Emergent Cardiopulmonary Bypass for a 180 Kilogram Patient: Support with a Single Oxygenator

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Abstract: As obesity increases in prevalence, so will cases in which patients present at the boundaries of care. We report the support of a class III obese man, having a body mass index of 60.8 kg/m² and in acute renal failure, with a single Trillium™-coated Affinity® NT Hollow Fiber oxygenator in cardiopulmonary bypass for an emergent aortic valve replacement secondary to infective endocarditis. A maximum oxygen delivery of 807.51 mL of oxygen per minute is reported for this oxygenator in this case report.

Keywords: cardiopulmonary bypass, oxygen transfer, obesity.

DESCRIPTION

A 49-year-old African American male, weighing 180 kg with a height of 172 cm, and a body surface area (BSA) of 2.73 m², with a recent medical history of a urinary tract infection and significant medical history of morbid obesity, obstructive sleep apnea, and hyperlipidemia presented to an outside hospital for angina. One week before the onset of angina, the patient was treated for a urinary tract infection at a separate medical facility. The patient reported increasing severity of angina and electrocardiographic (ECG) analysis demonstrated ST segment elevation in inferior ECG leads. The patient was then treated with a tenecteplase regimen with mild improvement of angina and was then transferred to our facility for subsequent cardiac catheterization. Results of the cardiac catheterization demonstrated only luminal irregularities of both coronary artery systems but a left ventricular end-diastolic pressure of 43 mmHg. After cardiac catheterization, the patient reported tachypnea and dyspnea, and nitroglycerin therapy was initiated but slowly reduced as a result of hypotension. The patient was then given 100 mg furosemide intravenously with a 10 mg per hour intravenous infusion and 1000 mg intravenous bolus of chlorothiazide. Despite several hours of diuretic therapy, the patient remained anuric.

Approximately 15 hours before surgery, the patient went into cardiac arrest in the intensive care unit followed by clonic movement and agonal breaths. The patient was resuscitated with basic life support of external chest compressions, epinephrine, atropine, sodium bicarbonate, calcium gluconate, and magnesium boluses. After these therapies, the patient went into ventricular fibrillation with 200 of direct current cardioversion, immediate endotracheal intubation, and mechanical ventilation. Multiple
ventricular fibrillations continued to occur with increasing intravenous boluses of epinephrine and amiodarone. Maximal inotropic support continued with no improvement in hemodynamics. An emergency cardiac surgical consent was obtained and a transesophageal echocardiogram revealed severe aortic valve insufficiency. Serum creatinine was noted to be 5.4 mg/dL and circulating leukocyte count of 24,000/mm³ at this time. After this, the patient was immediately transferred to the operating room with maximal inotropic support. On transfer to the operating room, a central venous catheter and Swan-Ganz pulmonary artery catheter were inserted, and the patient's cardiac output (CO), cardiac index (CI), central venous pressure (CVP), pulmonary artery pressures (PAP), and systemic vascular resistance (SVR) were noted as: CO = 5.0 L/min, CI = 1.8 L/min/m², CVP = 31 mmHg, PAP = 50/44 mmHg, and SVR = 499 dynes-second/cm⁵. The patient's chest was rapidly prepared with Chloraprep (CareFusion, San Diego, CA) and the sterile surgical field prepared as quickly as possible. A midline incision was performed with subsequent division of the sternum with a reciprocating sternal saw. Shortly after dividing the sternum, the patient went into severe bradycardia and a heparin bolus of 300 units/kg was administered immediately intravenously through the central venous catheter. The pericardium was opened with no blood being observed in the pericardium, and the ascending aorta was free from significant calcification or dilatation. The ascending aorta was cannulated with a 24 Fr. arterial cannula (CalMed, Costa Mesa, CA), and the right atrium was cannulated with a 36/46 triple-stage venous cannula (Edwards LifeSciences, Irvine, CA).

