Historically, cardiac catheterization has served as an important diagnostic and therapeutic tool in patients undergoing staged palliation of single ventricle heart disease. For those with a superior cavopulmonary connection (SCPC), catheterization may be indicated either to assess candidacy for Fontan completion or to investigate abnormal clinical findings, such as hypoxemia or low cardiac output. Management decisions are then guided by the quantitative indicators of SCPC circuit function that result most notably, pulmonary (\(Q_p\)) and systemic (\(Q_s\)) blood flow estimates, \(Q_p:Q_s\) ratio, and pulmonary vascular resistance.

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In the presence of intracardiac mixing, catheter-based estimates of flow and resistance are derived from the Fick principle. Admittedly, the SCPC is not an ideal Fick system for several reasons. Systemic-to-pulmonary arterial collateral flows are nearly ubiquitous in these patients\(^1\)\(^-\)\(^4\) and provide an additional source of pulmonary blood flow that cannot be reliably accounted for in conventional oximetry calculations. In addition, the systemic venous circulation is completely separated into superior vena cava (SVC) and inferior vena cava (IVC) circuits, meaning that there is no true mixed venous blood available for sampling.

Oximetry is widely used, despite these drawbacks, primarily because of the historical lack of an alternative method to measure flow in complex anatomic circuits. Without a gold standard modality capable of accounting for systemic-to-pulmonary arterial collateral flow, it has been impossible to describe the degree of inaccuracy in the Fick calculations and therefore to understand whether the error is clinically relevant. Fortunately, recent developments in phase-contrast cardiac MRI (CMR) techniques have provided new insight by

Background—Cardiac catheterization is routinely used as a diagnostic tool in single ventricle patients with superior cavopulmonary connection. This physiology presents inherent challenges in applying the Fick principle to estimate flow. We sought to quantitatively define the error in oximetry-derived flow parameters using phase-contrast cardiac MRI (CMR) as a reference.

Methods and Results—Thirty patients with superior cavopulmonary connection who underwent combined CMR and catheterization between July 2008 and June 2012 were retrospectively analyzed. Estimates of flow and resistance calculated using the Fick equation were compared with CMR measurements. Oximetry underestimated CMR-measured pulmonary blood flow (\(Q_p\)) by an average of 1.1 L/min per m\(^2\) or 32% of the CMR value (\(P<0.0001\)). Oximetry overestimated systemic blood flow (\(Q_s\)) by an average of 0.5 L/min per m\(^2\) or 15% of the CMR value (\(P=0.009\)). There was no correlation between the \(Q_p:Q_s\) ratio derived by Fick and that measured by CMR (\(\rho=0.01\)). The error in the Fick \(Q_p\) correlated moderately with the measured systemic-to-pulmonary arterial collateral flow (\(r=0.39\)). The median total oxygen consumption calculated using combined CMR and oximetry data was 173 mL/min per m\(^2\), higher than the assumed values used to calculate flows by the Fick equation. The upper body circulation received on average 51% of systemic blood flow while conducting only 39% of total body metabolism.

Conclusions—Fick-derived estimates of flow are inherently unreliable in patients with superior cavopulmonary connections. Integrating flows measured by CMR and pressures measured by catheter will provide the best characterization of superior cavopulmonary connection physiology. (Circ Cardiovasc Imaging. 2013;6:943-949.)

Key Words: bidirectional cavopulmonary shunt ■ catheterization ■ collateral circulation ■ congenital heart defects ■ magnetic resonance imaging
allowing us to quantify collateral burden in a reliable fashion. The accuracy of CMR for simple flow assessment has been previously validated in both in vivo and in vitro studies, including in patients with congenital heart disease.

The primary objective of this study was to compare Fick-derived estimates of pulmonary and systemic blood flow with direct measurements obtained using CMR. We hypothesized that \( Q_s \) derived by Fick would underestimate the measured CMR values because of the inability to account for systemic-to-pulmonary arterial collateral flow. In addition, we suspected that oximetry-based calculations of \( Q_s \) would be inaccurate compared with CMR measurements because of the lack of a true mixed venous saturation.

