The theoretical optimization of pulmonary-to-systemic flow ratio after a bidirectional cavopulmonary anastomosis

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Santamore, William P., Ofer Barnea, Christopher J. Riordan, Mitchell P. Ross, and Erle H. Austin. Theoretical optimization of pulmonary-to-systemic flow ratio after a bidirectional cavopulmonary anastomosis. Am. J. Physiol. 274 (Heart Circ. Physiol. 43): H694–H700, 1998.—A univentricular circulation characterized by parallel pulmonary and systemic circulations is inherently inefficient because mixing of pulmonary and systemic venous return occurs. Thus a cavopulmonary anastomosis is used as a staged palliative procedure to reduce volume overload in patients with cyanotic congenital heart disease. On the basis of oxygen uptake and consumption, an equation was derived that related cardiac output, pulmonary venous oxygen saturation, upper body oxygen consumption, and superior-to-inferior vena caval blood flow ratio (QSVC/QIVC) to oxygen delivery. The primary findings were as follows. 1) As QSVC/QIVC increases, total body oxygen delivery and arterial and superior vena caval oxygen saturations increase. 2) As QSVC/QIVC increases, lower body oxygen delivery and inferior vena caval oxygen saturation initially increase, then peak, and then decrease. 3) As the percentage of lower body oxygen consumption increases, oxygen delivery and saturation decrease. 4) A cavopulmonary anastomosis decreases the required cardiac output for a given oxygen delivery. Thus we concluded that a high systemic arterial oxygen saturation after cavopulmonary anastomosis requires a high percentage of upper body oxygen consumption and a high QSVC/QIVC and that the cavopulmonary anastomosis reduces the volume load on the single ventricle.

hypoplastic left heart syndrome; pulmonary blood flow; aortic blood flow; computer models; infants

HYPOPLASTIC LEFT HEART syndrome is currently the most common cardiac malformation that results in death of the newborn infant (17). Without treatment, 95% of these infants die during the first month of life and none survive beyond 4 mo (23). The management of neonates with hypoplastic left heart syndrome is complex. Palliative surgery, developed by Norwood, involves several staged operations (6, 19). The first surgery stabilizes the univentricular circulation by attaching the right ventricular outflow to the aorta and placing an aorta-to-pulmonary artery shunt. With this approach, survival beyond the first few weeks became possible (19).

Definitive long-term palliation requires a modified Fontan procedure, in which the aorta-to-pulmonary artery shunt is closed and venous blood flows directly into the pulmonary artery, completely bypassing the right heart. Because of an observed high mortality rate from the Fontan operation for hypoplastic left heart syndrome, a bidirectional cavopulmonary anastomosis or Glenn shunt procedure is now often performed before the Fontan operation (5, 8, 11, 12, 20). This procedure, in which the superior vena cava is anastomosed to the pulmonary artery, is thought to diminish the effects of volume overload on the single ventricle. Because natural animal models of this heart defect do not exist, management of infants with hypoplastic left heart syndrome has been derived primarily from clinical experience, determined by trial and error. Thus we previously developed a theoretical analysis to determine the effect of blood flow distribution between the two circulations on systemic oxygen delivery (4). In the present study, we performed a similar theoretical analysis to determine the effects of a bidirectional cavopulmonary anastomosis in the univentricular circulation on systemic oxygen delivery and on the required cardiac output to provide this systemic oxygen delivery.

METHODS

A model of this circulation is shown in Fig. 1. After the Glenn shunt, the blood that normally drains into the right atrium from the superior vena cava is redirected via an anastomosis to the pulmonary artery and flows passively through the pulmonary circulation. Oxygenated pulmonary venous blood is routed from the left atrium via an interatrial septal defect to the right atrium, where it mixes with systemic venous blood. Note that with hypoplastic left heart syndrome, the right ventricle does all the pumping of blood. The right ventricular outflow goes into a reconstructed aorta, where it is divided into flow to the upper and lower systemic circulations. In effect, this forms a parallel circulation: the combined upper systemic circulation and pulmonary circulation is in parallel with lower systemic circulation. Note that although Fig. 1 and the analysis below are for hypoplastic left heart syndrome, the analysis also is applicable to the other univentricular conditions.

