Cardiac Performance and Mortality Early After Intracardiac Surgery in Infants and Young Children

By Grant V. S. Parr, M.D., Eugene H. Blackstone, M.D., and John W. Kirklin, M.D.

SUMMARY

One hundred thirty-nine infants and small children less than 48 months old were studied during the first 72 hours after intracardiac surgery for a variety of lesions. The hospital mortality rate was 19.4% (27 patients); 16 of those dying succumbed from acute cardiac failure. Deaths from acute cardiac failure were commonest in patients with low cardiac index (CI), and a continuous probability curve relates the two. The mean CI for all patients was 2.51 ± 0.794 l.min⁻¹.m⁻², and that for individual patients varied between 0.6 and 4.9. Cardiac output normalized by surface area (cardiac index) correlated weakly with age; cardiac output normalized by weight did not correlate with age. Mean mixed venous oxygen partial pressure (PvO₂) varied between 17 and 60 mm Hg, and the weighted mean for the group of 80 patients in whom it was obtained was 33.1 ± 6.57 mm Hg. CI and PvO₂ were only slightly related. Acute cardiac deaths occurred more frequently in patients with low PvO₂. Acute cardiac death was more reliably predicted using CI and PvO₂ together than either alone. The average of the mean arterial pressure was 80.5 ± 2.33 mm Hg, and this did not correlate with CI. Mean average systemic vascular resistance was 30.0 ± 8.395 SBU. We conclude that in such patients treatment should be directed toward keeping CI > 2.0 l.min⁻¹.m⁻² and PvO₂ > 30 mm Hg; that the possibility of maintaining adequate CI and PvO₂ is not related to the age of the patient but is related to the malformation treated; and that a therapeutic trial of reducing left ventricular afterload is indicated in some of these patients.

Additional Indexing Words:
Cardiac output 
Cardiac output normalization 
Mixed venous oxygen partial pressure 
Probability of an event 
Systemic vascular resistance

THE RECENT EMPHASIS on primary corrective intracardiac surgery in infants and young children has resulted in several series of such cases with good early results. Some early deaths occur, however, and in the case of a few malformations, the mortality rates have been disturbingly high.

Previous studies of cardiac performance early postoperatively in this age group have been limited to those of a few specific malformations. We have studied cardiac performance in 139 infants and small children undergoing a variety of intracardiac operations, and analyzed the deaths in these patients in detail. The results indicate the central role of inadequate cardiac performance in most of the hospital deaths.

Materials and Methods

Patients

In the 18 month period July 1, 1972, to December 31, 1973, 167 patients less than 48 months of age underwent definitive repair of a variety of types of congenital heart disease in our center. Cardiac output measurements were obtained during the first 72 hours after operation on 142 of these. Two patients, both survivors, were excluded from the present analysis because of residual shunts rendering the dye curves inadequate for measuring cardiac output. One patient who died after a single injection of a proven contaminated batch of indocyanine green was also excluded. The study group was thus 139 patients.

Twenty-eight of the patients were less than 6 months old, and 25 were between 6 and 12 months of age; thus 53 patients were less than one year old. Forty patients were between 12 and 24 months old, and 46 between 24 and 48 months of age.

Operative Techniques

Hypothermia to about 30°C C was induced by surface cooling prior to sternotomy in patients less than two years of age or 15 kg in weight. Two venous cannulae, hypothermia, and a period of low flow and occasionally total circulatory arrest were used except as indicated below. The longer the anticipated duration of the intracardiac procedure, the deeper was the hypothermia used and the longer the duration of the period of low flow. The temperature varied between 21°C and 28°C (nasopharyngeal) and the low flow periods from 50 to 10 minutes. Generally the heart was thoroughly cooled by the perfusate, and at times by external
cardiac cooling with ice-cold saline, and the repair done with a single period of aortic cross-clamping less than one hour in duration. In some infants less than 9 months of age or less than 6 kg, the alternative technique of profound hypothermia, limited cardiopulmonary bypass, and total circulatory arrest was employed. The foramen ovale was inspected and if patent, it was closed. The techniques of intracardiac repair varied according to the malformation.

