Estimation of Oxygen Delivery in Newborns With a Univentricular Circulation

Ofer Barnea, PhD; William P. Santamore, PhD; Anthony Rossi, MD; Ellis Salloum, MD; Sufan Chien, MD; Erle H. Austin, MD

**Background**—The management of neonates with complex congenital anomalies depends on careful interpretation of arterial blood gas values. Improved interpretation of these oxygen parameters may allow clinicians to avoid unexpected cardiovascular events. This study examined whether systemic oxygen delivery (DO₂) can be maximized by the use of indices derived from oxygen saturation measurements in neonates with hypoplastic left heart syndrome.

**Methods and Results**—For the single-ventricle heart with both circulations in parallel, we used a previously developed computer simulation to obtain DO₂ as a function of systemic arterial (SaO₂) and venous (SvO₂) oxygen saturation, arteriovenous oxygen difference (Sa–Vo₂), or pulmonary-to-systemic flow ratio (Qp/Qs). We also examined the oxygen excess factor, SaO₂/Sa-vo₂ (Ω). We found that (1) slight increases in SaO₂ may be associated with large decreases in DO₂, (2) low values for SvO₂ indicate low values for DO₂, (3) Curves for Sa–Vo₂ and Qp/Qs are redundant in the data provided. (Qp/Qs, however, provides these data in more physiologically relevant terms.) (4) High values for Qp/Qs (>4) are associated with low DO₂, (5) Estimating Qp/Qs from oxygen saturation measurements may result in errors when pulmonary venous oxygen saturation is not available. (6) Maximizing DO₂ is extremely difficult using SaO₂, SvO₂, and Qp/Qs. (7) A linear relationship exists between Ω and DO₂, and this linear relationship is not altered by changes in cardiac output.

**Conclusions**—Patients with low SvO₂ values require attention. Ideally, after reducing Qp/Qs to <1.5, Ω might be a better index to guide further therapy and maximize DO₂. Interventions that increased Ω would be considered beneficial, whereas interventions that decreased Ω would be considered detrimental. (*Circulation. 1998;98:1407-1413*.)

**Key Words:** computers ■ hypoplastic left heart syndrome ■ hemodynamics ■ oxygen ■ pediatrics

Hypoplastic left heart syndrome is presently the most common cardiac malformation that results in death in newborns.¹ Without treatment, 95% of these infants die during the first month of life, and none survive beyond 4 months.²

Management of neonates with hypoplastic left heart syndrome is complex and controversial. Treatment generally commences with vigorous infusion of prostaglandin to prevent the ductus arteriosus from closing.³ This restores fetal parallel circulation, with the right ventricle as the only active pump. However, reduction in pulmonary resistance after birth may result in an unbalanced circulation in which most blood flows into the pulmonary circulation, thereby compromising systemic oxygen supply.

In a previous theoretical analysis,⁴ we examined the effects of the ratio of pulmonary to systemic blood flow (Qp/Qs) on systemic oxygen delivery (DO₂). We found that a Qp/Qs of <1 was optimal. However, maximizing oxygen delivery based on Qp/Qs was difficult. Further, when calculating Qp/Qs from blood gases, pulmonary venous oxygen saturation (SvO₂) is generally estimated. This can lead to substantial errors. Thus, in the present study, we examined whether DO₂ can be maximized by the use of indices derived from oxygen saturation measurements.

**Methods**

A model of flow in the univentricular circulation is shown in Figure 1. This diagram represents the circulation of the newborn with hypoplastic left heart syndrome that has been treated to maintain both atrial and arterial shunts open. Pulmonary venous return is forced into the right atrium, and right ventricular output is distributed between pulmonary and systemic circulations. This forms two parallel circulations.

In a previous study,⁴ we derived an equation for DO₂ based on the simple flow of oxygen uptake in the lungs and whole-body oxygen consumption (CVo₂). The analysis is based on movement of oxygen into the pulmonary circulation (uptake) and out of the systemic circulation (consumption). The basic equation is as follows:

\[
\text{CaO}_2 \times \text{Qs} - \text{CVo}_2 = \text{Cvo}_2 \times \text{Qs}
\]

Equation 1 states that the oxygen flow rate into the systemic circulation (CaO₂ × Qs) is reduced by (Cvo₂), leaving the reduced oxygen flow rate returning to the right ventricle (Cvo₂ × Qs).

\[
\text{CaO}_2 \times \text{Qp} + \text{SVo}_2 = \text{Cvo}_2 \times \text{Qp}
\]

Equation 2 states that the oxygen flow rate into the pulmonary circulation (CaO₂ × Qp) plus oxygen uptake in the lungs (SVo₂) gives...
Estimation of Qp/Qs in Newborns

Figure 1. A model of the circulation with hypoplastic left heart syndrome. The darkened left ventricle (LV) indicates that the ventricle is not functioning and that blood does not flow through it. P indicates pulmonary circulation; S, systemic circulation; and RV, right ventricle.