Methods

Patients

All patients with SCPC who had CMR quantification of systemic-to-pulmonary arterial collateral flow between July 2008 and June 2012 were reviewed. The subset that underwent combined CMR and catheterization (XMR) under the same general anesthetic was eligible for inclusion in this study. SCPC was defined as any operation that involved complete rerouting of the superior vena cava flow to the pulmonary arteries (PAs), including bidirectional Glenn anastomosis, bilateral bidirectional Glenn, or hemi-Fontan procedure (superior cavopulmonary anastomosis incorporating a portion of the right atrium). Patients with residual antegrade pulmonary blood flow and those with interrupted IVC (Kawashima-type procedures) were excluded. Patients with systemic-to-pulmonary venous collaterals, visible by MRI, or those with pulmonary vein (PV) saturations <95% were also excluded. A retrospective review of the medical record was conducted to extract the demographic and clinical variables of interest. The study was approved by the institutional review board.

Cardiac MRI

All patients underwent CMR immediately before catheterization. It is our current practice to perform both procedures with the patient mechanically ventilated on room air, minimizing variability in physiologic parameters, such as blood pressure and heart rate to the greatest extent possible. A minority (6 of 30) of patients whose studies were performed during our early experience did receive supplemental oxygen during the CMR portion.

Baseline CMR images were acquired on a 1.5-T MRI Scanner (Siemens Avanto). Localization of velocity mapping image planes was performed using multilayer reformating of a static balanced steady-state free precession axial stack gated to late diastole. Retrospectively gated, through-plane, phase-contrast cines (PC-MRI) were performed in the PAs, PVs, vena cavae (SVC and IVC), and aorta. Right and left PA measurements were obtained individually, and the right PA measurement was performed proximal to the origin of the right upper lobe PA. In patients with proximal right PA branching, the right upper lobe PA was measured separately. Typical parameters for the phase-encoded velocity mapping sequence for a typical R-R interval of 600 ms include a 220×165 mm field of view, 192×144 matrix, 3- to 4-mm slice thickness, echo time (TE) of 2.82 ms, bandwidth of 501 Hz/pixel, repetition time (TR) of 34 ms, 25° flip angle, 14 measured phases, 24 calculated phases, 3 segments, and 3 averages. Typical encoding velocities were 150 cm/s for the aorta; 60 cm/s for the SVC, IVC, right PA, and left PA; and 80 cm/s for PVs. Acquisition time generally ranged from 55 to 110 seconds per velocity map and 12 to 18 minutes overall. These parameters were chosen on the basis of clinical experience and unpublished flow phantom experiments, which suggest that spatial resolution is more critical than temporal resolution in resolving flow. This is particularly true with the low-frequency venous flows that predominate the protocol.

PC-MRI measurements are highly dependent on adequate spatial resolution, which, in general, requires ≥16 voxels in the contoured lumen to achieve <10% error. In 23, the PV measurements were measured at a single confluence, and in 37, the upper and lower veins were measured separately. The minimum number of voxels used on an individual upper or lower PV was, on average, 30 (range, 14–89). On average, the total number of voxels used to measure the PV flow on one side was 59 (range, 19–139). Only a single vessel acquisition had fewer voxels (14) than 16. There was 1 patient in this cohort for whom we had to measure a separate right upper lobe PA branch, in which the number of voxels contoured was 16.

\[ Q_s = \text{total PV flow,} \]
\[ Q_s' = \text{the measured aortic outflow.} \]

Cardiac Catheterization

All oximetry data used in Fick calculations were obtained with the patient mechanically ventilated on room air. If interventions were performed during the catheterization, only the preintervention hemodynamic data were analyzed. Saturation measurements were abstracted from the chart and used to calculate systemic and pulmonary blood flow according to the conventional Fick equation. The lowest SVC or PA saturation was used as the mixed venous saturation. Most patients had multiple PV saturations measured, which were averaged to produce the final value. In 2 patients with no direct measurement, the PV saturation of 96% assumed at the time of the procedure was used in our calculations.

Total body oxygen consumption (\( VO_2 \)) was assumed for each patient according to published weight-based formulae (patient age <3 years) or age-based tables (>3 years). Catheterization-derived PVR was calculated using the Fick \( Q_s \) and the recorded pulmonary arterial and PV atrial pressures. In the case of unequal left and right transpulmonary gradients, the higher gradient was used. It should be noted though that no patient in this cohort had more than a 1 mm Hg discrepancy between right and left transpulmonary gradients. An MRI-corrected total PVR was also calculated using left and right PV flows and their respective transpulmonary gradients according to the formula:

\[
\frac{1}{R_T} = \frac{1}{R_R} + \frac{1}{R_L},
\]

where \( R_T = \text{total PVR}, R_R = \text{right lung PVR}, \) and \( R_L = \text{left lung PVR}. \)

