of ECMO when pulse pressure was >16.5 mmHg; Group 3 = average values during Hours 51–100 of ECMO when pulse pressure was >16.5 mmHg.

* p value < .05 when compared with right radial PaCO₂ within the same group.

† p value < .05 when compared with right radial PaO₂ within the same group.
this correlated with a characteristic increase in ECMO PaCO₂ at those time points. Interestingly, this same decreased sweep rate was associated with a paradoxical decrease in right radial PaCO₂, again indicating that another factor (aside from sweep and minute ventilation) must be the cause of changes in PaCO₂ with increasing pulse pressure.

Essentially, the data from Groups 1 and 2 support our hypothesis of “dual,” or “independent,” circulations: the native circulation (i.e., blood ejected from the left ventricle) was responsible for regional perfusion of the proximal aorta, whereas the ECMO circulation was providing regional perfusion of more distal regions. After recognizing this phenomenon, the ventilator settings and ECMO settings were both adjusted properly to provide the patient with appropriately ventilated and oxygenated blood in both the native and ECMO circulatory systems.

Group 3 in Table 2 shows the average of 10 data points over the next 50 hours of ECMO support (Hours 51–100). We analyzed these data primarily for two reasons: 1) because the left ventricle had begun to recover, resulting in more consistent pulse pressures; and 2) because after 50 hours of support, we had conclusive enough evidence to support our hypothesis to treat the patient appropriately. During the first 50 hours of ECMO support, our treatment of the patient was very dynamic and inconsistent while we tried to determine the cause of the unique blood gas scenarios. After 50 hours, we treated the patient more appropriately by using the mechanical ventilator to manipulate blood traversing the lungs and left ventricle, whereas the ECMO settings were adjusted to provide appropriate ECMO blood gases. From Hours 51–100, there were no time points when the pulse pressure was 16 mmHg or less, so only data with higher pulse pressure are shown. Interestingly, from Hours 51–100 we still see significant differences in both the PaCO₂ and PaO₂ when comparing the right radial and ECMO gases. This is not surprising because we were treating the right radial gases and ECMO gases as separate entities, namely the native circulation and ECMO circulation. Despite the significant differences in blood gases between sampling sites, it should be noted that the PaCO₂ at both sampling sites was largely physiological (44.8 and 35.2 mmHg). Also, the difference in PaCO₂ between sampling sites was dramatically less between Hours 51 and 100 (9.6-mmHg difference) than was seen during the first 50-hour PaCO₂ (22.2-mmHg difference). Although the ECMO FiO₂ and blood flow values were statistically significantly different between the high pulse pressure groups at Hours 0–50 versus Hours 51–100, the differences in these values are likely the result of the low variance from the mean and likely not significant in terms of the technical and physiological aspects of ECMO support. On the other hand, the minute ventilation provided by the mechanical ventilator was significantly lower (p = .016) from Hours 51–100 than during Hours 0–50 (3235 versus 7600 mL). This decrease in minute ventilation correlated with an increase patient PaCO₂ from 30.5 mmHg to 35.2 mmHg in the high pulse pressure group. However, this difference in PaCO₂ was not statistically significant (p = .105).

After 10 days of ECMO support, the patient was successfully weaned from ECMO and managed with mechanical ventilation for another 10 days. Five days after ECMO weaning, echocardiogram revealed an ejection fraction of 61%. Repeat right heart catheterization showed a cardiac index of 2.7 L/m²/min. The patient continued to receive dialysis treatment for 34 days after ECMO weaning until renal recovery. During the hospital course, the patient did have acute limb ischemia of either the left upper or left lower extremity with compartment syndrome. The patient underwent fasciotomies to each of these extremities. The patient was discharged to a rehabilitation facility 36 days after weaning. Although the patient did require significant physical therapy, there were no apparent long-term renal, cardiorespiratory, or neurocognitive sequelae related to ECMO support.

