Oxygenation in Patients With a Functionally Univentricular Circulation and Complete Mixing of Blood
Are Saturation and Flow Interchangeable?

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Background—Perioperative management of patients with complete mixing of pulmonary and systemic blood centers on approximately equating pulmonary (Qp) and systemic (Qs) blood flow (Qp/Qs≈1). This empirically derived target is opposed by theoretical studies advocating a target Qp/Qs well below 1. We studied the cause of this persistent discrepancy.

Methods and Results—Classic theoretical studies have concentrated on maximizing 1 of many potential combination parameters of arterial oxygen content (CaO₂) and systemic blood flow: total oxygen delivery (DO₂)=CaO₂×Qs. We defined “useful” oxygen delivery as the amount of oxygen above a notional saturation threshold (Satₜhresh): D(u)O₂=carrying capacity×(SaO₂−Satₜhresh)×Qs. Whereas DO₂ peaks at Qp/Qs ratios <1, D(u)O₂ peaks at higher Qp/Qs ratios, nearer to (or exceeding) 1. Systemic venous saturation (which mirrors tissue oxygen tension) peaks at Qp/Qs=1.

Conclusions—First, the standard model of single-ventricle physiology can be reexpressed in a form allowing analysis by differential calculus, which allows broader conclusions to be drawn than does computer modeling alone. Second, the classic measure DO₂ fails to reflect the fact that proportional changes in saturation and flow are not clinically equivalent. Recognizing this asymmetry by using D(u)O₂ can give a target Qp:Qs balance that better represents clinical experience. Finally, to avoid an arbitrary choice of Satₜhresh, systemic venous oxygen saturation (SsvO₂) may be a useful parameter to maximize: this occurs at a Qp/Qs ratio of 1. Attempts to increase DO₂ by altering Qp/Qs away from this value will inevitably reduce SsvO₂ and therefore tissue oxygenation. Oxygen delivery is far from synonymous with tissue oxygen status. (Circulation. 1999;100:2198-2203.)

Key Words: oxygen ■ circulation ■ blood flow

Patients with complete mixing of pulmonary and systemic blood present a difficult clinical problem, particularly in the delicate perioperative period, when mortality can be very high. Given a limited cardiac output, clinicians on the critical care unit modify filling pressures and vary the administration of oxygen (a pulmonary vasodilator), carbon dioxide (a peripheral vasodilator), and inotropic and vasoactive agents to affect the relative flows through the systemic and pulmonary circulations.

A clinical target of an approximately equal division of flow between pulmonary (Qp) and systemic (Qs) arms of the circulation (Qp/Qs≈1) is a common one that has been developed largely by trial and error.

Theoretical studies have considered the effects of different Qp/Qs ratios with the aim of forming an analytical basis for the clinical manipulations. However, the standard mathematical model has persistently yielded target Qp/Qs ratios of <1.

We set out to study the reason for this discrepancy and consider possible ways of unifying theory and practice.

Choice of Variable to Maximize and Implicit Choice of Relative Merits of Flow and Saturation

Tissue oxygenation is difficult to measure directly. It is intuitively obvious that for any given peripheral blood flow Qs, the situation of the tissues would be improved if the arterial oxygen content (CaO₂) were to rise. Likewise for any given arterial oxygen content, an increase in Qs would clearly be beneficial. This much is not in contention.

In principle, one aims to combine these 2 variables into a single parameter that can be maximized theoretically and clinically in the hope of thus optimizing the clinical state of the patient. However, a wide variety of combination parameters are conceivable that demonstrate an increase when either CaO₂ or Qs alone increases. The combination parameters differ in the way they interpret the balance of benefit and harm when (for example) Qs increases while CaO₂ falls.

Recent theoretical studies have chosen the product of the 2 as the combination parameter. This is known as oxygen...
delivery: $D\alpha_2 = CaO_2 \cdot Qs$. Alongside this explicit selection, there is therefore an implicit assumption that any change in 1 variable, if it were accompanied by a reciprocal change in the other, would lead to zero net clinical effect. For example, by choosing to use $D\alpha_2$ as our combination parameter, we are saying that a fall in arterial oxygen saturation from 80% to 40% would have no net clinical effect if $Qs$ could somehow be doubled from 0.3 to 0.6 $L \cdot min^{-1} \cdot kg^{-1}$.

