Neonatal Critical Valvar Aortic Stenosis
A Comparison of Surgical and Balloon Dilation Therapy

Benjamin Zeevi, MD, John F. Keane, MD, Aldo R. Castaneda, MD, Stanton B. Perry, MD, and James E. Lock, MD, FACC

Balloon aortic valvotomy (BAV) is an alternative to surgical valvotomy in infants and children. We compared BAV in 16 consecutive neonates (1985–1988) to surgical valvotomy in a prior group of 16 consecutive neonates (1978–1984). Both groups were comparable in terms of age, weight, hemodynamic data, left ventricular size, and associated lesions. There were six early and one late deaths after surgery. Five out of six neonates requiring a second operation died. Left ventricular size (measured in 13 neonates) had some influence on survival after surgery: three of three with small or hypoplastic left ventricles and three of 10 with normal-sized left ventricles died. After BAV, there were three early deaths, two patients who underwent stage I palliation of hypoplastic left heart syndrome, and two late deaths. As with surgical valvotomy, left ventricular size seemed to influence survival after BAV: five of six with small or hypoplastic left ventricles died or underwent stage I palliation for hypoplastic left heart syndrome and two of nine with normal-sized left ventricles died. At follow-up (26 ± 17 months) in six patients in the surgical group, the peak systolic ejection gradient (PSEG) was 52.2 ± 23 mm Hg and left ventricular end-diastolic pressure (LVEDP) 18.2 ± 5.2 mm Hg. Aortic regurgitation was mild in five and moderate in the sixth patient. At follow-up (17.6 ± 7.8 months) in nine patients in the balloon dilation group, the PSEG was 45.6 ± 11 mm Hg in five patients at catheterization and 43.8 ± 22.9 mm Hg in four patients by echocardiography-Doppler. Aortic regurgitation was mild in three and absent in the other six patients. BAV may be an effective alternative to surgical valvotomy in unselected neonates with critical valvar aortic stenosis. (Circulation 1989;80:831–839)

Critical valvar aortic stenosis in neonates causes severe congestive heart failure and shock; death ensues in most patients within the first weeks of life with medical treatment alone.1,2 Operative mortality for neonatal valvar aortic stenosis is high and ranges widely between 9% and 86%.1,3–12 Contributing to the high mortality is the association of other anatomic abnormalities, including varying degrees of left ventricular hypoplasia.1–5

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Although surgical valvotomy can be an effective treatment, it must be considered palliative because restenosis and aortic regurgitation are not uncommon.7,9,13–15

Since the initial description of balloon aortic valvotomy in 1984,16 several studies have demonstrated that the severity of congenital valvar aortic stenosis can be reduced with minimal mortality and acceptable morbidity.17–22 If the efficacy, morbidity, and mortality of balloon dilation in neonates with critical valvar aortic stenosis are no worse than the results of surgical valvotomy, it might be the initial form of management. We, therefore, compared surgical valvotomy in 16 consecutive neonates (1978–1984) (no patient has been managed surgically since 1984) to balloon valvotomy in 16 consecutive neonates (1985–1988) with critical valvar aortic stenosis.

Methods

Patients

Surgical group. From January 1978 through December 1984, 16 consecutive neonates (less than 30 days old) underwent surgical valvotomy for critical valvar aortic stenosis at The Children’s Hospital, Boston. Data for analysis were obtained from the medical, echocardiographic, catheterization, surgical, and autopsy records.

These neonates ranged in age from 1 to 30 days (mean, 8.8 ± 9.1 days) and in weight from 2.4 to 4.1 kg (mean, 3.3 ± 0.5 kg). All were in severe conges-
The anatomy of the aortic valves, as determined by inspection at surgery or autopsy or by echocardiography, were unicommissural in nine and bicommissural in seven.

Aortic valvotomy was performed in two neonates without preoperative catheterization. Thus, preoperative hemodynamic data were available in 14 neonates. The peak-to-peak systolic ejection gradient (PSEG) ranged from 16 to 120 mm Hg (mean, 66.1±33.6 mm Hg) and the left ventricular end-diastolic pressure (LVEDP) from 8 to 27 mm Hg (mean, 18.1±5.9 mm Hg). No patient had aortic regurgitation before surgery. Left ventricular biplane cineangiograms were available for volumetric analysis by the area-length method in 13 neonates. Derived left ventricular diastolic volumes were expressed as a percentage of normal values established in our laboratory. Left ventricular end-diastolic volumes ranged from 11.5 to 55.1 ml/m² (mean, 30.9±12.9 ml/m²) and were 33–174% (mean, 94.6±38.3%) of the predicted normal. The left ventricle was small (60–80% of predicted normal) in one and hypoplastic (less than 60% of predicted normal) in two. The angiographic ejection fraction ranged from 23 to 73% (mean, 41±14%) (Table 1).

