Noninvasive Quantification of Systemic-to-Pulmonary Collateral Flow
A Major Source of Inefficiency in Patients With Superior Cavopulmonary Connections
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Background—Systemic-to-pulmonary collateral flow (SPCF) is common in single-ventricle patients with superior cavopulmonary connections (SCPC). Because no validated method to quantify SPCF exists, neither its hemodynamic burden nor its clinical impact can be systematically evaluated. We hypothesize that (1) the difference in total ascending aortic (Ao) and caval flow (superior vena cava [SVC]+inferior vena cava [IVC]) and (2) the difference between pulmonary vein and pulmonary artery flow (PV−PA) provide 2 independent estimators of SPCF.

Methods and Results—We measured Ao, SVC, IVC, right (RPA) and left (LPA) PA, and left (LPV) and right (RPV) PV flows in 17 patients with SCPC during routine cardiac MRI studies using through-plane phase-contrast velocity mapping. Two independent measures of SPCF were obtained: model 1, Ao−(SVC+IVC); and model 2, (LPV−LPA)+(RPV−RPA). Values were normalized to body surface area, Ao, and PV, and comparisons were made using linear regression and Bland-Altman analysis. SPCF ranged from 0.2 to 1.4 L/min for model 1 and 0.2 to 1.6 L/min for model 2, for an average indexed SPCF of 0.5 to 2.8 L/min/m²: 11% to 53% (mean, 37%) of Ao and 19% to 77% (mean, 54%) of PV. The mean difference between model 1 and model 2 was 0.01 L/min (P=0.40; 2-SD range, −0.45 to 0.47 L/min).

Conclusions—we present a noninvasive method for SPCF quantification in patients with SCPC. It should provide an important clinical tool in treating these patients. Furthermore, we show that SPCF is a significant hemodynamic burden in many patients with bidirectional Glenn shunt physiology. Future investigations will allow objective study of the impact of collateral flow on outcome. (Circ Cardiovasc Imaging. 2009;2:405-411.)

Key Words: single ventricle • collateral circulation • MRI • blood flow • superior cavopulmonary connection

It has long been recognized that single-ventricle patients with cavopulmonary anastomoses are susceptible to development of systemic-to-pulmonary collateral flow (SPCF).1,2 There has been much investigation and speculation into the etiology of these collaterals. Cyanosis, pleural effusion, and decreased pulmonary blood flow have all been implicated in the development of collateral flow to the lungs.2,3

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Controversy exists over the prevalence of collateral flow and more importantly, the significance of these collaterals. Some investigations have shown that the presence of significant SPCF is a risk factor for pleural effusion, poor outcomes, and heart failure.1,3,4 However, other investigators have failed to show a difference in outcome based on the amount of collateral flow.5 Some studies have demonstrated increased prevalence of collaterals in superior cavopulmonary connections (SCPC) compared with total cavopulmonary anastomoses.2 Collateral flow is potentially a significant source of power loss, and other research has suggested that collateral flow results in additional power loss in the Fontan pathway by transferring kinetic energy to the distal pulmonary vasculature and causing competitive flow losses.6

One of the major obstacles to investigating the importance of SPCF in single-ventricle physiology has been the inability to accurately quantify this flow. These collaterals have been classically identified and then graded qualitatively by angiography.2 However, the validity of the grading systems has never been verified. When a decision is made to coil collaterals thought to be hemodynamically significant, there is no good method of assessing the effect of the procedure.

One potential method to quantify collateral flow involves the use of MRI, using through-plane phase-contrast velocity mapping (PC-MRI), a technique that has been validated in multiple in vitro and clinical investigations.7−10 We recently published flow data obtained from PC-MRI velocity mapping...
on 105 Fontan patients in whom a significant difference was noted between the aortic flow and total caval flow. It was hypothesized that this difference was primarily due to SPCF. As a method of validating the difference between measured aortic and caval flow as an estimator of collateral flow, we propose a second estimator (the difference between measured pulmonary vein flow and pulmonary artery flow), providing 2 independent estimators of collateral flow.

