Cardiac Performance after Open Intracardiac Surgery

By John W. Kirklin, M.D., and Richard A. Theye, M.D.

The hemodynamic state, blood gas levels, blood volumes, and residual shunts soon after repair of atrial septal defect, ventricular septal defect, and tetralogy of Fallot have been described in previous publications.1-4 Similar data have now been obtained after open operation for acquired disease of the aortic valve, and additional measurements of atrial pressures after repair of ventricular septal defect and tetralogy of Fallot have been made. The present paper represents an attempt to summarize all these data in such a way as to permit generalizations concerning the relation of various factors to cardiac output, heart rate, stroke volume, and atrial pressure (in a word, to cardiac performance) after open intracardiac surgery. The variability in the relations of cardiac output to the arteriovenous difference in oxygen content of blood and in the percentage of oxygen saturation of mixed venous blood becomes apparent from examination of these data and, as would be expected, is related to the variability in the metabolic rate and the amount of hemoglobin in the blood after operation. These studies and observations made by other investigators have led to the gradual evolution of methods of accomplishing the operation and postoperative care so as to maintain optimal cardiac performance and blood gas levels in such patients. The incidence of deaths from low cardiac output, a problem originally stressed by Boyd and associates,5 has thereby been decreased.

Methods

Methods of studying the hemodynamic state, blood gas levels, and blood volume were presented in previous publications.1-4, 6 Oxygen uptake and carbon-dioxide elimination were measured by Sturridge, Fowler, and ourselves by analysis of timed samples of expired air, and metabolic rate was derived from the nomogram of Boothby and associates.7

In parts of this analysis, the levels and relations of right and left atrial pressures (which in these patients are believed to approximate respective ventricular filling pressures) are used to identify situations in which conditions affecting primarily one ventricle may be responsible for limitation of cardiac output. Experimental observations of Berglund8 indicate that the relation between right and left ventricular filling pressures, at output equality and with the thorax and pericardium open, is determined by the relative contractility, distensibility, and arterial resistance of the two ventricles (fig. 1).

Cardiac Output

Cardiac output, in reality systemic blood flow since a correction was made for residual shunts, was variable during the first 3 postoperative days. It ranged from 1.6 to 5.5 liters per minute per square meter of body surface (based on about 300 observations in 47 patients). It averaged 3.2, 2.7, and 2.9 on postoperative days 1 (2 to 4 hours after operation), 2, and 3 respectively. This tendency for cardiac output to be lower on day 2 than on day 1, and higher on day 3 than 2, was observed in most individual patients and is believed to be related to changes in ventricular function relative to filling pressure.2 Cardiac output tended to be lower after repair of tetralogy of Fallot than after other types of open intracardiac operations, averaging 2.9, 2.2, and 2.5 liters per minute per square meter on days 1, 2, and 3, respectively. In the group as a whole there was no consistent relation between cardiac output and heart rate nor between heart rate and temperature or age of the patient.

Three of the 47 patients died of progressive decline in cardiac output. Two others died late on the third postoperative day with low but rising cardiac indices (averaging 2.0 on day 2 and 2.3 on day 3). A number of pa-

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patients with as low a cardiac output survived, indicating the complexities in judging the adequacy of a given value for cardiac output after operation.

**Factors Affecting Cardiac Performance Postoperatively**

**Anatomic Lesion**

Observations made during open intracardiac operations indicate that size of the cavity and thickness of the wall of one ventricle relative to the other vary according to the anatomic lesion, a fact which probably affects some aspects of cardiac performance after repair of the various lesions.

The relation between right and left atrial pressures (chest and pericardium open) differed after repair of uncomplicated atrial septal defect, uncomplicated ventricular septal defect, and calcific aortic stenosis (fig. 2).

