Criteria for Two-Staged Arterial Switch Operation for Simple Transposition of Great Arteries

Makoto Nakazawa, MD, Kotaro Oyama, MD, Yasuharu Imai, MD, Keiko Nojima, MD, Hiroyuki Aotsuka, MD, Gengi Satomi, MD, Hiromi Kurosawa, MD, and Atsuyoshi Takao, MD

We analyzed hemodynamic variables in patients with transposition of the great arteries with intact ventricular septum (simple transposition) after pulmonary artery banding and Blalock-Taussig anastomosis to establish the criteria for two-staged arterial switch operation. The study included 35 patients who underwent the switch operation after banding and anastomosis; two died shortly after surgery, and one died of tachycardia 31 days after the operation. The left-to-right ventricular pressure ratio was above 0.83 in all surviving patients, 0.68 and 0.63 in the two operative deaths, and 0.84 in the other death. In the 32 surviving patients, angiographically determined left ventricular (LV) end-diastolic volume and ejection fraction averaged 147 ± 46(SD)% of normal and 0.64 ± 0.12, respectively. We also measured LV mass using angiographic LV semiaxes and end-diastolic LV posterior wall thickness obtained from an echocardiogram. These variables of the patients who died were not different from those of the surviving patients. The wall thickness was greater than 4 mm in the surviving patients, while it was 3.5 mm in the two patients who died early and 5 mm in the patient who died later. From the semiaxes, the wall thickness, and aortic diastolic pressure, all of which were obtained before the anatomic repair, we calculated the predictive LV wall stress. This wall stress would indicate the LV wall stress at the time of aortic valve opening immediately after the arterial switch operation. Predictive wall stress was less than 120 × 10^3 dynes/cm^2 in all but one of the surviving patients, whereas it was 151, 153, and 186 × 10^3 dynes/cm^2 in the three patients who died. From these data, we propose the following criteria for the safe two-staged arterial switch operation: left-to-right ventricular pressure ratio greater than 0.85; LV end-diastolic volume greater than 90% of normal; LV ejection fraction greater than 0.5, or it may be greater than 0.4; posterior wall thickness greater than 4 mm or very safely greater than 4.5 mm; and the predictive wall stress less than 120 × 10^3 dynes/cm^2. (Circulation 1988;78:124–131)

With regard to postoperative physiology, it is now increasingly accepted that the arterial switch operation is better than the atrial switch procedures in the surgical management of transposition of the great arteries (TGA). The basic concept is that the left ventricle (LV) supports the systemic circulation after the arterial switch operation; thus, systemic ventricular function should be normal in those patients, whereas the right (systemic) ventricular (RV) function is known to be compromised after the atrial switch operation.1–4 In fact, it has been reported that LV function is normal or near normal in patients after the arterial switch operation.4–7

The arterial switch operation was initially applied to TGA with a ventricular septal defect because the arterial switch operation resulted in a high mortality in this type of TGA and because the LV is unquestionably suitable for supporting the systemic circulation immediately after the repair. Recently, several reports have indicated that the anatomic repair is feasible in TGA with intact ventricular septum (simple TGA) with an acceptably low mortality during the neonatal period when LV pressure is still high.8–10

It is obvious that the arterial switch operation cannot be applied in patients whose LV pressure is
low after pulmonary artery pressure decreases beyond the neonatal period. In such patients, Yacoub et al\textsuperscript{11} performed pulmonary artery banding for the purpose of elevating LV pressure to the systemic level and for the arterial switch operation later. Even when the switch operation is feasible in the neonatal period, this two-staged procedure is still an important choice of surgical management of simple TGA in patients who are to be treated beyond the neonatal period. In this approach, it is mandatory that the LV is prepared suitably to support the high-pressure systemic circulation at the time of the anatomic repair. The details of well-prepared LV, however, have not been well defined.\textsuperscript{12,13} Therefore, the purpose of the present study was to investigate the relation between preoperative hemodynamic data and surgical outcome and to establish criteria of two-staged arterial switch operation for simple TGA after the palliation.

