“Watch your sweep”

Case report; Peripheral ECMO patient with a ventricular septal defect, loss of left ventricular ejection triggered by gas blender flow.

Summary

During peripheral ECMO therapy on a patient with a persistent VSD, ejection from the left ventricle was found to be impaired by transient hypocapnic responses. loss of LV ejection was first identified during a routine membrane “sigh” where gas flow was increased to 10l/min for 30-60 seconds to blow condensed moisture from the gas phase of the membrane oxygenator. During this “sigh” procedure the patient’s arterial systolic pressures dropped form 110mmHg down to 50mmHg. After full diagnostics of ECMO circuit the gas flow blender was found to be capable of delivering 30l/min to the oxygenator without the operator’s full knowledge. Although theorised; it is thought that delivery of massively hypocapnic blood to the arterial system resulted in peripheral vasoconstriction, failure of the LV to eject via the aortic valve and preferential offloading to the right ventricle.

Introduction

Extracorporeal membrane oxygenation (ECMO), is a therapy which provides prolonged cardiac and/or respiratory support to patients whose heart/lungs are unable to provide an adequate amount of gas exchange and/or blood delivery to sustain life.

ECMO works by temporarily diverting blood from the body through an artificial circuit to allow oxygenation and removal of carbon dioxide before returning blood to the patient. When returned to the patient's arterial side (VA ECMO) this delivered blood augments the cardiac output of the failing heart increasing perfusion (Fig 1).

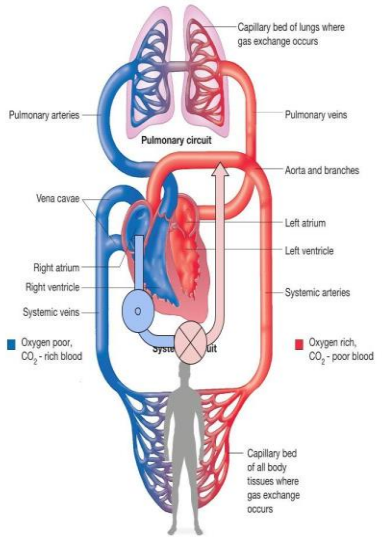

Figure 1. Central VA ECMO cannulation sites and direction of flow

A Ventricular septal defect (VSD) (Fig 2) is a defect or opening in the ventricular septum, the wall dividing the left and right ventricles of the heart. The extent of the opening may vary from pin size to complete absence of the ventricular septum, creating one common ventricle. Flow across any opening between the ventricles is determined by several factors but is principally determined by relative pressure gradients across the opening.

Case History

Several hours after undergoing extensive abdominal and thoracic surgery for repair of several stabbing wounds including what was thought to be a superficial injury to the apex of the right ventricular wall, the patient began to show signs of haemodynamic instability. Upon examination a large VSD was identified.

The patient was sent to theatre for surgical closure of the VSD however the repair was complicated and despite weaning successfully from cardiopulmonary bypass (CPB) and a return to intensive care unit (ICU), the patient soon deteriorated once again. It was clear that VSD closure had only been partially successful however concerns over tissue viability ruled out further surgery at that time. The decision was made to rest the heart on ECMO for possible device closure of residual VSD in the future.

Course of ECMO Therapy

Right femoral arterial cannulation was achieved through an 8mm chimney graft and 22fr EOPA cannula, right femoral venous cannulation was achieved using a multistage 25fr Biomedicus cannula with distal end

seated in the right atria/superior vena cava junction. Peripheral VA ECMO was initiated at 3500rpm giving a flow of 4.5L/min (80% of full flow, as calculated by DuBois derived BSA x CI of 2.4) and a FiO2 mixture of 60% delivered at 2.5L/min. Concurrent Intra-aortic balloon pump therapy via the left femoral artery was also initiated at this time.

The course of ECMO therapy continued unchanged for the next 24hours with the only point of note being a delay in initiation of heparin for 12hours to allow haemostasis of surgical intervention sites. The circuit remained fibrin / clot free during this period. After approximately 48hrs of ECMO therapy the amount of inotropic support had steadily been reduced and the patient appeared mostly stable though still precarious and very sensitive to fluid challenges.

This is Strange, what is happening?

