Percutaneous Balloon Valvuloplasty in Patients With Severe Aortic Stenosis and Low Ejection Fraction

Immediate Results and 1-Year Follow-up

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The efficacy, morbidity, and 1-year follow-up of balloon aortic valvuloplasty in patients with low ejection fraction (<40%) were studied on a consecutive series of 55 patients (mean age, 77 years) treated from September 1985 to February 1987. Because of their age (20 patients >80 years old), poor left ventricular function, and associated diseases, 45 patients were definitely not surgical candidates. Balloon dilatation with 15–23-mm diameter balloon catheters decreased the transvalvular gradient from 66±24 to 28±14 mm Hg (p<0.001) and increased the valve area from 0.47±0.15 to 0.83±0.27 cm² (p<0.001). Immediately after dilatation, ejection fraction mildly increased from 29±7% to 34±9% (p<0.001) in 38 patients who had undergone a second left ventricular angiogram after dilatation. No significant change in the degree of aortic regurgitation was found after the procedure. Three patients died in hospital (femoral arterial complications in two, septicemia in one). Immediate clinical improvement was noted in 80% of the patients. During the follow-up (mean, 11 months), 22 patients died (heart failure in 15 patients, sudden death in five patients, myocardial infarction in one patient, cancer in one patient). Thirty patients survived, 21 with persistent clinical improvement. Repeat cardiac catheterization was performed at 6 months in 20 patients, of whom eight had recurrence of symptoms. Nine patients had restenosis: their hemodynamic indexes had returned to prevalvuloplasty values. In the 11 patients with no restenosis, aortic valve area was 0.73±0.14 versus 0.48±0.13 cm² before dilatation, and the ejection fraction had increased from 39±10% to 52±10% (p<0.05). From this series, which represents our early experience in balloon aortic valvuloplasty, we conclude that when the increase in valve area is maintained for several months, clinical improvement in functional status is substantial and that ejection fraction may increase to almost normal levels in some patients. One-year follow-up shows a high secondary mortality rate that can be related to the severity of left ventricular impairment and to either insufficient valve dilatation or subsequent restenosis. (Circulation 1989;79:1189–1196)

Patients

From September 1985 to March 1987, balloon aortic valvuloplasty was performed in 218 consecutive patients in our institution. Of this population,
55 consecutive patients (38 men and 17 women) were reviewed for this study because of a prevalvuloplasty left ventricular angiographic ejection fraction below 40% (mean, 29±7%). Their mean age was 77±9 years, (mean, 20 patients older than 80 years of age.

All patients were markedly disabled. Fifty-two patients had marked dyspnea (New York Heart Association functional Class III or IV). Angina was present in 24 patients, and syncope with exercise was present in 15 patients.

Twenty-one patients exhibited refractory congestive heart failure despite intensive medical therapy, including an infusion of dobutamine in two patients at the time of catheterization. Balloon valvuloplasty was proposed to the patient in the same setting of the diagnostic cardiac catheterization.

Coronary arteriography was performed after the valvuloplasty in 17 of 24 patients with angina. Seven patients had normal results on the coronary angiogram, two exhibited single-vessel disease, one had double-vessel disease, and seven had triple-vessel disease. Because of their critical status or because of their age (>80 years), coronary angiography was deliberately not performed in the 31 patients without angina and in seven patients with angina.

**Catheterization Protocol**

The protocol has been previously published. Briefly, right heart catheterization was performed with a 7F Swan-Ganz thermodilution catheter placed in the left femoral vein, and left heart catheterization was done in most cases with a Sones catheter (USCI, Billerica, Massachusetts) placed in the femoral artery (n=52) or right humeral artery (n=3).

After placement of all catheters, right atrial, pulmonary arterial, systemic arterial, and left ventricular pressures were measured. Aortic and left ventricular pressures were recorded simultaneously on a Siemens Mingograph for evaluation of the peak to peak gradient and for further computation of the mean aortic valve systolic gradient. After pressures were recorded, the cardiac output was determined by the thermodilution technique (mean of at least three consecutive measurements), and right anterior oblique left ventricular angiography was subsequently performed.

