The changes in systemic arterial oxygen saturation and interatrial mean pressure difference as a consequence of balloon atrial septostomy in 75 infants with transposition of the great arteries have been reported. The technique is effective in raising the arterial oxygen saturation and lowering the interatrial pressure difference. There was no relation between survival and the arterial oxygen saturation after septostomy. However, there was a suggestive relation between the occurrence of cerebrovascular accidents and arterial desaturation. This underlines the need for early corrective operation in infants with transposition of the great arteries.

Case material and methods

Of the total of 80 infants, 75 had systemic arterial oxygen saturations measured immediately before and after septostomy; 74 had both left and right atrial mean pressures measured at these times.

A subsequent cardiac catheterization was performed in 57 cases. The age at which this was performed ranged from 3 to 176 weeks (mean 50 weeks). The systemic arterial oxygen saturation was measured in 57 cases, the interatrial mean pressure difference in 53.

Oxygen saturation was measured using the Kipp Haemorefractor M.O.I, an individual calibration line being plotted for each investigation. Pressures were measured using either a Statham P23Gb strain gauge or an Eleta Schonander EMT pressure transducer. Recordings were made on an Elema Schonander Mingograph EM 81 direct-writing recorder.

The grouped oxygen saturation data were analysed by comparing the means using Student's $t$ test, two-tailed with appropriate corrections for samples of unequal size and unequal variance. A separate analysis of variance was performed with the oxygen saturation data grouped in eight diagnostic groups. These groups were: transposition of the great arteries alone, transposition with persistent ductus arteriosus, transposition with ventricular septal defect, transposition with ventricular septal defect and pulmonary ductus arteriosus, transposition with ventricular septal defect and pulmonary stenosis, transposition with pulmonary stenosis, transposition with coarctation of the aorta, and a miscellaneous group (one case only).

The arterial pressures were analysed by the $X^2$ test. In all cases the null hypothesis was retained when its probability exceeded 0.05.

Results

Systemic arterial oxygen saturation Fig. 1 shows the distribution of systemic arterial oxygen saturation of the whole group. Immediately before septostomy the distribution was bimodal (Fig. 1A); analysis of variance in the anatomical groups confirmed that more than one population was present ($P < 0.01$). Immediately after septostomy (Fig. 1B) the distribution was unimodal; analysis of variance confirmed this ($P > 0.05$). The mean value was 65 per cent with a standard deviation (SD) of 14. The later investigations showed a fall in arterial oxygen saturation of the group as a whole from a mean of 65 per cent to one of 59 per cent, SD 10. This represents a significant fall ($P = 0.013$).
The bimodal distribution of the presystostomy saturations made it impossible to compare the whole group before and after treatment. It seemed reasonable to divide the population into two groups, those with only a patent atrial septum and those with additional communications between the pulmonary and systemic circuits, such as ventricular septal defect or persistent ductus arteriosus.

Fig. 2A shows the breakdown of observations into these two groups before septostomy, the mean values being 43 per cent, SD 12, for the group with an atrial communication only, and 56 per cent, SD 12, for the group with additional communications. Immediately after septostomy the mean values were 62 per cent, SD 12, and 67 per cent, SD 12, respectively (Fig. 2B). These results show a statistically significant rise after septostomy, \( P < 0.001 \) in both instances. The late results (Fig. 2C) gave mean values of 58 per cent, SD 9, for the group with an atrial communication only, and 59 per cent, SD 11, for those with additional communications. Though lower than the immediate post-septostomy values, they are still significantly higher than their respective preoperative value, \( P < 0.005 \) in both cases.

**Interatrial mean pressure** These were grouped into the four classes shown in Table 1. The balloon septostomy series was compared with a control group of 47 patients with transposition of the great arteries and a known atrial septal defect, either surgical or congenital.

There was a significant difference between the mean atrial pressures before and after septostomy (\( P < 0.001 \)). The change was in the direction of equalizing the two atrial pressures. Both groups were significantly different from the control group. In the follow-up investigation atrial pressures also differed from the control (\( P < 0.02 \)), but not from the immediate post-septostomy group (\( P > 0.6 \)). There were some changes in atrial pressure in individual patients between the immediate and late postoperative studies.

**Relation of systemic arterial oxygen saturation to survival and to cerebrovascular accidents (Table 2)** Immediate post-septostomy results There was no difference between the hospital death group (those dying during the first admission) and the survivors (\( P > 0.05 \)). The late and total death groups had higher mean values than the survivors. It did not appear reasonable that a higher arterial oxygen saturation should adversely affect survival. For this reason 6 patients whose death was considered unavoidable and unrelated to their arterial oxygen saturation were withdrawn. These were 4 with preductal coarctation of the aorta, ventricular septal defect, and persistent ductus arteriosus; one who died from erosion of the pulmonary artery after banding and one who had a coronary artery embolus. The