After approximately 60hrs of ECMO therapy it was noticed by nursing staff that the patient was very sensitive to changes in gas flow. This was especially evident during the routine membrane "sigh" procedure when the patients arterial systolic pressure would "crash" after increasing gas flow delivery through the oxygenator.

A membrane sigh is performed at least twice per 24hrs, typically at the start of the responsible nurse's shift however this may be increased if the membrane oxygenator is losing efficiency. Efficiency is lost due to build-up of condensation on the gas phase impeding gas exchange. The membrane sigh is performed at 10l/min gas flow for 30-60 seconds, in simple terms increasing the flow of gas through the oxygenator physically blows condensation out of the vent port of the oxygenator.

This sensitivity to changes in gas flow was reported to perfusion staff who performed a full examination of the ECMO system components. Examination of the gas blender revealed that above 10l/min there was effectively no regulation of gas flows; meaning that if nursing staff increased gas flow delivery to above the 10l/min mark (as indicated by the built-in gas flow meter on the gas blender) then actual gas flow delivery could be as high as 30L/min. During testing by perfusion staff, gas flow of 28-30l/min was recorded at very small increments above the 10l/min marking. The effect of delivering such high gas flows to blood transiting the oxygenator would drive massive diffusion of CO_2 from blood to gas phase (as demonstrated by ficks equation of diffusion across a membrane).

Having arterial blood delivered with a theorised $PaCO_2$ close to atmospheric concentrations could have been causing a high degree of transient endothelial and peripheral chemoreceptor activation. This activation could manifest in massive

peripheral vasoconstriction leading to a spike in left ventricular afterload. This spike in LV afterload could have resulted in the LV offloading preferentially through the residual ventricular septal defect into the right ventricle meaning ejection through the aortic valve would be suspended, leading to the observed loss in arterial systolic pressures.

ECMO was terminated after XXhrs of therapy when it became clear that the patient had suffered ischaemic damage to a large portion of his bowel which was unsalvageable.

Discussion

As readers may note; the above case report make a few theoretical assertions, primarily that the loss of left ventricular ejection was as a result of preferential offloading to the RV through the VSD, driven by a hypocapnic vasoconstrictive response. The short time period between identifying the issue and the end of ECMO therapy meant that there no time to fully investigate the how and why of what was observed. In an ideal world echocardiographic examination with Doppler colour shift during the "sigh" procedure would have demonstrated what was happening within the heart.

Septal defects in ECMO patients are not uncommon. Several case studies have been published demonstrating successful closure of VSD's after a course of ECMO therapy (primarily to allow time for post-infarct tissue stabilisation). Surgically creating a hole in the septum has even been proposed as a means of ensuring the left ventricle is sufficiently offloaded, helping to protect against ventricular distention and thrombus formation, though most of the literature advocates for this "man-made" septal defect to be made between the atria.

Pahuja *et al* (2018) demonstrated that the haemodynamic effect of ECMO therapy on patients with a VSD worsens Left to right shunting and pulmonary wedge pressure, however this study didn't look at how the ability of ECMO therapy to alter blood gas balance could affect haemodynamics.

While the basic haemodynamic responses to PaCO2 levels are fairly well understood, it is less well understood how these mechanisms would respond to extreme deviations in PaCO2. As previously mentioned in this case report the model of gas blender used was capable of delivering up to 30L/min gas flow to the oxygenator without the operator's full knowledge. Due to the nature of this blender's design (being a type of gate valve), opening the gas flow regulator just a little above the indicated markings means that gas flow is essentially unregulated and will be delivered at whatever flow/pressure the main supply is operating at.

How much CO2 is removed from the blood transiting the oxygenator is driven by Fick's equation of diffusion and given that the

pressure gradient of CO2 between blood and atmosphere (or less than atmosphere in the case of ECMO gas delivery) is very large, it is not unreasonable to assume that the concentration of CO2 in blood which has experienced gas flows of up to 30L/min is severely reduced. How endothelial activation and peripheral chemoreceptors would respond to blood with PaCO2 levels close to atmospheric is unknown to this author but given the dramatic loss of ejection seen in this case I suspect that vasoconstriction of peripheral arteries is more marked than would be observed in normal circumstances.

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