**Postoperative Care**

During the first 18 hours after operation, blood was infused to keep left atrial mean pressure between 11 and 14 mm Hg. Packed red blood cells (100 ml·m⁻² body surface area) were administered hourly to patients with blood hemoglobin values less than 10.5 grams percent and low atrial pressures. Twenty-five percent albumin (20 ml·m⁻²) was administered hourly to such patients with hemoglobin greater than 15 grams percent. Other interventions were employed as indicated by the measured cardiac index, the mixed venous oxygen partial pressure (PvO₂), and other hemodynamic and metabolic determinations. Generally, when measured cardiac index was less than about 2 l·min⁻¹·m⁻², or PvO₂ below about 30 mm Hg, catecholamines were infused (52 patients). Atrial pacing was employed in ten patients to keep heart rate above 125 beats/min in infants and above 110 beats/min in the small children. Digoxin was employed in 39 patients and furosemide in 49. The patients remained intubated for varying periods postoperatively, usually about 36 hours. The majority of patients were maintained on a volume ventilator initially and then spontaneous breathing with continuous positive airway pressure was used for a period before extubation. No patients received tracheostomy. When bleeding was excessive, reoperation was performed, with the average interval between the initial operation and reoperation being 5.7 hours (11 patients).

**Study Methods**

Prior to sternotomy an 18 or 20 gauge teflon needle was inserted into the radial or brachial artery. Prior to the initiation of cardiopulmonary bypass a vinyl catheter was placed into the left atrium via the right superior pulmonary vein. At the conclusion of bypass a similar catheter was placed into the right atrium and in many patients a third catheter was placed into the pulmonary artery.

Cardiac output postoperatively was determined by the indicator-dilution technique, using in each instance the means of duplicate or triplicate measurements. All interventions needed by the patient, including catecholamines, were continued during the measurements. In all, 683 dye curves in duplicate or triplicate were obtained. The concentration of the indicator (indocyanine green) was 0.625 mg/ml for patients weighing less than or equal to 10 kg and 1.25 mg/ml for patients weighing between 10 and 20 kg. One ml of indicator solution was injected rapidly by hand through the catheter in the right atrium or pulmonary artery. Blood was simultaneously withdrawn at a rate of 7.64 ml/min from the radial or brachial artery through a cuvette densitometer by a constant rate pump. Five to seven ml of blood were withdrawn for each indicator dilution curve and the blood was not reinfused. Cardiac output calculations were performed on a digital computer using Sekelj, Tait, and Nathanson’s adaptation of the Stewart-Hamilton formula. Each curve was examined, and those without a good exponential decay of the indicator were rejected.

Arterial and left and right atrial pressures were continuously monitored, and measured at end-expiration. Pulmonary artery pressure was measured intermittently. The zero pressure reference point was midway between the anterior and posterior surfaces of the chest at the level of the manubrium.

Arterial blood pressure measurements immediately before and after indicator dilution curves were available in 76 of the last 80 patients, and were used to calculate the correlations with cardiac index. Systemic vascular resistance normalized to body surface area could be calculated in 72 of the 76 patients as the difference between mean arterial blood pressure and mean right atrial pressure divided by cardiac index, and was expressed as resistance units·m⁻² (SRU). The pressures used were the average of the determination of each immediately preceding and following the indicator dilution curve.

The body surface area (m²) was calculated from Boyd’s modification of the DuBois equation, using weight (W) in grams and height (H) in centimeters obtained one or two days before operation:

\[
BSA = 3.107 \cdot W^{0.385} \cdot H^{0.425}
\]

Heparinized arterial, and in the last 80 of the 139 patients, mixed venous blood (from pulmonary artery or right atrium), was analyzed immediately after withdrawal for pH, PO₂, PCO₂, hemoglobin, and oxygen saturation on automated assemblies. Samples of mixed venous blood were obtained immediately after cardiac output measurement in 419 of these 80 patients.

Oxygen consumption (VO₂) normalized to body surface area was calculated by the formula:

\[
VO₂ = CI [(SVO₂ - SvO₂) Hgb] \cdot 1.34 + 0.0032 (PvO₂ - Po₂)
\]

in which CI = cardiac index (l·min⁻¹·m⁻²); SVO₂ = arterial oxygen saturation (%); SvO₂ = mixed venous oxygen saturation (%); Po₂ = arterial partial pressure of oxygen (mm Hg); Pvo₂ = mixed venous partial pressure of oxygen (mm Hg); Hgb = hemoglobin (grams/100 ml).