**Table I** Mean pressure difference between left atrium (LA) and right atrium (RA)

<table>
<thead>
<tr>
<th></th>
<th>Before septostomy</th>
<th>After septostomy</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Immediate</td>
<td>Late</td>
<td></td>
</tr>
<tr>
<td>Class 4 (LA &gt; 3 mmHg higher than RA)</td>
<td>29</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Class 3 (LA 1–3 mmHg higher than RA)</td>
<td>24</td>
<td>17</td>
<td>9</td>
</tr>
<tr>
<td>Class 2 (LA up to 1 mmHg higher than RA)</td>
<td>13</td>
<td>18</td>
<td>12</td>
</tr>
<tr>
<td>Class 1 (LA not higher than RA)</td>
<td>8</td>
<td>34</td>
<td>29</td>
</tr>
<tr>
<td>Totals</td>
<td>74</td>
<td>74</td>
<td>53</td>
</tr>
</tbody>
</table>
Haemodynamic effects of balloon atrial septostomy in infants

![Graph showing Systemic Arterial Oxygen Saturation of Cases with Communication at atrial level only and additional communications.]

**TABLE 2** Systemic arterial oxygen saturation related to survival and to cerebrovascular accidents

<table>
<thead>
<tr>
<th></th>
<th>No. of cases</th>
<th>Mean per cent</th>
<th>Standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Immediate post-septostomy results</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital deaths</td>
<td>15</td>
<td>67%</td>
<td>14</td>
</tr>
<tr>
<td>Late deaths</td>
<td>15</td>
<td>70%</td>
<td>14</td>
</tr>
<tr>
<td>Total deaths</td>
<td>30</td>
<td>68%</td>
<td>15</td>
</tr>
<tr>
<td>Survivors</td>
<td>45</td>
<td>61%</td>
<td>10</td>
</tr>
<tr>
<td><strong>Late post-septostomy results</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths</td>
<td>8</td>
<td>53%</td>
<td>11</td>
</tr>
<tr>
<td>Survivors</td>
<td>38</td>
<td>60%</td>
<td>10</td>
</tr>
<tr>
<td><strong>Cerebrovascular accidents</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Immediate post-septostomy</td>
<td>10</td>
<td>71%</td>
<td>15</td>
</tr>
<tr>
<td>Late post-septostomy results</td>
<td>7</td>
<td>52%</td>
<td>7</td>
</tr>
</tbody>
</table>

Discussion

Balloon atrial septostomy causes a statistically significant rise in systemic arterial oxygen saturation. This is true for those cases with existing communications between the pulmonary and systemic circulations as well as those with only a persistent foramen ovale. Before septostomy two ‘populations’ of arterial oxygen saturation are discernible, but after septostomy there is only one. This suggests that, as a group, all patients after septostomy have similar capabilities for cross-flows between the two circulations. The immediate post-septostomy arterial oxygen saturations are higher than those measured later. Thus, though a rise in saturation above the immediate post-septostomy value (Rashkind and Miller, 1968) may occur in individual cases, in general it is likely that infants will become more cyanosed with time. The inter-atrial pressure difference is lowered by balloon septostomy, but this effect is not so uniform as that after operation. However, the immediate postoperative atrial pressures compare well with those measured later. This accord between the early and late atrial pressures and the presence of only one ‘population’ in the late arterial oxygen saturations suggest that the increase in cyanosis with time is not due, in the group as a whole, to inadequate septostomy. The individual changes in mean interatrial pressure differences and the fact that before septostomy 21 patients had little difference between the left atrial and right atrial mean pressures suggest that atrial pressure is, taken alone, not a suitable criterion when assessing the adequacy...
of the atrial septal defect. However, it is unlikely that an adequate defect is present if the atrial mean pressure is more than 3 mmHg higher than that in the right atrium (Shaher and Kidd, 1966).

The immediate post-septostomy results are similar to those reported by Rashkind and Miller (1968), but the arterial oxygen saturations are lower than those after surgical septostomy (Plauth et al., 1968; Shaher and Kidd, 1966; Deverall et al., 1969). In our Unit (Deverall et al., 1969) the mean late arterial oxygen saturation was 75 per cent. However, as reported previously (Tynan, 1971a), the mortality in that series was much higher than after the balloon technique. Our experience with the balloon followed by surgical septostomy suggests that the lower arterial oxygen saturations in this report are not due to an inadequate atrial septal defect (Tynan, 1971b).

It is somewhat surprising that no relation was shown between arterial oxygen saturation and survival. The probable relation between cerebrovascular accidents and low late arterial oxygen saturations is reasonable and underlines the unreliability of the immediate results in predicting the final outcome, both clinical and haemodynamic. This relation reinforces the need for early corrective surgery in transposition.

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Requests for reprints to Dr. Michael Tynan, Cardiovascular Department, Newcastle General Hospital, Westgate Road, Newcastle-upon-Tyne, NE4 6BE.
Haemodynamic effects of balloon atrial septostomy in infants with transposition of the great arteries.

M Tynan

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