Plateauing Oxygen Consumption


In 1979 in our Journal, Joe Mandl and Richard Motley claimed and demonstrated that the “endpoint of good perfusion is argued to occur when $V\dot{O}_2$ plateaus” despite further interventions (1). Our classic article authors recommended that the continuous calculation of $V\dot{O}_2$ enables the perfusionist to construct an individualized oxygen supply-consumption curve that responds to patient metabolic needs and perfusion conditions.

We are probably familiar with the fact that most textbooks cite the 1982 work of Fox, Blackstone, Kirklin and coworkers to illustrate the effect of increasing patient cardiac index leading to rising and plateauing patient oxygen extraction (2). Fox’s team taught us in the 1980’s that the CPB blood flow index required to cause plateauing $V\dot{O}_2$ reduces with patient tissue temperature and that cerebral blood flow is somewhat preserved at the same lower blood flows.

In 1979, the devices to measure and trend CPB patient $V\dot{O}_2$ were not available so Mandl and Motley showed how one could execute the algorithms with a programmable calculator in the operating room or at the bedside. Today, the instruments to continuously measure the necessary parameters to calculate $V\dot{O}_2$ are available and are going through design improvements. The use of computerized electronic patient records make it less complex to calculate and trend $V\dot{O}_2$, providing continuous surveillance of $V\dot{O}_2$, improving the clinicians awareness which should result in avoidance of metabolic acidosis and suboptimal tissue perfusion.

Recent publications like the work of Inoue, Kuro and Furuya demonstrate that type A hyperlactatemia (anaerobic metabolism from inadequate perfusion) during cardiopulmonary bypass appears to be coupled with the choice of arterial pump flow, hypotension, CPB time, and the delivery of arterial blood oxygen and metabolic production of $CO_2$ (3,4). Inoue and colleagues further reported that the duration of cardiopulmonary bypass, and especially the occurrence of hypotension at the start of the bypass period, were related to the development of lactic acidosis (3). Ranucci, his coworkers, and other groups, have published clinical data that demonstrate the predictive value of maintaining a minimal oxygen delivery rate ($D\dot{O}_2$) and the $CO_2$ production ($VCO_2$) are predictive of lactic acidosis during CPB (4,5).

When reviewing recent oxygen and carbon dioxide monitoring data, Mandl and Motley’s 1979 hypothesis from their classic article seems to have been right on target. Why then today is it not the standard of practice to calculate, record or use oxygen consumption for patient management during CPB? The answers are numerous, starting with clinicians’ lack of knowledge regarding how to monitor $DO_2$, $V\dot{O}_2$, and $VCO_2$, to the complexity of the computers required to present real-time information to care-givers.

Then there is the fact that most patients today are adequately perfused by selecting pragmatic cardiac index and monitoring Svo2, as illustrated by Engoren and Evans (6). They prospectively measured lactic acid levels three times, up to 65 minutes after aortic cross-clamping in twenty CPB patients, and failed to correlate lactic acid levels with $O_2$ consumption or $CO_2$ production, despite low oxygen consumption levels. At the same time, Ranucci, De Somers and many other teams are advancing CPB monitoring. Groom and his coworkers have worked to redesign continuous perfusion monitoring methods to reduce our most challenging negative outcomes (7). Current CPB lactic acidosis research like that described by De Somers, that shows the monitoring weakness of CPB Svo2 and mixed venous pO2, will likely drive the resurgence of the continuous monitoring of oxygen delivery and gas ($O_2$ and $CO_2$) transfer monitoring (8).

The perfusionist’s knowledge and skills associated with calculating and monitoring oxygen delivery, as well as oxygen and carbon dioxide transfer, remain fundamental to perfusion education and the future trends in clinical CPB patient management. Twenty-seven years after our classic article was published, we are still debating the definition and methods to use to avoid hypoperfusion in complex perfusion procedures. Mandl and Motley’s classic
article is an historic reference that will renew your knowledge of the *how and why* of VO$_2$ monitoring.

Jeffrey B. Riley, MHPE, CCT
Mayo Clinic, Rochester MN
Riley.Jeffrey@Mayo.edu

REFERENCES


