Progressive Improvement in Pulmonary Vascular Resistance After Percutaneous Mitral Valvuloplasty

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Percutaneous mitral valvuloplasty has been proposed as a nonsurgical technique for treating high-risk patients with mitral stenosis who are deferred from mitral valve replacement. The effect of this technique on patients with pulmonary hypertension, however, has not been fully evaluated. Accordingly, serial assessment of pulmonary vascular resistance was made in 14 patients with critical mitral stenosis and pulmonary hypertension (pulmonary vascular resistance >250 dynes · sec/cm² or mean pulmonary artery pressure >40 mm Hg or both) who underwent percutaneous balloon dilatation of the mitral valve. Balloon valvuloplasty was performed with either one (n=10) or two (n=4) balloons through the transseptal approach, and it resulted in significant improvement in mean mitral gradient (from 18±4 to 9±4 mm Hg, p<0.001), systemic blood flow (from 3.7±1.2 to 5.0±2.2 l/min, p<0.001), and calculated mitral valve area (from 0.7±0.2 to 1.6±0.7 cm², p<0.001). Immediately after balloon mitral valvuloplasty, pulmonary vascular resistance fell from 630±570 to 447±324 dynes · sec/cm². Repeat catheterization 7±4 months after valvuloplasty showed further improvement of pulmonary hypertension in 12 of the 14 patients, with a mean pulmonary vascular resistance for the group as a whole of 280±183 dynes · sec/cm², p<0.005. In two patients, mitral valve restenosis to a mitral valve area less than 1.0 cm² was associated with a return of pulmonary hypertension to predilatation values. We conclude that balloon mitral valvuloplasty results in an immediate decrease in pulmonary vascular resistance, followed by progressive improvement in pulmonary hypertension in most patients. The improvement in pulmonary hypertension, however, may be transient in some patients who develop critical valvular restenosis. (Circulation 1989; 79:1061–1067)

Pulmonary hypertension frequently complicates mitral stenosis and may significantly influence long-term prognosis.1–7 The increase in pulmonary arterial pressure is often out of proportion to the degree of left atrial hypertension, reflecting a major increase in pulmonary vascular resistance. Surgical decompression of the left atrium through mitral commissurotomy or mitral valve replacement has often yielded marked regression of pulmonary hypertension.2,7–15

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Supported in part from research training Grant HL-07394 from the US Public Health Service.

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Percutaneous balloon mitral valvuloplasty has been proposed as a technique for treating high-risk patients with mitral stenosis who were deferred from surgical intervention.16–23 However, the long-term effects of this technique on pulmonary hypertension have not been well established. Accordingly, the purpose of this study was to assess the immediate and long-term effects of percutaneous balloon mitral valvuloplasty in patients with mitral stenosis and severe pulmonary hypertension.

Methods

Between October 1, 1985, and March 1, 1988, 73 patients underwent percutaneous balloon mitral valvuloplasty. Fourteen of these patients (eight women and six men) with longstanding calcific mitral stenosis had pulmonary hypertension (pulmonary vascular resistance >250 dynes · sec/cm² or mean pulmonary artery pressure >40 mm Hg or both) before
valvuloplasty and underwent repeat catheterization a mean of 7±4 months after the valvuloplasty. The mean age of this subset was 63±14 years (range, 40–81 years). Each patient had undergone baseline cardiac catheterization and coronary angiography. The origin of mitral stenosis was presumed to be rheumatic in all cases. One patient had concurrent critical aortic stenosis, and a second patient had previously undergone aortic valve replacement. All patients had severe mitral stenosis with fluoroscopic evidence of extensive mitral valve calcification. Eight patients had normal coronary arteries; two patients had one-vessel coronary disease; three patients had two-vessel coronary disease; and one had three-vessel coronary disease. All patients were symptomatic with heart failure, fatigue, and weakness. Clinical characteristics of the patients are summarized in Table 1.