How can we have arrived at such an untenable result? The answer lies in the choice of combination parameter. $D\alpha_2$ recognizes no special nature in saturation that distinguishes it from flow: it in effect considers them to be interchangeable.

What if they are not interchangeable? The question arises because not all the $O_2$ molecules that arrive in the systemic circulation are of equal value. The first molecules to be removed from the oxygenated blood are unloaded at a high capillary $P_0_2$. Once these valuable molecules are transferred into the tissue, those that remain are of lower value, because they will only pass into tissue of low $P_0_2$.

**Total Delivery or Useful Delivery?**
Any given total delivery is more helpful to the tissue (ie, demands a less severe tissue hypoxia for the transfer to occur) if it is delivered as a smaller quantity of high-saturation blood than if it is delivered as a large quantity of low-saturation blood. Assessment of $D\alpha_2$ (which treats flow and saturation symmetrically) unfortunately does not recognize this fact. One simple solution is to assert an arbitrary saturation threshold, $Sat_{thresh}$, below which we consider the tissues to be “scraping the bottom of the barrel.” The modified parameter, which we propose to call “useful oxygen delivery” ($D(u)\alpha_2$), can be calculated by a simple modification of the formula for $D\alpha_2$.

We placed a notional threshold in capillary oxygen saturation ($Sat_{thresh}$) below which any further oxygen remaining (carrying capacity $\times Sat_{thresh}$) should be considered not useful for maintaining normal function. The amount of oxygen being delivered in a useful form to the body could therefore be considered to be not $D\alpha_2 = CaO_2 \cdot Qs$ but rather $D(u)\alpha_2 = (CaO_2 - Carrying Cap \cdot Sat_{thresh}) \cdot Qs$.

**Capillary Contents: Quantity or Quality?**
The term “oxygen delivery” may itself be a misnomer, because its formula represents the total amount of oxygen that passes through the systemic capillary tree. Not all of this is actually delivered into the tissues: indeed, true delivery into tissues must equal total tissue utilization of oxygen ($V\alpha_2$).

Using the measurable values of blood oxygen saturations, the closest we can get to determining tissue oxygen levels is to examine the blood that was in equilibrium with it most recently, ie, mixed blood in the systemic veins. Thus, systemic venous $O_2$ saturation may be our best available capillary indicator of mean systemic capillary $O_2$ saturation and thus capillary $P_0_2$ and, indirectly, tissue $P_0_2$.

**Methods**
A single functioning ventricle receives blood from both the pulmonary and systemic veins and pumps this mixture into both pulmonary and systemic circulations. The content of oxygen is the same ($CaO_2$, measured in $L O_2$ per L blood) in both pulmonary and systemic arteries, whereas in venous blood the contents differ ($CpvO_2$ and $CsvO_2$, respectively). The cardiac output is divided between the pulmonary and systemic circulations, with flow rates of $Qp$ and $Qs$, respectively (in $L/min$). At steady state, metabolic utilization of oxygen must be matched by pulmonary oxygen uptake, so both can be represented by the symbol $V\alpha_2$, measured in $L/min$. In both systemic and pulmonary circulations, oxygen transfer rate is by definition blood flow multiplied by the arteriovenous difference in oxygen content.

1. $V\alpha_2 = Qs(CaO_2 - CsvO_2) = Qp(CpvO_2 - CaO_2)$.

For a given oxygen concentration in the blood returning from the lungs ($CpvO_2$), this same double equation can be rewritten to describe the concentrations in the arterial and systemic venous circulations:

$$\begin{align*}
\frac{CaO_2 - CpvO_2}{Qp} &= \frac{V\alpha_2}{Qp}, \\
\frac{CsvO_2 - CaO_2}{Qs} &= \frac{V\alpha_2}{Qs}
\end{align*}$$

In critically ill patients, there is a need to maximize tissue oxygen tension. An increase in cardiac output or a decrease in total body metabolism would improve tissue oxygen tension. However, there is a limit to the extent to which these 2 variables can be manipulated. The main free variable is therefore the balance of distribution of the limited cardiac output between pulmonary and systemic circulations ($Qp:Qs$ balance).

We used 2 independent methods to study the effect of changes in this balance.