Metabolic Calculations

Although it is common practice to assume a total body oxygen consumption for the purposes of the Fick equation, the actual \( VO_2 \) for each patient can be calculated using combined MRI and catheterization data. Upper body oxygen consumption can be defined as the difference between oxygen delivery to the SVC distribution and SVC oxygen return:

\[
(1) \quad VO_2(\text{SVC}) = h \cdot Q_{\text{SVC}} \cdot (S_{S_A} - S_{SVC}).
\]

where \( h = 13.6 \times \text{hgb (g/dL)}, \) and \( S_{S_A} \) and \( S_{SVC} \) are systemic arterial and SVC saturations, respectively. \( Q_{\text{SVC}} \) is defined as the SVC flow measured just above the cavopulmonary anastomosis. In the case of bilateral SVCs, Equation 1 is calculated separately for each SVC and the
results summed to produce the total upper body VO$_2$. Similarly, the lower body oxygen consumption can be defined as the difference between oxygen delivery and return in the IVC distribution. Because we do not typically measure an IVC saturation, the IVC oxygen return must be calculated indirectly from the conservation of mass around the heart. In patients with SCPC, total oxygen enters the heart via the IVC and PVs (excluding a small amount of coronary sinus flow). This must be equal to oxygen leaving the heart through the aortic valve. IVC oxygen return is therefore the difference between aortic oxygen delivery and PV oxygen return. The following can then be extrapolated:

$$\text{VO}_2_{	ext{IVC}} = h(S_{\text{IVC}}Q_{\text{IVC}} - (hQ_{\text{ao}}S_{\text{ao}} - hQ_{\text{pv}}S_{\text{pv}})) = h(S_{\text{IVC}}Q_{\text{IVC}} + Q_{\text{pv}}S_{\text{pv}} - Q_{\text{ao}}S_{\text{ao}})$$

where $S_{\text{IVC}}$ and $S_{\text{pv}}$ are IVC and PV saturations, respectively, and $Q_{\text{ao}}$ is the total aortic outflow measured just above the aortic or neoaortic valves. The total body oxygen consumption is then defined as the sum of Equations 1 and 2.

### Statistical Considerations

Baseline demographic and clinical variables were summarized using standard descriptive statistics. Normally distributed variables were reported as mean±SD, and skewed variables were reported as median with range. The concordance correlation coefficient ($\rho_c$), as described by Lin,$^3$ was used to quantify agreement between CMR and catheterization measurements of flow and to evaluate intraobserver and interobserver reliability. Means were compared using the paired Student’s t test for normal variables or the Wilcoxon signed-rank test when variables had significant positive skew. Comparisons of pulmonary vascular resistance were performed after natural logarithmic transformation, which resulted in normally distributed data. All $P$ values reported are 2-sided. Statistical significance was established a priori at $P<0.05$. Analyses were conducted with STATA, v. 12.0 (StataCorp; College Station, TX).

### Results

Thirty patients met inclusion criteria and were included in the analyses. Baseline characteristics are outlined in Table 1. Patients were between 11 months and 7 years of age at XMR (median, 2.6 years), with a median of 23 months elapsed from the time of SCPC. The predominant anatomic diagnoses were hypoplastic left heart syndrome and unbalanced common atroventricular canal defect, and many patients (77%) had a systemic right ventricle. Approximately half of the subjects had bidirectional Glenn anastomoses, 20% had hemi-Fontan, and 33% had bilateral cavopulmonary connections. One third of patients carried a diagnosis of heterotaxy syndrome.

### Table 1. Baseline Characteristics of Included Patients (n=30)

<table>
<thead>
<tr>
<th>Age at XMR</th>
<th>2.6 y (11 mo–7 y)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BSA, m$^2$</td>
<td>0.56±0.12</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>16 (53%)</td>
</tr>
<tr>
<td>Female</td>
<td>14 (47%)</td>
</tr>
<tr>
<td>Age at SCPC</td>
<td>6.4 mo (3.6 mo–2 y)</td>
</tr>
<tr>
<td>Time from SCPC to XMR</td>
<td>23 mo (3 mo–6.8 y)</td>
</tr>
<tr>
<td>Type of SCPC</td>
<td></td>
</tr>
<tr>
<td>Bidirectional Glenn</td>
<td>14 (47%)</td>
</tr>
<tr>
<td>Hemi-Fontan</td>
<td>6 (20%)</td>
</tr>
<tr>
<td>Bilateral SCPC</td>
<td>10 (33%)</td>
</tr>
<tr>
<td>Systemic ventricle</td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>23 (77%)</td>
</tr>
<tr>
<td>Left</td>
<td>5 (17%)</td>
</tr>
<tr>
<td>Both</td>
<td>2 (6%)</td>
</tr>
</tbody>
</table>