The primary goal of this study was to validate the described noninvasive method to quantify SPCF. We hypothesize that collateral flow can be accurately estimated noninvasively in patients with superior cavopulmonary anastomoses by cardiac MRI, using phase-contrast velocity mapping. Furthermore, we hypothesize SPCF is a significant hemodynamic burden in many patients with superior cavopulmonary anastomoses.

**Methods**

**Patients**

We retrospectively investigated 17 consecutive patients who underwent superior cavopulmonary connections at the Children’s Hospital of Philadelphia and subsequently underwent a cardiac MRI from April to September of 2008. Patients were between 0.7 and 3.4 years of age (mean, 2.1 years). There were 8 patients with hypoplastic left heart syndrome, 5 patients with tricuspid atresia, 3 patients with heterotaxy, and 1 with double-inlet left ventricle. Ten patients had dominant right ventricles, 6 had dominant left ventricles, and 1 patient had 2 good-sized ventricles. There were 13 patients with superior cavopulmonary anastomoses (3 with bilateral SVCs, 2 with bilateral cavopulmonary connections, and 1 with a small left SVC still connected to the left atrium) and 4 patients with hemi-Fontan.

**Control Subjects**

To establish a control group, we retrospectively reviewed 13 2-ventricle patients (6 patients with arch anomalies and no prior surgery, as well as 7 postoperative 2-ventricle repair patients with no known residual shunts) who had complete pulmonary vein flow measurements. Seven of these patients also had vena caval velocity maps. These patients ranged in age from 1.7 years to 29 years.

**MRI Studies**

All patients underwent cardiac MRI consisting of balanced steady-state free precession (bright blood) and half-Fourier single-shot turbo spin-echo (dark blood) axial stacks, balanced steady-state free precession cine imaging for anatomy and to quantify ventricular function, gadolinium angiography, and through-plane PC-MRI cines as part of their routine clinical management. Multiplanar reformatting was used to set the position and angle of the imaging plane for anatomic and PC-MRI cines. Retrospectively gated, through-plane PC-MRI cines were performed in the aorta (native and/or neoaorta), superior and inferior vena cava (SVC and IVC), right and left pulmonary arteries (RPA and LPA), and right and left pulmonary veins (RPV and LPV). We have previously described the typical parameters and positions that we use to obtain the velocity mapping sequences. Figure 1 demonstrates the typical positions that were used to obtain the velocity maps. The aortic flow was measured at the sinuses. When 2 outflows were present (aorta and neoaorta), they were measured separately and added for the total aortic flow. Care
was taken to obtain the right pulmonary flow close to the SVC-RPA anastomosis to include the right upper lobe branch, which is often very close. The pulmonary veins were measured using an encoding velocity of 50 to 100 cm/s. When possible (based on whether all the pulmonary veins on 1 side formed a common vein of sufficient length before entering the atrium), all the pulmonary veins on 1 side were measured in 1 velocity map. This was more common for the left pulmonary veins, which often form a single confluence before joining the left atrium. When necessary, the upper and lower pulmonary veins, and occasionally a middle pulmonary vein, were measured separately.

### Analysis and Statistics

Velocity mapping sequences were previously analyzed using Argus flow analysis software on a Leonardo workstation (Siemens, Inc) to obtain the aortic, SVC, IVC, RPA, LPA, RPV, and LPV flows (Q). These flow values were obtained from the patient’s MRI report. The collateral flow was then calculated for each patient by the 2 different methods of collateral flow (Qcoll-syst and Qcoll-pulm) from equations 1 and 2:

1. \( Q_{\text{coll-syst}} = Q_{\text{aorta}} - (Q_{\text{SVC}} + Q_{\text{IVC}}) \)
2. \( Q_{\text{coll-pulm}} = (Q_{\text{RPV}} - Q_{\text{RPA}}) + (Q_{\text{LPV}} - Q_{\text{LPA}}) \)

where \( Q_{\text{coll-syst}} \) and \( Q_{\text{coll-pulm}} \) represent the estimated collateral flow by comparing supply and return of the systemic and pulmonary systems, respectively.