**DISCUSSION: BLOOD MIXING AND EXTRACORPOREAL MEMBRANE OXYGENATION**

Proper understanding of how the two circulations interacted in this patient contributed to a successful patient outcome that may not have occurred without this level of expertise. Although the concept of dual circulations is described in ECMO-related textbooks, we feel that this concept is poorly understood by many ECMO caregivers. In the fourth edition of “ECMO: Extracorporeal Cardiopulmonary Support in Critical Care,” authors Jonathan Haft, MD, and Richard Firmin, MD, dedicate a paragraph to describe the potential for unique blood mixing patterns similar to those seen in this case study (6). The authors state: “A frequently overlooked complication of V-A ECMO support is cerebral hypoxia. Fully saturated blood from the ECMO circuit will meet blood ejected from the native ventricle. The location of the mixing point depends upon the amount of ECMO support provided and the degree of left ventricular ejection. If there is severe myocardial dysfunction, the mixing point will typically be in the proximal ascending aorta or aortic root. As myocardial function improves, the mixing point may migrate more distally into the aortic arch. Blood ejected by the left ventricle relies upon adequately functioning lungs for oxygenation. If significant pulmonary edema is present, hypoxic blood may perfuse the proximal aortic branches, including the coronaries and innominate artery. The patient’s head will appear blue, while the lower
Extremities will be pink. Arterial blood gases sampled from a femoral arterial line will reveal fully saturated blood, while blood samples from the right radial artery will be hypoxic.

Also, a review of peer-reviewed manuscripts reveals the impact of blood mixing and regional perfusion during ECMO. Animal studies have shown that during ECMO, the coronary arteries are preferentially perfused by blood being ejected from the left ventricle and not from the ECMO cannula (7–9), even when the cannula is placed in the brachiocephalic trunk (9). Wada et al. (10) investigated cerebral tissue oxygen saturation during percutaneous cardiopulmonary support and found that cerebral oxygen saturation was higher when using axillary arterial cannulation (82.3%) versus femoral artery cannulation (54.2%). These manuscripts suggest that statements made by authors Haft and Firmin indeed represent a significant challenge to patients undergoing ECMO support.

Because of this “frequently overlooked complication,” we sought to more thoroughly educate our ECMO team on unique blood mixing patterns. In conjunction with a graphic illustrator, animation segments were created to facilitate learning by comparing and contrasting V-A ECMO cannulation modes. The finalized educational modules focus on clouds of mixing between the patient’s own cardiac output and the ECMO arterial blood flow for cardiac and respiratory support.

Armed with a novel new teaching tool, our immediate goal was to use it to unify our institutional knowledge base and multidisciplinary approach to managing patients on V-A ECMO. The modules were incorporated into yearly ECMO classes for perfusionists, RN ECMO specialists, and respiratory therapists and were electronically distributed to intensivists and the cardiothoracic surgery team. We also posted them on our intranet for easy reference and accessibility. Based on our own observations as well as feedback from learners, the use of multiple teaching tools including a case study, patient data, and original illustrations/animations greatly facilitated learning and comprehension among all of the workgroups.

**EDUCATIONAL MODULES**

To simplify the educational experience for learners, we first developed diagrams clearly illustrating each circulation independently. Figure 1 illustrates the patient’s native circulation (Figure 1A), the ECMO circulation (Figure 1B), and also the concept of “dual circulations” (Figure 1C). Figure 1C shows how two diverging parallel circulations later merge together again, creating a mixing cloud where blood represents a dynamic mixture of the two circulations. Hypothetically, this mixing cloud can occur anywhere in the aorta depending on: 1) cannulation site; 2) ventricular...
function; and 3) ECMO pump flow. It is important to note that if ventricular function is so severely depressed that the aortic valve is not opening, the concept of “dual circulations” is not pertinent because the patient is being completely supported by ECMO and not by blood traversing the heart and lungs.

After designing the diagrams showing parallel circulations (Figure 1C), we then proceeded to development of animations to illustrate the concept of blood mixing during ECMO. In conjunction with a graphic illustrator, educational animation segments were created to facilitate the learning, comparing, and contrasting of V-A ECMO cannulation modes. Still shots of the animations are shown in this article and the animations can be found online at the following link (11): www.youtube.com/playlist?list=PLSWR1ylG_6JYR6Nu3aYvlcNFgM2EbYDVk.