**Surgical Evaluation Before Valvuloplasty**

All patients included in this study were evaluated by a cardiac surgeon and were offered surgical intervention and an estimation of their expected operative mortality. Ten of the 14 patients were considered high-risk candidates with an expected operative mortality greater than 15%. Factors that contributed to the elevated risk in these 10 patients included heavily calcified mitral valve with extensive subvalvular calcification (n=10), severe pulmonary hypertension with pulmonary vascular resistance greater than 500 dynes · sec/cm$^5$ (n=7), weakened or debilitated condition (n=6), age greater than 70 years (n=5), the need for extensive additional surgery including coronary artery bypass grafting or aortic valve replacement or both (n=6), prior aortic valve replacement (n=1), and the need for urgent abdominal surgery for tumor resection (n=1). The remaining four patients were considered excellent surgical candidates and had an expected operative mortality in the range of 2–3% despite modest elevation of pulmonary vascular resistance greater than 200 dynes · sec/cm$^5$. After being informed of the potential risks and complications of mitral valvuloplasty, all patients subsequently declined surgical intervention and elected to undergo percutaneous valve dilatation.

**Mitral Valvuloplasty Protocol**

After administration of a local anesthetic, the left femoral artery and left and right femoral veins were instrumented with 8F Hemokit sheaths. Right and left heart catheterization with a 7F balloon flotation catheter and a 7F pigtail catheter, respectively, were performed from the left groin. During the right heart catheterization, an oximetry run was done with blood obtained from the superior vena cava and pulmonary artery. After placement of the right and left heart catheters, measurements were made of systemic arterial, left ventricular, pulmonary capillary wedge and pulmonary artery pressures, and pulmonary artery and left ventricular oxygen saturations. Pulmonary capillary wedge pressure was confirmed by aspirating blood from the wedge position and subsequently documenting an arterial oxygen saturation. Oxygen consumption was measured with a metabolic rate meter (Waters Instruments, Rochester, Minnesota). Baseline arterial PO$_2$ was recorded (Table 1).

After prevavuloplasty measurements were made, transseptal catheterization was accomplished by a standard technique with an 8F Mullins transseptal sheath and dilator (USCI, Billerica, Massachusetts) and Brockenbrough needle. After entry into the left atrium, the needle and dilator were removed, and a
7F balloon-tipped end-hole catheter (Critikon, Tampa, Florida) was advanced through the sheath to the left atrium. Left heart pressures and transvalvular gradient were then measured directly to corroborate the previously obtained wedge pressure measurements. The 7F balloon catheter was then advanced through the mitral valve to the left ventricular apex, followed by insertion of an 0.038 in. (0.097 cm), 300 cm long Teflon-coated exchange wire through the flow-directed catheter. The sheath and balloon were then removed, leaving only the guide wire in place.

Next, an 8F angioplasty catheter (Mansfield, Mansfield, Massachusetts) with an 8-mm balloon was advanced over the guide wire to the level of the interatrial septum and inflated with saline under pressure to dilate the septal opening. After removal of this catheter, a 9F balloon dilation catheter with a 25-mm balloon was advanced across the septum to the left ventricle and positioned across the mitral anulus. The balloon was subsequently inflated for 10–15 seconds with a mixture of saline solution and radiographic contrast medium (Angiovist, Berlex Laboratories, Wayne, New Jersey) by hand pressure. Repeat inflations were made until a waist in the balloon at the level of the mitral valve was noted to disappear. In four patients, single-balloon dilatation was followed by a double-balloon dilatation with two 20-mm balloons. Two guide wires were placed across the mitral valve with the use of a double-lumen Block catheter (Mansfield). Single 20-mm balloon catheters were advanced over each guide wire to the level of the mitral apparatus and were inflated simultaneously.

Immediately after balloon valvuloplasty, all hemodynamic measurements were repeated, including determination of transvalvular gradient and cardiac output. A right heart oximetry series was repeated to search for evidence of a new left-to-right shunt. Mitral valve area before and after valvuloplasty was calculated by the Gorlin formula with systemic flow for cardiac output, when appropriate. Pulmonary vascular resistance was calculated with the formula 

\[(PA - LA)/PBF\] \times 80, \]

where PA is mean pulmonary arterial pressure; LA is left atrial pressure, and PBF is pulmonary blood flow. The results are expressed in dynes \( \cdot \) sec/cm\(^5\).

Dilatation was regarded as successful if there was disappearance of a waist in the balloon(s) and a 50% or greater increase in the calculated mitral valve area.