All neonates underwent open aortic valvotomy, using inflow occlusion in 14 and cardiopulmonary bypass in two. During the study, all neonates with critical aortic stenosis underwent surgery regardless of ventricular size or function (i.e., no patient with patent mitral and aortic valves underwent stage I palliation for hypoplastic left ventricle as the initial procedure on the basis of left ventricular size alone).

**Balloon dilation group.** From January 1985 through March 1988, all 16 neonates admitted to The Children’s Hospital, Boston, with the diagnosis of critical valvar aortic stenosis underwent attempted balloon dilation. We did not exclude any neonate on the basis of clinical condition, valve morphology, or left ventricular size.

The neonates ranged in age from 1 to 30 days (mean, 10.1±9.9 days) and in weight from 2.2 to 4.6 kg (mean, 3.5±0.8 kg). All were in severe congestive heart failure or shock at presentation, and five received prostaglandin E₁ to maintain patency of the ductus arteriosus (Table 1).

Associated lesions were present in 14 patients, including patent ductus arteriosus in seven, coarctation of the aorta in one, and mitral regurgitation in 11 (moderate or severe in seven and mild in four). The anatomy of the aortic valves, as determined by inspection at surgery or autopsy or by echocardiography, were unicommissural in nine and bicommissural in seven.

**TABLE 1. Comparison of Patient Characteristics**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Surgical group (n=16)</th>
<th>Balloon dilation group (n=16)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at intervention (days)</td>
<td>8.8±9.1</td>
<td>10.1±9.9</td>
<td>NS</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>3.3±0.5</td>
<td>3.5±0.8</td>
<td>NS</td>
</tr>
<tr>
<td>Associated lesions (n)</td>
<td>14</td>
<td>14</td>
<td>NS</td>
</tr>
<tr>
<td>PDA</td>
<td>7</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Coarctation</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Mitral regurgitation</td>
<td>11</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Moderate-severe</td>
<td>7</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>4</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Small left ventricle (60–80% NL)</td>
<td>1</td>
<td>3</td>
<td>NS</td>
</tr>
<tr>
<td>HLV (60% NL)</td>
<td>2</td>
<td>3</td>
<td>NS</td>
</tr>
<tr>
<td>Valve morphology</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unicommissural</td>
<td>9</td>
<td>10</td>
<td>NS</td>
</tr>
<tr>
<td>Bicommissural</td>
<td>7</td>
<td>6</td>
<td>NS</td>
</tr>
<tr>
<td>PSEG (mm Hg)</td>
<td>66.1±33.6</td>
<td>54.7±30.1</td>
<td>NS</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>18.1±5.9</td>
<td>18.9±5.1</td>
<td>NS</td>
</tr>
<tr>
<td>LVEDV (ml/m²)</td>
<td>30.9±12.9</td>
<td>33.2±16.1</td>
<td>NS</td>
</tr>
<tr>
<td>LVEDV (% NL)</td>
<td>94.6±38.3</td>
<td>104.5±54.2</td>
<td>NS</td>
</tr>
<tr>
<td>EF (%)</td>
<td>41±14</td>
<td>33±14</td>
<td>NS</td>
</tr>
</tbody>
</table>

PDA, patent ductus arteriosus; HLV, hypoplastic left ventricle; PSEG, peak systolic ejection gradient; LVEDP, left ventricular end-diastolic pressure; LVEDV, left ventricular end-diastolic volume; EF, ejection fraction.
18.9±5.1 mm Hg). No patient had aortic regurgitation before balloon dilation. Left ventricular biplane cineangiograms were available for volumetric analysis in 15 neonates. The left ventricular end-diastolic volumes ranged from 10.4 to 69 ml/m² (mean, 33.2±16.1 ml/m²) being 30–202% (mean, 104.5±54.2%) of the predicted normal. The left ventricle was small (60–80% of predicted normal) in three and hypoplastic (less than 60% of predicted normal) in three. The angiographic ejection fraction ranged from 23% to 73% (mean, 33±14%) (Table 1).

For both surgical and balloon dilation groups, early death was defined as death occurring within 30 days of the initial procedure. Patients undergoing stage I palliation for hypoplastic left heart syndrome were considered failures and excluded from further follow-up.