**Patients and Methods**

**Surgical Protocol**

Our current protocol for surgically managing simple TGA beyond the neonatal period is that pulmonary artery banding is performed at 2–3 months of life (or as soon as possible after patients have been referred to us later than that age) with Blalock-Taussig anastomosis, which is followed by the Lecompte modification of the Jatene procedure\textsuperscript{14} 1–4 months later or when LV pressure reaches a satisfactory level (see the later discussion). We have found that the palliation is most effectively and easily performed within this age range, probably because the cardiovascular system has completed the perinatal transition from the fetal to adult circulation. At the time of palliation, the shunt operation is now instituted first, which is followed by artery banding. When first developing this protocol, the pulmonary artery was banded first, but because the patients developed very severe hypoxemia and metabolic acidosis that resulted in profound circulatory insufficiency, we were forced to loosen the banding. After this experience, we have adopted the current protocol.

Our previous echocardiographic study\textsuperscript{15} suggests that the anatomic repair could be performed as early as 1 month after palliation if the criteria are fulfilled. The limited capacity of our ward and operating rooms, however, does not enable us to follow this time schedule. Nevertheless, the longer interval between the palliation and the anatomic repair has not proven to be adverse in any way, unless LV pressure becomes excessively high after pulmonary artery banding, which will be discussed later. Obviously, balloon atrioseptostomy is to be performed during the neonatal period in all patients.

**Patients**

Since we started the arterial switch operation in August 1982, 42 patients with simple TGA have been treated under the protocol described above, and by the end of June 1987, 36 patients had undergone the arterial switch operation. The remaining six patients underwent Senning procedure because of inadequate elevation of LV pressure in one, severe pulmonary artery hypertension after palliation in two,\textsuperscript{16} deformation of the sinus of Valsalva of the pulmonic (future aortic) root due to banding in two, and severe LV dysfunction in one. These were all palliated for the first 2-year period. None of the six patients died.

Patients' age at palliation was 1–14 months, averaging $6.7 \pm 2.9$ (SD) months, and was 5–37 months (mean, $15 \pm 6.8$ months) at the time of anatomic repair. The interval between the two operations was 1–36 (8 ± 6.8) months. Patients' body weight was $6.3 \pm 1.4$ kg at palliation and $7.7 \pm 1.6$ kg at intracardiac repair. The hemodynamic data were taken at 3–9 days before the palliation and 4 days to 3 months before the arterial switch operation.

Four of the 36 patients died after the arterial switch operation. Two patients (33 and 34) died 16 days and 1 day after repair, respectively. They had very high left atrial pressure and signs of severe low cardiac output but had no electrocardiographic or enzymatic evidence of myocardial ischemia. The echocardiogram, taken immediately after operation, showed marked dilatation of the LV and a generalized, but not localized, decrease of wall motion with fractional shortening less than 0.1 in both patients. The third patient (35) died of tachycardia 31 days after repair. Initially, the arrhythmia was supraventricular in origin, but after a few hours, the QRS complex became wide, and it could not be controlled by antiarrhythmic procedures including direct-current countershock. The final patient had an unusual pattern of the coronary arteries (Figure 1), for which we believe the anatomic repair was not indicated. This last patient was eliminated from the study because we believed that the cause of death was not related to LV

![Figure 1](image-url)  
**Figure 1.** Schematic of coronary artery pattern in a patient who died of low cardiac output secondary to poor left ventricular function, probably caused by myocardial ischemia after the arterial switch operation. Left anterior descending artery (LAD) might have kinked at its origin after implantation. Ao, aorta; RCA, right coronary artery; PA, pulmonary artery; LCX, circumflex branch.
TABLE 1. Clinical Features of Patients