The relation between output resistances of the two ventricles was normal and the output of the two ventricles equal after repair in the three groups. Some deductions as to relative right and left ventricular function (contractility and distensibility) after repair can therefore be made, using Berglund's concepts. The large differential pressure (left atrial pressure minus right atrial pressure) observed in patients after repair of atrial septal defect (larger than in normal dogs) seemed to result from increased right ventricular function relative to left. This probably was related to the large size of the cavity and the normal thickness of the wall (and consequent increased distensibility) of the right ventricle and the normal size of the left ventricle in cases of atrial septal defect. Differential pressure after repair of uncomplicated ventricular septal defect via the atrial approach seemed normal, a finding consistent with the increased size of the cavities and consequently greater distensibility of both ventricles in these patients. The abnormally small differential pressure often observed in such patients after repair via transverse ventriculotomy presumably results from selective impairment of right ventricular function secondary to transverse ventriculotomy. Differential pressure appears larger than normal after repair of a stenotic calcareous aortic valve. It is suggested that left ventricular distensibility relative to right may be decreased in these patients, presumably because of increased thickness of the wall and decreased size of the cavity of the left ventricle and normal size of the right ventricle.

After repair of tetralogy of Fallot (fig. 3), atrial pressure relations are particularly variable, probably because of the unusual lack of homogeneity in the relations between output resistances of the right and left ventricles, stroke volumes (because of incompetence of the pulmonary valve), and function. There is a suggestion that in patients in whom pericardial enlargement over the right ventricular outflow tract was not done, and in whom transverse ventriculotomy was used, different-

![Figure 1](http://circ.ahajournals.org/)

*Replot of data obtained by Berglund during experimental study of response of dog to rapid infusion of solution of dextrose or dextran. When the pericardium was open, left atrial pressure rose more rapidly than right, resulting in increasing Δ pressure (left atrial pressure minus right atrial pressure) as right atrial pressure increased. Δ Pressure was less marked at high right atrial pressures when pericardium was closed.*

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Figure 2

Relations of Δ pressures to right atrial pressures in operating room (thorax and pericardium open): a. after complete repair of atrial septal defect in patients without significant pulmonary vascular disease, b. after complete repair of ventricular septal defect in patients without significant pulmonary vascular disease, and c. after replacement of aortic valve with three Bahnson cusps in cases of calcareous aortic valve stenosis. Wide variation in atrial pressures resulted from rapid augmentation of blood volume by intra-arterial infusion of heparinized blood immediately after cardiopulmonary bypass and subsequent changes related to factors enumerated in text.

A comparison of cardiac output and stroke volume at various atrial pressures after repair of atrial septal defect by a variety of methods (including the atrial well) gave only equivocal evidence of a deleterious effect of normothermic cardiopulmonary bypass (or of 30 minutes of myocardial ischemia with the heart thoroughly cooled) on ventricular function. Data from a patient whose ventricular septal defect could be closed digitally...
Prior to operative repair (table 1) suggest immediate mild depression of ventricular function by this operative procedure with the ventriculotomy probably being the major etiologic factor. Cardiac output in the operating room after repair of a stenotic calcareous aortic valve was the same or higher than immediately before the repair (table 2). Estimations of stroke work, admittedly unreliable in this situation where heart rate is variable and the possibility of regurgitant flow through the aortic valve not eliminated, and comparison of this with atrial pressures suggest some reduction in ventricular function immediately after this type of intracardiac procedure.

Ventricular-Outflow Resistance

A previous publication\(^2\) tabulated the data showing that patients with postoperative elevation of pulmonary arterial pressure secondary to pulmonary vascular disease had lower cardiac outputs and higher right atrial pressures than did those without significant pulmonary vascular disease. This was true whether the defect repaired was atrial septal defect, total anomalous pulmonary venous connection, or ventricular septal defect. In these patients, there is in general an inverse relation between ventricular-outflow resistance and cardiac output, probably because of the limitation in cardiac adaptability resulting from decreased ventricular function.

Table 1

<table>
<thead>
<tr>
<th>Condition</th>
<th>Cardiac index, L./min./M.(^2)</th>
<th>Heart rate, beats/min.</th>
<th>Stroke index, ml./M.(^2)</th>
<th>Atrial pressure mm. Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Right</td>
</tr>
<tr>
<td>Closed digitally(^1)</td>
<td>3.1</td>
<td>80</td>
<td>39</td>
<td>6</td>
</tr>
<tr>
<td>75 Minutes postrepair</td>
<td>3.1</td>
<td>80</td>
<td>38</td>
<td>10</td>
</tr>
<tr>
<td>85 Minutes postrepair</td>
<td>3.6</td>
<td>80</td>
<td>45</td>
<td>11</td>
</tr>
</tbody>
</table>

\(^a\)Repair made through transverse ventriculotomy with heart thoroughly cooled and ischemic for 25 minutes, during hypothermic, whole-body perfusion.