**Balloon Dilation Technique**

Informed consent was obtained from the parents in accordance with the guidelines of the Committee on Clinical Investigation. The methodology outlined below is our current approach and has been modified in response to our initial experience.

All patients are premedicated with morphine sulfate (0.1 mg/kg body wt) and receive local anesthesia with 1% xylocaine for percutaneous cannulation of the femoral vessels. In neonates less than 72 hours old, we attempt to use the umbilical artery. If the aortic valve cannot be crossed within 30 minutes by umbilical artery access, the femoral artery is used. The femoral artery was used in 10 and the umbilical artery in six. The arteries are cannulated with 3.2F or 4F cut-off Pigtail catheters. Heparin was administered intravenously (100 μ/kg body wt). Complete right and left heart catheterization includes measurement of the PSEG across the aortic valve with simultaneous measurement of left ventricular (antegrade) and ascending aortic (retrograde) pressures. Biplane cineangiograms of the left ventricle and the ascending aorta allowed measurement of left ventricular size and function, the aortic annulus, evaluation of associated lesions, and the degree of aortic regurgitation (Figure 1).

For dilation, the aortic valve is initially crossed with a soft 0.015–0.021 in. guidewire; over this wire, the cut-off Pigtail catheter is positioned in the left ventricle. The small wire is exchanged for a 0.021–0.028 in. guidewire with a preformed curve that is curled in the apex of the left ventricle. The Pigtail catheter is then exchanged for a balloon dilation catheter (Cook Inc or Mansfield Scientific), which is positioned across the aortic valve and inflated by hand to 3–8 atm or until the waist in the balloon produced by the valve disappears. The actual diameter of the inflated balloon will vary with inflation pressure; small (less than 1 mm) increases in balloon diameter can be achieved by increasing pressure rather than changing to a larger balloon. As a waist is commonly absent in neonates with
aortic stenosis (Figure 2), two to four inflations are performed to ensure that the balloon is properly positioned. After each inflation, the balloon is deflated rapidly and withdrawn to the descending aorta. Inflation-deflation cycles lasted, at most, 15 seconds. After balloon dilation, left ventricular and ascending aorta pressures are measured. Finally, an aortogram is performed to evaluate the severity of aortic regurgitation after the dilation. The neonates were observed, at least overnight, in the cardiovascular intensive care unit.

Data Analysis

Measured and calculated data are expressed as mean±1 SD. Comparison of the two groups was performed using the unpaired Student’s t test. A p value of less than 0.05 was considered significant.

Results

No significant differences were found between the clinical, hemodynamic, and anatomic characteristics of the surgical and balloon valvotomy groups (Table 1).

Surgical Group

In the first 30 days after the initial surgical valvotomy, there were six early deaths (Table 2). Of these, three occurred within 5 days of surgery and were secondary to low cardiac output, and two occurred in patients with small or hypoplastic left ventricles (33% and 61% of predicted normal). At autopsy, the third patient had severe residual aortic stenosis with extensive endocardial fibroelastosis. The other three early deaths occurred during or after repeat operation prompted by severe residual aortic stenosis (PSEG of 60, 84, and 90 mm Hg) and left ventricular dysfunction. In addition to these six deaths, one patient underwent stage I palliation for hypoplastic left heart syndrome and was, therefore, excluded from further follow-up.

Thus, nine patients were available for follow-up. For this group, the length of the hospitalization ranged from 10 to 20 days (mean, 13.5±3.3 days). Of these nine patients, one patient was lost to follow-up. Another patient died at 60 days of age at his third operation, an attempted left ventricular to descending aorta conduit placement; two open valvotomies failed to relieve a peak systolic ejection gradient of 140 mm Hg. Of the remaining seven, six underwent cardiac catheterization 4–48 months (mean, 26±17 months) after surgical valvotomy. The residual PSEG ranged from 20 to 72 mm Hg (mean, 52.2±23 mm Hg) and the LVEDP from 12 to 20 mm Hg (mean, 18.2±5.2 mm Hg). Aortic regurgitation was mild in five and moderate in the sixth; mitral regurgitation was mild in one and moderate in another. Balloon aortic valvotomy decreased the peak systolic ejection gradient from 60 to 40 mm Hg.
in one child 4 years after surgery. In the patient who has not been recatheterized, Doppler echocardiography 14 months after surgery demonstrated a peak instantaneous systolic gradient of 60 mm Hg and moderate aortic regurgitation.