CPB was immediately initiated after venous cannulation with a custom CPB circuit containing a Trillium™ Affinity NT oxygenator, an Affinity® 38-µm arterial filter, MYotherm XP blood cardioplegia unit (Medtronic, Inc., Minneapolis, MN), an X-Coated cardiomyocyte reservoir (Terumo Cardiovascular, Elkton, MD), and a Dideco hemoconcentrator DHF0.6 (Sorin Group, Arvada, CO). Inline mixed venous saturation (SvO₂) and hematocrit were measured with a Medtronic BioTrend (Medtronic, Eden Prairie, MN). Autologous blood recovery was achieved with a Medtronic Autolog (Medtronic, Inc.). Anticoagulation monitoring was achieved with ACT Plus (Medtronic, Inc.).

Incomplete decompression of the right atrium and right ventricle were observed immediately after initiation of CPB, and vacuum-assisted venous drainage was used in an effort to better decompress the heart at a regulated suction pressure of –30 mmHg. However, despite this technique, the right atrium and right ventricle retained venous blood. A 16 Fr. pulmonary artery cannula (Medtronic, Inc.) was inserted into the anterior aspect of the pulmonary artery, just distal to the pulmonic valve, and complete decompression of the heart was achieved. Next, a 12 gauge (9 Fr.) aortic root cannula (Medtronic, Inc.) was inserted into the aortic root and then a 14 Fr. retrograde cardioplegia cannula (Edwards Lifesciences) into the coronary sinus for diastolic arrest with blood cardioplegia. The patient was cooled to a bladder temperature of 34°C as the aortic crossclamp was applied and secured. Immediately an arresting dose of 1000 mL of blood cardioplegia (1:1 ratio) was delivered retrograde at 4°C, over approximately 4 minutes, and as this dose was delivered, a 20 Fr. left ventricular vent catheter (Medtronic, Inc.) was inserted into the right superior pulmonary vein and advanced into the left atrium and across the mitral valve for adequate decompression of the left ventricle. Four subsequent maintenance doses of 300 mL of blood cardioplegia were delivered retrograde with time between doses ranging from 12 minutes to 20 minutes and ranging from 4°C to 6°C.

Correction of acidosis and the excessive base deficit of sodium bicarbonate while the patient was supported by CPB was achieved through the administration of sodium bicarbonate in conjunction with the administration of .45% solution of sodium chloride and intermittent ultraltrafiltration to prevent any potential hypernatremia. Total sodium bicarbonate administered into the patient during CPB was 600 mEq of an 8.4% solution, whereas total volume of .45% sodium chloride solution administered was 4.0 L and a final ultrafiltration volume of 10 L.

On creating the aortotomy and exposure of the aortic valve, the left coronary leaflet was found to be virtually eroded and absent. The valve leaflets and remnants of them were excised, the abscess cavity was débrided, and an autologous piece of pericardium that had been harvested and prepared by immersion in para-aldehyde for several minutes was used to cover the abscessed cavity with a running Prolene 4-0 suture. Seventeen Ti-cron 2-0 (Covidien, Mansfield, MA) sutures were placed in the annulus of the aortic valve in a noneverting fashion with no pledgeted sutures in the pericardium. These 17 sutures were then placed through the sewing ring of a 27 mm Edwards Lifesciences Perimount Magna Ease Bovine Pericardium Aortic Valve Bioprosthesis (Edwards Lifesciences). Once these 17 sutures were placed, rewarming of the patient to a bladder temperature of 37°C ensued. The bioprosthetic valve was lowered onto the annulus; the Ti-cron sutures were then tied and cut. The aortotomy was repaired with a Prolene 4-0 suture (Ethicon, Inc., Bridgewater, NJ) in a double-layer fashion. While the aortotomy was closed, a final retrograde reperfusion with warm blood at approximately 35°C was administered for several minutes during aortic root deairing and before removal of the aortic crossclamp. The patient resumed a normal sinus rhythm after crossclamp removal and the retrograde cardioplegia cannula was removed. After complete rewarming of the patient to a bladder temperature of 36.5°C, weaning from
CPB was attempted but reinitiated for an 4 additional minutes for adequate equilibration of systemic and pulmonary hemodynamics. Final separation from CPB occurred with the following inotropes and vasopressors: 1 μg/kg/min of epinephrine, 4 μg/kg/min of Levophed, and 16 units/h of vasopressin. Total time on CPB was 125 minutes with an aortic crossclamp time of 93 minutes. After stable hemodynamics, complete deairing of the left ventricle and aortic root, and as adequate volume status was observed, the venous cannula and aortic root cannula were removed and protamine sulfate (3 mg/kg) administration began. Once all protamine sulfate was administered, the aortic cannula was removed and as hemostasis was observed to be sufficient in the mediastinum, with subsequent verification with an activated clotting time, the sternum was re-approximated with stainless steel wires. The patient received no transfusions in the operating room and once the subcutaneous tissue was reapproximated and then closed along with the skin incision, a surgical dressing was applied in a sterile fashion to the incision site. The patient was then transferred out of the operating room with stable hemodynamics.