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (months)</th>
<th>Weight (kg)</th>
<th>Age (months)</th>
<th>Weight (kg)</th>
<th>Body surface area (m²)</th>
<th>Interval (months)</th>
<th>LV:RV pressure ratio</th>
<th>d-PWT (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>10</td>
<td>8.5</td>
<td>18</td>
<td>9.3</td>
<td>0.50</td>
<td>8</td>
<td>1.12</td>
<td>6.0</td>
</tr>
<tr>
<td>2</td>
<td>12</td>
<td>7.7</td>
<td>20</td>
<td>9.7</td>
<td>0.48</td>
<td>7</td>
<td>1.67</td>
<td>8.5</td>
</tr>
<tr>
<td>3</td>
<td>5</td>
<td>...</td>
<td>9</td>
<td>5</td>
<td>0.26</td>
<td>3</td>
<td>0.95</td>
<td>5.5</td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>3.8</td>
<td>10</td>
<td>5.9</td>
<td>0.32</td>
<td>7</td>
<td>0.93</td>
<td>7.0</td>
</tr>
<tr>
<td>5</td>
<td>4</td>
<td>4.7</td>
<td>12</td>
<td>6.4</td>
<td>0.30</td>
<td>8</td>
<td>1.37</td>
<td>5.0</td>
</tr>
<tr>
<td>6</td>
<td>6</td>
<td>...</td>
<td>13</td>
<td>6.9</td>
<td>0.37</td>
<td>7</td>
<td>1.32</td>
<td>5.0</td>
</tr>
<tr>
<td>7</td>
<td>6</td>
<td>7.5</td>
<td>13</td>
<td>8.8</td>
<td>0.38</td>
<td>6</td>
<td>1.00</td>
<td>5.0</td>
</tr>
<tr>
<td>8</td>
<td>14</td>
<td>...</td>
<td>19</td>
<td>10.6</td>
<td>0.48</td>
<td>4</td>
<td>0.97</td>
<td>5.5</td>
</tr>
<tr>
<td>9</td>
<td>12</td>
<td>...</td>
<td>16</td>
<td>8.3</td>
<td>0.42</td>
<td>4</td>
<td>0.93</td>
<td>4.0</td>
</tr>
<tr>
<td>10</td>
<td>4</td>
<td>...</td>
<td>23</td>
<td>9.3</td>
<td>0.45</td>
<td>18</td>
<td>1.06</td>
<td>5.0</td>
</tr>
<tr>
<td>11</td>
<td>7</td>
<td>4.5</td>
<td>15</td>
<td>8.5</td>
<td>0.41</td>
<td>8</td>
<td>1.17</td>
<td>5.0</td>
</tr>
<tr>
<td>12</td>
<td>7</td>
<td>6.5</td>
<td>14</td>
<td>6.8</td>
<td>0.36</td>
<td>7</td>
<td>1.04</td>
<td>4.5</td>
</tr>
<tr>
<td>13</td>
<td>7</td>
<td>7.0</td>
<td>10</td>
<td>6.3</td>
<td>0.34</td>
<td>3</td>
<td>1.03</td>
<td>6.0</td>
</tr>
<tr>
<td>14</td>
<td>5</td>
<td>6.0</td>
<td>10</td>
<td>6.4</td>
<td>0.34</td>
<td>5</td>
<td>1.41</td>
<td>5.5</td>
</tr>
<tr>
<td>15</td>
<td>6</td>
<td>...</td>
<td>12</td>
<td>6.2</td>
<td>0.37</td>
<td>6</td>
<td>0.84</td>
<td>5.0</td>
</tr>
<tr>
<td>16</td>
<td>8</td>
<td>7.6</td>
<td>18</td>
<td>8.8</td>
<td>0.38</td>
<td>9</td>
<td>0.83</td>
<td>5.5</td>
</tr>
<tr>
<td>17</td>
<td>8</td>
<td>...</td>
<td>14</td>
<td>6.2</td>
<td>0.36</td>
<td>5</td>
<td>...</td>
<td>5.0</td>
</tr>
<tr>
<td>18</td>
<td>6</td>
<td>...</td>
<td>14</td>
<td>8.1</td>
<td>0.44</td>
<td>8</td>
<td>1.18</td>
<td>4.5</td>
</tr>
<tr>
<td>19</td>
<td>8</td>
<td>5.6</td>
<td>12</td>
<td>5.5</td>
<td>0.30</td>
<td>3</td>
<td>1.18</td>
<td>5.0</td>
</tr>
<tr>
<td>20</td>
<td>7</td>
<td>5.1</td>
<td>9</td>
<td>5.9</td>
<td>0.32</td>
<td>2</td>
<td>0.89</td>
<td>5.0</td>
</tr>
<tr>
<td>21</td>
<td>4</td>
<td>4.1</td>
<td>5</td>
<td>4.6</td>
<td>0.28</td>
<td>1</td>
<td>1.34</td>
<td>5.0</td>
</tr>
<tr>
<td>22</td>
<td>8</td>
<td>8.0</td>
<td>13</td>
<td>7.7</td>
<td>0.37</td>
<td>4</td>
<td>1.07</td>
<td>7.0</td>
</tr>
<tr>
<td>23</td>
<td>8</td>
<td>6.3</td>
<td>11</td>
<td>5.9</td>
<td>0.32</td>
<td>3</td>
<td>1.56</td>
<td>6.7</td>
</tr>
<tr>
<td>24</td>
<td>5</td>
<td>6.5</td>
<td>12</td>
<td>7.5</td>
<td>0.39</td>
<td>7</td>
<td>1.00</td>
<td>5.3</td>
</tr>
<tr>
<td>25</td>
<td>7</td>
<td>7.0</td>
<td>11</td>
<td>6.8</td>
<td>0.35</td>
<td>4</td>
<td>0.98</td>
<td>6.5</td>
</tr>
<tr>
<td>26</td>
<td>1</td>
<td>...</td>
<td>37</td>
<td>10.6</td>
<td>0.50</td>
<td>36</td>
<td>0.88</td>
<td>5.0</td>
</tr>
<tr>
<td>27</td>
<td>4</td>
<td>4.7</td>
<td>11</td>
<td>6.8</td>
<td>0.35</td>
<td>7</td>
<td>0.98</td>
<td>4.0</td>
</tr>
<tr>
<td>28</td>
<td>5</td>
<td>7.4</td>
<td>10</td>
<td>8.5</td>
<td>0.40</td>
<td>5</td>
<td>1.29</td>
<td>6.0</td>
</tr>
<tr>
<td>29</td>
<td>4</td>
<td>6.1</td>
<td>10</td>
<td>7.6</td>
<td>0.41</td>
<td>6</td>
<td>1.20</td>
<td>4.3</td>
</tr>
<tr>
<td>30</td>
<td>7</td>
<td>6.4</td>
<td>23</td>
<td>10.5</td>
<td>0.44</td>
<td>16</td>
<td>0.85</td>
<td>5.0</td>
</tr>
<tr>
<td>31</td>
<td>5</td>
<td>5.2</td>
<td>12</td>
<td>7.6</td>
<td>0.38</td>
<td>7</td>
<td>1.00</td>
<td>4.6</td>
</tr>
<tr>
<td>32</td>
<td>8</td>
<td>7.3</td>
<td>14</td>
<td>7.3</td>
<td>0.38</td>
<td>6</td>
<td>1.10</td>
<td>5.0</td>
</tr>
<tr>
<td>33</td>
<td>13</td>
<td>8.0</td>
<td>35</td>
<td>10</td>
<td>0.48</td>
<td>22</td>
<td>0.68</td>
<td>3.5</td>
</tr>
<tr>
<td>34</td>
<td>7</td>
<td>6.9</td>
<td>14</td>
<td>8.1</td>
<td>0.44</td>
<td>8</td>
<td>0.63</td>
<td>3.5</td>
</tr>
<tr>
<td>35</td>
<td>6</td>
<td>7.5</td>
<td>25</td>
<td>8.5</td>
<td>0.46</td>
<td>19</td>
<td>0.84</td>
<td>5.0</td>
</tr>
</tbody>
</table>