The data were analyzed for means, standard deviation, and standard error of the mean. Unless otherwise specified, the data are presented as mean ± 1 standard deviation. For regression analyses the BMD02R Stepwise Regression computer program (forward stepwise with elimination) was employed using as its input time averaged data whenever multiple measurements were made. All regressions are presented as the intercept and the regression coefficient ± 1 standard error, the P value associated with the coefficient, and the standard error of the estimate (Sy.x). The interrelationship of simultaneously measured cardiac index, Pvo₂, SVO₂, and mean arterial blood pressures, and systemic vascular resistance was determined by weighted least squares regression, in which the number of observations was used to weight the average over all observations on a given patient. Since systemic vascular resistance is a ratio, the geometric mean was employed.

The records of all patients dying during the postoperative period were reviewed to determine inasmuch as possible the cause of death. A group of cases was easily identified, characterized by the sudden appearance of bradycardia progressing rapidly to cardiac asystole or ventricular fibrillation. Arterial blood gas abnormalities were not present and adequate ventricular filling pressures existed. Cardiac tamponade was not present. These patients were considered to
have died of acute cardiac failure. Other causes of death were relatively easily identified.

Probability of acute cardiac death, \( P(D|x_i) \), both uni- and multivariate, was generated by nonlinear weighted least squares fitting to the model:

\[
P(D|x_i) = (1 + e^a)^{-b}
\]

where \( Z = -\beta_0 - \beta_1 x_1 - \beta_2 x_2 \ldots - \beta_k x_k; \) \( \beta_i \) = regression coefficient fitted by the analysis; \( x_i \) = independent variable. Each value was weighted by the factor \( [P(1-P)]^{-1} \). Tests of the \( \beta \)'s for significance were by Student's \( t \) using the ratio of \( \beta \) to its standard deviation as \( t \). Two tests for lack of fit of the model to the data were performed: a Chi-square test and a test of observed versus calculated incidence of death. For the latter test, the patients were ranked according to calculated probability of death, then grouped in deciles for which a decile probability and the expected number of deaths (probability times number of patients in the decile) were calculated.

Decile probability = \( 1/(1 + e^\gamma) \)

\( \gamma_i = \ln[(1 - P_i)/P_i] \)

\( \gamma = \) average of \( \gamma_i \)

where \( P_i \) is the probability of death for each patient in the decile. The average value during the first 72 hours postoperatively for each patient was used when calculating the relation of cardiac index and \( \text{PVO}_2 \) to death from acute cardiac failure. In all analyses the natural logarithm (ln) of the variable was employed to constrain the model to positive numbers only.

**Results**

**Survival Data**

Twenty-seven of the 139 patients died postoperatively, a hospital mortality rate of 19.4%. The mortality according to age and procedure is shown in Table 1. (The miscellaneous procedures were repair in two patients, each of double outlet right ventricle with subpulmonary ventricular septal defect (VSD), VSD with pulmonic stenosis, and VSD and debranching of pulmonary artery; and in one patient each, of VSD with interrupted aortic arch, VSD and partial anomalous pulmonary venous connection, pseudotruncus arteriosus with pulmonary valve atresia using an outflow patch, tetralogy of Fallot with pulmonary valve incompetence using a heterograft valve in the outflow tract, banded pulmonary artery with spontaneously closed VSD, pulmonary venous stenosis, cor triatriatum, anomalous coronary artery, and mitral incompetence.) Since only 139 of the 167 patients operated upon during this period are included in the study group, the data do not indicate over-all mortalities for the various lesions and ages. In general, however, they are representative.

Sixteen (11.5%) of the 139 patients died of acute cardiac failure. This represented 59.3% of the 27 deaths. Fourteen of these patients died within 24 hours of operation, one during the second 24 hours, and one during the third 24 hours. Five patients (3.6% of the total group) were considered to have died from hemorrhagic pulmonary edema; left atrial pressures were below 15 mm Hg in all. Death in the other six patients dying of causes other than acute cardiac failure was considered to be preventable by present knowledge (Table 2).

Deaths from acute cardiac failure were commonest in patients with a low value for mean cardiac output early postoperatively (Table 2). The continuous probability curve of acute cardiac death versus cardiac index is shown in Figure 1.