COMMENT

Some important considerations for the management of a patient this size during CPB must be entertained and undertaken. Primarily, a patient of this mass and BSA gives rise to considerations for the insertion of a second oxygenator in parallel to provide adequate gas exchange. However, considering the patient’s normal BMI for the corresponding height, 18.5–25 kg/m², the corresponding mass of the patient would be in the range of 55–74 kg and a lower BSA. In light of this estimate, and the emergent arrival and acuity of the patient in the operating room, a single oxygenator was selected. In selecting a single oxygenator for a patient of this size, it is imperative that there be clear communication among the perfusionist, cardiac surgeon, and anesthesiologist of the limits of performance of the oxygenator, heat exchanger, and venous reservoir selected for use in a patient of this size before the procedure begins and a remediating strategy discussed and agreed on should these components be unable to meet the demands of the patient.

Two strategies can be used to account for and remediate the deficit in oxygenator performance. One strategy is to initiate CPB under normothermia and before crossclamp placement, sample and compare arterial and venous blood gases to determine if oxygen delivery is greater than oxygen consumption. Should oxygen delivery be less than adequate, and if the patient has stable hemodynamics, termination of CPB should be done and a second oxygenator inserted in parallel into the extracorporeal circuit (ECC). In this strategy, there must be native cardiac output and stable hemodynamics to support the patient safely should the need arise to terminate CPB. Although this method adds time to the duration of CPB and has limitations, it allows an accurate evaluation of a single oxygenator to sustain the needs of the patient. A second and more advisable method involves electively inserting a second, fluid-primed oxygenator in parallel into the ECC isolated with tubing clamps. Once CPB has been initiated, and sampling of arterial and venous blood gases under normothermia reveals a deficit in oxygen delivery, blood flow into the second oxygenator could be opened without termination of CPB and subsequent arterial and venous blood gases performed to adjust necessary blood flow and ventilation of both oxygenators. An ancillary benefit to this method is access to a second heat exchanger in the ECC should the need arise.

Also, consideration of the circulating blood volume of a patient of this mass must be given due diligence. Classical estimations of the circulating blood volume of an adult may refer to 60–70 mL/kg, yielding an estimate of 10.8–12.6 L of total blood volume (TBV) for a patient of this size. Although crude, this method takes no consideration of the influence of the height of the patient. Nadler’s equation for blood volume estimation of males, \( BV_{male} = 0.3369 \times (h)^3 + 0.03219 \times w + 0.6041 \), where \( h \) is height in meters and \( w \) is mass of the patient, yields a TBV of 8.27 L. Irrespective of the estimating equation used, preparations for alternate reservoirs for the excessive volume should be made to accommodate the patient’s blood volume for the duration of the procedure (Figure 2).

In emergent salvage situations in which considerable metabolic acidosis and electrolyte imbalances are observed in patients of this size with acute renal failure or compromised renal function, correction of the acidosis and electrolyte imbalances must account for the potential of

<table>
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<td>[SVO₂] (%)</td>
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ongoing anuria. At the time of surgery, a nationwide pharmaceuti-
cal shortage of Tris(hydroxymethyl)aminomethane (THAM) had occurred and although THAM is consider-
able effective in correcting acidosis and is a well-suited buffer, it was not available for use. Therefore, different strategies to correct the severe metabolic acidosis had to be used. These strategies had to account for using large volumes of the only available buffer, sodium bicarbonate, without causing critical hypernatremia given the acute renal failure of the patient. It is possible to conceive that conventional hemodialysis could accomplish this, but given the emergent nature of the procedure and potentially exorbitant blood volume, ultrafiltration while the patient was supported by CPB was the best option available. This was achieved through intermittent ultrafiltration and replacement of volume with 4 liters of .45% sodium chloride solution while adding sodium bicarbonate as indicated. Initial arterial blood gas for the procedure revealed a base deficit of –8 mEq/L and the final arterial blood gas on CPB revealed a mild alkalosis and a small base excess (Table 1).