**Serial Echocardiographic Studies**

Two-dimensional and Doppler echocardiograms were obtained 24 hours before and 48 hours after mitral valvuloplasty with an ATL Mark 600 echocardiograph (Bellevue, Washington). Severity of mitral regurgitation was assessed with pulsed Doppler echocardiography by interrogating the left atrium above both mitral leaflets with parasternal long-axis and apical long-axis, four-chamber and two-chamber views. The severity of mitral regurgitation was graded as trace, mild, moderate, or severe depending on the extension of the signal of mitral regurgitation from the mitral leaflets to the back wall of the left atrium.

**Follow-up Cardiac Catheterization**

Combined right and left heart catheterization in 12 patients and right heart catheterization alone in the remaining two patients was performed a mean of 7±4 months after the mitral valvuloplasty. Transseptal catheterization was not repeated (because entry into the left atrium was not necessary). In addition to making repeat measurements of oxygen consumption, an oximetry run was repeated to permit calculation of systemic and pulmonary blood flows in all patients. Mitral valve area was calculated with the Gorlin equation in the 12 patients with combined right and left heart catheterization, substituting systemic blood flow for cardiac output where appropriate. Pulmonary vascular resistance was calculated in all patients with the formula described above.

**Statistical Evaluation**

Mean values and standard deviations were determined for all variables. Variables measured before, immediately after mitral valvuloplasty, and at repeat catheterization were compared by analysis of variance. A \( p \) value less than 0.05 was considered significant.

**Results**

**Short-term Hemodynamic Changes Associated With Percutaneous Valvuloplasty**

Balloon dilatation was accomplished successfully in all 14 patients as evidenced by a decrease in mean mitral gradient from 18±4 to 9±4 mm Hg (\( p < 0.001 \)), an increase in calculated mitral valve area from 0.7±0.2 to 1.6±0.7 cm\(^2\) (\( p < 0.001 \)), and an increase in cardiac output (systemic blood flow) from 3.7±1.2 to 5.0±2.2 l/min (\( p < 0.001 \)) (Table 2). Immediately after valve dilatation, there was a decrease in left atrial pressure (from 26±6 to 15±5 mm Hg, \( p < 0.001 \)), pulmonary vascular resistance (from 630±570 to 447±324 dynes \( \cdot \) sec/cm\(^5\), \( p = \text{NS} \)), and mean pulmonary arterial pressure (from 51±17 to 40±14 mm Hg, \( p < 0.001 \)).

**Follow-up Hemodynamic Measurements**

At follow-up catheterization 7±4 months later, the calculated mitral valve area remained improved compared with area before valvuloplasty (1.3±0.7 vs. 0.7±0.2 cm\(^2\), \( p < 0.001 \)), as did the mitral valve gradient (11±5 vs. 18±4 mm Hg, \( p < 0.001 \)) and cardiac output (systemic blood flow) (4.3±1.2 vs. 3.7±1.2 l/min, \( p < 0.05 \)). Mean pulmonary capillary wedge pressure was 19 mm Hg compared with a mean left atrial pressure of 15 mm Hg immediately after valve dilation. Ten of the 12 patients who had repeat mitral valve area determinations showed some reduction in mitral valve area compared with
area immediately after valvuloplasty, with two patients (patients 2 and 3) showing mitral valve areas less than 1.0 cm². For the group as a whole, pulmonary vascular resistance fell to 280 dynes · sec/cm². Of note, there was a progressive decrease in pulmonary vascular resistance in 11 patients, with a return to predilatation values in the two patients who showed restenosis to a mitral valve area less than 1.0 cm². Repeat hemodynamic data before and after valvuloplasty and at follow-up are shown in Table 2.

### Presence of an Interatrial Shunt After Valvuloplasty

Serial right heart oximetry samples documented the presence of a right atrial oxygen “step-up” in three of the 14 patients (patients 1, 2, and 6) immediately after valvuloplasty; calculated pulmonary to systemic flow (Qp/Qs) ratios were 1.8, 1.1, and 1.2, respectively. These shunts were still present at follow-up catheterization.

### Serial Echocardiography

After percutaneous mitral valvuloplasty, the degree of mitral regurgitation assessed by Doppler echocardiography increased in five patients: from mild to moderate in patient 2, from not detectable to mild in patient 3, from trace to moderate in patient 6, from not detectable to mild in patient 8, and from mild to mild-to-moderate in patient 11.