By guest on July 26, 2017 http://circ.ahajournals.org/ Downloaded from

the oxygen flow rate returning to the right ventricle (CpvO₂ · Qp).

Equation 3 relates blood flow in the two circulations to total cardiac output (CO):

(3) \[ \text{CO} = Q_s + Q_p \]

The analysis assumes steady-state conditions, and thus oxygen uptake and consumption must be equal:

(4) \[ \text{SV}O_2 = \text{CVO}_2 \]

By combining equations 1 to 4, \( \text{DO}_2 \) (or \( \text{CaO}_2 \) · \( Q_p/Q_s \)) equals

(5) \[ \frac{\text{CaO}_2 \cdot Q_s}{Q_p/Q_s} = \frac{1}{1 + \frac{Q_p}{Q_s}} \frac{\frac{1}{\text{CO} \cdot \text{CpvO}_2}}{\text{CpvO}_2} \]

Thus, \( Q_p/Q_s \) is a complex function of CO, pulmonary venous blood oxygen content \( \text{CpvO}_2 \), rate of \( \text{CVO}_2 \) and \( Qp/Qs \). \( Qp/Qs \) based on the Fick principle, can be expressed in a single-ventricle heart with parallel circulation as

(6) \[ \frac{Q_p}{Q_s} = \frac{\text{Sao}_2 - \text{SvO}_2}{\text{SpvO}_2 - \text{Sao}_2} \]

where \( \text{SpvO}_2 \), \( \text{SvO}_2 \), and \( \text{Sao}_2 \) are pulmonary venous, systemic venous, and systemic arterial oxygen saturation, respectively. \( \text{Sao}_2 \) and \( \text{SvO}_2 \) levels can be measured. However, \( \text{SpvO}_2 \) is difficult to measure and is generally estimated.\(^5\) To determine the error involved in calculating \( Qp/Qs \) based on this estimation, the derivative of equation 6 with respect to \( \text{SpvO}_2 \) is taken. Thus, the sensitivity (\( \Psi \)) of \( Qp/Qs \) to \( \text{SpvO}_2 \) (or the relative change in \( Qp/Qs \) that is generated by a small change in \( \text{SpvO}_2 \)) is

(7) \[ \Psi = \frac{\text{SpvO}_2 \cdot (Qp/Qs)}{Qp/Qs \cdot (\text{SpvO}_2)} = -\frac{\text{SpvO}_2}{\text{Sao}_2 - \text{SvO}_2} \]

for example, when \( \text{SpvO}_2 \) is 87.3% and arterial oxygen saturation is 77%, \( \Psi = 87.3/(87.3 - 77) = 8.5 \). This means that a 10% overestimation of \( \text{SpvO}_2 \) will result in a 85% underestimation of \( Qp/Qs \) and vice versa. For example, with a \( \text{Sao}_2 \) of 77% and a \( \text{SvO}_2 \) of 45%, one may assume that \( \text{SpvO}_2 \) is 96% when it is actually 87.3%. The calculated values of \( Qp/Qs \) would be 1.68, whereas the actual value for \( Qp/Qs \) is 85% greater, or 3.11. This indicates that calculating \( Qp/Qs \) by the use of equation 6 and assuming a value for \( \text{SpvO}_2 \) involves a risk of large errors.

Buheitel et al\(^6\) proposed that an index of oxygen delivery/oxygen consumption is helpful in managing infants with critical cardiovascular problems. We refer to this index as the oxygen excess factor \( (\text{SaO}_2/\text{Sa-vO}_2) \cdot Q_p/Q_s \), which can be calculated on the basis of 2 measurements of oxygen saturation:

(8) \[ \Omega = \frac{\text{Oxygen Delivery}}{\text{Oxygen Consumption}} = \frac{\text{CaO}_2 \cdot Q_s}{(\text{CaO}_2 - \text{CvO}_2) \cdot Q_s} = \frac{\text{SaO}_2}{\text{SaO}_2 - \text{SvO}_2} \]