In summary, 16 patients had surgical valvotomies as the initial procedure. Of these, seven patients have undergone at least one additional procedure: three underwent left ventricular—descending aorta conduits (one “temporary”), one stage I palliation for hypoplastic left heart syndrome, two a second surgical valvotomy, and one balloon dilation. A total of eight additional procedures in five patients were performed because of residual aortic stenosis. There were eight deaths. Of the eight remaining patients, the left ventricle supplies systemic output through the native left ventricular outflow tract in seven.

**Balloon Dilation Group**

The diameter of the aortic anulus ranged from 4.5 to 8.0 mm (mean, 6.1±1 mm), and the diameter of the balloon from 4 to 6 mm, resulting in a balloon-to-anulus diameter ratio (BAR) of 67–120% (mean, 86±13%) (Table 3). The immediate postdilation peak systolic ejection gradient ranged from 1 to 100 mm Hg (mean, 26.5±23.9 mm Hg), representing 0–95% (mean, 55.5±24.2%) reduction in the gradient. The immediate postdilation LVEDP ranged from 6 to 22 mm Hg (mean, 14.2±4.9 mm Hg) compared with 18.9±5.1 mm Hg predilation (p<0.02).

New aortic regurgitation developed in six neonates, being mild in four and severe in two. Loss of pulse occurred in nine neonates but returned to normal in seven after anticoagulation, thrombolytic therapy, or both. The length of the hospitalization for the 10 patients who were discharged ranged from 3 to 50 days (mean, 13.7±14.7 days). Among those is a patient with hypoplastic left ventricle (30% of predicted normal) who underwent a stage I repair for hypoplastic left heart syndrome and subsequently a successful modified Fontan operation at the age of 2 years old.

In the first 30 days after the initial balloon dilation, there were three early deaths. (In addition to the three early deaths, two patients underwent stage I palliation for hypoplastic left heart syndrome.) The three early deaths were probably related to the procedure. One neonate died from *Staphylococcus aureus* sepsis 2 days after a prolonged (5 hours), unsuccessful dilation attempt performed via an umbilical artery and subsequent surgical aortic valvotomy. Autopsy showed numerous small infected vegetations on all four heart valves and in the great arteries. A second neonate developed severe aortic regurgitation after dilation with a BAR of 120%, and at surgery was found to have avulsion of the right and noncoronary cusps from the anulus. Although at initial catheterization the left ventricu-
lar volume was normal, repeat catheterization showed a hypoplastic left ventricle and the patient died 2 weeks after dilation, during an attempt to perform stage I palliation for hypoplastic left heart syndrome. The third neonate, a patient with uncorrectable acidosis, a unicommissural valve, and a small left ventricle (66% of predicted normal), developed severe aortic regurgitation after dilation of a cusp rather than the valve orifice after passage of a 0.025 in. guidewire through the valve tissue (Figure 3). He died 3 hours later from low cardiac output.

There were two late deaths, 6–16 weeks after the procedure. Both patients had small left ventricles (71% and 63% of predicted normal) and died secondary to low cardiac output after late repair of coarctation of the aorta.

Thus, excluding the five early failures and two late deaths, there were nine patients available for long-term follow-up. Of these, five were recatheterized 2 weeks to 13.5 months (mean, 5.8±5.4 months) after the initial balloon dilation. The PSEG ranged from 35 to 60 mm Hg (mean, 45.6±11 mm Hg) and the LVEDP from 7 to 23 mm Hg (mean, 15.8±6.1 mm Hg). Of these five, two who had symptoms of congestive heart failure and were dependent on mechanical ventilation underwent successful repeat balloon dilation and were subsequently discharged home. None of these five patients had aortic regurgitation and one had moderate mitral regurgitation. The other four survivors had Doppler two-dimensional echocardiography studies 3 weeks to 24 months (mean, 9.9±10.6 months) after dilation.

