Changes in Pulmonary Vascular Resistance in Infants and Children with Left-to-Right Intracardiac Shunts

By P. A. M. Auld, M.D., A. L. Johnson, M.D., J. E. Gibbons, M.D., and M. McGregor, M.D.

Of the several factors that must be assessed in selecting the optimum time for surgery in infants and children with left-to-right intracardiac shunts, the extent of elevation of pulmonary vascular resistance is one of the more important. Opinion is divided as to whether pulmonary vascular resistance in these cases can progressively rise and if so with sufficient frequency to influence the optimum age for surgery. To obtain further information on this problem the records of all such patients who had had two or more successful catheterization studies in our laboratory were reviewed.

Material and Method

The data in this report are based on catheterization studies performed on 17 patients with intracardiac left-to-right shunts and significant pulmonary hypertension. The subjects were not randomly selected. Among other reasons, repeat catheterization studies were performed to re-evaluate cases with known elevation of pulmonary vascular resistance or because of clinical x-ray and electrocardiographic evidence suggesting a change in pulmonary vascular resistance. Catheterization studies were performed in the conventional manner. Blood samples were analyzed for oxygen by the manometric technic of Van Slyke and Neill or by a whole blood oximeter technic. In certain cases systemic arterial saturation was measured by an ear oximeter. For the purpose of calculation of pulmonary (Qp) and systemic (Qs) flows, oxygen consumption was assumed from the values of Rudolph and Cayler. Pulmonary vascular resistance was computed by the formula

\[ PVR = \frac{PAm - PCm (or LAm)}{Qp L./min./M^2} \]

where PAm, PCm, and LAm are mean pressures in the pulmonary artery, pulmonary capillary, and left atrium respectively. In some instances the pulmonary capillary pressure was assumed. None of the cases demonstrated a systolic pressure gradient between right ventricle and pulmonary artery.

The diagnosis in seven cases was confirmed by autopsy and in the remainder was based on operative findings or on clinical and catheterization data (table 1). The two cases diagnosed as atioventricular communis resembled an isolated ventricular septal defect functionally.

Results

Eleven patients in the study showed a decrease in pulmonary flow together with a rise in pulmonary artery mean pressure, indicating a rise in pulmonary vascular resistance (fig. 1). Between the first and subsequent studies no significant change in either parameter was observed in one patient, and in five patients a rise in pressure only was noted. The pulmonary vascular resistance standardized for body surface area is related to age at time of study in figure 2. In all but one there was some increase in calculated resistance between the first and second study. It should be particularly noted that in six children the pulmonary vascular resistance rose from a normal to an elevated level, in some instances in the early months of life. To remove the influence of body size from the assessment, as well as the assumed oxygen consumption, the change in PAm pressure was related to the change in the Qp/Qs ratio between first and second studies (fig. 3). The majority of the cases were arranged in the area representing a rise in pulmonary vas-
Table 1

Diagnosis, Catheterization Data, and Resistance Calculations in Seventeen Patients with Repeat Studies