**Computer Model**
We formed a model of the system described above using commercial mathematical modeling software (Mathlab, Mathwork) to assess the effect of changes in distribution of cardiac output ($Qp:Qs$) on the value of $D\alpha_2$ and $D(u)\alpha_2$, as well as the ability of $CaO_2$ and $CsvO_2$ to predict the peak levels of $D\alpha_2$ and $D(u)\alpha_2$.

In the acute postsurgical situation of neonates, clinical work has suggested a cardiac output of 0.3 $L \cdot min^{-1} \cdot kg^{-1}$ and an oxygen consumption of 0.009 $L \cdot min^{-1} \cdot kg^{-1}$ as suitable approximations, although it should not be forgotten that patients with single-ventricle physiology may develop the ability to survive on even lower oxygen consumption rates. Oxygen-carrying capacity of blood was taken as 207 $L O_2/L$ blood, based on 1.38 $\times 10^{-6} L O_2/g$ hemoglobin and a hemoglobin concentration of 150 g/L. We studied the effects of $Sat_{thresh}$ values of 10%, 20%, 30%, 40%, and 50% on the behavior of $D(u)\alpha_2$ in relation to changes in balance between $Qp$ and $Qs$.

**Calculus Analysis**
Differential calculus provides simple and well-validated methods for analyzing the behavior of such systems of equations without the aid (and restrictions) of a computer. Entirely independently of the computer model, we applied differential calculus to the formulas above to seek to understand the relationships between $Qp/Qs$ distribution and $D\alpha_2$, $D(u)\alpha_2$, and systemic venous oxygen saturation.
The avoidance of computer modeling enabled us to consider these relationships independently of any particular oxygen consumption or cardiac output.

Results

Computer Model of Total and Useful Oxygen Delivery: Impact of O₂ Saturation Threshold

We studied the effect of varying Qp:Qs balance on both total DO₂ (Figures 2 and 3, thick line, marked 0%) and useful delivery D(u)O₂ (thin lines). We examined the impact of different notional minimal acceptable capillary O₂ saturation thresholds (Satₜhresh): 10%, 20%, 30%, 40%, and 50% (thin lines). Crosses mark the points at which Qp=Qs, that is, half of the cardiac output goes to each part of the circulation.

In Figure 2, we can see that total oxygen delivery (thick line) peaks at a Qp/Qs ratio of clearly <1. However, considering useful oxygen delivery instead reveals 2 interesting facts. First, D(u)O₂ is of course less than DO₂: the absolute difference is greatest at low Qp/Qs ratios. Second, the peak in D(u)O₂ occurs at higher Qp/Qs ratios than the peak in DO₂. Indeed, so great is this shift in optimal Qp/Qs that it becomes >1 at higher Satₜhresh values.

The effect can be seen more clearly by eliminating the distorting nonlinearity introduced by the Qp/Qs ratio and considering instead Qp itself (with Qs understood to be CO-Qp), as shown in Figure 3.

More striking is the implication for the interpretation of arterial blood gases. In Figure 4, we show the relationship of useful oxygen delivery to arterial oxygen saturation. At Qp/Qs=1 (crosses on graph), arterial saturation is 60%. Clearly, total oxygen delivery peaks at an oxygen saturation at which Qp/Qs is <1 (to the left of the cross on the thick line). However, evaluation of useful oxygen delivery reveals that its maximum coincides with higher oxygen saturations than those of maximum total DO₂. Note also that for a Satₜhresh of 30% (central thin line), useful oxygen delivery falls to zero when arterial saturation is 30%, because below this threshold, blood flow (however vigorous) cannot contribute oxygen above Satₜhresh.

In Figure 5, we can see that systemic venous saturation levels peak at a Qp/Qs ratio of 1 (as shown in the previous section). What is interesting is that 1 particular venous saturation may indicate 2 completely different DO₂ values and similarly, 2 different D(u)O₂ values, even when the same Satₜhresh is used. The only exception is when systemic venous saturation is maximal, which corresponds to a unique Qp/Qs ratio of 1, and consequently an unambiguous DO₂ or D(u)O₂.