The analysis is based on movement of oxygen into the pulmonary circulation (uptake) and out of the systemic circulation (consumption). The basic equations are as follows

\[
V_{O2} = k \cdot V_{O2} + (1 - k) \cdot \dot{V}_{O2}
\]

(1)
ventricular output (cardiac output), respectively; CPVO2, CSVCO2, CIVCO2, and CaO2 are oxygen content (ml O2/ml blood) in pulmonary venous blood, superior vena cava blood, inferior vena cava blood, and mixed blood ejected from right ventricle, respectively. VO2 is the rate of oxygen supply or uptake in the lungs and VO2 is whole body oxygen consumption. RV, right ventricle; RA, right atrium; LA, left atrium; SVC-PA anastomosis, superior vena cava-to-pulmonary artery anastomosis; Ao, aorta; k, fraction of whole body oxygen consumption used by the upper body.

Equation 1 states that whole body oxygen consumption (VO2, in ml O2/min) is divided between the upper body [blood draining into the superior vena cava (Q SVC)] and lower body oxygen consumption [(1 - k)VO2], where k is the fraction of whole body oxygen consumption used by the upper body. For the lower systemic circulation

\[ \text{CaO}_2 \cdot Q_{\text{SVC}} - (1 - k) \cdot \dot{V}_{O2} = C_{\text{IVC}}O_2 \cdot Q_{\text{IVC}} \]  

Equation 2 states that the oxygen flow rate into the lower systemic circulation is a product of arterial oxygen content (CaO2 in ml O2/ml blood) and lower body blood flow [blood draining into the inferior vena cava (QIVC)]. This oxygen flow rate into the lower systemic circulation is reduced by the lower body oxygen consumed (ml O2/min), leaving the reduced oxygen flow rate returning to the right atrium (CIVCO2·QIVC), where CIVCO2 is the oxygen content of the inferior vena cava blood. Similarly

\[ \text{CaO}_2 \cdot Q_{\text{SVC}} - k \cdot \dot{V}_{O2} = C_{\text{SVC}}O_2 \cdot Q_{\text{SVC}} \]  

Equation 3 states that the oxygen flow rate into the upper systemic circulation (CaO2·Q SVC) is reduced by the upper body oxygen consumed (k·VO2), leaving the reduced oxygen flow rate returning to the superior vena cava and then to the pulmonary artery (CSCVCO2·Q SVC), where CSCVCO2 is the oxygen content of the superior vena cava blood

\[ Q_p = Q_{\text{SVC}} \]  
\[ C_{\text{SVC}}O_2 \cdot Q_{\text{SVC}} + \dot{V}_{O2_{\text{a}}} = CPV_{O2} \cdot Q_p \]  

Equation 4a states that pulmonary artery blood flow (Qp) is equal to blood flow to the upper systemic circulation, i.e., Q SVC. Equation 4b states that the oxygen flow rate into the pulmonary circulation (CSCVCO2·Q SVC) plus the oxygen uptake in the lungs (VO2a) gives the oxygen flow rate returning to the atrium from the pulmonary circulation (CPVO2·Qp), where CPVO2 is the oxygen content of the superior vena cava blood.

Equation 5 relates blood flow in the upper and lower systemic circulations to total cardiac output (CO)

\[ CO = Q_{\text{IVC}} + Q_{\text{SVC}} = Q_{\text{IVC}} + Q_p \]  

The analysis assumes a steady-state condition. At the cellular level, on the basis of the law of mass conservation, oxygen uptake and oxygen consumption must be equal. Therefore

\[ \dot{V}_{O2_{\text{a}}} = k \cdot \dot{V}_{O2} + (1 - k) \cdot \dot{V}_{o2} \]

By replacing C SVC O2·Q SVC in Eq. 4b with Eq. 3 and replacing Qp with Q SVC by using Eq. 4a, and then using Eq. 6, we obtain

\[ \text{CaO}_2 \cdot Q_{\text{SVC}} + (1 - k) \cdot \dot{V}_{o2} = CPV_{O2} \cdot C_{\text{SVC}}O_2 \]  

Using Eq. 5 gives

\[ \text{CaO}_2 \cdot Q_{\text{SVC}} + (1 - k) \cdot \dot{V}_{o2} = CPV_{O2} \cdot C_{\text{SVC}}O_2 \]  

Using Eqs. 3, 4, and 5, note that

\[ \text{CaO}_2 \cdot \text{Q}_{\text{IVC}} - \text{CPV}_{O2} \cdot \text{Q}_{\text{IVC}} \]

Combining Eqs. 8 and 9, systemic arterial oxygen delivery (CaO2·CO) equals

\[ \text{CaO}_2 \cdot \text{CO} = \text{CPV}_{O2} \cdot \text{CO} - (1 - k) \cdot \dot{V}_{o2} \]

Thus systemic oxygen delivery is a complex function of cardiac output, pulmonary venous blood oxygen content, lower body oxygen consumption [(1 - k)·VO2], and Q SVC/QIVC.