After removal of the left ventricle catheter, a valvuloplasty balloon catheter (Mansfield, Boston, Massachusetts) was introduced transcutaneously and was positioned across the valve. The diameter of the first balloon used was 8 mm in one patient, 12 mm in two patients, 15 mm in 35 patients, 18 mm in eight patients, and 20 mm in three patients. A newly designed dilatation catheter with a double size step balloon (15 then 20 mm) was used in six patients. Three successive inflations, of 60-seconds duration if blood pressure was maintained above 60 mm Hg, were performed by hand injection. After the third inflation, the first balloon catheter was exchanged for a larger one, except when the first catheter was 18 or 20 mm in diameter. To obtain a further decrease of the gradient, the largest balloon diameter used was 15 mm in five patients, 18 mm in 16 patients, 20 mm in 30 patients, and 23 mm in one patient. In three patients, two balloons (12 and 15 mm) were inflated together side by side. After the last inflation, the balloon catheter was withdrawn, and pressures were repeatedly measured, and a second left ventricular angiogram was performed in 38 patients. The catheter was subsequently pulled back to the ascending aorta, and supravalvular angiography was again performed.

**Follow-up**

The patients’ follow-up status was determined either during an outpatient examination or by phone (or questionnaire) twice a year during January and July. Because of this systematic follow-up schedule, all patients were contacted approximately between 1 and 18 months twice a year. All questionnaires included information on early balloon aortic valvuloplasty (first 2 months) and present clinical status. For patients who were dead at the time of the survey, the attending physician or the family was contacted to identify the exact date of death and its probable cause. For these patients, the mean interval between valvuloplasty and death was 6±5 months. For the surviving patients, the clinical status was assessed 12±5 months after the valvular dilatation.

The severity of dyspnea was assessed by the NYHA Classification, and the severity of angina was evaluated according to the Canadian Heart Association Classification.

Twenty patients were recatheterized 6±3.7 months after the procedure (range, 2–15 months). Twelve patients were systematically restudied despite persistent clinical improvement, whereas eight patients had recurrence of symptoms, which was the reason for the second hemodynamic study.

**Hemodynamic and Angiographic Measurements**

Aortic and left ventricular pressure recordings at 100 mm/sec on the same scale were digitized on a HIPAD digitizing table connected to a PD P11 Digital computer. Mean systolic gradient was computed to calculate the valve area by the Gorlin formula.

Left ventricular end-diastolic and end-systolic silhouettes were traced from the 35-mm film and subsequently digitized to compute the volumes by a program derived from the Simpson rule, and the ejection fraction was then calculated.

**Statistical Analysis**

Mean±SD was calculated for all variables. The paired t test was used to compare variables before and after valve dilatation. In patients who underwent repeat catheterization, an analysis of variance was done to compare data before balloon aortic valvuloplasty immediately after and at
TABLE 1. Hemodynamic Variables Before and After Balloon Aortic Valvuloplasty

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>After valvuloplasty</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>RAP (mm Hg)</td>
<td>6.9±5.4</td>
<td>6.4±5.0</td>
<td>NS</td>
</tr>
<tr>
<td>SAP (mm Hg)</td>
<td>59±25</td>
<td>43±17</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PCW (mm Hg)</td>
<td>22±11</td>
<td>16±9</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>25±12</td>
<td>19±10</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVS (mm Hg)</td>
<td>185±29</td>
<td>159±24</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AoSP (mm Hg)</td>
<td>120±24</td>
<td>134±26</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Peak gradient (mm Hg)</td>
<td>66±24</td>
<td>28±14</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CI (l/min/m²)</td>
<td>2.26±0.7</td>
<td>2.40±0.7</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>AoVA (cm²)</td>
<td>0.47±0.15</td>
<td>0.83±0.27</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVEDV (ml/m²)</td>
<td>118±30</td>
<td>104±33</td>
<td>NS</td>
</tr>
<tr>
<td>EF (%)</td>
<td>29±7</td>
<td>34±9</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

RAP, mean right arterial pressure; SAP, systolic arterial pulmonary pressure; PCW, mean pulmonary capillary wedge pressure; LVEDP, left ventricular end-diastolic pressure; LVS, left ventricular systolic pressure; AoSP, aortic systolic pressure; Peak gradient, peak to peak left ventricular-aortic systolic pressure gradient; CI, cardiac index; AoVA, aortic valve area computed by the Gorlin formula; LVEDV, angiographic left ventricular end-diastolic volume; EF, ejection fraction.