**Cardiac Index**

The mean cardiac index for the first 72 postoperative hours was highly variable from patient to patient, despite use of a postoperative protocol designed to optimize preload, myocardial contractility, and heart rate during that time and despite a relatively standardized approach to myocardial preservation during surgery. Thus, the mean cardiac index of individual patients varied between 0.6 and 4.9 l/min-1·m-2, with the mean value for all 139 patients being 2.51 ± 0.774 l/min-1·m-2. For the 80 patients in whom \( \text{PVO}_2 \) and \( \text{SVO}_2 \) were also available, the mean value for cardiac index (2.49 ± 0.616 l/min-1·m-2) was not significantly different from that of the group as a whole.

![Graph](http://circ.ahajournals.org/Downloaded from http://circ.ahajournals.org)
### Table 1

**Mortality According to Age and Procedure**

<table>
<thead>
<tr>
<th>Operative procedure</th>
<th>Age &lt; 6 months</th>
<th></th>
<th>6 mos ≤ age &lt; 12 mos</th>
<th></th>
<th>12 mos ≤ age &lt; 24 mos</th>
<th></th>
<th>24 mos ≤ age &lt; 48 mos</th>
<th></th>
<th>Total</th>
<th>No.</th>
<th>Hosp. deaths</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Repair of aorto-pulmonary window</td>
<td>1</td>
<td>0</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Repair of atrial septal defect</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Repair of ventricular septal defect</td>
<td>3</td>
<td>1</td>
<td>8</td>
<td>2</td>
<td>10</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>24</td>
<td>3</td>
<td>12.5%</td>
<td></td>
</tr>
<tr>
<td>Insertion of valved external conduit</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>7</td>
<td>10</td>
<td>4</td>
<td>6</td>
<td>4</td>
<td>40.0%</td>
<td></td>
</tr>
<tr>
<td>Repair of complete A-V canal</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>7</td>
<td>2</td>
<td>0</td>
<td>6</td>
<td>4</td>
<td>66.7%</td>
<td></td>
</tr>
<tr>
<td>Repair of TGA + VSD + PS</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Repair of tetralogy of Fallot</td>
<td>2</td>
<td>1</td>
<td>4</td>
<td>0</td>
<td>14</td>
<td>1</td>
<td>28</td>
<td>3</td>
<td>10.7%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Repair of TGA with intact ventricular septum</td>
<td>2</td>
<td>0</td>
<td>8</td>
<td>2</td>
<td>6</td>
<td>0</td>
<td>7</td>
<td>0</td>
<td>23</td>
<td>2</td>
<td>8.7%</td>
<td></td>
</tr>
<tr>
<td>Repair of pulmonary stenosis</td>
<td>—</td>
<td>—</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>6</td>
<td>0</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Repair of TGA + PS</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Repair of aortic stenosis</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Repair of TGA + VSD</td>
<td>1</td>
<td>0</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>5</td>
<td>2</td>
<td>40.0%</td>
<td></td>
</tr>
<tr>
<td>Repair of miscellaneous lesions</td>
<td>6</td>
<td>4</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>3</td>
<td>0</td>
<td>6</td>
<td>15</td>
<td>5</td>
<td>33.3%</td>
<td></td>
</tr>
<tr>
<td>Repair of TAPVC</td>
<td>10</td>
<td>4</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>10</td>
<td>4</td>
<td>40.0%</td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>28</strong></td>
<td><strong>11</strong></td>
<td>25</td>
<td><strong>7</strong></td>
<td>40</td>
<td><strong>5</strong></td>
<td><strong>46</strong></td>
<td><strong>139</strong></td>
<td><strong>27</strong></td>
<td>19.4%</td>
<td>4.0%</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: TGA = transposition of the great arteries; VSD = ventricular septal defect; PS = pulmonary stenosis; TAPVC = total anomalous pulmonary venous connection.
Table 2
Mortality vs. Measured Cardiac Index

<table>
<thead>
<tr>
<th>Total cases</th>
<th>Total mortality</th>
<th>Acute cardiac death</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>No.</td>
<td>Percent</td>
</tr>
<tr>
<td>CI &lt; 1.0</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>1.0 ≤ CI &lt; 1.5</td>
<td>10</td>
<td>4</td>
</tr>
<tr>
<td>1.5 ≤ CI &lt; 2.0</td>
<td>18</td>
<td>4*</td>
</tr>
<tr>
<td>2.0 ≤ CI &lt; 2.5</td>
<td>42</td>
<td>10</td>
</tr>
<tr>
<td>2.5 ≤ CI &lt; 3.0</td>
<td>30</td>
<td>13</td>
</tr>
<tr>
<td>3.0 ≤ CI</td>
<td>33</td>
<td>15</td>
</tr>
<tr>
<td>Total</td>
<td>139</td>
<td>27</td>
</tr>
</tbody>
</table>

