Results of surgical treatment of ventricular septal defects with pulmonary hypertension

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ABSTRACT Two hundred and twenty-five consecutive patients with interventricular septal defect and associated pulmonary hypertension have undergone corrective surgery at the Christian Medical College Hospital. The mean preoperative systolic pulmonary artery pressure was 70-5 (range 31–136) mm Hg and the calculated pulmonary vascular resistance ranged from 300 to 1680 dyns cm⁻⁵. A paracoronary right ventriculotomy was the approach of choice. Profound hypothermia and circulatory arrest were not used, even in 12 patients weighing under 10 kg. Among the older children and young adolescents there were 27 who had a calculated pulmonary vascular resistance of over 800 dyns cm⁻⁵ and their mortality was 22%, which is good when compared with that of other series. It is evident that both the early and the late death rate after surgery increase with the age of the patient, especially in those with associated pulmonary hypertension. In 69 patients studied after repair recatheterisation showed no residual defect by oximetry. The fall in the pulmonary artery pressures after surgery has been striking in most patients. The late death rate was 2-5%. The surviving patients are leading normal, active lives.

In 1879 Roger described the clinical signs of ventricular septal defect and indicated the underlying pathology. 1 Maude Abbott 2 gave credit to Dalrymple as being the first to describe Eisenmenger’s complex in 1874, by reporting a postmortem study on a 45-year-old woman who had cyanosis for several years before her death. Lillehei et al in 1954 was the first to use cardiopulmonary bypass with controlled cross-circulation to close the defect. 3 Several studies since have documented the benefits that accrue from closure of a ventricular septal defect, even in those with pulmonary hypertension. 4–7 From 1966 to April 1982, of 293 patients undergoing surgical correction of ventricular septal defects in our department of cardiothoracic surgery, 225 had complicating pulmonary hypertension. The results in this group form the basis of our report.

Patients and methods

The youngest of the 225 patients was aged 6 months and the oldest 33 years (mean 9.1 years)—see figure 1 and table 1 for the age distribution.

All patients had pulmonary hypertension. The mean preoperative systolic pulmonary artery pressure was 70-5 (range 31–136) mm Hg. The calculated pulmonary vascular resistance ranged from 300 to 1680 dyns cm⁻⁵ (3.75–21 units (Wood)). In 27 patients the pressures were equal in the right and left ventricles with a pulmonary vascular resistance higher than 800 dyns cm⁻⁵. During the last two years the response to inhalation of 100% oxygen was determined in 30 patients in whom the pulmonary artery systolic pressure was 50–100% of the systemic pressure. The fall in pulmonary artery pressure and pulmonary vascular resistance is shown in table 2.

Most of the ventricular septal defects were infra-cristal, in the membranous portion of the septum (type II), 8 but they were supracristal (type I) in 33 cases and in the muscular septum in five. In most cases the defect was large, with a mean diameter of...
2.25 cm. The commonest associated anomalies were patent ductus arteriosus (in 21 patients) and mild aortic incompetence (in 16).

In all cases closure of the ventricular septal defect was performed with cardiopulmonary bypass and moderate total body hypothermia. Even in 12 patients weighing under 10 kg we used conventional bypass rather than profound hypothermia with circulatory arrest. The ventricular septal defect was exposed in all patients through a paracoronary right ventriculotomy that avoided major coronary artery branches. In all but 21 cases the defect was closed with a Dacron patch that was anchored in place with buttressed interrupted mattress sutures. In the posteroinferior portion of type II and type III defects the sutures were placed caudad and to the right of the edge of the defect to avoid injury to the conduction system.

In seven patients early in the series a patent ductus was ligated intrapericardially before cardiopulmonary bypass was established. More recently, in 14 patients, we have adopted the transpulmonary approach to obliterate the ductus, as we have already reported.9

Postoperative care included careful maintenance of blood volume and replacement of blood losses from the chest. Many of the patients, including infants, required inotropic support. Ventilation was assisted overnight in all cases and tracheostomy, with prolonged ventilatory assistance, was carried out in those with severe pulmonary vascular disease.