The collateral flow was normalized to aortic flow to determine the percentage of cardiac output, to body surface area to obtain an indexed flow, and to total pulmonary vein flow (\( Q_{\text{RPV}} + Q_{\text{LPV}} \)) to determine the percent of pulmonary flow from collateral flow. In addition, the total venous return to the heart was calculated (\( Q_{\text{IVC}} + Q_{\text{RPV}} + Q_{\text{LPV}} \)) to compare with the aortic flow as an indicator of internal consistency.

To establish interobserver and intraobserver variability, all the flow measurements of 4 patients were repeated by the same observer (28 flow measurements in all), and all the measurements of 4 different patients were repeated by a different observer (additional 28 flow measurements). The percent difference between the 2 measurements was calculated for each of the 7 measured flows. In addition, the mean and SD of the difference between the 2 observations of the same collateral flow estimator were calculated. Reliability coefficients were calculated using single-measure intraclass correlation for the intraobserver and interobserver agreement for \( Q_{\text{coll-pulm}} \) and \( Q_{\text{coll-syst}} \).

Venous volumes, including the absolute and indexed end-diastolic volume, end-systolic volume, and ejection fraction, were also obtained from the clinical report. Estimated indexed collateral flow was compared with indexed end-diastolic venous volumes by linear regression.

There were 7 patients who had cardiac catheterizations with aortic angiography adequate for collateral grading by angiography as described by Spicer et al.4 Collateral grading by angiography was compared with the quantification of collateral flow by the Spearman rank correlation coefficient for nonparametric parameters.

The 2 methods of calculating collateral flow were compared using linear regression, Bland-Altman analysis, intraclass correlation, and paired Student t test. Control and SCPC parameters were compared using unpaired Student t tests. The authors had full access to the data and take responsibility for its integrity. All authors have read and agree to the manuscript as written. The study was approved by the institutional review board.

### Results

Table 1 summarizes the 17 patients and the relevant data. Estimated collateral flow ranged from 0.2 to 1.4 L/min for \( Q_{\text{coll-syst}} \) and 0.2 to 1.6 L/min for \( Q_{\text{coll-pulm}} \), which corresponds to an indexed collateral flow of 0.5 to 2.9 L/min/m², with a mean of 1.8 L/min/m². Collateral flow accounted for 11% to 54% of the aortic flow, with a mean of 37%. The estimated percent contribution of collateral flow to total pulmonary blood flow ranged from 19% to 77%, with a mean of 54%. The measured pulmonary-to-systemic flow ratio (Qp/Qs), when corrected for collateral blood flow, was on average 1.1, with a range of 0.6 to 1.7.