### Clinical Follow-up

At the time of follow-up catheterization, 12 patients remained symptomatically improved; recurrence of symptoms occurred in the two patients (patients 2 and 3) with calculated mitral valve areas less than 1.0 cm² at 1 and 12 months after valvuloplasty, respectively. Patient 2 subsequently underwent mitral valve replacement, whereas patient 3 was treated with repeat balloon valvuloplasty, with symptomatic improvement.

### Discussion

Between October 1, 1985, and March 1, 1988, percutaneous mitral valvuloplasty was performed successfully in our laboratory in 73 patients with critical mitral stenosis with an improvement in mean mitral valve area from 1.0±0.4 to 1.9±0.7 cm² and an improvement in cardiac output from 4.2±1.2 to 5.0±1.6 l/min. Mean pulmonary artery pressure and pulmonary vascular resistance also improved in this group of patients from 39±15 to 31±12 mm Hg and from 335±351 to 277±232 dynes · sec/cm², respectively. The present study describes further changes in pulmonary vascular resistance in a subset of 14 patients with pulmonary hypertension who underwent repeat catheterization 7±4 months after dilatation.

The results of the present study suggest that there may be progressive improvement in pulmonary vascular resistance in certain patients with pulmonary hypertension who undergo successful balloon dilatation for treatment of mitral stenosis. The extent of resolution of pulmonary hypertension in any given individual is variable, with some patients showing normal pulmonary vascular resistances at follow-up catheterization, whereas others show a decreased but still abnormal pulmonary vascular resistance.

### Table 2. Hemodynamics Before, Immediately After, and at Follow-up Valvuloplasty

<table>
<thead>
<tr>
<th>Patient</th>
<th>MVA (cm²)</th>
<th>LA (mm Hg)</th>
<th>CO (l/min)</th>
<th>RA (mm Hg)</th>
<th>Mean PA (mm Hg)</th>
<th>PVR (dynes · sec/cm²)</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>0.6</td>
<td>1.4</td>
<td>1.2</td>
<td>34</td>
<td>26</td>
<td>9</td>
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<tr>
<td>2</td>
<td>0.6</td>
<td>1.0</td>
<td>0.7</td>
<td>22</td>
<td>19</td>
<td>21</td>
</tr>
<tr>
<td>3</td>
<td>0.8</td>
<td>1.2</td>
<td>0.5</td>
<td>25</td>
<td>18</td>
<td>34</td>
</tr>
<tr>
<td>4</td>
<td>0.6</td>
<td>1.8</td>
<td>1.0</td>
<td>21</td>
<td>19</td>
<td>18</td>
</tr>
<tr>
<td>5</td>
<td>0.5</td>
<td>1.1</td>
<td>NA</td>
<td>30</td>
<td>14</td>
<td>26</td>
</tr>
<tr>
<td>6</td>
<td>0.6</td>
<td>1.0</td>
<td>1.0</td>
<td>26</td>
<td>13</td>
<td>25</td>
</tr>
<tr>
<td>7</td>
<td>0.5</td>
<td>1.1</td>
<td>NA</td>
<td>28</td>
<td>17</td>
<td>15</td>
</tr>
<tr>
<td>8</td>
<td>1.0</td>
<td>2.2</td>
<td>2.0</td>
<td>18</td>
<td>15</td>
<td>13</td>
</tr>
<tr>
<td>9</td>
<td>0.5</td>
<td>1.2</td>
<td>1.5</td>
<td>25</td>
<td>10</td>
<td>13</td>
</tr>
<tr>
<td>10</td>
<td>0.3</td>
<td>1.3</td>
<td>1.3</td>
<td>37</td>
<td>10</td>
<td>15</td>
</tr>
<tr>
<td>11</td>
<td>0.7</td>
<td>1.9</td>
<td>1.2</td>
<td>22</td>
<td>11</td>
<td>26</td>
</tr>
<tr>
<td>12</td>
<td>0.8</td>
<td>2.3</td>
<td>1.4</td>
<td>33</td>
<td>11</td>
<td>24</td>
</tr>
<tr>
<td>13</td>
<td>0.9</td>
<td>1.6</td>
<td>1.1</td>
<td>21</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>14</td>
<td>1.2</td>
<td>3.6</td>
<td>3.1</td>
<td>23</td>
<td>18</td>
<td>20</td>
</tr>
<tr>
<td>Mean</td>
<td>0.7</td>
<td>1.6</td>
<td>1.3</td>
<td>26</td>
<td>15</td>
<td>19</td>
</tr>
<tr>
<td>SD</td>
<td>0.2</td>
<td>0.7</td>
<td>0.7</td>
<td>6</td>
<td>5</td>
<td>8</td>
</tr>
</tbody>
</table>

Cardiac output reported here represents systemic blood flow calculated by the Fick method.