In patient 17, left ventricular pressure was not available. Patients 33–35 died after surgery (see text for details).

PAB, pulmonary artery banding; BT, Blalock-Taussig shunt; LV:RV, left to right ventricular pressure ratio; d-PWT, end-diastolic left ventricular posterior wall thickness on echocardiogram.

Functional variables, bearing in mind that the purpose of this study was to clarify functional aspects. Thus, 35 patients were included in the present study (Table 1).

Methods

Intracardiac and vascular pressures were obtained with a fluid-filled catheter system. Because the LV:RV pressure ratio is critically important, LV pressure measurement was always followed immediately by RV pressure measurement, and this procedure was repeated two or three times in each patient. The lowest ratio was taken. LV volume was calculated by the area-length method as described by Graham et al., and LV end-diastolic volume (EDV) was expressed as a percentage of the normal expected value. We also measured semiaxes of the LV at end diastole. We found that it was very difficult or even impossible to obtain LV wall thickness on the angio gram because the left-side border of the cardiac silhouette is often confined by the RV but not by the LV. We used echocardiography, therefore, to measure end-diastolic LV posterior wall thickness according to the standard of the American Society of Echocardiography. The data were taken within a few days of the invasive examinations. With posterior wall thickness, LV muscle mass was calculated.
according to the method of Rackley et al.\textsuperscript{20} and it was again expressed as a percentage of the normal expected value as reported by Graham et al.\textsuperscript{17}