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Surface area M²</th>
<th>Age, yr.</th>
<th>Pulm. artery pressure mm. Hg</th>
<th>Systemic artery pressure mm. Hg</th>
<th>PCmpl. mm. Hg</th>
<th>Pulm. Flow L./min. M²</th>
<th>Systemic flow L./min. M²</th>
<th>P.V. Res. L. mm. Hg</th>
<th>Total Pulm. Res. L. mm. Hg</th>
<th>Q/Q</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. VSD female</td>
<td>1.42</td>
<td>3</td>
<td>54/25 (35)</td>
<td>75/---</td>
<td>13</td>
<td>20.0</td>
<td>4.7</td>
<td>1.1</td>
<td>1.8</td>
<td>3.4</td>
</tr>
<tr>
<td>2. VSD female</td>
<td>0.51</td>
<td>6</td>
<td>73/23 (40)</td>
<td>85/40 (55)</td>
<td>10*</td>
<td>12.3</td>
<td>3.1</td>
<td>5.4</td>
<td>2.5</td>
<td>3.3</td>
</tr>
<tr>
<td>3. VSD male</td>
<td>0.102</td>
<td>1</td>
<td>102/60 (76)</td>
<td>106/72 (88)</td>
<td>3</td>
<td>3.6</td>
<td>4.5</td>
<td>20.0</td>
<td>21.1</td>
<td>0.8</td>
</tr>
<tr>
<td>4. VSD+ male</td>
<td>0.58</td>
<td>1</td>
<td>62/20 (37)</td>
<td>---</td>
<td>3</td>
<td>22.5</td>
<td>5.0</td>
<td>1.5</td>
<td>1.6</td>
<td>4.6</td>
</tr>
<tr>
<td>5. VSD &amp; PDA male</td>
<td>1.74</td>
<td>8</td>
<td>71/36 (48)</td>
<td>86/48 (61)</td>
<td>5*</td>
<td>6.7</td>
<td>3.2</td>
<td>6.5</td>
<td>7.1</td>
<td>2.1</td>
</tr>
<tr>
<td>6. VSD &amp; PDA male</td>
<td>0.34</td>
<td>7</td>
<td>72/25 (43)</td>
<td>95/60 (72)</td>
<td>7</td>
<td>5.5</td>
<td>3.3</td>
<td>12.5</td>
<td>13.6</td>
<td>1.7</td>
</tr>
<tr>
<td>7. VSD female</td>
<td>0.67</td>
<td>5</td>
<td>79/56 (63)</td>
<td>10</td>
<td>3</td>
<td>8.8</td>
<td>2.5</td>
<td>6.5</td>
<td>7.2</td>
<td>3.5</td>
</tr>
<tr>
<td>8. VSD male</td>
<td>0.9</td>
<td>1</td>
<td>85/55 (72)</td>
<td>106/72 (88)</td>
<td>10</td>
<td>10.8</td>
<td>2.7</td>
<td>5.2</td>
<td>6.7</td>
<td>4.0</td>
</tr>
<tr>
<td>9. VSD female</td>
<td>0.87</td>
<td>9</td>
<td>95/51 (70)</td>
<td>97/67 (62)</td>
<td>5*</td>
<td>4.0</td>
<td>2.5</td>
<td>16.4</td>
<td>17.5</td>
<td>1.6</td>
</tr>
<tr>
<td>10. VSD male</td>
<td>0.34</td>
<td>7</td>
<td>69/38 (52)</td>
<td>93/48 (66)</td>
<td>6</td>
<td>9.9</td>
<td>4.7</td>
<td>4.6</td>
<td>5.3</td>
<td>2.1</td>
</tr>
<tr>
<td>11. VSD female</td>
<td>0.53</td>
<td>1</td>
<td>77/29 (56)</td>
<td>86/50 (67)</td>
<td>6</td>
<td>9.8</td>
<td>3.5</td>
<td>5.3</td>
<td>5.0</td>
<td>2.8</td>
</tr>
<tr>
<td>12. VSD male</td>
<td>0.35</td>
<td>1</td>
<td>72/28 (45)</td>
<td>130/70 (95)</td>
<td>12</td>
<td>9.0</td>
<td>4.1</td>
<td>3.7</td>
<td>5.0</td>
<td>2.2</td>
</tr>
<tr>
<td>13. VSD female</td>
<td>0.55</td>
<td>2</td>
<td>70/26 (49)</td>
<td>96/55 (58)</td>
<td>5</td>
<td>9.3</td>
<td>5.0</td>
<td>4.7</td>
<td>5.3</td>
<td>1.7</td>
</tr>
<tr>
<td>14. VSD male</td>
<td>0.46</td>
<td>1</td>
<td>125/75 (90)</td>
<td>10</td>
<td>3</td>
<td>8.2</td>
<td>3.4</td>
<td>7.2</td>
<td>7.6</td>
<td>2.4</td>
</tr>
<tr>
<td>15. AVCS male</td>
<td>0.46</td>
<td>1</td>
<td>80.34 (62)</td>
<td>87/50 (68)</td>
<td>10</td>
<td>13.8</td>
<td>5.0</td>
<td>2.9</td>
<td>3.6</td>
<td>2.7</td>
</tr>
<tr>
<td>16. AVCS female</td>
<td>0.60</td>
<td>3</td>
<td>100/50 (75)</td>
<td>112/62 (87)</td>
<td>13</td>
<td>8.0</td>
<td>8.0</td>
<td>7.7</td>
<td>9.4</td>
<td>1.0</td>
</tr>
<tr>
<td>17. VSD female</td>
<td>0.50</td>
<td>2</td>
<td>83/43 (63)</td>
<td>---</td>
<td>10</td>
<td>3.6</td>
<td>5.1</td>
<td>3.6</td>
<td>4.0</td>
<td>0.7</td>
</tr>
</tbody>
</table>