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age, y</th>
<th>BSA, m²</th>
<th>Disease</th>
<th>SCPC Type</th>
<th>DV</th>
<th>( Q_{a} )</th>
<th>( Q_{coll-syst} )</th>
<th>( Q_{coll-pulm} )</th>
<th>( Q_{coll}/(Q_{RPV} + Q_{LPV}) )</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>1.44</td>
<td>0.43</td>
<td>HLHS</td>
<td>Hemi</td>
<td>R</td>
<td>1.3</td>
<td>0.56</td>
<td>0.47</td>
<td>0.69</td>
</tr>
<tr>
<td>2</td>
<td>2.61</td>
<td>0.50</td>
<td>DILV, hypoplastic RV</td>
<td>BDG</td>
<td>L</td>
<td>2.8</td>
<td>0.90</td>
<td>0.80</td>
<td>0.47</td>
</tr>
<tr>
<td>3</td>
<td>3.18</td>
<td>0.54</td>
<td>HLHS</td>
<td>BDG</td>
<td>B</td>
<td>2.7</td>
<td>1.34</td>
<td>1.07</td>
<td>0.68</td>
</tr>
<tr>
<td>4</td>
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<td>0.54</td>
<td>HLHS</td>
<td>Hemi</td>
<td>R</td>
<td>3.1</td>
<td>1.40</td>
<td>1.00</td>
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</tr>
<tr>
<td>5</td>
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<td>0.41</td>
<td>HLHS</td>
<td>BDG</td>
<td>R</td>
<td>2.1</td>
<td>0.41</td>
<td>0.51</td>
<td>0.35</td>
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<tr>
<td>6</td>
<td>3.45</td>
<td>0.64</td>
<td>TA</td>
<td>Hemi</td>
<td>L</td>
<td>3.1</td>
<td>1.39</td>
<td>1.47</td>
<td>0.65</td>
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<td>7</td>
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<td>0.50</td>
<td>TA, DORV, Dextrocardia</td>
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<td>0.57</td>
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<td>8</td>
<td>1.38</td>
<td>0.44</td>
<td>Heterotaxy, Unbalanced canal</td>
<td>BBDG</td>
<td>R</td>
<td>2.4</td>
<td>0.68</td>
<td>0.65</td>
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<tr>
<td>9</td>
<td>1.18</td>
<td>0.40</td>
<td>HLHS</td>
<td>BDG</td>
<td>R</td>
<td>1.9</td>
<td>0.23</td>
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<td>0.19</td>
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<td>10</td>
<td>1.62</td>
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<td>Heterotaxy (L,L)</td>
<td>BDG</td>
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<td>2.6</td>
<td>0.30</td>
<td>0.8</td>
<td>0.25</td>
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<tr>
<td>11</td>
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<td>TA, PA</td>
<td>Hemi</td>
<td>L</td>
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<td>1.11</td>
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<td>0.69</td>
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<td>BDG</td>
<td>L</td>
<td>2.1</td>
<td>0.90</td>
<td>0.74</td>
<td>0.53</td>
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<td>13</td>
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<td>HLHS</td>
<td>BDG</td>
<td>R</td>
<td>2.8</td>
<td>0.90</td>
<td>0.90</td>
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<td>14</td>
<td>2.21</td>
<td>0.56</td>
<td>TA, PA, RV aorta</td>
<td>BDG</td>
<td>L</td>
<td>2.4</td>
<td>1.23</td>
<td>1.55</td>
<td>0.59</td>
</tr>
<tr>
<td>15</td>
<td>0.70</td>
<td>0.33</td>
<td>Heterotaxy, MA, DORV</td>
<td>BBDBG</td>
<td>R</td>
<td>2.0</td>
<td>1.05</td>
<td>0.86</td>
<td>0.77</td>
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<td>16</td>
<td>1.74</td>
<td>0.52</td>
<td>HLHS</td>
<td>BDG</td>
<td>R</td>
<td>2.6</td>
<td>1.09</td>
<td>1.38</td>
<td>0.68</td>
</tr>
<tr>
<td>17</td>
<td>1.45</td>
<td>0.50</td>
<td>HLHS</td>
<td>BDG</td>
<td>R</td>
<td>2.3</td>
<td>0.77</td>
<td>0.72</td>
<td>0.47</td>
</tr>
</tbody>
</table>

Mean ± SD: 2.09 ± 0.82, 0.50 ± 0.08

BSA indicates body surface area; DV, dominant ventricle; HLHS, hypoplastic left heart syndrome; DILV, double-inlet left ventricle; TA, tricuspid atresia; DORV, double-outlet right ventricle; PA, pulmonary atresia; MA, mitral atresia; Hemi, hemi-Fontan; BDG, bidirectional Glenn shunt; BBDBG, bilateral bidirectional Glenn shunt; R, right-dominant ventricle; L, left-dominant ventricle; B, 2 good-sized ventricles.

### Table 1. Summary of Patients and Collateral Flow

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age, y</th>
<th>BSA, m²</th>
<th>Disease</th>
<th>SCPC Type</th>
<th>DV</th>
<th>( Q_{a} )</th>
<th>( Q_{coll-syst} )</th>
<th>( Q_{coll-pulm} )</th>
<th>( Q_{coll}/(Q_{RPV} + Q_{LPV}) )</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td>2.41 ± 0.47</td>
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<tr>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.91 ± 0.36</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.90 ± 0.36</td>
</tr>
<tr>
<td>4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.54 ± 0.16</td>
</tr>
</tbody>
</table>

Control and SCPC parameters were compared using unpaired Student t tests. The authors had full access to the data and take responsibility for its integrity. All authors have read and agree to the manuscript as written. The study was approved by the institutional review board.
There was a strong correlation between the 2 methods of estimating collateral flow (Figure 2), with a correlation coefficient of 0.80 and a regression slope of 0.78. The intraclass correlation coefficient was 0.81 \((P < 0.001)\), indicating good agreement. Looking at the Bland-Altman analysis in Figure 2, there was little bias, with a mean difference between the 2 methods of only 0.01 L/min, which was not statistically significant \((P = 0.40)\). The SD of the difference between the 2 methods was 0.23 L/min, for a 2-SD range of 0.45 to 0.47 L/min.