From the data before the anatomic repair, we calculated meridional LV wall stress by the following equation\textsuperscript{21}: \( \text{LV wall stress} = \frac{Pb}{h}(2b + h) \), where \( P \) is aortic diastolic pressure, \( h \) is end-diastolic LV posterior wall thickness, and \( b \) is end-diastolic semiaxis of the LV. We calculated \( b \) from two semiaxes as follows: \( b^2 = L_1 \times L_2 \), where \( L_1 \) and \( L_2 \) are semiaxes in the frontal and lateral views of the LV, respectively. This variable would predict LV wall stress at the time of aortic valve opening immediately after the arterial switch operation was completed. Thus, in the following discussion, this variable was termed the "predictive LV wall stress." The problems and applicability of this variable are described further in the "Discussion." Data are expressed as mean ± SD, and the interrelation between LV ejection fraction and LV pressure or the LV:RV pressure ratio was tested by least-squares linear regression analysis.

**Results**

The LV:RV pressure ratio ranged from 0.83 to 1.67, averaging 1.1 ± 0.21 in the 32 surviving patients. It was 0.63 and 0.68 in the two patients who died of low cardiac output after surgery and was 0.84 in the patient who died of tachycardia 31 days after operation. The posterior wall thickness in the surviving patients was 4 mm or greater, which was greater than the lower limit of our normal range, whereas it was 3.5 mm in the two patients who died early postoperatively and 5 mm in the patient who died late (Table 1 and Figure 2).

Among the surviving patients, LVEDV ranged from 65% to 286% of normal, averaging 147 ± 46%, and LV ejection fraction ranged from 0.33 to 0.83, averaging 0.64 ± 0.12. The variables of the nonsurviving patients were not different from patients who survived (Figure 3). LV ejection fraction was weakly correlated with LV pressure (Figure 4; \( r = -0.56, p<0.001 \)) and the LV:RV pressure ratio (\( r = -0.43, p<0.02 \)). LV mass and the LV mass: LVEDV ratio also did not distinguish surviving from nonsurviving patients (Figure 5). The predictive LV wall stress was below \( 120 \times 10^3 \) dynes/cm\(^2\) in all but one surviving patient and was \( 131 \times 10^3 \) dynes/cm\(^2\) in the last surviving patient, while it was 151, 153, and \( 186 \times 10^3 \) dynes/cm\(^2\) in the patients who died, respectively (Figure 6).

**Discussion**

It has been indicated that the RV is not suitable as a systemic pumping chamber for the duration of the human life span. This view has been substantiated by the observations of Masden and Franch,\textsuperscript{22} who reviewed the literature describing the clinical features of patients with isolated, congenitally corrected TGA and found that many of the patients developed congestive heart failure of unknown etiology in the 3rd to 4th decade, which could be attributable to systemic RV dysfunction.\textsuperscript{23,24} It is also reported that systemic ventricular function is compromised in patients after the atrial switch repair,\textsuperscript{1-4} whereas it is normal or near normal after the arterial switch operation.\textsuperscript{4-7} Although the arterial switch operation may be complicated with stenosis of the anastomosed sites, perioperative or postoperative myocardial ischemia, or aortic regurgitation,\textsuperscript{25,26} this operation is being increasingly accepted in many institutions throughout the world,
FIGURE 4. *Plot of left ventricular ejection fraction (LVEF) plotted against LV pressure (LVP). LVEF was inversely correlated to LVP, but the correlation coefficient was only −0.56. Relation secured largely affected by two patients with excessively high LVP and low LVEF.*

largely because of the superiority of postoperative physiological conditions.

The arterial switch operation was initially applied to TGA with a large ventricular septal defect in which LV pressure is at the systemic level; thus, the LV is readily prepared to support the high-pressure systemic circulation. In simple TGA, however, LV pressure decreases soon after birth when the pulmonary artery pressure drops, which results in regression of LV wall thickness and muscle mass. These changes preclude the arterial switch operation in this setting. Alternatively, one would naturally consider that the primary anatomic repair might be performed before the LV regresses during the early neonatal period. With recent advances of the techniques in neonatal cardiac surgery, several institutions have now shown that the arterial switch operation for simple TGA is feasible with an acceptable mortality during the early neonatal period. Surgical correction in early life will shorten the period of hemodynamic load to the ventricles and of hypoxia in the myocardium, thereby preserving ventricular function. Thus, if technical problems are solved, the neonatal switch will be the best choice of surgical management of simple TGA.