Results

Initial Hemodynamic and Angiographic Evaluation

In all patients, hemodynamic measurements have confirmed the presence of severe aortic stenosis with advanced heart failure (Table 1). Peak to peak left ventricular and aortic systolic gradient was 66±24 mm Hg. Cardiac index was 2.25±0.67 l/min/m² and below 2 l/min/m² in 20 patients. Aortic valve area was 0.47±0.15 cm². Nineteen patients had a gradient below 50 mm Hg, but because their cardiac output was very low, their aortic valve area was severely reduced (0.51±0.15 cm²). Mean left ventricular end-diastolic volume index was 102±28 ml/m², and left ventricular angiographic ejection fraction was 29±7%. Supravalvular aortograms obtained in 51 patients showed no or grade 1 aortic regurgitation in 45 patients, grade 2 in five patients, and grade 3 in one patient. Grade 1 mitral regurgitation was shown in 18 patients and grade 2 in three patients.

Results of Valvuloplasty

After valvuloplasty, peak to peak systolic gradient decreased from 66±24 to 28±14 mm Hg (p<0.001) (Figure 1, Table 1). In only two patients, the gradient remained above 50 mm Hg. Cardiac output increased slightly but significantly from 2.26±0.70 to 2.40±0.65 l/min/m² (p<0.01). Aortic valve area increased by 82% from 0.47±0.15 to 0.83±0.27 cm² (p<0.001).

Hemodynamic improvement was also confirmed by the significant decrease in systolic pulmonary pressure from 59±25 to 43±17 mm Hg (p<0.001). In the 38 patients who could undergo postvalvuloplasty left ventricular angiography, there was a significant increase in angiographic ejection fraction from 29±6% to 35±10% (p<0.001). Eighteen patients had an increase in ejection fraction greater than 5%, and 20 patients had no change or a decrease in ejection fraction. Among the 48 patients who had a supravalvular aortogram performed after valvuloplasty, no change in the severity of aortic regurgitation was seen in 40. Four patients had a grade 3 aortic insufficiency when they had either grade 1 (n=2) or grade 2 (n=2) before.

![Figure 1. Plots of changes in pressure gradient, valve area, cardiac index, and ejection fraction after balloon aortic valvuloplasty (BAV).](http://circ.ahajournals.org/)

follow-up. Actuarial survival rate was calculated by the life table method.
Complications

There was no death during the procedure. In one patient, a transient stroke occurred during the procedure, but the patient fully recovered 3 days later. Seven patients exhibited femoral arterial complications, and four of these required surgery. Three patients died during the hospital stay, one from endocarditis (76 years old) and two after arterial surgical repair (79 and 91 years old). No post-mortem examination could be performed.

Clinical Follow-up

Follow-up was obtained in all 52 patients discharged from the hospital. Mean follow-up was 11±5 months. The longest follow-up was 18 months. Twenty-one patients had more than 1 year follow-up.

Early follow-up (first 2 months). In 47 of 52 patients, marked improvement in dyspnea or angina was noted in the first weeks after valvuloplasty. Six patients died within 2 months. Five of these patients who had no improvement in their functional status died because of persistent heart failure. For four of these five patients, because of their age (77, 79, 81, and 85 years) and associated severe coronary disease in two, respiratory insufficiency in one, and severe peripheral artery disease in one, surgery could not be considered. The fifth patient was 75 years old and could have been operated on. The last patient who died within 2 months had a previous history of anterior myocardial infarction and multi-vessel nongraftable coronary disease; his ejection fraction was 30%. He died of a second myocardial infarction 15 days after the procedure.

Late follow-up. Forty-six patients were observed after 2 months (Figure 2). There were 16 deaths due to cardiac insufficiency (10 patients) or sudden death (five patients). One death was related to cancer. Two of these 16 patients had had repeat aortic valve dilatation. The actuarial survival curve (Figure 3) shows a survival rate of 85% at 1 month, 70% at 6 months, 60% at 1 year, and 55% at 18 months.

