Optimal Perfusion Flow Rate Debate:
“The patient does need that much flow!”

Kenneth G. Shann, CCP
Department of Cardiothoracic Surgery,
Montefiore-Einstein Heart Center, New York, NY

Introduction

One of the primary objectives of cardiopulmonary bypass (CPB) is to provide sufficient perfusion flow to meet the metabolic demand of vital organs and tissues. Unfortunately, defining sufficient perfusion flow has been particularly challenging since traditional adequacy of perfusion monitoring have proven inadequate. Clinical teams often reference measured flow rates from healthy, unanesthetized volunteers to determine optimal flow rate for CPB patients. For example, Kirklin and Barratt-Boyces recommend a CPB flow rate of 2.2 L/min/m² in adults at 28°C or warmer to be an adequate perfusion flow rate. Since cardiac surgery and CPB is performed on heterogeneous patient populations who present with very high-risk profiles it is unlikely that fixed flow rates will be appropriate for all patients. As new evidence is generated and new monitoring technology is developed the opportunity to customize perfusion flow rates to meet the metabolic needs of individual patients will be possible. CPB flow rates customized to meet metabolic needs will likely improve patient outcomes.

Monitoring Adequacy of Perfusion During CPB

Traditionally, in-line mixed venous saturation monitors and blood gas analyzers have been used to estimate tissue oxygenation during CPB. However, the oxygen content of mixed venous blood is an average of the oxygen content of multiple organs and tissues. It is possible that organ specific changes in tissue oxygen consumption (VO₂) and an increase in the ratio of oxygen consumption and delivery (VO₂/DO₂) will go unrecognized through monitoring of only mixed venous oxygen content. Further, it has been shown that oxygen-derived values are unreliable for identifying anaerobic metabolism and lactic acidosis. Another monitoring tool gaining popularity in recent years is the direct measurement of lactic acid in whole blood. New blood gas analyzers have allowed lactate measurement to become a more common strategy to monitor the presence or absence of anaerobic metabolism during CPB. Unfortunately, this strategy has limitations since it confirms the accumulation of lactate but does not permit prophylactic interventions.

Ranucci and colleagues recently utilized capnography to measure exhaled CO₂ levels from the oxygenator of the CPB circuit. They identified an association with CO₂ derived parameters and hyperlactatemia during CPB that is consistent with the work done by Mekontso-Dessap and colleagues in critically ill patients. The ratio of DO₂/VCO₂ (CO₂ production) was the best predictor of exceeding the lactate threshold (> 3 mMol/L) with a cutoff value of 5. The next best predictor was VCO₂ with a cutoff value of 60 mL/min/m² predictive of exceeding the lactate threshold. The authors also concluded that
venoarterial CO2 tension gradient did not correlate with arterial blood lactate concentrations in patients on CPB. They hypothesize that current membrane oxygenators for CPB are so efficient at removing CO2 that the artificial lung blunts venous CO2 accumulation secondary to critical DO2. The excess CO2 that is anaerobically produced is therefore found at the exhaled site of the oxygenator rather than the venous blood.

In recent years near infrared spectroscopy (NIRS) has gained popularity in congenital cardiac surgery with CPB to non-invasively monitor brain and lower body tissue perfusion in order to detect regional malperfusion.4 The adoption of NIRS in adult cardiac surgery with CPB has been much slower. Some explanation for this may be the paucity of data supporting the use of NIRS in the adult population. However, recently Murkin and colleagues5 performed a randomized trial using NIRS in adult patients on CPB. They showed an improvement in a composite of patient outcomes in patients where interventions were performed to maintain NIRS values versus patients who were only monitored.

**Understanding the Concept of Critical Oxygen Delivery**

In a healthy resting adult at sea level with a cardiac output of 5.0 L/min oxygen delivery (DO2) is approximately 1,000 mL/min and oxygen consumption (VO2) is approximately 250 mL/min.6 Thus, approximately 25% of the oxygen delivered to tissues is extracted every minute under normal conditions. The delivery of oxygen to peripheral tissues and vital organs (DO2) by the blood can be calculated using the Fick Cardiac Output (CO) equation:

\[
DO2(mL/min) = 10 \times CO (L/min) \times CaO2
\]