**Results**

Of the 225 patients, 38 died within 30 days of operation, giving a hospital death rate of 16.9% for the group. During the last six years, however, the death rate has been only 10%. The causes of death are shown in table 3. Technical imperfections in the cardiopulmonary bypass were responsible for 10 deaths. Low cardiac output with congestive failure was another frequent cause. The mortality in relation to age and pulmonary vascular resistance is shown in table 4. Of those with non-fatal complica-

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**Table 1** Age distribution in the 225 patients with pulmonary hypertension undergoing surgical correction of ventricular septal defects

<table>
<thead>
<tr>
<th>Age (y)</th>
<th>No of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>6/12-5</td>
<td>74</td>
</tr>
<tr>
<td>6-10</td>
<td>66</td>
</tr>
<tr>
<td>11-20</td>
<td>70</td>
</tr>
<tr>
<td>21-35</td>
<td>15</td>
</tr>
</tbody>
</table>

**Table 2** Preoperative response to inhalation of 100% oxygen

<table>
<thead>
<tr>
<th>Age group (y)</th>
<th>No of cases</th>
<th>Systolic pulmonary artery pressure range (and mean) (mm Hg)</th>
<th>Pulmonary vascular resistance range (and mean) (units (Wood))</th>
</tr>
</thead>
<tbody>
<tr>
<td>6/12-2</td>
<td>4</td>
<td>68-85 (77)</td>
<td>7-19 (13-1)</td>
</tr>
<tr>
<td>3-6</td>
<td>12</td>
<td>65-101 (91-2)</td>
<td>4-1-21 (12-1)</td>
</tr>
<tr>
<td>7-10</td>
<td>12</td>
<td>78-104 (83-7)</td>
<td>5-4-16-1 (13-6)</td>
</tr>
<tr>
<td>11-20</td>
<td>8</td>
<td>70-118 (86-1)</td>
<td>3-3-16-8 (10-2)</td>
</tr>
<tr>
<td>21-35</td>
<td>2</td>
<td>108-130 (119)</td>
<td>8-18-1 (13-0)</td>
</tr>
</tbody>
</table>

**Table 3** Causes of early postoperative death

<table>
<thead>
<tr>
<th></th>
<th>No of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low cardiac output</td>
<td>8</td>
</tr>
<tr>
<td>Unsatisfactory perfusion</td>
<td>10</td>
</tr>
<tr>
<td>Disseminated intravascular coagulation</td>
<td>3</td>
</tr>
<tr>
<td>Septicaemia</td>
<td>3</td>
</tr>
<tr>
<td>Ventricular arrhythmia</td>
<td>4</td>
</tr>
<tr>
<td>Fungal encephalitis</td>
<td>1</td>
</tr>
<tr>
<td>Aspiration</td>
<td>1</td>
</tr>
<tr>
<td>Cerebral hypoxia</td>
<td>3</td>
</tr>
<tr>
<td>Convulsions</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>38</td>
</tr>
</tbody>
</table>
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Table 4  Hospital mortality among the 225 patients by age and pulmonary vascular resistance (PVR)

<table>
<thead>
<tr>
<th>PVR (units (Wood))</th>
<th>Age (years)</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3-75- 6-10</td>
<td></td>
</tr>
<tr>
<td>No of cases</td>
<td>No of deaths</td>
<td>No of cases</td>
</tr>
<tr>
<td>61/12-5</td>
<td>93</td>
<td>7</td>
</tr>
<tr>
<td>4-1- 7-9</td>
<td>75</td>
<td>7</td>
</tr>
<tr>
<td>5-8-9-9-9</td>
<td>58</td>
<td>7</td>
</tr>
<tr>
<td>&gt;10-0</td>
<td>137</td>
<td>7</td>
</tr>
</tbody>
</table>

Table 5  Preoperative systolic pulmonary artery (PA) pressure and response to oxygen and postoperative pressure for six patients

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Age (y)</th>
<th>Preoperative PA pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Before oxygen</td>
</tr>
<tr>
<td>1</td>
<td>5</td>
<td>94</td>
</tr>
<tr>
<td>2</td>
<td>6</td>
<td>101</td>
</tr>
<tr>
<td>3</td>
<td>9</td>
<td>104</td>
</tr>
<tr>
<td>4</td>
<td>17</td>
<td>108</td>
</tr>
<tr>
<td>5</td>
<td>19</td>
<td>112</td>
</tr>
<tr>
<td>6</td>
<td>26</td>
<td>130</td>
</tr>
</tbody>
</table>