Thirty patients were alive with a mean delay of 12±5 months. Nine patients had worsening or recurrence of symptoms (dyspnea class III or IV in nine patients, angina in five, and syncope in one patient). Twenty-one patients exhibited persistent, clinical improvement. Comparisons of clinical, initial hemodynamic, and valvuloplasty results between patients who died and surviving patients are listed in Table 2. Valve areas or ejection fractions before and after dilatation were not different in these two groups, and cardiac index was the only variable significantly different between these two groups; patients died having the lower cardiac index (2.04±0.6 vs. 2.43±0.7 l/min/m², p<0.05).

Repeat Cardiac Catheterization

A second hemodynamic evaluation could be performed in 20 patients 6±4 months after the initial dilatation. Eight patients were recatheterized for symptoms and 12 for angina pectoris. Valvuloplasty was performed in 15 patients, and surgical replacement was indicated in nine.

TABLE 2. Clinical and Hemodynamic Variables of Dead and Alive Patients

<table>
<thead>
<tr>
<th>Variable</th>
<th>Deaths (n=25)</th>
<th>Alive (n=30)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥75</td>
<td>19</td>
<td>17</td>
<td>NS</td>
</tr>
<tr>
<td>&lt;75</td>
<td>6</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td>5/8</td>
<td>5/9</td>
<td>NS</td>
</tr>
<tr>
<td>AVA base (cm²)</td>
<td>0.44±0.27</td>
<td>0.50±0.16</td>
<td>NS</td>
</tr>
<tr>
<td>AVA after BAV (cm²)</td>
<td>0.81±0.27</td>
<td>0.84±0.27</td>
<td>NS</td>
</tr>
<tr>
<td>EF base (%)</td>
<td>30±7</td>
<td>29±6</td>
<td>NS</td>
</tr>
<tr>
<td>EF post BAV (%)</td>
<td>34±9</td>
<td>34±8</td>
<td>NS</td>
</tr>
<tr>
<td>CI base (l/min/m²)</td>
<td>2.04±0.60</td>
<td>2.43±0.70</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

AVA, aortic valve area computed by the Gorlin formula; base, baseline; BAV, balloon aortic valvuloplasty; EF, ejection fraction; CI, cardiac index.
ized because they were symptomatic again, and 12 patients were systematically restudied, though they were doing well. Restenosis was defined as more than 50% loss of the gain in valve area obtained by valvuloplasty. By that definition, nine patients (45%) had restenosis (six of the eight patients with symptoms again), and 11 did not have restenosis.

In patients with restenosis, the aortic valve area had almost returned to prevalvuloplasty values (0.50±0.27 cm² initially vs. 0.58±0.20 at control, NS) (Table 3). Despite restenosis, the ejection fraction had increased from 34±3% to 43±11% (NS). The hemodynamic variables of the patients with no restenosis are shown in Figure 4. Aortic valve area at control (0.73±0.17 cm²) was comparable to the postvalvuloplasty result (0.74±0.15 cm², NS). Peak to peak systolic pressure gradient had increased from 38 mm Hg after valvuloplasty to 54 mm Hg at control (p<0.01). This increase was due to the improvement in ventricular function as shown by the increase in cardiac index from 2.6±0.4 to 3.1±0.8 l/min/m² (p<0.05) and by the marked rise in ejection fraction from 39±10% after valvuloplasty to 52±10% at control (p<0.01) in the eight patients with a left ventricular angio gram. No significant difference in immediate postvalvuloplasty variables could be found between patients that did and did not have restenosis, in particular, the aortic valve area measured immediately after dilatation was not different (0.88±0.40 in patients with restenosis vs. 0.74±0.15 cm² in patients without restenosis, NS).

Ten patients had a redilatation procedure performed, five with restenosis and five with no restenosis who underwent the procedure because of insufficient primary results. For the whole group, the aortic valve area increased from 0.65±0.22 to 0.77±0.21 cm² (p<0.05) after the second valvuloplasty, and the postdilatation area was similar to the valve area obtained after the first dilatation (0.82±0.38 cm², NS). Patients with and without restenosis had similar results.