Venous return to the heart demonstrated excellent correlation with the measured aortic flow (Figure 2), with a correlation coefficient of 0.88 and a regression slope of 1.06 (Figure 3). It also showed little bias, with a mean difference between the 2 measures of only 0.01 L/min and a SD of 0.27 L/min (Figure 3), for a 2-SD range of −0.53 to 0.55.

**Comparison With Control Subjects**

Table 2 summarizes the comparisons between the control group and the study population. In the control group, collateral flow was on average 0.2 L/min/m², which is an order of magnitude less than the SCPC group and statistically significant. The measured collateral flow average, 5% of both aortic and pulmonary blood flow, was significantly less than for the SCPC group. There was also excellent correlation between the pulmonary venous return and the aortic flow (Figure 3). Because the control group is retrospective, there is a significant difference between the age and body surface area, making it less than ideal. However, there is significant overlap in the ages, and collateral flow was not a function of age in this group, making this less of a concern.

### Table 2. Summary of Patients With SCPC Versus Control Subjects

<table>
<thead>
<tr>
<th></th>
<th>SCPC Patients ((n=17))</th>
<th>Control Patients ((n=13))</th>
<th>(P) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>2.1 ± 0.82</td>
<td>11.4 ± 8.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BSA, m²</td>
<td>0.50 ± 0.08</td>
<td>1.1 ± 0.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>(Q_{coll}), L/min/m²</td>
<td>1.8 ± 0.61</td>
<td>0.18 ± 0.18</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>100×(Q_{coll}/Q_{Ao}), %</td>
<td>37% ± 11%</td>
<td>5% ± 5%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>100×(Q_{coll}/Q_{PV}), %</td>
<td>54% ± 16%</td>
<td>5% ± 4%</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Control subjects were unoperated patients with arch anomalies and no intracardiac shunts and postoperative patients with 2-ventricle repair with no residual intracardiac shunts. BSA indicates body surface area.

Values listed are mean ± SD.
Relationship Between Collateral Flow and Ventricular Volumes

When using the entire cohort, there was no significant correlation between collateral flow and ventricular volumes. However, 2 patients, 1 with severe tricuspid regurgitation and 1 with moderate RV dysfunction, had very dilated ventricles for other reasons. When these 2 were excluded, indexed end-diastolic volume correlated significantly with indexed collateral flow (Figure 4) ($r = 0.59$, $P = 0.02$).

There was no relationship between collateral flow and SCPC type, underlying type of heart disease, or ventricular morphology. Collateral flow was slightly greater to the left lung on average (56% versus 44%), but the difference was not significant ($P = 0.25$). Indexed collateral flow increased with time from the SCPC operation: The correlation was weak but significant (Figure 5).

Angiographic grading of collateral flow did not correlate with collateral flow indexed to body surface area, aortic flow, or pulmonary blood flow.

Interobserver and Intraobserver Variabilities

Intraobserver variability for all the flow measurements combined was 4%, with an SD of 4%. The interobserver variability was 7%, with an SD of 7%. Both intraobserver and interobserver differences between both measures of collateral flow were $<0.1$ L/min on average, with an SD of 0.06 L/min.

The intraclass correlation coefficient for intraobserver agreement was 0.986 for $Q_{coll-syst}$ ($P = 0.0001$) and 0.960 for $Q_{coll-pulm}$ ($P = 0.001$). The coefficient of interobserver agreement was 0.836 for $Q_{coll-syst}$ ($P = 0.05$) and 0.986 for $Q_{coll-pulm}$ ($P = 0.001$).