\(^1\)Defect temporarily closed digitally while circulation was intact.
Table 2
Observations Made before and after Replacement of Calcified Stenotic Aortic Valve with Three Bahnson Cusps (Thorax and Pericardium Open)*

<table>
<thead>
<tr>
<th>Case</th>
<th>Condition</th>
<th>Cardiac index, L./min./M.²</th>
<th>Heart rate, Stroke index, ml./M.²</th>
<th>Pressures mm. Hg</th>
<th>Radial artery L. ventricle (systolic)</th>
<th>Atrium</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Before repair</td>
<td>3.1</td>
<td>54</td>
<td>58</td>
<td>84/48</td>
<td>162</td>
</tr>
<tr>
<td></td>
<td>25 Minutes after repair</td>
<td>3.1</td>
<td>72</td>
<td>43</td>
<td>76/40</td>
<td>84</td>
</tr>
<tr>
<td>2</td>
<td>Before repair</td>
<td>1.8</td>
<td>60</td>
<td>30</td>
<td>104/44</td>
<td>189</td>
</tr>
<tr>
<td></td>
<td>40 Minutes after repair</td>
<td>2.5</td>
<td>78</td>
<td>32</td>
<td>152/69</td>
<td>152</td>
</tr>
<tr>
<td>3</td>
<td>Before repair</td>
<td>2.2</td>
<td>66</td>
<td>33</td>
<td>68/40</td>
<td>165</td>
</tr>
<tr>
<td></td>
<td>105 Minutes after repair</td>
<td>2.1</td>
<td>90</td>
<td>23</td>
<td>103/62</td>
<td>114</td>
</tr>
<tr>
<td>4</td>
<td>Before repair</td>
<td>2.2</td>
<td>60</td>
<td>37</td>
<td>98/87</td>
<td>184</td>
</tr>
<tr>
<td></td>
<td>5 Minutes after repair</td>
<td>3.7</td>
<td>70</td>
<td>53</td>
<td>100/51</td>
<td>35</td>
</tr>
<tr>
<td></td>
<td>15 Minutes after repair</td>
<td>3.5</td>
<td>66</td>
<td>53</td>
<td>99/51</td>
<td>102</td>
</tr>
</tbody>
</table>

*Operation included hypothermic whole-body perfusion for about 100 minutes, direct perfusion of left and right coronary arteries, and total replacement of aortic valve.

Table 3
Response to Rapid Intra-arterial Infusion of Heparinized Blood from Pump-Oxygenator Immediately after Cardiopulmonary Bypass (Thorax and Pericardium Open)

<table>
<thead>
<tr>
<th>Case</th>
<th>Lesion repaired</th>
<th>Amount of blood transfused, ml./M.²</th>
<th>Right atrial pressure, mm. Hg</th>
<th>Δ Pressure,*</th>
<th>Cardiac index, L./min./M.²</th>
<th>Heart rate, beats/min.</th>
<th>Stroke index ml./M.²</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A.S.D.†</td>
<td>1000</td>
<td>6</td>
<td>+ 1</td>
<td>2.2</td>
<td>96</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>14</td>
<td>+13</td>
<td>4.9</td>
<td>76</td>
<td>62</td>
</tr>
<tr>
<td>2</td>
<td>A.S.D. with moderate pulmonary vascular disease</td>
<td>400</td>
<td>7</td>
<td>+ 9</td>
<td>2.4</td>
<td>84</td>
<td>28</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>12</td>
<td>+ 9</td>
<td>2.5</td>
<td>72</td>
<td>34</td>
</tr>
<tr>
<td>3</td>
<td>A.S.D.</td>
<td>325</td>
<td>7</td>
<td>+ 7</td>
<td>2.5</td>
<td>90</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>8</td>
<td>+13</td>
<td>2.5</td>
<td>78</td>
<td>32</td>
</tr>
<tr>
<td>4</td>
<td>V.S.D.†</td>
<td>400</td>
<td>8</td>
<td>+ 2</td>
<td>2.5</td>
<td>90</td>
<td>36</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>12</td>
<td>+ 5</td>
<td>3.7</td>
<td>90</td>
<td>46</td>
</tr>
<tr>
<td>5</td>
<td>V.S.D.</td>
<td>700</td>
<td>12</td>
<td>+ 3</td>
<td>2.7</td>
<td>70</td>
<td>38</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>16</td>
<td>+ 5</td>
<td>3.9</td>
<td>80</td>
<td>48</td>
</tr>
<tr>
<td>6</td>
<td>V.S.D.</td>
<td>350</td>
<td>15</td>
<td>0</td>
<td>4.9</td>
<td>96</td>
<td>57</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>17</td>
<td>0</td>
<td>5.6</td>
<td>110</td>
<td>57</td>
</tr>
<tr>
<td>7</td>
<td>V.S.D.</td>
<td>350</td>
<td>9</td>
<td>+ 8</td>
<td>2.8</td>
<td>66</td>
<td>42</td>
</tr>
<tr>
<td>8</td>
<td>Calcareous aortic valve stenosis</td>
<td>400</td>
<td>14</td>
<td>+10</td>
<td>2.9</td>
<td>69</td>
<td>48</td>
</tr>
<tr>
<td>9</td>
<td>Calcareous aortic valve stenosis</td>
<td>400</td>
<td>15</td>
<td>+ 5</td>
<td>2.6</td>
<td>66</td>
<td>39</td>
</tr>
</tbody>
</table>