One patient, aged 66 years, with restenosis had elective valve replacement without complications 6 months after the initial valvuloplasty.
Discussion

Left ventricular failure is a common complication in patients with severe aortic stenosis. A mild decrease in ejection fraction does not necessarily imply severe myocardial dysfunction, but an ejection fraction below 40% is most often a sign of severely impaired left ventricular function. Most studies are in agreement regarding the increased risk of these patients at surgery, although it is also established that the depression of ejection fraction may be reversible after aortic valve replacement. However, because of the severity of the clinical status of our 55 patients, their advanced age (77±9 years), and associated diseases (diabetes, coronary atherosclerosis, and renal or respiratory insufficiency), 45 of these patients could not be considered for aortic valve replacement, and the 10 others were at high surgical risk. Therefore, aortic valvuloplasty was offered as the only therapeutic possibility.

Immediate Results

In this series, the percent increase in valve area was 82%, and the post valvuloplasty valve area was 0.83±0.27 cm² with 20 patients having a valve area less than 0.7 cm². This is comparable to the 90% increase obtained in our first 66 patients with normal left ventricular function. However, in patients with a low cardiac output, the accuracy of the Gorlin formula is decreased and in that situation, this formula seems to systematically underestimate the true valve area. Therefore some of the valve areas after dilatation could actually be larger.

The low mortality rate during the procedure despite the severe condition of these patients has to be emphasized. The 5% mortality rate in hospital (three patients) should compare favorably with the surgical mortality rate. In fact, no real comparison with surgery is possible because 45 of these patients were not considered suitable for valve replacement, whereas the 10 remaining patients were high risk. Concerning age only, in one recent published surgical series, 30% of octogenarian patients died early after aortic valve replacement. Concerning patients with aortic stenosis and low ejection fraction, in four surgical series, though the mean ages of patients were clearly lower in three (59, 62, and 65 years) than in our series and although their condition was less critical, the mortality rate was 15%, 21%, 25%, and 20%, respectively. Undoubtedly, however, the surgical risk has decreased in recent years. The most recently published surgical series on aortic stenosis with low ejection fraction (20 patients) gives a 5% mortality rate, but the patients were clearly younger (mean age, 66 years), and all had normal coronary arteries.

In 18 out of our 38 patients who had a second left ventricular angiogram performed immediately after valvuloplasty, ejection fraction significantly rose immediately after dilatation from 28±6% to 39±9%, which is a 40% increase (p<0.001). This seems to confirm that in these cases afterload mismatch was the principal cause of the decreased left ventricular ejection fraction. For the other patients in whom no significant change in ejection fraction could be found, other mechanisms for alteration of myocardial function (i.e., myocardial hypertrophy, diffuse coronary disease, coexistent cardiomyopathy) and insufficient valvular dilatation could explain the absence of immediate improvement in systolic function.
Clinical Follow-up Results

The spontaneous course of hemodynamically severe aortic stenosis is known to be malignant. A 40% survival rate 1 year after cardiac catheterization in patients much younger (mean age, 44 years) than those of our series has been reported.2 More recently, O’Keefe et al22 reported that in a group of 50 elderly candidates for aortic valvuloplasty of whom only 22 were considered high surgical risk, the 1 year spontaneous death rate was 43%. Taking into consideration that our patients were either not operable (40 of 55 patients) or operable only at a very high risk (10 of 55 patients) because of age (mean, 77 years), advanced condition, and associated disease, our 60% survival rate at 12 months and the 74% reported by Safian et al23 suggests that valvuloplasty may give encouraging results compared with spontaneous evolution of their valve disease.

Long-term follow-up after aortic valve replacement has been extensively studied in patients younger than 70 years old with normal left ventricular function, and a 1 year survival rate of 85% is commonly cited.24,25 Preoperative left ventricular dysfunction seems not to impair the long-term prognosis of patients younger than 70 years old18,20,26 because complete reversibility of the dysfunction is obtained in more than 70% of the patients.4,18,20 Conversely, the postoperative prognosis in elderly patients seems to be related to the preoperative clinical status. In a recent surgical series of patients with a mean age of 73 years, 42% of the patients in NYHA Class IV (which indicates a poor left ventricular contractility in most cases) died later after valve replacement compared with 12% of the patients who were in NYHA Class II.27 The high cardiac-related mortality rate in our series may be related to the old age of 77 years of our patients. It can also be speculated that the increase in valve area obtained by valvuloplasty was not sufficient to ensure recovery of long-term, severely impaired left ventricular function, but this may not be a sufficient reason because the patients who died had the same post-valvuloplasty area and a similar increase in ejection fraction than did the patients who survived.

