A Quantitative Analysis of Hemodynamic Effects of the Right Ventricle Included in the Circulation of the Fontan Procedure

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Background. Right heart hemodynamics were analyzed with a catheter-mounted velocity meter in seven patients after direct atrioventricular anastomosis for Fontan procedure (RV group) and were compared with those obtained in eight patients after direct atripulmonary anastomosis (RA group).

Methods and Results. In the RV group, cardiac output was 2.7±0.6 l/min/m²; mean right atrial and pulmonary artery pressures were both 13±3 mm Hg; mean pulmonary artery wedge pressure was 7±5 mm Hg; left ventricular end-diastolic volume, determined angiographically, was 129±40% of normal; and its ejection fraction was 0.50±0.09. In the RA group, data were similar to those of the RV group except that right heart pressures were lower in the RV group, which was related to the preoperative condition of the pulmonary circulation. In the RV group, the fraction of ventricular forward flow to the total forward flow in the main pulmonary artery ranged from 0.21 to 0.46 and was not correlated with cardiac output or with any other parameter. The backward flow into the inferior vena cava at ventricular systole was greater than the atrial flow in two patients in whom cardiac output was less than 2.2 l/min/m², whereas caval backward flow at atrial contraction was greater than ventricular flow in the other five patients, of whom four had a cardiac output greater than 3.1 l/min/m².

Conclusions. We conclude that the inclusion of the right ventricle in the circulation of the Fontan procedure does not necessarily improve overall hemodynamics in most patients. (Circulation 1991;83:822–826)

The dynamics of right heart blood flow have been studied in patients after undergoing a Fontan procedure, in which the right ventricle is completely excluded from the circulation.1,2 Bjork's modification, which includes the rudimentary right ventricle,3 is the other surgical choice for tricuspid atresia. The rationale for the modification is that the right ventricle should augment forward flow to the pulmonary circulation. In fact, contraction of the ventricle raises its pressure1,4 and produces a forward flow,1 and the volume of the ventricle increases after the operation in some cases.5,6 Coles et al6 concluded that a nonvalved atrioventricular connection is the choice of surgery; their conclusion was based on their own experience with postoperative hemodynamics, survival, and reoperative risks. These observations favor the inclusion of the right ventricle in Fontan operations. Our initial experience, however, showed that total hemodynamics of patients after Bjork's procedure were not necessarily better than those after direct atropulmonary anastomosis.1 Many recent reports7–9 indicate that surgeons have adopted a variety of procedures, probably based on their own experiences, and this variety indicates a lack of agreement on whether the right ventricle should be included in the circulation in Fontan procedures. The major reason for the uncertainty is that the hemodynamic effect of including the right ventricle has not been precisely studied. Therefore, we investigated the role of right ventricular contraction in right heart flow in patients after Bjork's modification.

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and compared these hemodynamics with those in patients after direct atroventricular anastomosis.

**Methods**

We included seven patients after undergoing Bjork’s modification of the Fontan procedure in whom satisfactory flow data were obtained on routine catheterization that was performed 3–4 weeks after operation; patients’ ages ranged from 3 to 23 years. Diagnoses were tricuspid atresia of type 1b in six patients and double inlet left ventricle with normally related great arteries and a narrow tricuspid valve ring in the remaining patient. Four patients had had palliative operations (Table 1). Preoperative hemodynamic data are also presented in Table 1. Operation was completed by a direct atroventricular anastomosis with closure of a ventricular septal defect in six patients and with atrial partitioning in one patient (patient 3). Pulmonary valve commissurotomy was performed in two patients and annuloplasty in one patient. We did not use artificial valves in any patient.

Right heart pressures were recorded by a fluid-filled catheter system, and cardiac output was measured by the thermodilution method. Blood flow velocity at the right heart was obtained by a catheter-mounted velocity probe (model VPC-663A, Millar Instruments, Houston, Tex.). Then, angiography was performed in anterolateral projections, from which left ventricular volume was calculated by the area-length method, and end-diastolic volume was expressed as percentage of normal.