*Δ Pressure = left atrial pressure minus right atrial pressure.
†A.S.D. = atrial septal defect; V.S.D. = ventricular septal defect.

Postperfusion Transfusion
Rapid augmentation of blood volume by the intra-arterial infusion of heparinized blood shortly after termination of cardiopulmonary bypass usually resulted in increased atrial pressures, cardiac output, and stroke.

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Table 4
Response to Intravenous Infusion of Citrated Blood in the Postoperative Period

<table>
<thead>
<tr>
<th>Case</th>
<th>Lesion repaired</th>
<th>Amount of blood transfused, ml/M.²</th>
<th>Right atrial pressure, mm. Hg</th>
<th>Δ Pressure, mm. Hg</th>
<th>Cardiac index, L./min./M.²</th>
<th>Heart rate, beats/min.</th>
<th>Stroke index ml./M.²</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A.S.D.*</td>
<td></td>
<td>7</td>
<td>0</td>
<td>3.7</td>
<td>114</td>
<td>32</td>
</tr>
<tr>
<td></td>
<td>Day 1</td>
<td>180</td>
<td>8</td>
<td>0</td>
<td>3.7</td>
<td>108</td>
<td>34</td>
</tr>
<tr>
<td></td>
<td>Day 2</td>
<td>180</td>
<td>9</td>
<td>+1</td>
<td>3.0</td>
<td>108</td>
<td>28</td>
</tr>
<tr>
<td>2</td>
<td>A.S.D.</td>
<td></td>
<td>190</td>
<td>5</td>
<td>+2</td>
<td>108</td>
<td>19</td>
</tr>
<tr>
<td></td>
<td>Day 1</td>
<td>320</td>
<td>7</td>
<td>3</td>
<td>+1</td>
<td>114</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td>Day 2</td>
<td>320</td>
<td>-1</td>
<td>+1</td>
<td>2.4</td>
<td>108</td>
<td>22</td>
</tr>
<tr>
<td>3</td>
<td>A.S.D.</td>
<td></td>
<td>190</td>
<td>5</td>
<td>+4</td>
<td>108</td>
<td>37</td>
</tr>
<tr>
<td></td>
<td>Day 1</td>
<td>190</td>
<td>5</td>
<td>+4</td>
<td>5.3</td>
<td>102</td>
<td>52</td>
</tr>
<tr>
<td></td>
<td>Day 3</td>
<td>95</td>
<td>7</td>
<td>+3</td>
<td>4.0</td>
<td>96</td>
<td>41</td>
</tr>
<tr>
<td>4</td>
<td>T.A.P.V.C.†</td>
<td></td>
<td>85</td>
<td>5</td>
<td>+4</td>
<td>3.7</td>
<td>96</td>
</tr>
<tr>
<td></td>
<td>Day 1</td>
<td>12</td>
<td>5</td>
<td>+4</td>
<td>3.8</td>
<td>90</td>
<td>42</td>
</tr>
<tr>
<td>5</td>
<td>T.A.P.V.C. Severe pulmonary vascular disease</td>
<td>160</td>
<td>12</td>
<td>-1</td>
<td>2.0</td>
<td>138</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>Day 1</td>
<td>160</td>
<td>15</td>
<td>-1</td>
<td>2.0</td>
<td>108</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>Day 2</td>
<td>160</td>
<td>9</td>
<td>+1</td>
<td>2.1</td>
<td>126</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>Day 3</td>
<td>85</td>
<td>13</td>
<td>-2</td>
<td>2.6</td>
<td>126</td>
<td>21</td>
</tr>
<tr>
<td>5</td>
<td>V.S.D.*</td>
<td></td>
<td>85</td>
<td>7</td>
<td>-3</td>
<td>126</td>
<td>32</td>
</tr>
<tr>
<td></td>
<td>Day 1</td>
<td></td>
<td>10</td>
<td>-5</td>
<td>4.8</td>
<td>130</td>
<td>37</td>
</tr>
<tr>
<td>7</td>
<td>V.S.D.</td>
<td></td>
<td>12</td>
<td>+2</td>
<td>4.1</td>
<td>100</td>
<td>41</td>
</tr>
</tbody>
</table>