*1 patient died of massive hepatic necrosis (preventable)
†1 patient died of gram negative sepsis (preventable)
§3 patients died of hemorrhagic pulmonary edema
$3 patients died of hemorrhagic pulmonary edema
§1 patient died of transfusion reaction (preventable)

The mean values of cardiac index for patients grouped according to the operation performed (thus for the most part according to the malformation present) varied widely between the groups (table 3). Patients undergoing repair of aortopulmonary window and of atrial septal defect had, as a group, the highest cardiac indices for the 48 hour period, and patients undergoing repair of total anomalous pulmonary venous connection the lowest.

The effect of age on cardiac index early postoperatively for the group of 139 patients was not strong ($r = 0.355$). The weighted regression equation is $CI = 2.17 + [0.018 ± 0.004]$-Age (months), $P < 0.001$. The same statements are true for the correlation of cardiac index with weight ($CI = 1.976 + [0.063 ± 0.01826]$-weight, $P < 0.001$; $r = 0.283$) and height ($CI = 1.334 + [0.040 ± 0.0116]$-height, $P < 0.005$; $r = 0.281$). Cardiac output divided by weight (mean value for the 139 patients was $133 ± 41.4$ ml-ml min$^{-1}$kg$^{-1}$) did not correlate with age ($r = -0.001$) or height.

Mixed Venous Oxygen Levels

The mean $P_{O_2}$ during the first 72 hours postoperatively of the 80 individual patients in whom it was available varied between 17 and 60 mm Hg, and the weighted mean value for the group was $33.1 ± 6.57$ mm Hg. Correlation of the 419 simultaneous measurements of cardiac index and $P_{O_2}$ was weak ($r = 0.485$). The weighted regression equation is $CI = 0.914 + [0.0449 ± 0.00918]P_{O_2}$; $P < 0.00001$, weighted $S_{t-x} = 1.063$. Acute cardiac deaths occurred more frequently in patients with low $P_{O_2}$ compared with those with high $P_{O_2}$ (table 4 and fig. 2). Acute cardiac death was more reliably predicted when cardiac index and $P_{O_2}$ were used together (fig. 3). Table 5 summarizes the incidence of death from acute cardiac failure, comparing the observed incidence of such deaths and the incidence predicted by the probability model for patients grouped according to risk.

The weighted mean value of $S_{O_2}$ was 61.9 ± 10.62%. The relation of $S_{O_2}$ to cardiac index