Using a Compaq computer (Deskpro 486 66dx, Compaq Computer, Houston, TX), we studied this relationship by altering each variable individually while considering the following constraints. The relevant physiological and physical constraints were that the arterial oxygen content cannot be less than zero and that blood flow rates (Q SVC and Q IV C) must be positive, i.e., in the direction of the Q SVC and Q IV C arrows in Fig. 1. In RESULTS, blood oxygen content was converted to percent oxygen saturation by assuming a hemoglobin concentration ([Hb]) of 15 g/dl, giving an oxygen capacity of 22 ml O2/dl blood (1.38 × 15[Hb]).

RESULTS

Figure 2A shows oxygen delivery (top) and saturations (bottom) as a function of the ratio Q SVC/Q IV C. In this example, the whole body oxygen consumption was set to 9 ml O2/min kg-1, which represents a normal mean value for infants and young children (18). The percentage of oxygen consumed by the upper body, k, was set to 60%. Figure 2A, top, shows that as Q SVC/Q IV C increases, lower body systemic oxygen delivery initially increases. This is because Q SVC, which is equal to pulmonary flow, increases. However, as Q SVC/Q IV C in-

Fig. 1. A model of the hypoplastic left heart circulation after a cavopulmonary anastomosis. P is pulmonary circulation; S L and S U are lower and upper systemic circulations, respectively; Q P (or Q SVC), Q IV C, and CO are pulmonary flow (upper systemic or superior vena cava flow), slower systemic flow (inferior vena caval flow), and right ventricular output (cardiac output), respectively; CPVO2, CSVCO2, CIVCO2, and CaO2 are oxygen content (ml O2/ml blood) in pulmonary venous blood, superior vena cava blood, inferior vena cava blood, and mixed blood ejected from right ventricle, respectively. VO2 is the rate of oxygen supply or uptake in the lungs and VO2 is whole body oxygen consumption. RV, right ventricle; RA, right atrium; LA, left atrium; SVC-PA anastomosis, superior vena cava-to-pulmonary artery anastomosis; Ao, aorta; k, fraction of whole body oxygen consumption used by the upper body.
creases further, lower body systemic oxygen delivery reaches a maximum and then decreases. This decrease is due to the decrease in \(Q_{IVC}\). At the peak of the lower body oxygen delivery, the optimal value for \(Q_{SVC}/Q_{IVC}\) is \(1\) (i.e., \(Q_{IVC} = Q_{SVC}\) or \(Q_p\)). In contrast, upper body and whole body (total) systemic oxygen delivery increase continuously as \(Q_{SVC}/Q_{IVC}\) increases.

Figure 2A, bottom, shows the relationship of oxygen saturations vs. \(Q_{SVC}/Q_{IVC}\). As \(Q_{SVC}/Q_{IVC}\) increases, systemic arterial (\(S_{A\text{O}_2}\)) and superior vena caval oxygen saturation (\(S_{SVC\text{O}_2}\)) continually increase. The changes in inferior vena caval oxygen saturation (\(S_{IVC\text{O}_2}\)) resemble the changes in lower body systemic oxygen delivery: as \(Q_{SVC}/Q_{IVC}\) increases, \(S_{IVC\text{O}_2}\) initially increases, reaches a maximum, and then decreases.