*See footnotes to table 3.
†T.A.P.V.C. = total anomalous pulmonary venous connection.

Table 5
Comparison of Hemodynamic State in Operating Room and in Early Postoperative Period in 17 Patients Operated on for Various Conditions

<table>
<thead>
<tr>
<th>Condition</th>
<th>Right atrial pressure, mm. Hg</th>
<th>Δ Pressure,* mm. Hg</th>
<th>Cardiac index, L./min./M.²</th>
<th>Heart rate, beats/min.</th>
<th>Stroke index ml./M.²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Just prior to closure of chest</td>
<td>11</td>
<td>+1</td>
<td>3.0</td>
<td>88</td>
<td>32</td>
</tr>
<tr>
<td>(pericardium open)</td>
<td>(7 to 16)</td>
<td>(−2 to +7)</td>
<td>(1.7 to 4.2)</td>
<td>(66 to 120)</td>
<td>(16 to 45)</td>
</tr>
<tr>
<td>2 Hours postoperatively</td>
<td>6</td>
<td>+1</td>
<td>3.1</td>
<td>114</td>
<td>28</td>
</tr>
<tr>
<td></td>
<td>(−1 to 12)</td>
<td>(−2 to +5)</td>
<td>(2.1 to 4.1)</td>
<td>(78 to 140)</td>
<td>(17 to 41)</td>
</tr>
</tbody>
</table>

*See footnote to table 3.

volume and decreased heart rate, except after repair of a stenotic calcareous aortic valve (table 3). Data in cases 2 and 3 suggest that during the late stages of the infusion the rise in cardiac output is less than that of stroke volume and of atrial pressures, particularly of the left atrium. The increased cardiac output with rapid infusion of blood under these circumstances probably is a response to increased ventricular diastolic volume resulting from increased filling pressure. The lack of response of cardiac output to increased filling.

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pressure after repair of calcific aortic stenosis may be due to the limited distensibility of the markedly hypertrophied left ventricle.

Postoperative Transfusion

Postoperatively, the rapid infusion intravenously of smaller amounts of citrated blood, when atrial pressures were low, resulted in elevation of cardiac output and of right and left atrial pressures. When right atrial pressure was between 5 and 12 mm. of mercury, prior to transfusion, the increase in cardiac output occurred in about half of the observations (table 4). Heart rate was generally unchanged or slightly reduced.

Stage of Convalescence

Cardiac output in the operating room, with chest and pericardium open, varied after repair. The atrial pressures usually declined gradually from the peak values existing after augmentation of blood volume immediately after cardiopulmonary bypass. Improving myocardial function, incomplete replacement of blood lost, variations in the levels of anesthetic gases, and increasing distensibility of the venous bed probably contribute to these changes in atrial pressure and cardiac output. During the first few postoperative hours, cardiac output is usually about the same or higher than during the operation, pulse rate is usually more rapid, and observed atrial pressures are lower (table 5). The lower level of observed atrial pressures postoperatively seems related in large part to the negative intrathoracic pressure prevailing at that time. Data on changes in effective (or transmural) atrial pressure between the time of operation and the early postoperative period are not available.