Table 3
Cardiac Index According to Procedure

<table>
<thead>
<tr>
<th>Operative procedure</th>
<th>N</th>
<th>Mean value</th>
<th>sd</th>
<th>Mean value</th>
<th>sd</th>
</tr>
</thead>
<tbody>
<tr>
<td>Repair of aorto-pulmonary window</td>
<td>2</td>
<td>17.5</td>
<td>20.51</td>
<td>3.213</td>
<td>0.1676</td>
</tr>
<tr>
<td>Repair of atrial septal defect</td>
<td>3</td>
<td>34.0</td>
<td>14.00</td>
<td>2.181</td>
<td>0.0517</td>
</tr>
<tr>
<td>Repair of ventricular septal defect</td>
<td>24</td>
<td>13.9</td>
<td>9.17</td>
<td>2.801</td>
<td>0.8303</td>
</tr>
<tr>
<td>Insertion of valved external conduit</td>
<td>10</td>
<td>31.0</td>
<td>16.09</td>
<td>2.858</td>
<td>1.0581</td>
</tr>
<tr>
<td>Repair of complete A-V canal</td>
<td>6</td>
<td>14.8</td>
<td>8.35</td>
<td>2.740</td>
<td>1.1271</td>
</tr>
<tr>
<td>Repair of TGA + VSD + PS</td>
<td>1</td>
<td>13.0</td>
<td>2.555</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Repair of tetralogy of Fallot</td>
<td>28</td>
<td>24.5</td>
<td>14.74</td>
<td>2.453</td>
<td>0.5674</td>
</tr>
<tr>
<td>Repair of TGA with intact ventricular septum</td>
<td>23</td>
<td>18.9</td>
<td>12.53</td>
<td>2.438</td>
<td>0.5248</td>
</tr>
<tr>
<td>Repair of pulmonary stenosis</td>
<td>6</td>
<td>23.2</td>
<td>14.08</td>
<td>2.417</td>
<td>0.3537</td>
</tr>
<tr>
<td>Repair of TGA + PS</td>
<td>3</td>
<td>20.0</td>
<td>7.94</td>
<td>2.367</td>
<td>0.6465</td>
</tr>
<tr>
<td>Repair of aortic stenosis</td>
<td>3</td>
<td>2.8</td>
<td>2.26</td>
<td>2.295</td>
<td>0.7979</td>
</tr>
<tr>
<td>Repair of TGA + VSD</td>
<td>5</td>
<td>17.4</td>
<td>14.22</td>
<td>2.137</td>
<td>0.4639</td>
</tr>
<tr>
<td>Repair of miscellaneous lesions</td>
<td>15</td>
<td>2.2</td>
<td>1.36</td>
<td>2.027</td>
<td>0.8732</td>
</tr>
<tr>
<td>Repair of TAPVC</td>
<td>10</td>
<td>18.7</td>
<td>14.29</td>
<td>2.508</td>
<td>0.7739</td>
</tr>
<tr>
<td>Total</td>
<td>139</td>
<td>18.7</td>
<td>14.29</td>
<td>2.508</td>
<td>0.7739</td>
</tr>
</tbody>
</table>

Abbreviations: sd = standard deviation; se = standard error; TGA = transposition of the great arteries; VSD = ventricular septal defect; PS = pulmonary stenosis; TAPVC = total anomalous pulmonary venous connection.
Table 4  
Mortality vs. Mixed Venous PO₂

<table>
<thead>
<tr>
<th>PO₂ Range</th>
<th>Total Deaths</th>
<th>Acute cardiac death</th>
</tr>
</thead>
<tbody>
<tr>
<td>PO₂ &lt; 20</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>20 ≤ PO₂ &lt; 25</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>25 ≤ PO₂ &lt; 30</td>
<td>13</td>
<td>2*</td>
</tr>
<tr>
<td>30 ≤ PO₂ &lt; 35</td>
<td>34</td>
<td>16</td>
</tr>
<tr>
<td>35 ≤ PO₂</td>
<td>25</td>
<td>12</td>
</tr>
<tr>
<td>Total</td>
<td>80</td>
<td>14</td>
</tr>
</tbody>
</table>

*1 patient died of hemorrhagic pulmonary edema  
†1 patient died of hemorrhagic pulmonary edema  
3 patients died of hypoxemia  
1 patient died of gram negative sepsis  
‡2 patients died of hemorrhagic pulmonary edema  
1 patient died of transfusion reaction

was similar to that of PO₂; the weighted regression equation is CI = 0.757 + [0.0262 ± 0.0056]·SVR, P < 0.00002, weighted S_y,x = 1.075; r = 0.466.

Calculated Oxygen Consumption

The product of cardiac index and arteriovenous oxygen difference (calculated oxygen consumption) averaged 145 ± 39.3 l·min⁻¹·m⁻² for all patients during the 72-hour period. Its regression against cardiac index was not significant.

Systemic Arterial Pressures and Resistance

There was poor but significant correlation of the systolic pressure (SAP) with cardiac index (r = 0.260; CI = 1.135 + [0.0119 ± 0.0054]·SAP; P < 0.025). The average systemic arterial pressure of all patients was 107.2 ± 14.64 mm Hg. Mean arterial pressure (MAP) did not correlate with cardiac index. The average of the mean arterial pressures for all patients was 80.5 ± 2.53 mm Hg.

The mean of the average systemic vascular resistance of each patient was 30.0 ± 8.39 SRU. The resistance was highest in patients with low cardiac indices (ln(SRU) = 4.426 - [0.424 ± 0.0316]·CI; P < 0.000001; weighted S_y,x = 0.344).

Normalization of Cardiac Output

Apropos of normalization, the correlation coefficients and regression coefficients of cardiac output against body surface area, weight, height, and age are shown in figure 4.