Educational Model: Central Cannulation

Figure 2 illustrates blood circulation during ECMO with central cannulation. Figure 2A shows that when the left ventricle is not ejecting, there is no blood mixing, and the patient is supported entirely by ECMO support. However, when the left ventricle begins to eject (Figure 2B), blood from the left ventricle is ejected into the proximal aorta where it mixes with blood from the centrally placed aortic ECMO cannula. With proper and secure placement of the central aortic cannula, the relative position of this mixing cloud in the aorta is somewhat stationary in the proximal aorta. Also, with proper cannula placement, blood samples at different sites throughout the patient (i.e., right radial, left radial, etc.) should look quite similar to each other because the blood mixes proximally in the aorta. The coronary arteries are the exception because they are exposed primarily to blood from the left ventricle that may be poorly oxygenated. Regardless, the blood downstream from the mixing cloud will represent a mixture of blood from the left ventricle and the aortic cannula, and variability will exist based on the amount of blood being pumped by the left ventricle versus the ECMO circuit. Blood samples drawn from the ECMO arterial line may or may not look similar to blood samples drawn from patient sites (i.e., right/left radial) depending on the contribution of the patient’s native cardiorespiratory system. It is important to note that even when the left ventricle is contributing significantly to the overall circulation, this is only problematic if the lungs are not providing adequate gas exchange or if ventilator settings are too low. With functioning lungs and an ejecting ventricle, ventilator settings can be adjusted so that blood traversing the aortic valve is adequately oxygenated, even if the patient is being partially supported with ECMO.

Peripheral (femoral) Cannulation Educational Module

When performing ECMO with femoral cannulation, any blood being ejected from the left ventricle will eventually meet blood flowing retrograde from the femoral cannula. The meeting point, or “mixing cloud,” of these independent circulations will depend on the contribution of each individual circulatory pathway (Figure 3). If the left ventricular dysfunction is so severe that the aortic valve is not opening, the ECMO circuit provides full circulatory
As the heart begins to contract enough to open the aortic valve, with high ECMO flows, the location of the “mixing cloud” will be proximal in the aorta, closer to the aortic valve (Figure 4B). However, as the heart begins to recover and/or as ECMO flows decrease, the mixing cloud is pushed more distally in the aorta (Figure 4C–D).

It is important to note that although we believe these figures and animations are very useful in the training of ECMO specialists, they quite possibly represent an oversimplification. Further studies with ultrasound, for example, could investigate more directly how blood interacts in the aorta. It is reasonable to think that the mixing cloud could move in a pulsatile manner with each cardiac cycle, shifting distally during systole and then proximally during diastole. Also, it is not well understood how big the mixing cloud really is. Does blood mix in a relatively small area or is it widely distributed? Our limited data suggest that there are somewhat finite borders of blood mixing.

The location of the mixing cloud with peripheral cannulation is crucial in understanding regional perfusion throughout the body. In our case study, for example, we found that blood sampled from the right radial artery looked markedly different from blood sampled in the left radial artery. Furthermore, the blood in the left radial artery was quite similar in composition to blood being pumped from the ECMO arterial line. Therefore, we hypothesized that the “mixing cloud” was somewhere in the aortic arch (Figure 5). Fortunately, this patient had adequately functioning lungs and we were able to manipulate the blood being ejected from the left ventricle using mechanical ventilator settings. In other words, the patient was demonstrating independent circulations. The blood from the left ventricle was reaching only the proximal aortic arch and could only be manipulated using the ventilator, whereas the blood from the ECMO circuit was reaching only the distal aortic arch and could only be manipulated using the ECMO settings. In patients with severe respiratory dysfunction, it may be necessary to discourage the left ventricle from ejecting by minimizing inotropes or considering use of a left ventricular vent to decompress the ventricle.

**Figure 4.** Illustration of arterial blood mixing with central cannulation during venoarterial extracorporeal membrane oxygenation (V-A ECMO). (A) The absence of arterial blood mixing when the aortic valve remains closed. (B–D) The variable location of blood mixing when the left ventricle is ejecting.

**Figure 5.** Hypothesized location of “mixing cloud” of case study patient.
CONCLUSION

During V-A ECMO support, a full understanding of the relationship between oxygenated blood from the ECMO circuit and the patient’s native systemic circulation with varied cannulation schemes is critical for optimal care of the patient on ECMO. With peripheral cannulation, there may be dramatic variations in regional perfusion depending on the contributions of both the native circulatory system and the level of ECMO support. Our case report supports the idea that with peripheral cannulation, blood being pumped from the ECMO circuit may be forced to compete with blood being ejected from the left ventricle resulting in important differences in regional gas exchange. In cases such as this, it may be necessary to either minimize the contribution of the left ventricle or optimize ventilator settings to adequately ventilate the blood traversing the lungs and the aortic valve. This case report demonstrates the importance of monitoring the right radial arterial blood gas to confirm effective proximal and cerebral oxygenation in patients with a pulsatile blood pressure during peripheral ECMO.

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REFERENCES