Maximizing DO₂ and D(u)O₂ by Altering Qp:Qs Balance: Results From Application of Differential Calculus

To determine the flow balance at which DO₂ or D(u)O₂ reaches its peak, we can exploit the fact that Qp+Qs=CO:

\[ (2) \quad D(u)O₂ = \left( \frac{\text{CpVO₂}}{\text{Qp}} \cdot \text{CarryingCap} \cdot \text{Satₜhresh} \right) \cdot (\text{CO}-\text{Qp}) \]

Of course, total DO₂ is simply the special case of D(u)O₂ at which Satₜhresh=0, and so it need not be considered separately at this stage. To identify the Qp at which D(u)O₂ is maximal, we differentiate:

\[ (3) \quad D(u)O₂' = \left( \frac{\text{CpVO₂}}{\text{Qp}} \cdot \text{CarryingCap} \cdot \text{Satₜhresh} \right) \cdot \left( 1 - \frac{\text{Qp}}{\text{CO}} \right) \]

We can now solve this for Qp to determine the conditions that maximize D(u)O₂. This is demonstrated in Figure 5.
The maximum value of $dD(u)O_2$ is obtained when this expression is zero, i.e., when

$$Qp = \frac{CO \cdot V_O2}{Cpvo_2 - CarryingCap \cdot Sat_{thresh}}.$$  

Perhaps the most important result of all is that useful oxygen delivery $D(u)O_2$ is maximal when the pulmonary flow fraction is

$$Qp = CO \cdot \frac{1}{Cpvo_2 - CarryingCap \cdot Sat_{thresh}}.$$  

In contrast, traditional total oxygen delivery $D(O2)$ is maximal at this flow fraction:

$$Qp = \frac{V_O2}{CO \cdot Cpvo_2}.$$  

$D(u)O_2$ clearly peaks at a different $Qp/Qs$ balance than does $D(O2)$. The effect of choosing successively higher putative minimal useful oxygen saturation thresholds ($Sat_{thresh}$) is to progressively raise the $Qp/CO$ ratio at which $D(u)O_2$ is maximal. The target $Qp/CO$ will reach one half (and therefore target $Qp/Qs$ will reach 1) when a $Sat_{thresh}$ of $(Cpvo_2 - 4V_O2/CO)/CarryingCap$ is selected, which for our case is 37%. Above this value of $Sat_{thresh}$, a $Qp/Qs$ ratio of $>1$ will be necessary to maximize $D(u)O_2$.

**Impact of Qp:Qs Balance on Systemic Arterial and Venous Saturations**

Leaving aside the derived variables $D(O2)$ and $D(u)O_2$, we turned to address the relationship of $Qp/Qs$ balance on the systemic arterial and venous saturations, which are more concrete physical variables. Using Equation 1, we studied the effect of changing $Qp/Qs$ balance on arterial and systemic venous saturations (Figure 6). Cardiac output ($Qs + Qp$) was kept constant at 0.3 L·min$^{-1}$·kg$^{-1}$, as was $V_O2$ at 0.009 L·min$^{-1}$·kg$^{-1}$. Pulmonary venous $O_2$ saturation was taken as 95%.

As can be seen from the equation $CasO2 = Cpvo2 - (V_O2/Qp)$ and the graph, at high $Qp$, $CasO2$ is only slightly below $Cpvo2$. However, the consequence of a high $Qp$ with a limited $CO$ is that $Qs$ is small. Hence, from the equation $CsvO2 = CasO2 - (V_O2/Qs)$ or the graph, it can be seen that $CsvO2$ is forced considerably below the arterial level. In contrast, at low $Qp$, $CasO2$ is a long way below $Cpvo2$, but $CsvO2$ is not far below that level. Either extreme of flow distribution leads to a low $CsvO2$.

What flow balance gives the highest $CsvO2$? From Equations 1, we derive

$$CsvO2 = Cpvo2 - V_O2 \left( \frac{1}{Qp} + \frac{1}{CO-Qp} \right),$$

which is maximal when $Qp = CO/2$. The maximum attainable systemic venous oxygen concentration is therefore $Cpvo2 - (4 \cdot V_O2/CO)$. These relationships all hold true regardless of the level of $V_O2$ or $CO$.