In Fig. 2A, top and bottom, there are two vertical dashed lines. These lines represent the effects of early development. The line on the right has a \(Q_{SVC}/Q_{IVC}\) of 1.56, which was the average value found in children (mean age 2.95 yr) after a Glenn shunt procedure (21). The line on the left has a \(Q_{SVC}/Q_{IVC}\) of 0.65, which is the average value for 6-yr-old children (21). As the child develops, the lower body grows proportionately more than the upper body. Thus \(Q_{SVC}/Q_{IVC}\) will decrease. Thus normal early growth will decrease upper body oxygen delivery and \(S_{A\text{O}_2}\).}

Figure 2B plots \(S_{SVC\text{O}_2}\) and \(S_{IVC\text{O}_2}\) vs. \(Q_{SVC}/Q_{IVC}\). The solid lines were obtained with the percentage of oxygen consumed by the upper body, \(k\), set to 60%, whereas for the dashed lines \(k\) was set to 30%. \(S_{IVC\text{O}_2}\) is affected by this percentage. Lowering the percentage of oxygen consumed by the upper body (or conversely increasing the percentage of oxygen consumed by the lower body) decreases \(S_{IVC\text{O}_2}\). However, \(S_{IVC\text{O}_2}\) is only minimally affected by the percentage of oxygen consumed by the upper body. The decrease in upper body oxygen consumption and the decrease in arterial oxygen content tend to cancel each other, leading to only minimal changes in \(S_{IVC\text{O}_2}\). Also note in Fig. 2B the points of intersection at which superior and inferior oxygen saturations are equal (\(S_{SVC\text{O}_2} = S_{IVC\text{O}_2}\)). At these points, blood flow is matched to oxygen demands \((Q_{SVC}/Q_{IVC} = k/(1 - k))\). When \(S_{SVC\text{O}_2} > S_{IVC\text{O}_2}\), the superior vena cava blood flow is disproportionately higher than oxygen demands and/or inferior vena cava blood flow is disproportionately lower than oxygen demands. Conversely, when \(S_{SVC\text{O}_2} < S_{IVC\text{O}_2}\), the superior vena cava blood flow is disproportionately lower than oxygen demands and/or inferior vena cava blood flow is disproportionately higher than oxygen demands. Note in Fig. 2A and B, pulmonary venous oxygen saturation (\(S_{P\text{VO}_2}\)) = 98%, CO = 200 ml·min\(^{-1}\)·kg\(^{-1}\).

Figure 3 plots the maximum systemic oxygen delivery possible at any given cardiac output. The results are shown for the present Glenn shunt simulation, for our previously published hypoplastic left heart syndrome circulation simulation (4), and for the nonfenestrated Fontan circulation. Figure 3 shows the equations used to calculate the maximal systemic oxygen delivery. For the Glenn shunt, because there is no obvious peak oxygen delivery, we tied \(Q_{SVC}/Q_{IVC}\) to the ratio of upper to lower body oxygen consumption \((Q_{SVC}/Q_{IVC} = k/(1 - k))\).

For all circulations, a higher peak systemic oxygen delivery requires a higher cardiac output. In the nonfenestrated Fontan circulation, deoxygenated blood is not recirculated, and thus this is the most efficient circulation. For the same oxygen delivery, the nonfenes-
trated Fontan circulation requires considerably less cardiac output than the hypoplastic left heart syndrome circulation does. The Glenn shunt is between the nonfenestrated Fontan and the hypoplastic left heart syndrome circulations. The slope of oxygen delivery vs. cardiac output is the same for the Glenn shunt and the nonfenestrated Fontan circulation. The improvement in oxygen delivery after the Glenn shunt operation depends on the $k$ value: the higher the percentage of oxygen consumed by the upper body, the lower the cardiac output required for the same systemic oxygen delivery. Both early development, with its relative increase in lower body size compared with the upper body, and exercise (walking, running) will decrease $k$ and thereby decrease the efficiency of the Glenn shunt circulation.

Figure 4, A and B, plots whole body oxygen delivery and arterial and venous oxygen saturations, respectively. These variables are plotted against $k$. Again, we tied $Q_{SVC}/Q_{IVC}$ to the ratio of upper to lower body oxygen consumption [$Q_{SVC}/Q_{IVC} = k/(1 - k)$]. As the percentage of upper body oxygen consumption decreases, whole body oxygen delivery and arterial and venous oxygen saturations decrease. Exercise (walking, running) will increase lower body oxygen consumption and thus reduce $k$. In younger children, growth will increase the relative size of the lower body and thus reduce $k$. Both of these factors (exercise and growth) are additive. Hence, early development and exercise, by themselves, will decrease oxygen delivery and decrease blood gases. This situation is depicted in Fig. 4, A and B. As the infant grows (dashed line A to dashed line B), oxygen delivery and arterial and venous oxygen saturations will decrease. If the child starts to walk or run now (dashed line B to dashed line C), exercise will decrease further.