Haemodynamic studies were carried out in 69 patients who agreed to recatheterisation one to three years after repair. None showed any residual defect by oximetry; one patient had a very small residual jet shown on cineangiography. Systolic pulmonary artery pressures are shown in figure 3; in three subjects the pressures had fallen by 40-60 mm Hg from preoperative levels, but remained above 60 mm Hg. In six of the 69 patients...
who were recatheterised the preoperative response to 100% oxygen inhalation had been determined, and table 5 shows how far below these levels the systolic pulmonary artery pressure fell after operation. Pulmonary vascular resistance was calculated before and after correction in 14 patients with a preoperative level above 800 dyn/s cm⁻⁵. In these patients there was a considerable fall in all except four patients and no case of a rise in pressure (fig 4). All these patients are symptom free.

**Discussion**

In this series of patients ranging in age from 6 months to 33 years an appreciable number were in the second decade of life. The possibility that many of these patients would have developed Eisenmenger's complex if surgery had not been performed is fairly strong, perhaps 15–20%. Pulmonary vascular disease may regress completely up to the age of 2 years if the causal defects are corrected, but if surgery is deferred until after childhood this is less likely. It is noteworthy that in 27 patients the large ventricular septal defects were associated with equal systolic pressures in the two ventricles, while in 130 patients the calculated pulmonary vascular resistance was over 320 dyn/s cm⁻⁵ (4 units (Wood)). Recatheterisation in 69 patients after corrective repair has shown a drop in pressure in all. In three cases, however, the pressures were still appreciably raised, though considerably reduced from preoperative levels. In 14 of those restudied the calculated pulmonary vascular resistance had been over 800 dyn/s cm⁻⁵ before operation. We suggest that in some of these borderline cases serial postoperative recatheterisations are mandatory. Lillegard et al. in a large group, reported only two cases that showed progression of their pulmonary vascular disease after closure of a ventricular septal defect. Grosse-Brockhoff and Logan reported that all patients in their study who had a normal pressure after closure of the defect had had a pressure of less than 50 mm Hg before operation. Our results are at variance with this. Incomplete surgical closure of the defect has been reported to occur in 4–29% of cases in different series. We have been gratified that residual defects were not evident from oximetry in any of our 69 patients who were recatheterised.

Although uncommon, the combination of patent ductus arteriosus with ventricular septal defect should be recognised. Hallman et al. found an incidence of only 2.6% in 500 surgically treated patients, while in our series there was an incidence of 10%.

The operative mortality in this series was 16.9%. Sigman et al. and Allen et al. reported a higher mortality early in their experience. Hallman et al. in their study of 500 patients with ventricular septal defects and pulmonary hypertension noted 26% early deaths in patients with a systolic pulmonary artery pressure greater than 80% of the systemic pressure. From 1976 to 1981 the mortality was only 10% in our series. Of the 27 subjects with a pulmonary vascular resistance of over 800 dyn/s cm⁻⁵ there was a 22% early mortality, which is better than in other series. The use of cardioplegia, with better intraoperative myocardial preservation, and meticulous postoperative care with assisted ventilation have contributed greatly to these good results. We have not routinely carried out lung biopsies either before or during operation.

Complete atrioventricular block occurred in only two patients; one died four months after discharge from hospital, while the other is carrying out normal activities, with a heart rate of 60 beats/min, 12 years after repair. A higher incidence of complete atrioventricular block has been reported in other series. Eight patients (3%) showed evidence of right bundle-branch block with left anterior hemiblock. This should be viewed with concern and such
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patients need to be followed up carefully. Cas-
taneda\textsuperscript{10} reported a 14\% incidence of this conduc-
tion disturbance in his series.

Tricuspid incompetence was not seen in this study
after repair. Sigman \textit{et al}\textsuperscript{4} noted tricuspid incom-
tence in three subjects after operation, one of whom
required repair at the time of reoperation for closure
of a residual ventricular septal defect.

Follow-up in 181 long-term survivors for one to
16 years has shown that clinical benefit parallels
haemodynamic improvement and all surviving patients
are leading normal, active lives. There is
still much to be learned, however, about the precise
management of the borderline patient and the
degree of regression possible in the pulmonary vas-
cular disease in various age groups.

We acknowledge gratefully the help of Dr NK Kej-
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