The area under the forward flow of the velocity signal in the main pulmonary artery was integrated and divided into three parts. The atrial systolic (A) wave was separated from the diastolic (D) wave by a vertical line drawn to the zero line at the beginning of the upstroke of the A wave. The ventricular systolic (V) wave was defined as beginning at its upstroke and ending when the velocity signal crossed the zero line (Figure 1). Or, it was defined as being separated from the D wave by a line at 37 msec after the dicrotic notch on the pressure tracing in patients in whom the flow was continuous through this period. Then, the ratios of the area of the A and V waves to the total area (Fa and Fv) were calculated. From Fv and cardiac output, an absolute value of forward flow by the V wave was computed. To evaluate backward flow due to right ventricular contraction into the systemic vein, we obtained a and v wave pressures and the peak of the A and V waves of flow velocity in

**Table 1. Clinical Features of the Patients**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Diagnosis</th>
<th>Age at operation (yr)</th>
<th>Palliations</th>
<th>PA index</th>
<th>PAR (units/m²)</th>
<th>PAP (mm Hg)</th>
<th>Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1b</td>
<td>19</td>
<td>C shunt</td>
<td>353</td>
<td>2.3</td>
<td>17</td>
<td>Bjork</td>
</tr>
<tr>
<td>2</td>
<td>1b</td>
<td>7</td>
<td>Left BT</td>
<td>302</td>
<td>2.2</td>
<td>7</td>
<td>Bjork</td>
</tr>
<tr>
<td>3</td>
<td>SLV, TS</td>
<td>3</td>
<td>None</td>
<td>450</td>
<td>2.0</td>
<td>17</td>
<td>RA–RV, atrial partitioning</td>
</tr>
<tr>
<td>4</td>
<td>1b</td>
<td>4</td>
<td>None</td>
<td>480</td>
<td>1.2</td>
<td>29</td>
<td>Bjork</td>
</tr>
<tr>
<td>5</td>
<td>1b</td>
<td>8</td>
<td>Left BT</td>
<td>320</td>
<td>2.0</td>
<td>18</td>
<td>Bjork</td>
</tr>
<tr>
<td>6</td>
<td>1b</td>
<td>19</td>
<td>Left BT</td>
<td>188</td>
<td>0.6</td>
<td>10</td>
<td>Bjork</td>
</tr>
<tr>
<td>7</td>
<td>1b</td>
<td>6</td>
<td>None</td>
<td>455</td>
<td>0.5</td>
<td>18</td>
<td>Bjork</td>
</tr>
</tbody>
</table>

PA index (Nakata et al\textsuperscript{14}), pulmonary artery index; PAR, pulmonary artery resistance; PAP, mean pulmonary artery pressure; C shunt, central shunt; RVOTR, right ventricular outflow tract reconstruction; BT, Blalock–Taussig shunt; SLV, single left ventricle; TS, tricuspid stenosis; RA, right atrium; RV, right ventricle.

**Figure 1.** Tracing showing areas under the forward flow of the velocity signal recorded at the main pulmonary artery, which were integrated. Areas under the A and V waves were measured separately, and each was divided by the total area of forward flow. Thus, fractions of the A and V waves to the total flow (Fa and Fv, respectively) were obtained.
**TABLE 2. Postoperative Hemodynamic and Volume Data**

<table>
<thead>
<tr>
<th>Patient</th>
<th>CI (l/min/m²)</th>
<th>RAP (mm Hg)</th>
<th>PAP (mm Hg)</th>
<th>PAWP (mm Hg)</th>
<th>LVEDV (%)</th>
<th>EF</th>
<th>Fa</th>
<th>Fv</th>
<th>P: a/v</th>
<th>Q: A→V</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2.1</td>
<td>8</td>
<td>8</td>
<td>3</td>
<td>128</td>
<td>0.45</td>
<td>0.23</td>
<td>0.44</td>
<td>0.8</td>
<td>−8</td>
</tr>
<tr>
<td>2</td>
<td>2.2</td>
<td>14</td>
<td>14</td>
<td>10</td>
<td>131</td>
<td>0.37</td>
<td>0.50</td>
<td>0.21</td>
<td>1.0</td>
<td>−3</td>
</tr>
<tr>
<td>3</td>
<td>3.2</td>
<td>13</td>
<td>11</td>
<td>4</td>
<td>116</td>
<td>0.66</td>
<td>0.54</td>
<td>0.30</td>
<td>1.3</td>
<td>+10</td>
</tr>
<tr>
<td>4</td>
<td>3.4</td>
<td>14</td>
<td>13</td>
<td>3</td>
<td>209</td>
<td>0.50</td>
<td>0.57</td>
<td>0.29</td>
<td>1.5</td>
<td>+20</td>
</tr>
<tr>
<td>5</td>
<td>3.1</td>
<td>15</td>
<td>14</td>
<td>8</td>
<td>105</td>
<td>0.42</td>
<td>0.42</td>
<td>0.46</td>
<td>1.7</td>
<td>+23</td>
</tr>
<tr>
<td>6</td>
<td>1.6</td>
<td>10</td>
<td>10</td>
<td>...</td>
<td>67</td>
<td>0.55</td>
<td>0.43</td>
<td>0.28</td>
<td>1.2</td>
<td>+30</td>
</tr>
<tr>
<td>7</td>
<td>3.1</td>
<td>17</td>
<td>18</td>
<td>16</td>
<td>145</td>
<td>0.52</td>
<td>0.39</td>
<td>0.32</td>
<td>1.1</td>
<td>+5</td>
</tr>
</tbody>
</table>