Data are reported as mean±SD, median (range), or count (% of total). BSA indicates body surface area; SCPC, superior cavopulmonary connection; and XMR, combined catheterization and cardiac MRI.

### Table 2. Catheterization Parameters (n=30)

<table>
<thead>
<tr>
<th></th>
<th>84±5%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic arterial saturation</td>
<td></td>
</tr>
<tr>
<td>Mixed venous saturation</td>
<td>63±8%</td>
</tr>
<tr>
<td>SVC pressure, mmHg</td>
<td>12.7±2.8</td>
</tr>
<tr>
<td>PA pressure, mmHg</td>
<td>12.1±2.6</td>
</tr>
<tr>
<td>PV atrial pressure, mmHg</td>
<td>7.4±2.6</td>
</tr>
<tr>
<td>Ventricular EDP, mmHg</td>
<td>7.5±2.3</td>
</tr>
<tr>
<td>Hemoglobin, g/dL</td>
<td>15.5 (12.7–21.1)</td>
</tr>
<tr>
<td>$\text{aVO}_2$, mL/min per m$^2$</td>
<td>160 (130–190)</td>
</tr>
</tbody>
</table>

Data are reported as mean±SD, median (range), or count (% of total). $\text{aVO}_2$ indicates assumed oxygen consumption; EDP, end-diastolic pressure; PA, pulmonary artery; PV, pulmonary vein; and SVC, superior vena cava.
Table 3. Flow and Resistance Calculations—Oximetry Versus CMR

<table>
<thead>
<tr>
<th></th>
<th>Oximetry</th>
<th>MRI</th>
<th><em>p</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>$Q_s$, L/min per m²</td>
<td>2.3±0.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$Q_r$, L/min per m²</td>
<td>3.4±0.8</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>$Q_p$, L/min per m²</td>
<td>1.8±0.7</td>
<td>0.0002</td>
<td></td>
</tr>
<tr>
<td>$Q_s$ vs $Q_p$, L/min per m²</td>
<td>3.8±1.0</td>
<td>3.3±0.8</td>
<td>0.009</td>
</tr>
<tr>
<td>$Q_s/Q_p$</td>
<td>0.6 (0.4–0.8)</td>
<td>1.1 (0.5–1.6)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>PVR (iWU)</td>
<td>2.0 (0.8–5.6)</td>
<td>1.1 (0.6–3.7)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Data are reported as mean±SD for normal variables and median (range) for skewed variables. CMR indicates cardiac MRI; $Q_s$, pulmonary blood flow; $Q_r$, systemic blood flow; $Q_p$, pulmonary artery flow; $Q_v$, pulmonary vein flow; and iWU, indexed Wood units.

*Comparisons made by paired Student *t* or Wilcoxon sign-rank tests as appropriate.

When the $Q_s/Q_p$ ratio was calculated by oximetry, all values fell between 0.4 and 0.8. Furthermore, there was no correlation between catheterization $Q_s/Q_p$ and that measured by CMR (Figure 3A). The Fick-based pulmonary vascular resistance was higher than the CMR-corrected value in all but 1 patient, with a mean difference of 0.6 indexed Wood units ($P<0.0001$). Although there was a reasonable linear correlation between the 2 measurements of PVR (Figure 3B), the agreement was moderate at best ($\rho=0.66$) because of the systematic error.

Internal consistency of the PC-MRI measurements was excellent when comparing total venous return to the heart versus aortic outflow. The mean difference between these 2 measures was $0.2±0.4$ L/min per m², with excellent agreement ($\rho=0.87$) and no systematic bias.

Discussion

The physiology of the SCPC presents inherent challenges in applying the Fick principle to estimate flow. To better define the clinical utility of oximetry in this population, we describe the nature and degree of its error using phase-contrast CMR as a reference. In this cohort of 30 patients, we found that oximetry inaccurately estimated systemic and pulmonary blood flow, $Q_s$ vs $Q_p$, ratio, and pulmonary vascular resistance.