Figure 5 plots the ratio of Glenn to nonfenestrated Fontan circulation cardiac output ($CO_{Glenn}/CO_{nf-Fontan}$) vs. $k$. $CO_{Glenn}/CO_{nf-Fontan}$ indicates how much extra cardiac output is required in the Glenn shunt circulation for the same oxygen delivery as in the nonfenestrated Fontan or normal circulation. A ratio of 2 would indicate that the Glenn shunt circulation would require twice the nonfenestrated Fontan cardiac output for the same oxygen delivery. Figure 5 plots these relationships for resting conditions ($CO = 200 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$) and for mild exercise ($CO = 400 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$). As $k$ decreases, the efficiency of the Glenn circulation decreases. However, this decrease is not linear. For $k \leq 50\%$, the efficiency decreases rapidly. Figure 5 also shows the effects of early development and exercise on efficiency. As the infant grows (dashed line A to dashed line B), efficiency decreases. If the child starts to walk or run now (dashed line B to dashed line C), efficiency will decrease further.
ratios (QP/QS) in 29 children (mean age 2.95 yr) studied et al. (21) examined the pulmonary-to-systemic flow l·min
SSVCO₂ and SIVCO₂ are different.
oxygen delivery as the univentricular circulation.
creases the required cardiac output to achieve the same bidirectional cavopulmonary anastomosis greatly de-
for a high SaO₂ after the Glenn shunt operation, a high QSVC/QIVC, and a high
3
Q₅/SQ₅, and a high QSVC/QIVC to systemic oxygen delivery.
The key findings are as follows. 1) As QSVC/QIVC increases, upper body and total oxygen delivery in-
creases along with systemic SaO₂ and SsvCO₂. 2) As QSVC/QIVC increases, lower body oxygen delivery and SsvCO₂ initially increase, then peak, and then decrease.
3) For a high systemic SaO₂, after a bidirectional cavo-
polmonary anastomosis, a low percentage of oxygen consumed by the lower body and a high Qsv/Qivc are needed. 4) As the percentage of oxygen consumed by the lower body increases with early development or exer-
cise, whole body oxygen delivery and SaO₂ decreases. 5) A bidirectional cavopulmonary anastomosis greatly decreases the required cardiac output to achieve the same oxygen delivery as the univentricular circulation. 6) SsvCO₂ and SsvCO₂ are different.

The present analysis examined the balance between pulmonary and lower body systemic blood flows in univentricular circulation after a bidirectional cavopulmonary anastomosis. The model was based on the flow of oxygen uptake in the lungs and of whole body oxygen consumption. The analysis developed an equation relating the key variables of cardiac output, pulmonary venous oxygen saturation, lower body oxygen consumption, and QSVC/QIVC to systemic oxygen delivery.

DISCUSSION

The analysis indicates that the Glenn shunt is more efficient than the hypoplastic left heart circulation but less efficient than the nonfenestrated Fontan circulation. This implies that the systemic ventricle will decrease in size after the Glenn shunt operation but will still be larger than normal. Consistent with these ideas, several clinical studies have reported a significant decrease in the systemic ventricular volume after the Glenn operation. In patients with tricuspid atresia, LaCorte et al. (14) reported that two patients with a Glenn shunt had the least abnormal values for volumes and ejection fraction. Graham et al. (9) reported that exceed 80%. In our controls, we set pulmonary venous oxygen saturation at 98%, the same value reported by Salim et al. (98%) (21). Cardiac output was set to 200 ml·min⁻¹·kg⁻¹ in our analysis. Assuming that a 4-kg baby has a height of 53 cm for a body surface area of 0.19 m², this gives a cardiac output of 4.2 l·min⁻¹·m⁻², similar to the value reported by Salim et al. (21). As per Qp/QS, note that we calculated this as pulmonary flow divided by inferior vena caval flow and not as pulmonary flow divided by total systemic flow (Qsv/Qsv + Qivc). Thus a Qp/QS of 0.61 would equal a Qp/QS of 1.56, and Qp/QS can never be >1.