### Table 3. Details of Newborns With Valvar Aortic Stenosis Treated by Balloon Dilation

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (days)</th>
<th>Left ventricle volume (% predicted NL)</th>
<th>Gradient (mm Hg)</th>
<th>Reduction (%)</th>
<th>LVEDP BAR Pre (%)</th>
<th>New AR Type</th>
<th>Follow-up</th>
<th>AR (1–5): Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>11</td>
<td>130</td>
<td>100</td>
<td>60</td>
<td>0.75</td>
<td>16</td>
<td>0</td>
<td>C 6 40</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>130</td>
<td>55</td>
<td>0</td>
<td>0.67</td>
<td>16</td>
<td>1</td>
<td>Died 2 days after emergency surgery</td>
</tr>
<tr>
<td>3</td>
<td>2</td>
<td>30</td>
<td>130</td>
<td>23</td>
<td>0.83</td>
<td>12</td>
<td>12 0</td>
<td>Underwent stage I for HLHS at 16 years, modified Fontan at 2 years</td>
</tr>
<tr>
<td>4</td>
<td>30</td>
<td>196</td>
<td>53</td>
<td>42</td>
<td>0.86</td>
<td>25</td>
<td>12 1</td>
<td>Died at 6 weeks after stage I for HLHS</td>
</tr>
<tr>
<td>5</td>
<td>10</td>
<td>46</td>
<td>21</td>
<td>95</td>
<td>1.0</td>
<td>21</td>
<td>15 1</td>
<td>Died at 2 weeks of age after stage I for HLHS</td>
</tr>
<tr>
<td>6</td>
<td>2</td>
<td>100</td>
<td>65</td>
<td>92</td>
<td>1.2</td>
<td>13</td>
<td>6 5</td>
<td>Died at 4 months in multiple organ failure</td>
</tr>
<tr>
<td>7</td>
<td>3</td>
<td>202</td>
<td>10</td>
<td>70</td>
<td>0.75</td>
<td>20</td>
<td>20 0</td>
<td>Died 3 hours later; perforation of AV</td>
</tr>
<tr>
<td>8</td>
<td>1</td>
<td>71</td>
<td>20</td>
<td>50</td>
<td>1.0</td>
<td>23</td>
<td>22 0</td>
<td>Died at 6 weeks</td>
</tr>
<tr>
<td>9</td>
<td>19</td>
<td>103</td>
<td>60</td>
<td>67</td>
<td>0.86</td>
<td>17</td>
<td>22 0</td>
<td>Underwent second BD</td>
</tr>
<tr>
<td>10</td>
<td>18</td>
<td>65</td>
<td>20</td>
<td>69</td>
<td>0.71</td>
<td>20</td>
<td>15 0</td>
<td>Underwent second BD</td>
</tr>
<tr>
<td>11</td>
<td>2</td>
<td>64</td>
<td>30</td>
<td>33</td>
<td>1.0</td>
<td>5</td>
<td>4 0</td>
<td>Underwent second BD</td>
</tr>
<tr>
<td>12</td>
<td>16</td>
<td>63</td>
<td>50</td>
<td>50</td>
<td>0.83</td>
<td>26</td>
<td>18 0</td>
<td>Died 3 hours later; perforation of AV</td>
</tr>
<tr>
<td>13</td>
<td>29</td>
<td>171</td>
<td>70</td>
<td>71</td>
<td>0.83</td>
<td>18</td>
<td>10 0</td>
<td>Died after stage I for HLHS</td>
</tr>
<tr>
<td>14</td>
<td>12</td>
<td>119</td>
<td>60</td>
<td>50</td>
<td>0.77</td>
<td>17</td>
<td>10 1</td>
<td>Died after stage I for HLHS</td>
</tr>
<tr>
<td>15</td>
<td>1</td>
<td>41</td>
<td>36</td>
<td>47</td>
<td>0.83</td>
<td>11</td>
<td>9 0</td>
<td>Died after stage I for HLHS</td>
</tr>
<tr>
<td>16</td>
<td>3</td>
<td>99</td>
<td>50</td>
<td>64</td>
<td>0.89</td>
<td>28</td>
<td>16 0</td>
<td>Died after stage I for HLHS</td>
</tr>
</tbody>
</table>

BAR, balloon anulus ratio; LVEDP, left ventricular end-diastolic pressure; AR, aortic regurgitation; C, cardiac catheterization; E, Doppler echocardiography; HLHS, hypoplastic left heart syndrome; BD, balloon dilation.

### Table 4. Neonates With Critical Aortic Stenosis

<table>
<thead>
<tr>
<th>Neonates with surgical valvotomy (n=16)</th>
<th>Neonates with balloon valvotomy (n=16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early survivors (n=9)</td>
<td>Early failures (n=7): early deaths</td>
</tr>
<tr>
<td>(n=6)</td>
<td>(n=6)</td>
</tr>
<tr>
<td>(residual aortic stenosis [n=4]; stage I HLHS [n=1])</td>
<td>(technical errors [n=3]; stage I HLHS [n=2])</td>
</tr>
<tr>
<td>Late survivors (n=8)</td>
<td>Late death (residual aortic stenosis [n=1])</td>
</tr>
</tbody>
</table>
that showed a residual maximal instantaneous gradient ranging from 20 to 70 mm Hg (mean, 43.8±22.9 mm Hg). All had good left ventricular function and 3 had mild aortic regurgitation. The overall mean follow-up period for the balloon dilation group was 7.6±7.8 months, which is significantly shorter than the 26±17 months follow-up for the surgical group (p<0.02). The overall residual peak systolic ejection gradient was 44.8±16.1 mm Hg, which is not significantly different from the surgical group.