Discussion

We present a noninvasive method of quantifying SPCF in patients with cavopulmonary connections that provides 2 independent measures of collateral flow. There was excellent agreement between the 2 methods studied, providing internal validation for this noninvasive estimator.

In the 17 patients studied, the collateral flow averaged 54% of the total pulmonary blood flow and 37% of the cardiac output. In addition, in patients without other significant reasons for ventricular dilation, indexed collateral flow correlated with indexed end-diastolic volume. This suggests that collateral flow is a significant hemodynamic burden in this population and deserves further study. It may also explain previous findings by Fogel et al., which, despite the dogma that the superior cavopulmonary connection results in significant volume unloading, measured ventricular volumes were not significantly different after the cavopulmonary connection. The measured $Q_{p}/Q_{s}$ mean of 1.1 in our study is in significant contrast to data obtained from oximetric evaluation at cardiac catheterization in which typical measurements of $Q_{p}/Q_{s}$ range from 0.4 to 0.6.13,14 This suggests that cardiac catheterization significantly underestimates the actual pulmonary blood flow. This is not unexpected because the collateral connections enter the pulmonary tree distally, precluding accurate measurement of mixed pulmonary arterial saturation. Although indexed collateral flow demonstrated no correlation with angiographic grading of collateral flow, the limited number of patients with angiography prevents drawing conclusions.

Although the current population studied has superior cavopulmonary connections, the methodology should be valid for patients with total cavopulmonary connections as well. The data suggest that collateral flow is in general quite significant in patients with superior cavopulmonary connections. Our previously reported study of Fontan flows suggest that collateral flow is less but still significant in Fontan physiology.11 Further studies are required to quantify collateral flow in the Fontan population using the techniques described in this study.

Previous Studies

Few studies have focused on quantifying SPCF. In 2001, Bradley et al.5 measured collateral flow directly in 32 patients while on bypass and about to undergo a Fontan operation. Collateral flow was present in all patients and ranged from 9% to 49% of pump flow. These values are similar to the
range of 11% to 53% seen in our study. Systemic and pulmonary vascular resistances have significant effects on collateral flow. Given that the flows were measured on bypass, the measured collateral flows may not reflect the baseline physiological state. Additionally, this method has the obvious limitation of being invasive and thus not a practical means of tracking collateral flow over time.

Another notable study was performed by Inuzuka et al and quantified collateral flow in 10 patients using a combination of cardiac catheterization oximetry data and perfusion scintillography. Scintillography data from a lower extremity injection quantified the ratio of collateral-to-systemic flow. Oximetry data obtained from catheterization were then used to calculate absolute collateral flow and total pulmonary blood flow. They obtained a mean indexed collateral flow of 1.75 L/min/m², similar to our 1.8 L/min/m². The estimated mean systemic flow for the patients was 3.4 L/min/m² and the mean pulmonary flow was 3.0 L/min/m². This corresponds to collateral flow being on average 34% of cardiac output and 58% of pulmonary blood flow, which agrees very well with our current study. The obvious disadvantage to this method is that it requires an invasive procedure with ionizing radiation to obtain the data. It is also fairly cumbersome and assumes complete mixing in the IVC, which is often not a valid assumption. Finally, it involves 2 separate procedures that are performed under different conditions and thus may limit the accuracy of individual measurements.

Finally, Grosse-Wortmann et al recently described a method similar to the one presented here to quantify collateral flow in patients with both bidirectional cavopulmonary connections and Fontan. An important difference was the use of descending aortic flow as a surrogate to IVC flow. They obtained a mean indexed collateral flow of 0.73, compared with 0.81 in our study. These differences probably are accounted for by 2 important methodological differences: (1) their use of the descending aortic flow as a surrogate for IVC flow and (2) the measurement of aortic flow distal to the aortopulmonary anastomosis in patients with aortic reconstructions. We have demonstrated that the IVC flow can be reliably measured directly, and it will not be subject to inaccuracies caused by decompressing vessels or collateral vessels arising below the level of descending aortic measurement. The measurement of aortic flow distal to the aortopulmonary anastomosis of a reconstructed arch is not desirable secondary to the potential for disturbed flow from flow collisions between the native and neoaortic flows and swirling in the often dilated reconstructed arch. We therefore propose that the proper way to measure aortic flow in patients with reconstructed arches is to measure the flows just above the level of the semilunar valves. When there are 2 semilunar valves that contribute to total aortic flow, they should be measured separately and summed to provide the total aortic flow. The study also reported difficulty in accurately measuring pulmonary vein flows in patients with complicated pulmonary vein anatomy and excluded 7 of 36 patients from their analysis for this reason. We did not encounter this same difficulty despite 3 patients with heterotaxy and 2 with abnormal pulmonary venous connections. No patients in our study undergoing MRI during the study period were excluded secondary to abnormalities of pulmonary veins. Finally, the prior study reported increased collateral flow to the side of a prior Blalock-Taussig shunt, a finding that was not reproduced in our study. Contrary to the reported finding by Grosse-Wortmann et al of a trend between younger age and higher collateral flow, we found the opposite, with a significant correlation between elapsed time from SCPC surgery and indexed collateral flow.