MVA, mitral valve area; LA, left atrial pressure; CO, cardiac output; RA, right atrial pressure; PA, pulmonary artery pressure; PVR, pulmonary vascular resistance; Pre, before valvuloplasty; Post, after valvuloplasty; F/U, follow-up valvuloplasty.

*Pulmonary capillary wedge pressure was substituted for left atrial pressure.
resistance. It is unclear at this time if there will be further resolution of pulmonary hypertension with time in the patients who still have abnormal resistances at the time of repeat catheterization. It is clear, however, that restenosis of the mitral valve may be associated with a return of pulmonary hypertension to predilatation values.

Several mechanisms are believed to contribute to the development of pulmonary hypertension in patients with mitral stenosis. These include 1) passive retrograde transmission of elevated left atrial and pulmonary venous pressures into the pulmonary arterial vasculature, 2) reactive pulmonary arteriolar vasoconstriction, induced by pulmonary venous hypertension, and 3) induced morphologic changes in the pulmonary vasculature. The third mechanism has been described as an irreversible component of increased pulmonary vascular resistance. The improvement in pulmonary vascular resistance in the patients in this study is presumably related to the first two mechanisms, with balloon-induced improvement in mitral valve area and subsequent decompression of the left atrium and pulmonary venous beds. The persistence of abnormally high vascular resistances in some patients probably reflects incomplete valvar dilatation with persistence of moderate mitral stenosis but may be related to the third mechanism of irreversible morphologic changes in the pulmonary vasculature.

The hemodynamic findings associated with mitral valvuloplasty in the subgroup of 14 patients included in this study are similar to other previously reported studies in patients with critical mitral stenosis and heavily calcified valves. The predilatation valve area, the increase in mitral valve area, and the increase in cardiac output in this subgroup of patients are all smaller than have been reported previously in larger groups of patients including those with noncalcified, pliable valves. In part, this is related to the fact that balloon dilatation is less successful with heavily calcified valves. It may also be related to the fact that most patients in this study were treated with a single-balloon technique. In our study, three patients (21%) had greater than 50% reduction in the achieved increase in mitral valve area at follow-up catheterization. Only two of these patients had recurrence of symptoms (patients 2 and 3). However, nine patients (64%) had some reduction of calculated mitral valve area at follow-up catheterization compared with area immediately after valvuloplasty. The high restenosis rate of the mitral valve area at repeat catheterization has also been described previously and suggests that long-term valvular improvement is less likely in this subgroup of patients than in those with noncalcified, pliable valves.

Our results are consistent with the findings of Morrow and coworkers who reported in 1964 that closed mitral commissurotomy is most successful in patients with completely mobile valve leaflets. In their original report, 11 of 12 patients with completely mobile valve leaflets had normal postoperative left atrial pressure, whereas in seven patients with totally immobile valves, the left atrial pressure remained elevated in six of them because of significant residual stenosis or mitral regurgitation induced at operation. In our study, mitral regurgitation, assessed by Doppler echocardiography, increased in five patients (Table 3). In two of these patients (patients 2 and 3) there was recurrence of symptoms, reerelevation of left atrial pressure (assessed by pulmonary capillary wedge pressure at follow-up catheterization), restenosis of the mitral valve to the prevulvaloplasty calculated area, and failure of further regression (and, in fact, reerelevation) of pulmonary vascular resistance; the remaining three patients remained symptomatically improved and showed further regression of pulmonary vascular resistance despite the presence of some increase in mitral regurgitation compared with that before valvuloplasty.