Discussion

We have previously discussed the critical factors, accuracy, and reproducibility of the indicator-dilution method for estimating cardiac output in infants and small children. In this group of patients, cardiac

![Joint Probability of Acute Cardiac Death](image)

**Figure 3**

The probability of death from acute cardiac failure from the joint determinations of cardiac index and PO₂ in the last 80 patients is shown as isobars. Z = -2.038 + [3.481 ± 1.4922]·ln CI + [7.566 ± 2.4782]·[(ln PO₂) - 3]; P for the coefficient of CI < 0.025, for PO₂, P < 0.005. The Chi-square test for lack of fit of the model was not significant (x²(78) = 50, P > 0.98).

Circulation, Volume 51, May 1975
output correlated best with body surface area. In view of this, and the convention of normalizing cardiac output to body surface area, the data here have been presented for the most part as cardiac index.

Over half of the deaths in the patients studied were judged to be from what we have termed acute cardiac failure. The data indicate that the probability of this following intracardiac operations in infants and small children is greater than otherwise in patients whose cardiac index is less than about 2.0 l.min⁻¹.m⁻², and in those whose Pavo is less than about 30 mm Hg (the normal cardiac index in this age group is 4.15 ± 1.17² and normally Pavo is about 40 mm Hg). Although we have for several years been measuring these parameters, which cannot be predicted accurately from other more conventional observations and measurements, we had been uncertain as to the actual numerical values indicating special treatment. We believe that the data from this study and their conversion to the probability model shown in figures 1, 2, and 3 and table 5 indicate that pharmacologic and other types of intervention should be instituted in patients such as those studied when cardiac index is less than 2.0 l.min⁻¹.m⁻² or Pavo < 30 mm Hg, or if
both are available, when they together indicate a probability of death greater than 10% (fig. 3).

The possibility of maintaining adequate cardiac index and \( P_{\text{aO}_2} \) early postoperatively does not seem to be related to the age of the patient. From this standpoint, the inherent risk of intracardiac surgery does not seem to be greater in infants than in older children, and current experience in several centers supports this idea. The possibility of maintaining adequate cardiac index and \( P_{\text{aO}_2} \) early postoperatively does seem to be related to the malformation under treatment and thus to the operation itself. As examples, patients undergoing repair of aorto-pulmonary window have no ventriculotomy, have a left ventricle capable of generating a large stroke volume at a low left ventricular end-diastolic pressure, and tend to have good cardiac performance early postoperatively; and we have previously presented data suggesting that, following repair of total anomalous pulmonary venous connection, the left atrium, and following repair of transposition, the "right" atrium, is small and relatively noncompliant and may predispose to a low cardiac output.

Events in the operating room, including cardiopulmonary bypass and periods of induced myocardial ischemia, no doubt relate to the possibility of maintaining good cardiac output and mixed venous oxygen levels early postoperatively. As one example, previous studies in adult patients undergoing aortic valve replacement demonstrated a high incidence of myocardial necrosis early postoperatively. We have no reason to believe that this is not also occurring in infants with present techniques.

The interventions early postoperatively did not include manipulating arterial blood pressure to reduce left ventricular afterload, although mean arterial blood pressure and systemic vascular resistance were higher than the normal values of 60 to 80 mm Hg and 10 to 20 units-m\(^{-2}\) respectively for this age group. We have previously shown in some adults with low cardiac output and high mean arterial blood pressure that pharmacologically reducing arterial blood pressure increases cardiac output. This maneuver has also been shown to increase cardiac output in some patients with acute myocardial infarction.

We believe a trial of vasodilator therapy should be made in infants and small children with low cardiac output and high mean arterial blood pressure early postoperatively.

Acknowledgment

We wish to thank Drs. A. D. Pacifico, N. T. Kouchoukos and R. B. Karp for allowing patients operated upon by them to be included in the study; to acknowledge the assistance of Mr. Robert N. Brown in the data compilation and tabulation; and to thank Mrs. Anne McLeod for her editorial work.

References

Cardiac performance and mortality early after intracardiac surgery in infants and young children.
G V Parr, E H Blackstone and J W Kirklin

_Circulation._ 1975;51:867-874
doi: 10.1161/01.CIR.51.5.867

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1975 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
http://circ.ahajournals.org/content/51/5/867

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org//subscriptions/