It was indicated previously that cardiac output generally was lower on postoperative day 2 than on day 1, and higher on day 3 than on day 2. Changes in atrial pressures were usually inverse to those in cardiac output. Changes in blood volume did not parallel changes in atrial pressures (table 6) and are therefore not etiologic to them. Arterial blood gas levels and ventricular-outflow resistance were relatively steady throughout the 3-day period. Arterial blood pH was usually about 7.28 on day 1 and near normal on day 2 and day 3. It is suggested then that the phasic changes in cardiac output and atrial pressures are primarily related to changes in ventricular function as a result of local or neurohumoral factors, rather than to the variables mentioned above.

Ventilatory Mixture and Assisted Ventilation

No consistent change in cardiac output was noted when the breathing of an oxygen-enriched atmosphere was changed to the breathing of room air. Similarly, no consistent change was observed during the postoperative period when patients were switched from intermittent, positive-pressure breathing to spontaneous respiration.

Digitalis

Only two observations pertinent to the effect of digitalis on cardiac performance after surgery are available from this study. In a patient with ventricular septal defect and severe pulmonary vascular disease, there was no evidence of increase in cardiac output after administration of lanoxin, but atrial pressures were temporarily lowered. In another patient with total anomalous pulmonary venous connection and severe pulmonary vascular disease, cardiac output rose and pulse rate and atrial pressures fell after administration of the drug. These observations are in accord with the clinical impression of variability of response to digitalis in patients with sinus or nodal rhythm.

Table 6

<table>
<thead>
<tr>
<th>Case</th>
<th>Day</th>
<th>Blood volume, ml.</th>
<th>Cardiac index, L/min./M^2</th>
<th>Right atrial pressure, mm. Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1 (OR)</td>
<td>2460</td>
<td>3.8</td>
<td>7</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
<td>2490</td>
<td>5.5</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>2140</td>
<td>3.8</td>
<td>8</td>
</tr>
<tr>
<td>2</td>
<td>1 (OR)</td>
<td>3090</td>
<td>3.7</td>
<td>12</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
<td>3300</td>
<td>4.2</td>
<td>3</td>
</tr>
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Adequacy of Cardiac Output

Survival through the postoperative period is determined by many factors, a circumstance which makes difficult the identification of an exact value for a cardiac output which is adequate. Adequacy might be considered in terms of the level of oxygen in the tissue, as it is reflected in the oxygen saturation of mixed venous blood. However, the latter is also related to the oxygen content of arterial blood, its hemoglobin content, and the metabolic rate, all of which may change rapidly in these patients.

Postoperatively, the arteriovenous difference in oxygen content of the blood and cardiac output are, in general, inversely related, but the relation is not close (fig. 4). This is because of the wide variability in metabolic rate in these postoperative patients, a situation identified by the observations of Sturridge. Variability in the relation of cardiac output to the oxygen saturation of mixed venous blood is shown in figure 5. It is noteworthy that an oxygen saturation of 65 per cent was observed in mixed venous blood when cardiac indices varied from 1.6 to 4.5 liters per minute per square meter. In one patient subjected to total replacement of the aortic valve, oxygen saturation of mixed venous blood and cardiac index were 36 per cent and 3.8, respectively, on day 1, and 59 per cent and 2.3 on day 2.

Discussion

Understanding of the factors relating to cardiac performance after intracardiac surgery has allowed greater success in the prevention and treatment of low cardiac output after intracardiac surgery. Blood left in the pump-oxygenator is infused intra-arterially immediately after cardiopulmonary bypass, in increments of 50 to 100 mL, until the mean pressure in either the right or left atrium reaches 22 to 25 mm. of mercury. Cardiac output is usually brought to a maximum by this maneuver, and later in the operation little or no citrated homologous blood is administered except under circumstances of unusual blood loss. Atrial pressures gradually fall as the operation is being completed. Soon after surgery it seems prudent to maintain atrial pressures at about 10 to 14 mm. of mercury by administration of blood, unless the hemodynamic state is excellent at lower atrial pressures. No objective or clinical evidence of late desequestration and resultant hypervolemia has been observed. If atrioventricular valves are normal, only right atrial pressure need be monitored postoperatively.
since left atrial pressure is usually about 2
mm. and never more than 6 mm. of mercury
higher than right atrial pressure under these
circumstances.