In our controls, we set the percentage of upper body oxygen consumption to 60%, close to the value estimated from the data of Salim et al. (21). The mixing of the systemic venous blood and pulmonary venous blood lowers the resulting arterial oxygen tension. If the lower body uses more oxygen, systemic venous return oxygen tension decreases, thereby lowering the subsequent systemic arterial oxygen tension. Conversely, upper body oxygen consumption does not affect systemic arterial oxygen tension: the deoxygenated blood from the upper body flows from the superior vena cava into the lungs and is reoxygenated in the lungs. This is the advantage of the Glenn shunt.

Cardiac output and oxygen delivery. In the normal circulation, all the deoxygenated blood flows through the lungs, and only oxygenated blood is supplied to the systemic circulation. In the hypoplastic left heart circulation, the systemic and pulmonary circulations are in parallel. Thus, for the same systemic oxygen delivery, the univentricular circulation requires almost twice the normal cardiac output. The Glenn shunt is between the nonfenestrated Fontan and hypoplastic left heart circulations. The blood flow to the upper body is in series with the pulmonary circulation. The deoxygenated blood flows into the lungs, similar to the normal circulation. Thus there is no wasted energy or decreased efficiency in the upper circulation. However, the deoxygenated blood from the lower body is mixed with the oxygenated blood from the pulmonary veins, thereby reducing systemic oxygen saturation. This is the wasted energy in the Glenn shunt. Although physiologically impossible, the extremes provide insight. With no upper body oxygen consumption, the Glenn shunt becomes the hypoplastic left heart circulation again. With no lower body oxygen consumption, the Glenn shunt becomes a nonfenestrated Fontan circulation.

The analysis indicates that the Glenn shunt is more efficient than the hypoplastic left heart circulation but less efficient than the nonfenestrated Fontan circulation. This implies that the systemic ventricle will decrease in size after the Glenn shunt operation but will still be larger than normal. Consistent with these ideas, several clinical studies have reported a significant decrease in the systemic ventricular volume after the Glenn operation. In patients with tricuspid atresia, LaCorte et al. (14) reported that two patients with a Glenn shunt had the least abnormal values for volumes and ejection fraction. Graham et al. (9) reported that
three patients with a Glenn shunt had the smallest volumes among the patients with postshunt tricuspid atresia. Kobayashi et al. (13) showed that the systemic ventricular end-diastolic volume index decreased significantly, from 141 ml/m² before to 98 ml/m², 1 mo after bidirectional cavopulmonary shunt. Similarly, Allgood et al. (2) showed a significant decrease in the ventricular volume with the bidirectional Glenn shunt procedure.

Effects of early development and exercise. As stated above, the reduction in arterial oxygen tension is caused by lower body oxygen consumption. This is the extra cardiac output required in the univentricular circulation after the Glenn shunt. Thus anything that increases the percentage of lower body oxygen consumption will decrease systemic oxygen delivery and necessitate a higher cardiac output. Our analysis would predict that with walking or running or after a meal, the percentage of oxygen consumed by the upper body is increased. Another important and uncontrollable cause for increases in lower body oxygen consumption is early development or growth. As the child becomes older, the lower body grows proportionately more than the upper body (16). This will decrease the proportion of upper body oxygen consumption and result in a decrease in $\text{SaO}_2$.

Several clinical studies and observations are consistent with these predictions. The Glenn shunt palliation appears to be good for the first 5–7 yr and deteriorates after 7 yr (3, 7, 9, 15) with progressive cyanosis. Cyanosis may be due to collaterals between the superior and inferior vena cava system, due to a pulmonary arteriovenous shunt, and due to ventricular dysfunction. Independent of these effects, age also may be a factor. Gross et al. (10) analyzed the data from 45 patients with univentricular congenital heart disease after a bidirectional cavopulmonary anastomosis. They found that the patient’s age and body surface area were associated with postoperative desaturation independently of other known causes of cyanosis. Likewise, Allgood et al. (2) showed age to be independently associated with the ventricular volume status before and late after the Glenn procedure and with the early postoperative ventricular mass. Thus older age may be a risk factor for cyanosis, perhaps because of a smaller proportion of blood return from the superior vena cava relative to the inferior vena cava. For example, in the study of Gross et al. (10), $Q_s/Q_o$ was 0.69 for younger children (2.8 yr) but only 0.44 in the older children (9.6 yr). In 145 healthy children, Salim et al. (22) observed that the superior vena caval flow accounted for 49% of cardiac output in newborn infants, reached a maximum of 55% at age 2.5 yr, and then gradually decreased to the adult value of 35% by 6.6 yr. Obviously, additional clinical observations are needed to verify these theoretical predictions.