Then, what should be the choice when the infant with simple TGA is referred after the neonatal period: should the atrial switch simply be performed, or should the arterial switch be performed? Although the conclusion should be postponed until real long-term outcomes are clarified for both procedures, it can be said that the arterial switch operation is a more physiologically sound procedure than is the atrial switch operation. Thus, one would like to choose the arterial switch operation, but it is obvious that the primary switch cannot be performed in patients with low LV pressure and with regressed ventricular muscle mass. In these patients, Yacoub et al. banded the pulmonary artery to increase LV pressure, which prepared the LV for supporting the systemic circulation. They propose that the arterial switch operation can be performed within a few months after banding when LV pressure is within 10 mm Hg of the systemic pressure. When LV pressure is lower than this, they recommend performing the arterial switch operation after waiting for several months,6–7 during which time LV mass increases. We had initially adopted their criteria. But we later believed that these criteria were not satisfactory because our two patients who died of low cardiac output after surgery were operated upon according to these criteria. Thus, we had to establish more reliable criteria for the two-staged arterial switch operation.

If LV pressure is increased consistently to the systemic level after pulmonary artery banding, there will be no difficulty in selecting the candidate for the arterial switch operation. We found that there were patients in whom very tight banding, even a complete obliteration, of the main pulmonary artery did not bring about sufficient elevation of LV pressure on the operating table. In some of these patients, the pressure increased to the suprasystemic level several days to months after the palliation. Although the basic cause of this has not yet been clarified, the pressure would be determined not only by the degree of banding but also by the interaction of several factors such as the amount of flow through the Blalock-Taussig shunt, the size of interatral communication, which could be one of the major determinants of volume preload to the LV, the LV wall muscle mass, which generates the power, and myocardial contractility, which is affected by anesthetics and arterial oxygen content. Therefore, we found the LV pressure to be at various levels even though we banded the main pulmonary artery to increase LV pressure to 80% or more of the systemic pressure in each patient.

The present study demonstrates that low LV pressure was obviously a factor of mortality as indicated by the fact that two patients who had the lowest LV pressure died of poor LV pump function soon after surgery. The patient who died 31 days after correction had an LV pressure of 84% of the systemic pressure, but the other three patients (15, 16, and 30 in Table 1) survived at the same level of LV pressure (84%, 83%, and 85%, respectively). This indicates that the RV:LV pressure ratio between 0.8 and 0.9 is the lower limit for the survival of the arterial switch operation in the infant, but not in the newborn, after the palliation. It is, then, necessary to determine the risk factor or factors within this narrow range of pressure ratios. End-diastolic LV posterior wall thickness was another simple discriminator of mortality. It was greater than 4 mm or within the normal range for the body size of each patient. The body surface area of our patients fell within a rather narrow range (<0.5 m²) so that we may simply conclude that posterior wall thickness should be greater than 4 mm for the second-stage arterial switch operation in infants with this range in body size. The ventricular wall thickness and ventricular volume, however, influence each other; thus, one cannot discuss the former alone without
accounting for the latter. In this aspect, the wall thickness alone cannot be a single reliable factor. To solve these problems, we have considered calculating the predictive LV wall stress, which includes information on volume and wall thickness. The posterior wall thickness alone, however, will be a useful variable in screening patients.

The one major hemodynamic change resulting from the arterial switch operation is the change of afterload to both ventricles, and the change in LV afterload is vitally important for the survival of the patient. Systolic wall stress is one of the variables indicating the afterload and is a good indicator of pump performance of the LV with a given level of contractility. In the present study, we tried to calculate, with only preoperative data, the LV wall stress at the time when the great arteries were switched, that is, when the LV started to eject blood into the systemic circulation. Thus, the obtained value was not the actual one but only a predictive value. In the context of cardiac performance, the end-systolic wall stress or peak stress is usually used. It will be ideal if these values can be predicted by preoperative data, but it is not possible to estimate any numbers used for the calculation of these stress values from preoperative data.