Using a computer (Compaq Computer Corp), we used the above equations to determine the relationship of \( \text{SaO}_2 \), \( \text{SvO}_2 \), arterial-venous oxygen difference \( \text{Sa-vO}_2 \), \( Qp/Qs \), and \( \Omega \) versus \( \text{DO}_2 \). The blood oxygen content was converted to percent oxygen saturation by assuming a hemoglobin of 15 g/dL, giving an \( O_2 \) capacity of 22 mL O₂ per dL blood (1.38 × 15 per hemoglobin). \( \text{CVO}_2 \) was set at 9 mL oxygen per minute per kilogram, which represents an average value for neonates after cardiac surgery.\(^7,8\)

In computer simulations, a wide range of values can be examined. For the results section, we selected 2 very different levels of \( \text{DO}_2 \). Setting the CO to 300 mL · min⁻¹ · kg⁻¹ simulated the low level of \( \text{DO}_2 \), whereas setting the CO to 450 mL · min⁻¹ · kg⁻¹ simulated the high level of \( \text{DO}_2 \). Assuming a body surface area of 0.25 m² for a 3.0-kg newborn, this gave a range in cardiac index from 3.6 to 5.4 L · min⁻¹ · m⁻².

Results

Figure 2 plots \( \text{SaO}_2 \) against \( \text{DO}_2 \). Two curves are presented: 1 with a lower level of \( \text{DO}_2 \) and the other with a higher \( \text{DO}_2 \). Both curves demonstrate a nonlinear relationship between delivery and saturation. As \( \text{SaO}_2 \) increases, oxygen delivery increases, reaches a peak, and then decreases rapidly. Peak oxygen delivery occurs at \( Qp/Qs < 1 \). For \( Qp/Qs > 1 \), slight
increases in SaO₂ can be associated with large decreases in oxygen delivery. For example, for the higher oxygen delivery curve, increasing SaO₂ from 80% to 85% decreases oxygen delivery from 34.1 to 14.6 mL O₂ per minute per kilogram. However, this doesn’t mean that increasing SaO₂ is always associated with a decrease in oxygen delivery. For example, going from the lower oxygen delivery curve at an arterial oxygen saturation of 65% to the upper oxygen delivery curve at an arterial oxygen saturation of 70% increases oxygen delivery from 24.0 to 45.0 mL O₂ per minute per kilogram.

Figure 2 also shows that for any given SaO₂, a range of values exists for oxygen delivery. For example, at an SaO₂ of 70%, oxygen delivery can range from ≈21.9 mL to as much as 45.0 mL O₂ per minute per kilogram, depending on the CO. Additionally, no 1 value of SaO₂ exists at which oxygen delivery peaks. For the upper curve, oxygen delivery peaks at 45.4 mL O₂ per minute per kilogram at an arterial oxygen saturation of 64%, whereas for the lower curve, a peak oxygen delivery of 24.6 mL O₂ per minute per kilogram occurs at an arterial oxygen saturation of 60%.

Figure 3 plots Svo₂ against Do₂. Both curves show a nonlinear relationship between oxygen delivery and Svo₂. However, for Qp/Qs>1, increases in Svo₂ are associated with increases in oxygen delivery. Values of Svo₂<40% are indicative of severe derangements in oxygen transport (that is, the relationship between oxygen delivery versus demand). While potentially related to an increase in oxygen demand in the face of normal oxygen delivery in the immediate postoperative period, a Svo₂<40% usually reflects a critical decrease in Do₂. Higher Svo₂ values are associated with higher Do₂ values. However, for each curve, peak oxygen delivery does not occur at the peak Svo₂. Further, even without a change in CO, the same Svo₂ value can be associated with two possible values for oxygen delivery, depending upon the Qp/Qs value. For example, for the higher oxygen delivery curve, a Svo₂ of 50% can have a Do₂ of 44.9 mL O₂ per minute per kilogram when Qp/Qs=0.38 or 22.5 mL O₂ per minute per kilogram, when Qp/Qs=2.68.

Figure 3. Systemic oxygen (O₂) delivery against Svo₂.

