From Theory to Practice
Optimizing the Efficiency of an Inefficient Circulation

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In a study published in this issue of Circulation, Diller and colleagues1 applied differential calculus to the model derived by Santamore et al2 to explore the complex relationship between blood flow, arterial and venous oxygen content, and tissue oxygenation in patients with a bidirectional cavopulmonary shunt (BCPS). The BCPS is a unique and inherently inefficient circulatory arrangement that is used in the staged palliation of complex, functionally univentricular heart defects. In this circulation, pulmonary blood flow in the staged palliation of complex, functionally univentricular heart defects. In this circulation, pulmonary blood flow (Qp) is supplied directly by venous return from the upper body via the superior vena cava (SVC), while venous return from the inferior vena cava and coronary sinus mixes with pulmonary venous blood. This mixed venous return, which varies in oxygen content according to the absolute and relative rates of Qp and lower body blood flow, pulmonary and lower body systemic venous saturation, and oxygen carrying capacity, is then ejected to the systemic circulation. In this unique arrangement, upper and lower body systemic blood flow (Qs), oxygen delivery, and tissue oxygenation are related in a complex manner that does not necessarily lend itself to an intuitive understanding of circulatory efficiency and balance.

The novel and important contribution of the article by Diller et al is their distillation of this complex relationship into a simple and clear concept. Prior models of functionally univentricular circulations, which typically have focused on the parallel pulmonary:systemic circulation configuration characteristic of a first-stage palliation (including work by some of the authors of the Diller et al study3), have been predicated on the idea of and sought to define an optimal pulmonary-to-systemic blood flow ratio (Qp:Qs).3–5 Diller et al remind us that divergent upper and lower body systemic venous returns in the BCPS circulation preclude definition of a single optimal Qp:Qs.

The authors first focus on the important concept that systemic venous oxygen content is a more appropriate index of reserve than arterial oxygen content or oxygen delivery.3,5–8 They further develop the idea that the lower of the 2 systemic venous oxygen contents (upper and lower body) represents the best overall index in the BCPS circulation. On the basis of these 2 concepts, the authors reiterate and refine the finding of Santamore et al9 that the relationship between upper and lower body systemic venous saturations and upper and lower body blood flow distribution is largely proportional and complementary as long as upper body metabolism relative to blood flow exceeds that of the lower body (and thus upper body venous oxygen content is lower) but becomes increasingly uncoupled as the proportion of lower body metabolism and blood flow increase. In the context of this uncoupling, they argue, it is not appropriate to prioritize any single tissue in the pursuit of optimal tissue oxygenation.

In this way, they introduce the concept of SO2min, the lower of SVC and inferior vena cava saturations under any given set of conditions, as the single best measure of whole-body tissue oxygenation. They then use this index to demonstrate that, when the proportion of metabolism in the upper body exceeds that in the lower body, the maximal SO2min will occur when flow distribution matches metabolic distribution, as expected. However, when lower body metabolism is greater, SO2min will be maximized when upper and lower body flows are equal (ie, Qp:Qs=0.5:1). For the clinician, this can be restated as follows: Because the upper body delivers blood to the lungs directly, and thus most efficiently, for oxygen uptake, matching blood flow to metabolism is logical when upper body oxygen consumption predominates; however, when the lower body consumes most of the oxygen, there must be a different balance. Although the lower body needs the oxygen delivery more, its delivery of blood to the lungs for oxygen uptake is indirect, via the upper body; therefore, maximizing oxygen delivery is at odds with maximizing oxygen uptake. Some balance between upper and lower body flow must be achieved, and the model developed by the authors locates the optimal balance at 50:50.