*Assumed value.
†Double outlet right ventricle.
‡Results obtained 1 yr. after unsuccessful attempt at closure of VSD.
§Atrioventricularis communis.
pulmonary resistance. Four subjects appear in the area representing an increase in PA mean pressure and increase in Qp/Qs ratio. This finding was interpreted as indicating a passive increase in pressure and flow related to an increase in systemic resistance between the first and subsequent study.

Discussion

This study was not designed to present the natural history of ventricular septal defect, as evidenced by the exclusion of patients with systolic pressure gradients between right ventricle and pulmonary artery, and two patients whose data indicate spontaneous closure of a ventricular defect. The inclusion of the two patients with atrioventricularis communis is justified by the fact that hemodynamically they resembled a ventricular defect.

The difficulty of obtaining accurate data in studies such as this is considerable. Whereas the pressure values are relatively accurate, the flow data based on assumed values for oxygen consumption are not. Resistance values, particularly when left atrial pressure is assumed, must be less accurate still. However, all these errors should influence the results in a random way. One factor which might operate other than randomly is the level of sedation employed. Depression from sedation with resultant hypoxia, especially in the younger subjects, may well cause a misleading elevation of pulmonary vascular resistance. The need to sedate the younger children more heavily might influence the result of the initial study, but it is unlikely to be responsible for the observed higher resistance at the second study when the subjects were less likely to be depressed.

The tendency for pressure to rise and pulmonary flow index to fall with time strongly suggests progression of pulmonary vascular changes in these patients. However, no conclusion can be drawn from this study as to the frequency with which this occurs in pa-
tients with left-to-right intracardiac shunts. A proportion of the cases reported by Lucas et al.\textsuperscript{4} showed changes similar to those reported here. In contrast, other workers\textsuperscript{5,6} have considered rising pulmonary vascular resistance in childhood an unimportant occurrence. Some\textsuperscript{7} have based their conclusions on estimates of "total pulmonary resistance," a value which ignores the left atrial pressure and only approaches a valid reflection of resistance to blood flow in the pulmonary bed when left atrial pressure is assumed to be low and constant at each study. However, calculation of the present data expressed as "total pulmonary resistance" reveals the same result. The present study tends to support the conclusions of Dammann and Ferencz\textsuperscript{8} and Heath and Edwards,\textsuperscript{9} who suggested progression of pulmonary vascular changes on the basis of pathologic studies.

The data demonstrate that patients with left-to-right shunts and pulmonary hypertension may develop a progressive rise in pulmonary vascular resistance in the pediatric age group. Likewise, in considering the optimum age for surgical correction of such cases, the possibility of progression is a real one and should receive consideration.

Summary

The hemodynamic data of seventeen patients with left-to-right intracardiac shunts and pulmonary hypertension have been reviewed. A considerable rise in pulmonary vascular resistance was observed in 12 of these patients between the time of the first and second catheterizations. The results suggest that the possibility of progressive elevation of pulmonary vascular resistance with the passage of time should be a serious consideration in the selection of optimum age for operation in patients with left-to-right intracardiac shunts.

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