Figure 4. Systemic oxygen (O₂) delivery against Sa-vo₂.
Figure 4 plots \( \text{Sa-vo}_2 \) versus \( \text{Do}_2 \). As with the other plots, a nonlinear relationship exists between oxygen delivery and \( \text{Sa-vo}_2 \). An \( \text{Sa-vo}_2 > 40 \) (i.e., occurs at \( \text{Qp/Qs} > 3 \)) implies very low levels of oxygen delivery irrespective of the CO level. For \( \text{Qp/Qs} > 1 \), as \( \text{Sa-vo}_2 \) decreases, oxygen delivery increases. As \( \text{Sa-vo}_2 \) continues to decrease, oxygen delivery peaks. Beyond this peak, oxygen delivery decreases rapidly even though the \( \text{Sa-vo}_2 \) continues to decrease. For any given value for \( \text{Sa-vo}_2 \), a range of values exists for oxygen delivery. For example, at an \( \text{Sa-vo}_2 \) of 20, oxygen delivery ranges from 23.8 to 35.7 mL O\(_2\) per minute per kilogram depending on the CO. Also, no \( \text{Sa-vo}_2 \) value exists for peak \( \text{Do}_2 \). For the upper curve, oxygen delivery peaks at an \( \text{Sa-vo}_2 \) of 12.7 (45.4 mL \cdot min\(^{-1}\) \cdot kg\(^{-1}\)), whereas peak oxygen delivery for the lower curve lies at an \( \text{Sa-vo}_2 \) of 21.8 (24.6 mL \cdot min\(^{-1}\) \cdot kg\(^{-1}\)).

Figure 5A plots \( \text{Do}_2 \) versus \( \text{Qp/Qs} \). Once again, the relationship between oxygen delivery and \( \text{Qp/Qs} \) is nonlinear. For \( \text{Qp/Qs} > 4 \), oxygen delivery levels are very low and can only be partially compensated by higher levels of CO. As \( \text{Qp/Qs} \) decreases toward 1, oxygen delivery increases and peaks at values of \( \text{Qp/Qs} < 1 \). If \( \text{Qp/Qs} \) continues to fall, oxygen delivery precipitously decreases. For any given value of \( \text{Qp/Qs} \), a range of oxygen delivery values exists, and no single value for \( \text{Qp/Qs} \) can predict an absolute peak in oxygen delivery.

Compared with the other indices, \( \text{Qp/Qs} \) provides physiological information regarding the relative pulmonary (Qp) and systemic (Qs) flows. One disadvantage of \( \text{Qp/Qs} \) is that the value for \( \text{Spvo}_2 \) is generally estimated, and this estimation can cause errors in calculating \( \text{Qp/Qs} \). Based on Equation 7, the errors in the calculated values of \( \text{Qp/Qs} \) as a function of \( \text{Spvo}_2 \) are plotted in Figure 5B. The figure shows that for the same percentage error in \( \text{Spvo}_2 \), the calculation error of \( \text{Qp/Qs} \) is significantly larger at low values of \( \text{Spvo}_2 \). For example, at the highest level of \( \text{Spvo}_2 \), a minimal error of 5% (estimating \( \text{Spvo}_2 \) as 95% instead of the actual value of 90%) will yield a 25% error in the calculated \( \text{Qp/Qs} \). Estimating \( \text{Spvo}_2 \) as 88% when the actual value is 84% will result in a 60% error in \( \text{Qp/Qs} \). A more specific example is when \( \text{SaO}_2 \) is 77% and \( \text{Svo}_2 \) is 45%, with an assumed \( \text{Spvo}_2 \) of 96%; the calculated \( \text{Qp/Qs} \) would be 1.68. If, however, the actual value for \( \text{Spvo}_2 \) was 91.4% (a 5% overestimation), the actual \( \text{Qp/Qs} \) would be 2.22. For a 10% overestimation of \( \text{Spvo}_2 \) (87.3% versus 96%), the actual \( \text{Qp/Qs} \) would be 3.11.

Figure 6 plots the \( \text{Do}_2 \) versus \( \Omega \). Compared with the other relationships, a linear relationship exists between \( \text{Do}_2 \) and \( \Omega \). As \( \Omega \) increases, \( \text{Do}_2 \) increases in a linear fashion, and the higher \( \Omega \), the higher the \( \text{Do}_2 \). Further, despite the large differences in CO, all the points lie on the same line. For the
lower CO (300 mL min⁻¹ kg⁻¹), the points lie on the lower left side of the line at the lower levels of DO₂. For the higher CO (450 mL min⁻¹ kg⁻¹), the points lie on the upper right side of the line.