We assumed that LV wall thickness did not change from preoperative values immediately after surgery. The closure of the Blalock-Taussig shunt would possibly bring about the elevation of aortic diastolic pressure, but we considered that, even if there is some elevation, the prediction of LV wall stress in this way would be a very useful index. Thus, the preoperative values of wall thickness and the pressure were used for the calculation of the predictive LV wall stress. In fact, the postoperative pressure was not significantly different from preoperative values. After the correction when the cross-sectional shape of the LV becomes more circular, the semiaxis of the ventricle was calculated, as described in "Patients and Methods," with the assumption that the circumferential fiber length was not changed by surgery. The LV dimensions were those at end diastole, and the pressure was that at the beginning of ejection; thus, they were at different times. The dimensions, however, should not change greatly during the isovolumetric contraction; therefore, the stress obtained here would indicate the value at the instant of aortic valve opening. As shown in Figure 6, the predictive LV wall stress calculated in this way was less than \(120 \times 10^3\) dynes/cm\(^2\) in all but one surviving patient, and \(131 \times 10^3\) dynes/cm\(^2\) in the remaining surviving patient; it was \(151, 133,\) and \(186 \times 10^3\) dynes/cm\(^2\) in nonsurviving patients. Calculation of wall stress is detailed in text.

![Figure 5](http://circ.ahajournals.org/)

**Figure 5.** Plots of left ventricular mass (LVM) and LVM:LVEDV ratio. LVM in nonsurviving patients was not different from those of surviving patients. LVEDV, left ventricular end-diastolic volume.

![Figure 6](http://circ.ahajournals.org/)

**Figure 6.** Plot of predictive left ventricular (LV) wall stress. Wall stress was less than \(120 \times 10^3\) dynes/cm\(^2\) in all but one surviving patient, and \(131 \times 10^3\) dynes/cm\(^2\) in the remaining surviving patient; it was \(151, 133,\) and \(186 \times 10^3\) dynes/cm\(^2\) in nonsurviving patients. Calculation of wall stress is detailed in text.
up study showed a rather high LV fractional shortening, which is a usual finding in the post-Senning patient. Since we experienced this case, we perform the arterial switch operation as a semi-emergency as soon as we detect any signs of this adverse phenomenon. Two of the patients in this study had low LV ejection fraction along with high LV pressure (Figure 4). These patients and the others with a low ejection fraction did not experience an especially difficult postoperative course compared with the patients with normal LV function, and postoperative catheterization data indicated normal LV pump function.\textsuperscript{34} The long-term result of ventricular function in these patients, however, is not yet known and should be carefully observed.

In addition to pressure and stress, the volume and function of the LV are also important factors. Because the basic function of the ventricle is pumping out blood to the artery, the ventricle needs to have a potential for pumping out normal or at least subnormal stroke volume. When the ventricular volume is near normal, ejection fraction may be normal also; when the volume is large, ejection fraction may be a little low. One patient with an LVEDV of 65\% of normal went through a very difficult postoperative course and was very fortunate to have survived.

From the present data, we propose that criteria for the safe two-staged arterial switch operation include: an LV:RV pressure ratio greater than 0.85; LVEDV greater than 90\% of normal; LV ejection fraction greater than 0.5, or it may be greater than 0.4; end-diastolic LV posterior wall thickness greater than 4 mm or very safely greater than 4.5 mm (which are well above the lower limit of the normal range of most patients for this surgical protocol); and the predictive LV wall stress obtained as previously described less than 120 \times 10^3 \text{ dynes/cm}^2. These values may be very conservative since our major emphasis is the universal safety of this type of surgery. We have avoided setting any lower limit with which the operation can be successfully performed only by special teams or with which only an occasional survival or only a low survival rate can be achieved by a majority of institutions.

References
5. Aresman FW, Radley-Smith R, Yacoub MH, Lange P, Bernhard A, Sievers HH, Heintzen P: Catheter evaluation of left ventricular shape and function 1 or more years after anatomic correction of transposition of the great arteries. \textit{Am J Cardiol} 1983;52:1079–1083

KEY WORDS • Jatene operation • operative indication • left ventricular wall thickness • predictive left ventricular wall stress
Criteria for two-staged arterial switch operation for simple transposition of great arteries.
M Nakazawa, K Oyama, Y Imai, K Nojima, H Aotsuka, G Satomi, H Kurosawa and A Takao

_Circulation_. 1988;78:124-131
doi: 10.1161/01.CIR.78.1.124
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1988 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/78/1/124

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/