CI, cardiac index; RAP, mean right atrial pressure; PAP, mean pulmonary artery pressure; PAWP, mean pulmonary artery wedge pressure; LVEDV, left ventricular end-diastolic volume (% of normal); EF, ejection fraction; Fa and Fv, fraction of atrial and ventricular, respectively, forward flow in pulmonary artery total forward flow; IVC, inferior vena cava; P: a/v, pressure ratio of a to v wave; Q: A→V, difference of peak backward flow between A and V wave, expressed as cm/sec (see text).

the inferior vena cava. The relative contribution of right ventricular systole in regurgitation was evaluated by the pressure ratio of the a to v wave and by the difference of peak velocity, which was the A wave minus the V wave in centimeters divided by seconds. Thus, the difference was negative when the peak of the V wave was larger than that of the A wave. Although the area under each regurgitant flow should have been measured for the quantitative analysis, the time of pulmonary valve closure, thus, the end of ventricular systole, was not clearly identified in pressure records. This was probably because a small dicrotic notch did not transmit to the systemic vein so that only the peak of the flow was compared. All these data were obtained from three to five consecutive cardiac cycles.

Basic hemodynamics of these patients (RV group) were compared with those from eight patients (RA group) after direct atroipulmonary anastomosis. These were the eight of 10 patients who were included in the previous study. The other two patients (patients 3 and 9 in the previous study) were patients 6 and 7 in the present study. Data were expressed as mean±SD, and the differences were analyzed by Student’s t test. A probability less than 0.05 was considered significant.

**Results**

In the RV group, the cardiac index was 2.7±0.6 l/min/m². Mean right atrial and main pulmonary artery pressures were 13±3 and 13±3 mm Hg, respectively. Mean pulmonary artery wedge pressure was 7±5 mm Hg. Left ventricular end-diastolic volume was 129±40% of normal, and ejection fraction was 0.50±0.09 (Tables 2 and 3). The atrial and pulmonary pressures were significantly lower in the RV group than in the RA group, whereas cardiac output and ventricular volume were similar in these two groups (Table 3).

In the RV group, the forward flow in the pulmonary artery during ventricular contraction was 0.88±0.34 l/min/m², and the ventricular fraction of forward flow was 0.33±0.09, whereas the atrial fraction of forward flow was 0.44±0.11. The latter two parameters were not correlated with cardiac output or with pressure data. The backward flow into the inferior vena cava during ventricular contraction (V wave) was larger than that during atrial

**TABLE 3. Comparison Between RA→RV and RA→PA Anastomosis**

<table>
<thead>
<tr>
<th></th>
<th>Preoperative</th>
<th>Postoperative</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Age (yr)</td>
<td>PA index (mm²/m²)</td>
</tr>
<tr>
<td>RA→RV (n=7)</td>
<td>Mean</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>6</td>
</tr>
<tr>
<td>RA→PA (n=8)*</td>
<td>Mean</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>4</td>
</tr>
</tbody>
</table>

PA, pulmonary artery; PAR, pulmonary artery resistance; PAP, mean pulmonary artery pressure; CI, cardiac index; RAP, mean right atrial pressure; PAWP, mean pulmonary artery wedge pressure; LVEDV, left ventricular end-diastolic volume (% of normal); EF, ejection fraction; Fa, fraction of atrial forward flow in pulmonary artery forward flow; RA→RV, right atrial-right ventricular; RA→PA, right atrial-pulmonary artery.

*Data from Nakazawa et al.²
†p<0.05 RA→RV vs. RA→PA.
contraction (A wave) in two patients (Figure 2, right), in whom the a to v wave pressure ratio was 1.0 or less and cardiac output was 2.1 and 2.2 l/min/m². In contrast, the V wave was smaller than the A wave in the other five patients (Figure 2, left), in whom the a to v wave pressure ratio was 1.1 or higher. The cardiac index was larger than 3.1 l/min/m² in all but one of these patients (Table 2). Before operation, the pulmonary artery index was 379±110 mm²/m², and pulmonary artery vascular resistance was 2.0 units·m² or less in these five patients. By contrast, these values were 302 and 353 mm²/m² and 2.2 and 2.3 units·m² in the two patients with low cardiac output.