**Discussion**

In the management of patients with complete mixing of systemic and pulmonary blood, the clinical management has focused on attaining a $Qp/Qs$ ratio of $\approx 1$. Existing theoretical analyses have studied the relationship of controllable parameters (such as the pulmonary-to-systemic flow ratio, $Qp/Qs$) and measurable parameters (such as arterial and systemic venous saturations). One particular parameter that combines information about flow and saturation is $DO2$. This variable has been shown$^6$ to peak at $Qp/Qs$ ratios distinctly $<1$. It has not been clear how to reconcile this with the often-used clinical target of $Qp/Qs = 1$. Our observation is that not every molecule of oxygen passing through the systemic circulation is of equal value: those traveling in blood at higher saturations are able to be delivered at higher tissue partial pressures and are therefore more beneficial to tissue metabolism. Blood containing oxygen at lower saturations is of less value, even if the flow rate is correspondingly larger. The impact of these considerations on optimal $Qp/Qs$ ratios and
saturation measurements has not previously been assessed in detail.

Theoretical optimization of hemodynamics using $D_O^2$ alone can lead to some perverse choices when saturation is significantly $<100\%$. We believe that this contradiction between theory and clinical judgment is due to the insidious effect of choosing a simple (and at first sight, plausible) combination parameter for blood flow and arterial oxygen content. We believe that the tissue pressure at which the oxygen is made available is important rather than irrelevant.

These considerations are not purely academic, because the choice of parameter to optimize has a significant effect on the direction in which clinicians might attempt to influence changeable hemodynamic parameters such as $Qp:Qs$ balance.

**Factors Affecting Arterial and Systemic Venous Saturations**

Blood draining from the pulmonary veins undergoes 2 downward steps in oxygen content (Figure 7). First, at the time of mixing in the ventricle, it falls by $V_O^2/Qp$ from $CpvO_2$ to $CaO_2$. Blood traveling to the systemic circulation then transfers an amount of oxygen $V_O^2/Qs$ to the tissues and ends up with oxygen content $CsvO_2$.

High cardiac outputs allow large $Qp$ and $Qs$ to coexist, and so there are only small drops in oxygen content and consequently $CsvO_2$ is not very low. However, given a limited total cardiac output, there is a trade-off between the pulmonary–venous–to–arterial drop and the arterial–to–systemic-venous drop, because any change in $Qp$ results in a concomitant contrary change in $Qs$.

**Maximal Attainable Systemic Venous Saturation**

It can be seen from inspection of Equation 7 or Figure 6, or by application of calculus, that the situation that maximizes $CsvO_2$ for any given cardiac output is $Qp=Qs=CO/2$. The maximal systemic venous saturation that can be attained is $Spvo_2=4 \cdot V_O^2/(CarryingCap) \cdot CO$, which neatly summarizes the possible modalities (other than equalizing flow in systemic and pulmonary circulation) by which the oxygenation of such patients may be improved. $Spvo_2$ can often not be safely increased because this would lead to a fall in pulmonary vascular resistance and therefore an adverse change in $Qp:Qs$ balance. $V_O^2$ is not under the clinician’s direct control. It only remains to maximize the product of carrying capacity and cardiac output while realizing that if the carrying capacity is made too high by excessive blood transfusion, the increase in blood viscosity can impede flow and thus reduce cardiac output.

**Quantity Flowing Past or Quality at Point of Delivery?**

Oxygen delivery has been the center point of a variety of proposals for a choice of parameter to optimize: maximizing $D_O^2$ (Reference 6); maximizing $D_O^2/V_O^2$ (also called $\Omega^p$); or minimizing $V_O^2/D_O^2$ (also called oxygen extraction ratio).$^9$

Given a constant metabolic rate ($V_O^2$), we can see from Equation 5 that all of these targets are met simultaneously when the proportion of cardiac output sent to the pulmonary circulation is $Qp/CO=\sqrt{V_O^2/(CO \cdot CpvO_2)}$. For our case of $V_O^2=0.009 \text{ L} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$, $CO=0.3 \text{ L} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$, and $CpvO_2=0.95 \times 0.207=0.197 \text{ L} \text{O}_2/\text{L}$ blood, this optimal flow balance is 39% pulmonary, 61% systemic. This accords with the findings of the graph from the computer model. In fact, from the algebraic formulas, $D_O^2$ maximization will continue to recommend $Qp/Qs < 1$ unless cardiac output rises above $\sqrt{(V_O^2/CpvO_2)}$, ie, $0.065 \text{ L} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$.