Use of transverse ventriculotomy, preven-
tion of coronary air embolism, and protec-
tion by cold of the myocardium during
ischemia can be expected to minimize postop-
erative depression of myocardial function. The
volume of homologous blood used for priming
of the pump-oxygenator should be kept to a
minimum, in view of the possible ill effects
of large amounts of homologous blood. Con-
duct of the perfusion and operation so as to
minimize metabolic acidosis and the amount
of citrated blood administered after repair
should be beneficial to ventricular function.
Complete repair of the defect and avoidance
of heart block are obviously advantageous
to cardiac performance.

When cardiac performance is poor soon
after repair, NaHCO₃ (2 mEq. per kilogram
of body weight) is indicated, and usually
seems to improve cardiac output. Intracardiac
or intravenous injection of CaCl₂ has
an immediate although transient beneficial
effect on cardiac performance and is of value,
particularly in the operating room or when
large amounts of citrated blood have been
administered. In the postoperative patient,
digitalis is indicated when cardiac output is
low, even though its effectiveness under these
circumstances in man seems variable.

The importance of maintaining the oxygen
content of tissue at relatively high levels as
reflected in the oxygen saturation of mixed
venous blood is apparent. Avoidance of hyper-
metabolism, as much as possible, and atten-
tion to cardiac output, the level of hemoglobin
in the blood, and the level of arterial oxygen
are important in achieving this. Oxygen sat-
uration of arterial blood is usually brought
to a level of 98 per cent or more by simply
having the patient breathe oxygen-enriched
air. However, the studies of Dammann and
associates suggest that abnormally high lev-
els of oxygen in arterial blood (pO₂ of 200
to 300 mm. of mercury) may be advantageous
to cardiac performance after certain types
of intracardiac surgery. In view of this and
in order to reduce metabolic rate by reduc-
tion of work of breathing, tracheostomy
and intermittent positive-pressure breathing
(FI₀₂ 1.0) must be considered in patients
whose cardiac performance is unsatisfactory.

**Summary**

Cardiac output and related variables were
measured in patients after open intracardiac
surgery. Factors affecting cardiac perform-
ance during this period were the original
anatomic lesions, the details of the opera-
tive procedure, ventricular-outflow resistance,
transfusion of blood, the stage of convales-
cence, and digitalis. In postoperative patients,
metabolic rate, level of hemoglobin, and level
of oxygen in arterial blood varied more wide-
ly and acutely than in nonsurgical patients,
resulting in a less predictable relation be-
tween cardiac output and oxygen saturation
of mixed venous blood.

Therapeutic applications of these observa-
tions were discussed.

**Acknowledgment**

The contribution of Dr. Ara Doumanian in making
additional atrial pressure measurements after repair
of tetralogy of Fallot is appreciated. Remarks on
metabolic rate were included with the kind per-
mission of Doctor Marvin Sturridge. His findings
are the subject of a paper now in preparation
by Sturridge, Theye, Fowler, and Kirklin.

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Aortic Incompetence

In 1715 Raymond de Vieussens (1641-1716) gave an account of the morbid changes and the character of the pulse in a patient with this valvular lesion, William Cooper having in 1703 described the change in the valves. The Royal College of Surgeons' Museum contains a specimen of aortic incompetence described by John Hunter. In 1829 Thomas Hodgkin (1798-1866) published a modest reference to "retroversion of the aortic valves," disclaiming any originality and saying that C. Aston Key had drawn his attention to it; Laennec and Bertin had indeed described this retroversion of the aortic valves. Hodgkin gave a good pathological and clinical account of the disease, noting the murmurs, the dilatation and hypertrophy of the heart, and the arterial pulsation, but did not anticipate Corrigan in his more complete account. Hodgkin's contribution would have been even more completely forgotten had it not been for the dutiful loyalty of Samuel Wilks and William Hale-White. In 1832 Hope described the jerking character of the pulse in cases combined with adherent pericardium and other lesions. Soon after this Dominie Corrigan (1802-80) independently published his paper "On permanent Patency of the Mouth of the Aorta, or Inadequacy of the Aortic Valves" (1832), which has perpetuated his name in Corrigan's pulse. Sir Humphry Davy Rolleston The Harveian Oration. Great Britain, Cambridge University Press, 1928, p. 44.
Cardiac Performance after Open Intracardiac Surgery
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