Discussion

The management of neonates with complex congenital anomalies such as hypoplastic left heart syndrome depends on careful interpretation of arterial blood gas values. Improved interpretation of these oxygen parameters may allow clinicians to avoid unexpected cardiovascular events. Thus, we used a previously developed computer simulation to obtain DO₂ as a function of SaO₂, Svo₂, SvO₂, and Qp/Qs. Additionally, based on work by Buheitel et al. we examined Ω. The primary findings are as follows:

1. Slight increases in SaO₂ may be associated with large decreases in DO₂.
2. In the immediate post-operative period, low values for Svo₂ indicate low values for DO₂.
3. The curves for Sa-vo₂ and Qp/Qs are redundant in the data provided Qp/Qs, however, provides these data in more physiologically relevant terms.
4. High values for Qp/Qs (>4) are associated with low DO₂, which is only partially compensated by increases in CO.
5. Estimating Qp/Qs from oxygen saturation measurements may result in errors when Spvo₂ is not available.
6. Maximizing DO₂ is extremely difficult using just SaO₂, Svo₂, or Qp/Qs.
7. A linear relationship exists between Ω and DO₂, and this linear relationship is not altered by changes in CO and Spvo₂.

Clinical Implications

The Ω is based only on systemic oxygen saturation measurements and reflects relative excess oxygen delivery. The Ω is calculated as SaO₂ divided by Sa-vo₂, and it is the ratio of oxygen delivery to oxygen consumption (the reciprocal of the oxygen-extraction ratio). Compared with the other relationships, a linear relationship exists between DO₂ and Ω. As Ω...
increases, \( D_O_2 \) increases in a linear fashion, and the higher the \( \Omega \), the higher the \( D_O_2 \). Thus, irrespective of \( CO \) and \( SpvO_2 \), high \( \Omega \) values are associated with high levels of \( D_O_2 \). This suggests that \( \Omega \) may be a better index to use to maximize \( D_O_2 \).

The one disadvantage of \( \Omega \) is that the slope of \( \Omega \) versus \( D_O_2 \) varies with oxygen consumption (see Figure 7). Situations that increase \( D_O_2 \) but have greater percentage increases in oxygen consumption would reduce the \( \Omega \) even though total \( D_O_2 \) increased. Again, \( \Omega \) is the relative excess in \( D_O_2 \). The implications of an increase in \( D_O_2 \) with a decrease in \( \Omega \) needs further consideration.

Based on this analysis and discussion, one strategy for perioperative management of patients with hypoplastic left heart syndrome might be to measure \( SaO_2 \) and \( SvO_2 \). Patients with low \( SvO_2 \) values require immediate attention. An elevated estimated \( Qp/Qs \) ratio would indicate excessive \( Qp \). Ideally, after reducing the estimated \( Qp/Qs<1.5 \), \( \Omega \) would guide further therapy to maximize \( D_O_2 \). Interventions that increased \( \Omega \) would be considered beneficial, whereas interventions that decreased \( \Omega \) would be considered detrimental.

**Comparison to Literature**

We have recently developed a stable, closed heart model of a univentricular heart. We used neonatal pigs (3.5 to 6.0 kg) to anastomose a Gore-Tex graft from the innominate artery and to the pulmonary artery. We created an atrial septostomy by using a Rashkind septostomy catheter. Oclauding the right ventricular outflow tract completed a univentricular circuit in which all CO exited from the left ventricle and the pulmonary circulation was maintained via flow through the innominate artery-to-pulmonary artery shunt.9

We used this model to examine the response to inotropic agents in the univentricular circulation.10 The response to inotropic agents depended upon the agent. Dobutamine (15 \( \mu g \cdot kg^{-1} \cdot min^{-1} \)) increased the \( Qp/Qs \) ratio (1.03 to 2.52), while epinephrine (0.1 \( \mu g \cdot kg^{-1} \cdot min^{-1} \)) decreased the \( Qp/Qs \) ratio (1.23 to 0.82). Associated with these changes, dobutamine decreased \( D_O_2 \) from 50 to 36 mL O2 per minute, while epinephrine increased \( D_O_2 \) from 40 to 56 mL O2 per minute. If we apply the index \( \Omega \) to these data, we can derive the same conclusions: dobutamine decreased \( \Omega \) from 1.96 to 1.86, while epinephrine increased \( \Omega \) from 1.63 to 2.22.

Buheitel et al10 examined the ability of \( SvO_2 \) and \( \Omega \) to estimate cardiac index. They studied 25 infants and children with biventricular pathophysiology in the postoperative period after complete repair of congenital heart disease. Cardiac index was calculated using the Fick principle. \( SvO_2 \) provided a reasonable estimate of cardiac index. However, \( \Omega \) provided the best estimate of cardiac index.