All suffer from the limitation that the quality-blind parameter $D_O^2$ is used, rather than a quality-conscious one such as $D(u)O_2$, and so lower arterial saturation, however severe, is considered to be able to be compensated for simply by proportionately higher flows. Yet computation of $D(u)O_2$ rather than $D_O^2$ is not a panacea: it involves an arbitrary saturation threshold and still retains assumptions of a linear relationship of useful delivery to flow and saturation. The model could be refined further, although at the cost of substantially increased complexity.

The problem is that in tissues, oxygen extraction continues until metabolic requirements are met, even if this means that tissue oxygen tension is too low for satisfactory organ function. The oxygen tension in the tissue is determined not by the tension prevailing when the first molecule of $O_2$ is unloaded but rather by the tension at which the last molecule is transferred: this in turn is intimately related to $SsvO_2$ by the hemoglobin dissociation curve.

Maximizing $D_O^2$ and maximizing $SsvO_2$ will lead to different $SsvO_2$ values and hence different tissue oxygen tensions. The magnitude of the difference (shown in Figure 8) is small, at 3 to 8 percentage points in saturation, corresponding to...
<0.4 kPa in terms of PO₂. Yet it may be clinically important in the intensive care setting, given that it may represent a 10% difference in PO₂ and that the patients may be poised unstably at the extreme of physiological endurance.

Determination of SsvO₂ is difficult and can be subject to measurement error,10 but the substitution of SsvO₂-derived parameters such as D(u)O₂, D(u)O₂/V̇O₂, and VO₂/D(u)O₂ cannot ameliorate this. From a clinical point of view, it is often remarked that the venous sample is taken from a vein containing a biased sample of venous blood and therefore does not show true mixed venous saturation. However, it is likely that the difference in saturation between this sample and true mixed venous saturation is stable, so that maximizing the measured saturation is likely to maximize true mixed venous saturation. We suggest that adding, subtracting, multiplying, or dividing by other parameters, alone or together, cannot generate a parameter that more closely reflects changes in tissue oxygenation than does SsvO₂.

Indeed, clinical outcome studies in perioperative patients with hypoplastic left heart syndrome support a relationship between gradual elevation in SsvO₂ and survival.3 Particularly interesting is the observation in stable congenital heart disease patients11 that with low SsvO₂ values, oxygen extraction can no longer be increased (because tissue PO₂ values are too low) and consequently, aerobic metabolism becomes restricted.

The considerations outlined in this article are applicable not only to the neonatal patient but also to any patient with a functionally univentricular circulation and complete mixing of blood.

In patients without congenital cardiac disease who are undergoing supportive care in the intensive care unit, attempts have been made to maximize D(u)O₂ with conflicting results.12,13 Nor has maximization of mixed venous oxygen saturation14 been successful in conferring survival benefit among adults with normally connected hearts. However, their situation clearly is markedly different from that of the neonate with complete mixing, because the clinician is not adjusting the balance between pulmonary and systemic blood flow and trading off flow against saturation.

Conclusions
We contend that an excessive focus on quantity of delivered oxygen, as occurs in attempts to maximize D(u)O₂, necessarily involves depriving the body of quality of oxygen delivery. Instinct would prevent a clinician from believing the implication from D(u)O₂ calculations that a fall in SaO₂ from 90% to 15% could be adequately compensated for by an increase in blood flow from 0.15 to 0.90 L · kg⁻¹ · min⁻¹. When more subtle changes are involved, however, clinical judgment is less able to help.

We propose that maximizing D(u)O₂ is a more logical target than maximizing total D(u)O₂ and show how it gives a result that accords more closely with clinical judgment. We demonstrate the potential loss of capillary saturation, and hence tissue oxygen tension, that can arise from undue attention to D(u)O₂ despite a falling SsvO₂. Yet even D(u)O₂ cannot be said to be a perfect quantifier of oxygen traveling to tissues. A notional "perfect" parameter, taking into account the curvilinear nature of hemoglobin-oxygen dissociation and the potential for tissue metabolism to vary with oxygen delivery, may in the end closely mirror the simple parameter SsvO₂ or its partial-pressure counterpart.

We speculate that in these critically ill patients with complete mixing, it is tissue oxygenation, and hence SsvO₂, that we should be attempting to maximize. We show why apportioning blood flow equally between pulmonary and systemic circulations leads to this outcome.

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References
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