where CaO2 \((1.34 \times \text{Hemoglobin(Hb)} \times \text{SaO2} + 0.003 \times \text{PaO2})\) is the oxygen content of arterial blood. The amount of oxygen consumed globally (VO2) by peripheral tissues and vital organs can be calculated using the following equation:

\[
VO2(mL/min) = 10 \times CO (L/min) \times (CaO2 – CvO2)
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where CvO2 \((1.34 \times \text{Hb} \times \text{SvO2} + 0.003 \times \text{PvO2})\) is the oxygen content of venous blood. The ratio of VO2/DO2 can increase during clinical situations that commonly occur during cardiac surgery and CPB such as anemia and reduced CO since CO during CPB is directly proportional to CPB flow rate. In addition, each vital organ carries its own metabolic needs. For example, the brain, GI tract, and heart extract much more oxygen than other organs and are therefore very dependent on adequate oxygen delivery and are susceptible to oxygen deprivation.7 If the DO2 falls below a certain level, called the critical DO2, the VO2 cannot be maintained and metabolism shifts from aerobic to anaerobic. When metabolism becomes anaerobic blood lactate concentration rises. Blood lactate concentration can be considered a good marker of sub-optimal tissue perfusion.8 Hyperlactatemia during CPB occurs commonly (~ 18% of patients) and has been associated with increased morbidity and mortality.9, 10
During CPB, optimization of DO$_2$ is most efficiently achieved through increases in Hb and CPB flow rate. Many studies have identified an association between nadir hematocrit and morbidity and mortality during CPB.\textsuperscript{11-15} At the same time several studies have highlighted the risk of infection and short and long-term mortality with transfusion of allogeneic blood making this an undesirable treatment to increase DO$_2$.\textsuperscript{16-18} More recent work by Ranucci and colleagues\textsuperscript{10,19} has focused on the importance of CPB flow rate to optimize DO$_2$ and minimize the incidence of hyperlactatemia and acute renal failure. The risk of hyperlactatemia and acute renal failure increased at critical DO$_2$ limits of 260 mL/min/m$^2$ and 272 mL/min/m$^2$ respectively in normothermic patients on CPB. Von Heymann and colleagues\textsuperscript{20} similarly focused on CPB flow rate and randomized patients to either hematocrits of 20 or 25%. With a CPB flow rate of 3.0 L/min/m$^2$ the authors found no difference in outcomes.

**Applying the Concept of Critical Oxygen Delivery to CPB Practice**

The strategy to maintain DO$_2$ above critical limits requires close examination of CPB practice and the factors contributing to effective CPB flow or flow that actually results in tissue perfusion. For example, it is common practice to measure CPB flow prior to intra-circuit shunts such as those for ultrafiltration or arterial filter purging. Flow measurements taken from this location likely over-estimates the amount of effective CPB flow. This disparity may be worsened in smaller patients where differences in flow can make a larger difference in DO$_2$. This disparity can also be worsened in minimally invasive surgery where smaller arterial cannulas with higher resistance are often utilized. This issue can be addressed by the placement of a flow probe distal to any intra-circuit shunts in order to determine the effective CPB flow.

Similarly, the clinical team must consider the physiologic and anatomic shunting of blood that results in the need for active venting. Blood that is returned to the CPB circuit from left atrial, left ventricular, aortic root, or pulmonary artery venting is lost from effective CPB flow. Therefore, vent flow must be considered when estimating effective CPB flow.

Electronic data management systems (DMS) are quickly becoming a standard part of CPB practice. These systems will provide clinical teams the opportunity to implement real-time calculations such as DO$_2$ into clinical practice. It would then be possible to use DO$_2$ for the real-time management of CPB flow rather than a reference flow rate. These DMS systems will also provide the opportunity to collect continuous data for DO$_2$ during CPB. These data could then be used to further evaluate the association between DO$_2$ and patient outcomes in order to further improve the practice of CPB.

**Conclusion**

Traditional monitoring commonly utilized during CPB to evaluate the adequacy of perfusion are insufficient to identify imbalances in oxygen consumption and delivery in vital organs and tissues. The opportunity exists to improve the practice of CPB to minimize the incidence of hyperlactatemia and subsequent morbidity and mortality. These opportunities include the careful evaluation of local CPB techniques and
implementation of DO$_2$ calculations to maintain DO$_2$ above critical limits. The implementation of DMS systems will further enhance the ability to manage CPB by creating the opportunity for real-time DO$_2$ management.

References