In these subjects, the Fick-derived $Q_s$ always underestimated CMR PV flow, with a mean error of 1.1 L/min per m² (range, 0.1–2.5 L/min per m²). Although that average discrepancy is significant in and of itself, also important clinically is the wide range of the error across the cohort. For 7 of 30 patients, the Fick $Q_p$ was reasonably accurate, differing from the CMR-measured pulmonary blood flow by <20%. In 5 others, the error comprised >50% of the CMR value. This inconsistency can be explained physiologically by the varying contribution of systemic-to-pulmonary arterial collaterals to effective pulmonary blood flow. In patients with high absolute collateral burden or relatively higher systemic saturations, the ineffective collateral flow is greater, and the Fick estimate of $Q_p$ should become more inaccurate. Our data do support this because the error in catheterization $Q_p$ was found to correlate positively with absolute collateral flow. The substantial degree of scatter in that correlation (and the failure of the Fick $Q_p$ to agree with CMR pulmonary arterial flow) can then be at least partially attributed to varying systemic saturation.

Given the nature of the error in Fick $Q_p$, it was not surprising that oximetry-derived pulmonary vascular resistance overestimated the CMR-corrected values. There was a clear proportional error, with the discrepancy between the 2 modalities being most notable in patients with higher PVR. This could become clinically relevant if a patient was excluded from Fontan completion based on an erroneously high catheterization-derived resistance. However, the likelihood of that scenario could not be determined from this cohort because of a lack of subjects with markedly elevated PVR.

Somewhat less intuitive than the error in $Q_p$ was the observation that the Fick-derived $Q_s$ typically overestimated the CMR-measured total caval flow. Systemic-to-pulmonary arterial collaterals cannot be implicated in this case because they do not participate in systemic metabolism. The most likely sources of inaccuracy in calculated $Q_s$ would therefore be an erroneously high mixed venous saturation or a systematic overestimate of VO$_2$.

The conventional substitution of the SVC saturation for a true mixed venous sample is physiologically valid only if the SVC and IVC saturations are equal. In the typical biventricular circulation, the caval saturations are unequal, and their relationship may be influenced by factors such as patient age and size, cardiac output, and sedation strategy. It is not clear whether the same relationships would be observed after SCPC, although several groups have attempted to model this in a theoretical sense. Diller et al hypothesized that the SVC and IVC saturations would be equal in the subset of patients with SCPC whose upper body conducted >50% of the total body metabolism. Figure 4 plots SVC flow fraction...
against upper body metabolic fraction for our cohort. SVC flow increased linearly with metabolic fraction across the entire range, but flow almost always exceeded metabolic demand. Physiologically, this demands that the SVC saturation be higher than the IVC saturation, which we suspect contributes to the observed error in the Fick-derived $Q_s$.

Inaccuracies in assumed oxygen consumption may also contribute to error in Fick-derived flow parameters. In these patients, the assumed VO2 underestimated the calculated value by an average of 12%, which should translate into an underestimate of both $Q_p$ and $Q_s$ by Fick. Because Fick-derived $Q_p$ overestimated the CMR value, the error in VO2 cannot be directly implicated. It is likely that the discrepancy between Fick- and CMR-derived $Q_p$ would be even greater if the precise VO2 were known for each patient. The underestimate of VO2 almost certainly accounts for some of the error in the Fick $Q_p$. However, because the magnitude of the VO2 error is modest compared with a 33% error in the Fick $Q_p$, it seems that it is not the primary source of error in the oximetry calculation.

Having described significant (and opposite) errors in both pulmonary and systemic blood flows as calculated by oximetry, it follows that the Fick $Q_p/Q_s$ ratio was also found to be inaccurate. We observed that all oximetry-derived values of $Q_p/Q_s$ fell within a fixed range between 0.4 and 0.8, with no correlation to the actual ratio measured by CMR. As an example, 1 patient whose Fick $Q_p/Q_s$ ratio would have been deemed low at 0.6 had an actual $Q_p/Q_s$ by CMR of 1.6 because of extensive systemic-to-pulmonary arterial collateral flow. One can imagine that the management of a patient with low $Q_p$ and high PVR would differ substantially from that of a patient with high (but perhaps low effective) $Q_p$, low PVR, and a volume-loaded systemic ventricle.