In this model, both declines in $\text{SpV}_O_2$, and increases in oxygen consumption were associated with significant declines in systemic oxygen saturation, and these effects were additive. Alteration in $\text{SpV}_O_2$, related to intrapulmonary shunting of blood results in a lowering of the absolute ceiling for systemic oxygenation due to incomplete oxygenation of upper body systemic blood return. In contrast, given a fixed $\text{SpV}_O_2$, an increase in oxygen consumption will only influence the lower body contribution to systemic oxygenation (the second term on the right side of Eq. 10); therefore, the influence of increased oxygen consumption will be greatest if the percentage of oxygen consumed by the upper body is small. This influence will be greater as the child grows (decreasing k) and will be accentuated by physical activity such as running.

Timing of Fontan operation. The analysis also provides some insight into when to perform the Fontan procedure. Assuming blood flow and oxygen demands are matched [$Q_{\text{svc}}/Q_{\text{ivc}} = k(1 - k)$], our analysis shows that as $Q_{\text{svc}}/Q_{\text{ivc}}$ decreases, the advantages of the Glenn shunt decrease: i.e., mechanical efficiency, systemic oxygen delivery, and arterial and venous oxygen saturations all decrease. In healthy children, $Q_{\text{svc}}/Q_{\text{ivc}} = 0.96$ in newborn infants, reaches a maximum of 1.22 at age 2.5 yr, and then gradually decreases to the adult value of 0.54 by 6.6 yr (22). This would suggest that 2.5–3 yr of age would be the age to start to consider the Fontan procedure. Because these flow ratios were determined in normal children, another approach, if possible, would be to directly measure $Q_{\text{svc}}/Q_{\text{ivc}}$ in the patient. When this ratio is $\leq 1$ ($Q_{\text{svc}} \leq Q_{\text{ivc}}$), the Fontan operation should be considered. Obviously, many factors need to be considered. This analysis only considers the effects of $Q_{\text{svc}}/Q_{\text{ivc}}$ and the percentage of lower body oxygen consumption. Thus the suggested age may be too early when all the other relevant factors are taken into consideration.

Limitations. In Figs. 3–5, we assumed that blood flow and oxygen consumption were linearly related. If the upper body consumed 60% of total body oxygen, then $Q_{\text{svc}}$ equaled 60% of total cardiac output. This assumption allowed us to plot oxygen delivery and oxygen saturations vs. $k$ and relate this to changes in growth and exercise. If the upper body had a disproportionately higher flow ($Q_{\text{svc}}$) compared with its oxygen consumption, the effects of growth and exercise would be less than shown in Figs. 3–5. Conversely, if the lower body had a higher flow ($Q_{\text{ivc}}$) compared with its oxygen consumption, then the effects of growth and exercise would be greater. Without any data available from the literature on responses during exercise, the exact relationship between flow and oxygen consumption is impossible to determine. Thus we used a linear assumption.

Whether $Q_{\text{svc}}/Q_{\text{ivc}}$ can be acutely changed after Glenn shunt operation is unknown. One view is that because upper body systemic vascular resistance is in series with the pulmonary vascular resistance and because the upper body systemic vascular resistance is so much greater than pulmonary vascular resistance, small changes in pulmonary vascular resistance will have only minimal effects on $Q_{\text{svc}}/Q_{\text{ivc}}$. We believe that the systemic upper body vascular resistance is like a waterfall. The pulmonary circulation is downstream
from the systemic arterial capillary waterfall. Thus pulmonary blood flow is not affected by systemic arterial pressure (by the height of the waterfall). Rather, pulmonary blood flow is passively controlled by pulmonary artery pressure and pulmonary vascular resistance. If these ideas are correct, then changes in pulmonary vascular resistance will have a large effect on $Q_{SVC}/Q_{PV}$ and will also elevate superior vena caval pressure. The development of the superior vena caval syndrome and an increase in mortality after the Glenn shunt procedure in patients with elevated pulmonary vascular resistance have been well described clinically (1). Experimental studies and further clinical observations will be necessary to resolve this issue.

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