Discussion

This study demonstrates that the use of the subpulmonary right ventricle in a Fontan operation did not necessarily improve total hemodynamics, as shown by the data that cardiac output was not affected by the inclusion or exclusion of the ventricle as a whole. The lower right atrial pressure with the similar cardiac output in the RV group may reflect a beneficial role of the right ventricle in hemodynamics. The pulmonary artery pressure, however, was similar to the right atrial pressure, indicating that the ventricle did not provide any additional positive work to the right heart circulation. We considered that the lower right heart pressure in these patients was related to the fact that they had a larger pulmonary artery index and a lower arterial resistance than the other patients had, although the difference in the pulmonary artery index was not significant (Table 3). It would also be partially affected by the subtle difference in pulmonary artery wedge pressure.

We calculated ventricular output arbitrarily as the right ventricle functions with inflow and outflow valves. This supposition, however, is not complete. DiSessa et al., in a study using Doppler echocardiography, concluded that pulmonary arterial and caval flows were rather uniform regardless of the use of the right ventricle. They speculated that the major determinant of the flow pattern was the pressure gradient from the right heart to the left atrium, which supported an earlier study by Bull et al. It is obvious that pressure difference is the determinant of flow under any condition. We studied the relation of pulmonary blood flow patterns to pressure differences between the pulmonary artery and the left atrium in patients with congenital complete heart block after a Fontan procedure, which is a unique condition in which the atrial contraction is independent of ventricular contraction. The study confirmed that the blood flow pattern in the main pulmonary artery showed an instantaneous and totally direct relation to the pressure difference. Thus, ventricular contraction should produce forward flow as far as it raises the pressure that we previously reported, even if it is superimposed on other waves. The difference between our findings and those of DiSessa et al. may be related to variability in the size of the rudimentary ventricular chamber or differences in the residual functional capacity of the ventricle after surgery. The latter includes augmentation of the atrioventricular connection with a patch, release of outflow tract obstruction, possibly with an outflow patch, and closure of a ventricular septal defect with various sizes. In fact, some of our patients had only a small V wave in the pulmonary artery as indicated by a low ventricular fraction of forward flow.

The right ventricular contraction, however, did not function favorably in these patients on the whole, as mentioned above, and as shown by the lack of correlation of ventricular fraction of forward flow or right ventricular output to cardiac output. This finding supports the conclusion of Lee et al., who reported...
clinical results of medium-term follow-up of patients after direct atroio pulmonary or atroioventricular connection and found that there was no difference in physical improvement between these two groups. On the other hand, Fontan et al. and others recommended the incorporation of the ventricle with the inflow valve. They found that the ventricle worked as a pumping chamber, and it could grow later, thus giving nearly normal hemodynamics in some, but not most, of their patients. The disadvantage of using a valve with or without a conduit has, however, been increasingly clarified by the fact that such patients need reoperation for later pathway obstruction more frequently than those with direct anastomosis, 5,12 Based on their similar experience, Coles et al. recommended direct atroioventricular connection, although they did not support their conclusion with definitive data. The other reason for incorporating the ventricle is that the pulmonary valve may prevent regurgitation. We and others have demonstrated that retrograde flow from the pulmonary artery to the right atrium is negligible in patients after direct atroio pulmonary anastomosis.

Some of the patients in this study had a relatively low cardiac index, and the backward flow into the systemic vein during ventricular systole was comparatively large. It seemed that the backward flow impeded the venous return and decreased cardiac output in these patients. We considered that this was related to the power of pump function of the right ventricle. The ventricular fraction of forward flow and right ventricular output, however, were not correlated with the backward flow; in other words, the patients with a prominent ventricular caval backward flow did not necessarily have a high ventricular fraction of forward flow. This discrepancy can be explained by the difference in vascular impedance to right ventricular ejection between the pulmonary circulation and the systemic venous system. The pulmonary arterial vascular resistance was lower than 2.0 units \( \cdot m^{-2} \) before operation in patients whose preoperative cardiac index was higher than 3.0 l/min/m², whereas the resistance was higher in two of the three patients who had a relatively low cardiac index. Patient 6, an exceptional case, had a low preoperative pulmonary resistance, but his postoperative cardiac output was unexpectedly low. We considered that this was because he had the smallest pulmonary artery index among the study patients. Another possible explanation for the difference in the caval backward flow is that a well-developed venous valve functions as an inflow valve to prevent the backward flow. However, echocardiography did not clearly demonstrate the valve, and if it did so, this should have affected the A wave as well, which was not the case.

We conclude that good postoperative hemodynamics result from normal or larger-than-normal pulmonary artery size and low pulmonary artery vascular resistance, but not necessarily from the inclusion of the right ventricle, and that powerful contraction of the right ventricle may be disadvantageous in the circulation of the Fontan procedure.

References


Key Words • hemodynamics • tricuspid atresia • Bjork procedure • pulmonary artery index
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M Nakazawa, H Katayama, Y Imai, K Nojima, T Nakanishi, H Kurosawa, A Takao and H Okuda

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