Reversibility of left ventricular dysfunction does not always occur after valve replacement. Of 23 patients operated on for aortic stenosis with a low ejection fraction (<45%), Redeker et al20 showed no significant late improvement in ejection fraction in nine (40%). These investigators presumed that in this subgroup of patients, impaired myocardial function resulted from long-standing pressure-overload hypertrophy. The excessive myocardial hypertrophy as a reason for late failures of aortic valve replacement has been pointed out also by our group.4 Although we did not measure the left ventricular mass in the present study, the patients’ age and long-term evolution of their valve disease are strong arguments for the development of long-standing severe myocardial hypertrophy that could lead to an absence of improvement of myocardial contractility despite significant aortic valve enlargement by valvuloplasty.

Hemodynamic Follow-up

Postvalvuloplasty restenosis rate is not yet established at the present time. Of our 20 recatheterized patients, nine (45%) exhibited restenosis that is similar to the clinical restenosis rate of 44% at 6 months found by Safian et al.23 These nine patients had no improvement in ejection fraction, and six had recurrence of symptoms. In contrast, the 11 patients without restenosis exhibited at the time of recatheterization 6 months later a marked increase in ejection fraction (from 29% to 52%), which is comparable to that observed in the surgical series after aortic valve replacement in previous studies.4,5,17,18,21 Safian et al12 have also shown a significant rise in ejection fraction after balloon aortic valvuloplasty in almost 50% of their 28 patients with low ejection fraction. In contrast to our results, they could not find any differences in aortic valve areas between patients with a secondary rise in ejection fraction and patients without changes in ejection fraction. This discrepancy between our study and others may be due to the level of decrease in ejection fraction (<50% vs. <40%), the timing of repeat study (3 months vs. 6 months), the method of evaluation of ejection fraction (radionuclide angiography vs. contrast angiography) and the method of analyzing the valve area at follow-up (Doppler vs. hemodynamic analysis).

For the 10 patients who had readilation either because they had restenosis or because the initial result was not good, the increase of the valve area was slight, from 0.65 to 0.77 cm², and represented a 20% increase, which suggests that a second valvuloplasty procedure does not clearly improve the aortic valve area.

Limitations

Systematic invasive or noninvasive evaluation of the 35 patients who declined to return to repeat catheterization or died before 6 months would have strengthened the present study. However, their clinical follow-up records were examined. If we assumed that all the patients who died within 6 months (n = 17) and that all the patients with symptoms again during the same period of time (n = 8) had restenosis, our clinical restenosis rate would also be 47%, which is comparable to the hemodynamic restenosis rate in our 20 patients with repeat catheterization. Nevertheless, it should be recognized that the 20 patients restudied by cardiac catheterization may not be representative of the entire group.

Conclusion

These results indicate that aortic balloon valvuloplasty is a low-risk procedure in patients with aortic stenosis and severe left ventricular impairment who either are high surgical risk candidates or...
cannot be considered for surgery. With this technique, a significant enlargement in valve area is obtained, which is accompanied in most cases by an increase in ejection fraction and a decrease in pulmonary pressure. Consequently, immediate improvement in symptoms occurs in most of the patients and persists 1 year later in 40% of the patients. Late mortality remains high in these patients with advanced disease. Restenosis could explain some of the late deaths. Insufficient dilatation of the valve in this series, which represents the early experience of this new technique, could be also one of the reasons for these follow-up results.

Although the exact place of the valvuloplasty procedure in the treatment of aortic stenosis is yet undetermined, the immediate clinical improvement in most of the patients of this series suggests that in patients with poor left ventricular function for whom surgery is not definitely contraindicated, this technique should be considered at least as a palliative procedure to temporarily improve their status and perhaps decrease the operative risk. Therefore, further investigations in this indication need to be carried out.

References


Key Words: aortic stenosis • valvuloplasty • ejection fraction
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