In summary, of the 16 patients undergoing balloon dilation, six required a second procedure: two balloon dilations, one surgical valvotomy, and three stage I palliations for hypoplastic left heart syndrome. Excluding the five deaths and two additional patients who underwent stage I palliation for hypoplastic left heart syndrome, nine patients survive.

**Discussion**

Operative therapy for critical valvar aortic stenosis in neonates has a significant mortality, regardless of the surgical approach. Previous reports of balloon aortic valvotomy in small series of neonates have not compared the results directly with surgical results and have not described the technique in detail. In the largest reported series of neonates, there were five deaths among seven neonates. Our results suggest that in similar groups of neonates with critical valvar aortic stenosis, the efficacy and mortality of surgical and balloon valvotomy may be similar.

There are clearly limitations to our study, which retrospectively compares nonrandomized groups, including the fact that the patients are from different time periods and, therefore, preintervention and postintervention care may differ. It should also be emphasized that the balloon valvotomy group represents the first 16 neonates undergoing balloon dilation at our institution. Of the deaths in the balloon dilation group, three were due in part to technical errors (dilation through a cusp, use of an oversized balloon, and procedure-induced sepsis). It is likely that the incidence of these complications can be reduced with increased experience. Similarly, the surgical results are from an era when myocardial protection may have been suboptimal; current surgical results may have a lower incidence of residual aortic stenosis, especially if cardiopulmonary bypass is used.
The mortality in neonates with small hypoplastic left ventricles was high in both the surgical and balloon dilation groups (three of three and three of six, respectively). Of the three survivors in this group of nine patients, two underwent stage I palliation for hypoplastic left heart syndrome. A small hypoplastic left ventricle has previously been reported to be a high risk factor in this group of patients, but caution is required in assessing operability from left ventricular volumes alone; in the balloon dilation group, there was a survivor with a left ventricular volume of 41%. Obviously, survival after valvotomy is multifactorial; analysis of anatomic variables other than left ventricular volume and physiologic variables other than gradient may be needed to improve patient selection. Nonetheless, among the neonates with normal-sized left ventricles, there were only two of nine deaths (one of these two was found to have a hypoplastic left ventricle on follow-up and underwent stage I palliation) in the balloon dilation group compared with three of 10 in the surgical group.

Technique

Based on the above experience, in neonates less than 72 hours old, we initially attempted passage of a 4–5 mm, low-profile balloon dilation catheter. The aortic valves are soft and can be easily perforated. Thus, accurate assessment of the location of the valve orifice with two-dimensional echocardiography or angiography and the use of very soft wires to cross the valves are important. In almost all cases, the open commissure is between the left and non-coronary cusps, and valve perforation should be suspected whenever the wire crosses the valve anteriorly. In neonates, we initially use balloons that are at most 85–90% the anulus diameter, modifying inflation pressure to adjust the actual balloon diameter and allow improved gradient reduction. A BAR of more than 100% may adversely affect outcome and has been associated with increased aortic regurgitation.

Assessing the immediate results of balloon dilation in the catheterization laboratory can be difficult. In many neonates, no definite waist is seen. The peak-to-peak systolic ejection gradient in these neonates may not accurately reflect the severity of the obstruction due to impaired myocardial function, left-to-right atrial shunts, and right-to-left ductal shunts. Follow-up in both the surgical and balloon valvotomy groups demonstrated evidence of increasing valve gradients that probably reflects improved left ventricular function after decreasing the gradient across the obstruction. We currently consider the procedure successful when there is significant initial gradient reduction, LVEDP is reduced, no more than mild aortic regurgitation results, and clinical improvement is observed.

Conclusion

Our data suggest that percutaneous balloon valvotomy may be as effective as surgical valvotomy in newborns with critical valvar aortic stenosis. Follow-up, although brief to date, suggests that relief of stenosis and development of aortic regurgitation after balloon dilation or surgery are comparable.

Acknowledgments

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References


**KEY WORDS** • balloon dilatation • heart defects, congenital
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