Therefore, it seems that the best physiological characterization of these patients can be obtained by combining flow measurements obtained by CMR with catheterization-derived pressure data. Ultimately, a completely noninvasive strategy might be desirable, but the need to calculate PVR precludes this in many cases. Other groups have investigated using MRI indices alone to predict PVR21 or used MRI-guided catheterization as a means to reduce radiation exposure.22 These techniques warrant further study in the single ventricle population.

**Limitations**

This study is limited by its retrospective design. Most important, our cohort may not represent the SCPC population as a whole because all study patients had a clinical indication for XMR. This could produce a relevant selection bias if patients referred for CMR have more systemic-to-pulmonary arterial collateral flow than the general SCPC population. However, it is difficult to ascertain whether this is the case because to date, there are no prospective studies examining collateral flow in the general SCPC population.

The data on SVC/IVC flow and metabolic ratios must also contribute to error in Fick-derived flow parameters. In these patients, the assumed VO2 underestimated the calculated value by an average of 12%, which should translate into an underestimate of both $Q_p$ and $Q_s$ by Fick. Because Fick-derived $Q_p$ overestimated the CMR value, the error in VO2 cannot be directly implicated. It is likely that the discrepancy between Fick- and CMR-derived $Q_p$ would be even greater if the precise VO2 were known for each patient. The underestimate of VO2 almost certainly accounts for some of the error in the Fick $Q_p$. However, because the magnitude of the VO2 error is modest compared with a 33% error in the Fick $Q_p$, it seems that it is not the primary source of error in the oximetry calculation.

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Finally, the limitations of PC-MRI for flow assessment must be recognized. The target spatial resolution was achieved in 98% of acquisitions in this study, and internal consistency assessments showed excellent reproducibility of the velocity mapping data. In similar patients with SCPC, the coefficients of intraobserver and interobserver reliability for PV flow measurements were high at 0.98 and 0.86, respectively. Nevertheless, errors may occur, and anatomic features, such as turbulent flow around anastomotic sites, may exacerbate these errors in certain patients. However, there is no clear reason to suspect a systematic bias in PC-MRI measurements when compared with Fick.

Conclusions

In patients with SCPC presenting for XMR, oximetry consistently underestimates $Q_s$ and overestimates PVR, in part because of the presence of systemic-to-pulmonary arterial collateral flow. In contrast, Fick calculations of $Q_s$ overestimate CMR-measured $Q_s$ when conventional assumptions are applied. This may occur because the upper body consistently receives a disproportionate amount of blood flow relative to its metabolic demand, making the SVC saturation a systematic overestimate of the mixed venous saturation. Finally, the $Q_s/Q_c$ ratio generated by oximetry bears no relationship to the patient’s actual flow balance as measured by CMR.

We conclude that catheterization-derived flow measurements are generally unreliable in patients with SCPC. The addition of CMR can significantly enhance the understanding of an individual patient’s physiology. When CMR is contraindicated or unavailable, oximetry data should be interpreted with caution, understanding the nature and causes of its inaccuracy. Further study on combined cardiac catheterization and CMR techniques is warranted in the single ventricle population.

Sources of Funding

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Disclosures

None.

References

This research compares traditional oximetry-based methods for calculating pulmonary and systemic blood flows with phase-contrast MRI measurements in the population of single ventricle patients palliated with superior cavopulmonary connection. We found that oximetry significantly underestimates pulmonary blood flow, especially in patients with a greater burden of systemic-to-pulmonary arterial collaterals. This, in turn, leads to inaccurate calculation of pulmonary vascular resistance. Oximetry also produces a systematic overestimate of systemic blood flow and a $Q_p:Q_s$ ratio that does not correlate with that measured by MRI. Clinically, the implication is that oximetry, while still widely used, may yield information that inaccurately describes the true physiology of this patient population. Supplementing traditional hemodynamic catheterization with flow measurements by MRI will likely provide a better assessment of the superior cavopulmonary circuit, both for diagnosing suspected physiological derangements and for assessing patient candidacy for Fontan completion.
Accuracy of Conventional Oximetry for Flow Estimation in Patients With Superior Cavopulmonary Connection: A Comparison With Phase-Contrast Cardiac MRI
Tacy E. Downing, Kevin K. Whitehead, Yoav Dori, Matthew J. Gillespie, Matthew A. Harris, Mark A. Fogel, Jonathan J. Rome and Andrew C. Glatz

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