Seear et al11 examined the oxygen consumption-delivery relationship in children. In stable children with normal lactate levels, they found no significant rise in oxygen consumption when oxygen delivery was increased by erythrocyte transfusion. Conversely, infusion of adrenaline increased both consumption and \( D_O_2 \). Berman et al12 examined oxygen transport in patients with congenital heart disease. They found that \( D_O_2 \) varied in direct relation to \( CO \), but not with arterial oxygen content. \( D_O_2 \) varied with \( SvO_2 \).

The oxygen extraction ratio is also predictive of outcome. Rossi et al13 studied 49 infants after congenital heart surgery. The infants were intubated, paralyzed, and sedated with a continuous infusion of fentanyl. Arterial blood gas data and mixed venous oxygen saturation were measured on admission to the intensive care unit and at 6 hours and 24 hours. Severe derangements in the oxygen extraction ratio were present in nonsurvivors at 6 hours after admission to the intensive care unit.11 Infants with an oxygen extraction ratio >0.5 at 6 hours were at a much greater risk of dying.

**Limitations of Study**

This analysis is based on a few simple equations describing the flow of oxygen in the univentricular circulation. The analysis showed that \( D_O_2 \) is a complex function of \( CO \), \( CpvO_2 \), \( CV_O2 \), and the \( Qp/Qs \) ratio. For given values of \( Qp/Qs \), \( CO \), and \( SpvO_2 \), the analysis can predict the \( D_O_2 \). However, the analysis cannot describe how these values for \( Qp/Qs \), \( CO \), and \( SpvO_2 \) are achieved; nor can it predict the whole body response to a change in one variable. For example, a decrease in \( SpvO_2 \) may increase pulmonary vascular resistance, improve the \( Qp/Qs \) ratio, and lead to an increased \( D_O_2 \). A more complex model of the circulation is needed to predict whole-body responses to changes in the variables.14

Obviously, care should always be taken in extrapolating from a theoretical analysis to a clinical situation. One concern is the measurement of mixed venous oxygen saturation. To measure the saturation of mixed venous blood, samples must be recovered from a central site at which all systemic venous blood is fully mixed.15 In a theoretical model, this is easy to achieve. However, in a neonate with hypoplastic left heart syndrome, it is impossible to achieve. The pulmonary artery (or, after the Norwood procedure, the neoaorta) contains both oxygenated and deoxygenated blood. The right atrium also contains both oxygenated and deoxygenated blood. This necessitates sampling in the superior or inferior vena cava. The great veins generally are not used for central venous sampling, because the relative saturations of the superior and inferior vena cavae differ greatly with hemodynamic status.16 With no other choice remaining, blood samples must be obtained from the vena cava, and although this blood is not a truly mixed sample, it should represent trends. By convention, the saturation in the superior vena cava has been used by most pediatric cardiologists as a reflection of the mixed venous oxygen saturation.

**Summary**

This study examined whether \( D_O_2 \) can be maximized by the use of indices derived from oxygen saturation measurements in neonates with hypoplastic left heart syndrome. We used a previously developed computer simulation to obtain \( D_O_2 \) as a function of \( SaO_2 \), \( SvO_2 \), \( Sa VO_2 \), and \( Qp/Qs \). We also examined the \( \Omega \). Because of nonlinear relationships, maximizing \( D_O_2 \) is extremely difficult by \( SaO_2 \), \( SvO_2 \), or \( Qp/Qs \). However, a linear relationship exists between \( \Omega \) and \( D_O_2 \), and this linear relationship is not altered by changes in \( CO \) and \( SpvO_2 \). Ideally, after reducing \( Qp/Qs<1.5 \), \( \Omega \) might be a better index to guide further therapy and maximize \( D_O_2 \).
Acknowledgment
This study was supported in part by a grant from the Alliant Community Trust Fund.

References
Estimation of Oxygen Delivery in Newborns With a Univentricular Circulation
Ofer Barnea, William P. Santamore, Anthony Rossi, Ellis Salloum, Sufan Chien and Erle H. Austin

*Circulation*. 1998;98:1407-1413
doi: 10.1161/01.CIR.98.14.1407

*Circulation* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1998 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/98/14/1407

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Circulation* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to *Circulation* is online at:
http://circ.ahajournals.org/subscriptions/