Of noted observance during this procedure was the performance of the Medtronic Trillium®-coated Affinity® NT hollow fiber oxygenator. Arterial blood gas, CPB data, and oxygen delivery for the procedure are displayed in Table 1, and performance data provided by the manufacturer regarding oxygen transfer are reprinted and included for reference in Figure 1. However, despite the performance of this oxygenator, there are limitations to this case report. Although not primary, carbon dioxide (CO₂) transfer capacity for this oxygenator was not determined, because venous blood gas samples were not taken and therefore no accurate determination of the partial pressure of CO₂ in the venous blood. However, from observing the sweep gas [V] flows, one can reasonably speculate that a high amount of CO₂ was present and being removed by the oxygenator. Although SvO₂ were below tolerable limits during the normothermic portions of CPB, speculations of greater blood flow and hemoglobin concentrations during these portions of CPB could have produced mild or moderate increases in those SvO₂ values. Although certainly advisable and warranted, higher blood flow was not necessarily achievable throughout the duration of CPB. Another limitation conceded is that without the partial pressure of venous oxygen being measured, stating oxygen consumption extrapolated from a trending device regarding SvO₂ values may be less than accurate.

Previous studies have attempted to determine ideal blood flow rates of the obese while supported by CPB. Although a commonly ascribed determinant of CPB blood flow rate of 2.4 L/min/m², this method demonstrated no significant difference in SvO₂ or urine output for patients

\[
C_{\text{arterial}} = \{[\text{Hb}] \times 1.36(\text{mL O}_2/\text{g Hb}) \times \text{O}_2 \times \text{saturation fraction}\} + \{0.0032 \times \text{mL O}_2 \times P_{O_2}(\text{mmHg})\}
\]

\[
O_2\text{ delivery} = C_{\text{arterial}} \times Q
\]

Where \( Q \) is cardiac blood flow (L/minute)

\[
Nadler's\ Equation
\]

\[
BV_{\text{male}} = [0.3369 \times (h)^3] + (0.03219 \times w) + 0.6041
\]

\[
BV_{\text{female}} = [0.3561 \times (h)^3] + (0.03308 \times w) + 0.1833
\]

Where

- \( BV \) represents blood volume in liters
- \( h \) represents height in meters
- \( w \) represents mass in kilograms

\[
BMI = \text{[mass]/[height]^2}
\]

of BMI greater than 30 kg/m² when randomized and compared with flow rates calculated from a normal BMI of 25 kg/m² (4). Additionally, individualized determinants of an ideal blood flow rate for the obese such as intraoperative CI before normothermic CPB demonstrated no significant difference in regional cerebral oxygen saturation or SvO₂ when compared with the commonly ascribed flow rate of 2.4 L/min/m² (5). Separately, in a model that compared among other variables, height, weight, BSA, BMI, age, pump flow rate, SaO₂, and hemoglobin concentration, only lean body mass and nasopharyngeal temperature were predictors of SvO₂ on CPB (6). Admittedly, these studies did not involve patients of a corresponding mass or BMI as compared with that of the patient presented here, and so divergence from the aforementioned predictors could be possible. Although incongruence or speculation in the literature may still exist as to what the best indices are for determining blood flow rates of the obese on CPB, emergent salvage procedures involving the obese may yet be confined to the an arbitrary flow rate, perhaps 2.4 L/min/m², and adjusted according to SvO₂ and serum lactate concentrations once CPB is instituted. Furthermore, cardiac surgical procedures involving patients of this size, whether emergent or elective, should consider oxygen transfer capacity as well as the CO₂ transfer capacity of the oxygenator in use, our observation in this case report is suggestive that the latter may be of equal or even greater concern regarding the maximum threshold of support.

REFERENCES