Despite the novel insight into the BCPS circulation that the model of Diller et al affords us, it is arguably less valuable to the clinician than models of the first-stage parallel circulation. Such models predict that optimal metabolic status (as estimated by maximal systemic venous oxygen content) can be achieved by altering the relationship between Qp and Qs,3–5 which can be achieved by various means, including altering the diameter or length of the shunt, manipulating inspired gases to alter pulmonary vascular resistance, or pharmacologically manipulating systemic vascular resistance.9–12 Unfortunately, there are several factors that limit the direct clinical relevance of the model proposed by Diller et al in this regard. First, it assumes that Qp is derived solely from SVC return and that SVC return is committed completely to the
pulmonary circulation. This is not usually true in actual practice. Other sources of Qs, frequently exist, both intentionally, such as persistent flow across a partially obstructed pulmonary outflow tract, and unintentionally, most often in the form of systemic-to-pulmonary arterial collaterals; and at the same time, some SVC return is diverted away from the lungs via venovenous and venocameral connections. Second, when the circulation matches the model described, Qp is a function primarily of cerebral blood flow, with a minor contribution of upper extremity venous return, and although cerebral and pulmonary blood flow can be manipulated using pharmacological or ventilatory means, it is unlikely that such manipulation can be adjusted in an adequately controlled manner to optimize the circulatory balance, nor is it clear how that manipulation can be achieved in the BCPS circulation.

Control of Qp in the BCPS circulation is of great interest to the clinician, particularly in the immediate postoperative period when there is often transient but severe hypoxemia. Although there is equivocal evidence supporting the efficacy of endothelium-independent pulmonary vasodilators, such as inhaled nitric oxide in reducing SVC pressure and increasing systemic oxygenation after a BCPS,13,14 it is not clear that pulmonary vascular resistance has any role in the control of Qp when it is derived primarily from the cerebral circulation. Moreover, investigators have consistently found that endothelium-dependent mechanisms of pulmonary vascular regulation (eg, hypoxic vasoconstriction) are impaired in the setting of a BCPS.15,16 In addition, several recent studies have demonstrated that conditions leading to pulmonary vasodilation in the normal circulation, such as hyperoxia and hypercarbia, have no effect on or actually impair Qp and systemic arterial oxygenation in patients with a BCPS,17,18 whereas conditions that normally result in pulmonary vasoconstriction, such as hypoxia and hypercarbia, lead to increased Qp and systemic arterial oxygen content.18–21 Taken together, these findings indicate that attempts to modulate Qp by manipulation of pulmonary vascular resistance alone are unlikely to succeed, whereas decreasing cerebral vascular resistance, even through means that normally increase pulmonary vascular resistance, is likely to increase Qp.

According to the concept of SO2min propounded by Diller et al, a Qp:Qs <0.5 is suboptimal under any condition. Naturally, this conclusion assumes a constant cardiac output. As several of the aforementioned studies illustrate, however, the BCPS is part of a dynamic circulation in which alterations in regional blood flow may be coupled to physiological processes that invalidate steady-state assumptions.18–20 Alterations in blood flow to the upper body circulation in patients with a BCPS does not simply shift the relationship between upper and lower body blood flows but also may alter total Qs.18–20 For example, 2 studies performed in children with BCPS circulation have shown that induction of hypercarbia by adding carbon dioxide to the inspired gas mixture led to increased cerebral blood flow and thus Qp, with a concomitant increase in total Qs, such that flow to the lower body remained steady in one study18 and even increased in the other.19 Using patients from the latter study, Li et al20 observed that hypercarbia led to both increased systemic arterial oxygenation and decreased whole-body oxygen consumption, clearly evidence of improved oxygen reserve. This occurred despite the fact that Qp:Qs actually decreased from 0.5 to between 0.3 and 0.4, a ratio well below the minimum threshold stipulated by the Diller et al model.1

Findings such as these encourage critical evaluation of the notion of SO2min proposed by Diller,1 particularly with regard to its clinical utility. Such shortcomings, however, do not eliminate the clinical value of this study. Although the model cannot guide the clinician to simple therapeutic strategies, it may help clarify the appropriate focus for such strategies. The clinician must look beyond a straightforward approach to altering the relationship between Qp and Qs and consider the complex interrelationship between alterations in metabolic demands of the upper and lower body and the flow distribution between the upper and lower body. In such considerations, the goal of therapeutic manipulations should be maximizing metabolic stability, and the model of Diller et al suggests that this goal can best be achieved by optimizing SO2min.

Despite the inevitable limitations of a model that attempts to approximate a complex and, in many respects, incompletely understood physiological system, the work by Diller et al elucidates several important points and encourages us to think more critically and more deeply about the physiology of this unique condition. The BCPS is an inherently inefficient circulation. With critical analysis of the physiological principles and parameters governing this unique circulation and with increasing investigation of the pathophysiology that surrounds it, we can hope and expect eventually to optimize the efficiency of this inefficient circulation.

Disclosures

None.

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