Limitations
Although we were unable to find any difference in collateral flow based on differences in anatomy or surgical type, the study is underpowered to find such differences. The described method could not be compared with a gold standard, as is ideal when testing a new measurement, because no gold standard exists. However, the fact that this method incorporates 2 independent measures of collateral flow largely overcomes this limitation and is a significant advantage both in validating the technique and applying it clinically. The control population was not age-matched to the study group, which is a limiting factor. However, the age range did overlap significantly (youngest control, 1.6 years, with 3 under 3 years of age), and there was no relationship between age and collateral flow in the control population, partially alleviating this concern. Because there was angiographic data on only a limited number of patients, we are unable to draw conclusions regarding the validity of angiographic grading of collaterals.

Conclusions
We have presented a series of patients who underwent noninvasive quantification of SPCF using cardiac magnetic resonance phase-contrast velocity mapping. The excellent agreement between the 2 methods provides compelling evidence that this is an accurate method for quantifying collateral flow. Furthermore, this collateral flow represents a substantial hemodynamic burden to patients with SCPC (on average, 37% of the cardiac output and 54% of the pulmonary blood flow).

We present a methodology similar to that recently reported by Grosse-Wortmann et al, with important differences that result in improved accuracy and lower bias. Notably, our measurements do not appear to be subject to the occasional gross underestimation of collateral flow by the systemic collateral estimator noted in the other study. We propose that the IVC flow should be measured directly rather than indirectly by descending aorta flow and that the aortic flow should be measured just above the semilunar valves in patients with Norwood-type arch reconstructions, rather than distal to the aortic-to-pulmonary anastomosis. When there is
significant antegrade flow across 2 semilunar valves, they should be measured separately and summed together.

This study cannot address the important questions of why these collaterals develop, which patients are most susceptible to collateral formation, and perhaps most importantly what the long-term impact of this significant collateral flow is. However, it does provide a noninvasive tool that should allow us to answer these important questions in future investigations. By monitoring these patients prospectively, we may be able to determine which patients will have development of collaterals, what the impact of these collaterals is on both short-term and long-term Fontan outcomes, and what happens to the collateral flow after Fontan completion.

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Disclosures

None.

References


CLINICAL PERSPECTIVE

This research presents and validates a method of quantifying the systemic-to-pulmonary collateral flow in the single-ventricle population with superior cavopulmonary anastomoses. It used cardiac MRI through-plane velocity mapping to calculate 2 independent estimators of collateral flow—the difference between the aortic and caval flow and the difference in pulmonary vein and pulmonary artery flow. We found good agreement between the 2 estimators. In addition, this collateral flow was on average more than half of the total pulmonary blood flow and more than one third of the cardiac output (aortic flow), and ventricular end-diastolic volume correlated with collateral flow. This suggests that collateral flow can be a significant hemodynamic burden in this population. The proposed methodology could identify patients who may benefit from collateral embolization and determine whether embolization significantly affects total collateral flow and cardiac output. The implications of these findings for clinical outcomes of single-ventricle patients require further studies.
Noninvasive Quantification of Systemic-to-Pulmonary Collateral Flow: A Major Source of Inefficiency in Patients With Superior Cavopulmonary Connections
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