Mitral valve replacement has been long considered the reference standard for the treatment of mitral stenosis. A variety of artificial valves are available, with hemodynamic studies performed 6–17 months after mitral valve replacement showing effective orifice areas (calculated by the Gorlin equation) from 1.7±0.3 (Lillehei-Kaster valves) to 3.1±0.8 cm² (St. Jude Medical prostheses). Foltz and coworkers showed recently the effect of mitral valve replacement in patients with pulmonary hypertension. In their study, 15 patients with mitral stenosis and pulmonary hypertension underwent mitral valve replacement, with improvement in mean pulmonary artery pressure from 46±5.5 to 30±5.3 mm Hg, left atrial pressure (assessed by pulmonary capillary wedge pressure) from 29±5.5 to 14±4.3 mm Hg, and pulmonary vascular resistance index from 741±373 to 542±281 dynes · sec/cm⁵ · m². Similarly, McIlhuff and coworkers reported that mitral valve replacement in 26 patients with a pulmonary arterial pressure greater than 50 mm Hg resulted in

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**Table 3. Analysis of Mitral Regurgitation by Doppler Echocardiography Before and After Valvuloplasty**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>mild-moderate</td>
<td>mild</td>
</tr>
<tr>
<td>2</td>
<td>mild</td>
<td>moderate</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>mild</td>
</tr>
<tr>
<td>4</td>
<td>mild-moderate</td>
<td>mild to moderate</td>
</tr>
<tr>
<td>5</td>
<td>mild</td>
<td>mild</td>
</tr>
<tr>
<td>6</td>
<td>trace</td>
<td>moderate</td>
</tr>
<tr>
<td>7</td>
<td>mild-moderate</td>
<td>mild</td>
</tr>
<tr>
<td>8</td>
<td>0</td>
<td>mild</td>
</tr>
<tr>
<td>9</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>10</td>
<td>trivial-mild</td>
<td>0</td>
</tr>
<tr>
<td>11</td>
<td>mild</td>
<td>mild-moderate</td>
</tr>
<tr>
<td>12</td>
<td>mild</td>
<td>mild</td>
</tr>
<tr>
<td>13</td>
<td>mild-moderate</td>
<td>mild-moderate</td>
</tr>
<tr>
<td>14</td>
<td>mild-moderate</td>
<td>mild-moderate</td>
</tr>
</tbody>
</table>

Pre, 24 hours before mitral valvuloplasty; Post, 48 hours after mitral valvuloplasty.
improvement in pulmonary capillary wedge pressure from 29±1 to 15±1 mm Hg and pulmonary vascular resistance from 491±59 to 245±28 dynes·sec/cm². They observed an operative mortality of 7.7%, with an additional death within 2 months of mitral valve replacement. The hemodynamic results from both these studies were similar to those reported in earlier studies2-7-15 that showed regression of pulmonary hypertension after mitral valve surgery.

Thus, there is a striking similarity in the improvement in left atrial hypertension and pulmonary hypertension after both mitral valve replacement and percutaneous balloon mitral valvuloplasty in patients with severe mitral stenosis. However, there is no doubt that the final mitral valve area achievable by valve replacement is higher than that obtained by balloon valvuloplasty in our series of patients with calcific mitral stenosis. As discussed above, calcified, nonpliable valves are more difficult to dilate, although even a small increase in mitral valve area in a patient with critical mitral stenosis probably can lead to marked hemodynamic improvement.

Given the variable improvement in mitral valve function, the relatively high restenosis rate, and the variable improvement in pulmonary vascular resistance in the patients in our study, the utility of mitral valvuloplasty in this subgroup of patients may be questioned. One possible use of mitral valvuloplasty in this setting is that it may provide for a temporary improvement in the patient’s hemodynamic status and thereby decrease the risk of subsequent surgical intervention. Earlier reports of mitral valve surgery showed that the presence of pulmonary vascular disease greatly affected the mortality associated with corrective valve surgery.33-35 This may reflect the fact that most of the reported series were done before the era of cardioplegic arrest. More recently, Camara and coworkers36 reported a 5.6% operative mortality and a 7.2% late cardiac death rate in 88 patients with severe pulmonary hypertension undergoing mitral valve replacement or open mitral commissurotomy. Others have also shown that the presence of severe complicating pulmonary vascular disease does not by itself contraindicate mitral valve replacement,8 but improvement in pulmonary hypertension before valve surgery probably would reduce perioperative morbidity and mortality.

A significant complication that occurred in the subgroup of patients that underwent mitral valvuloplasty in this study was the creation of an interatrial shunt. The appearance of this shunt in our laboratory has been associated primarily with the use of a single 25-mm balloon technique and presumably is related to the large deflated profile of the balloon dilatation catheter used for valve dilatation. Theoretically, the creation of an interatrial shunt in patients with critical mitral stenosis and pulmonary hypertension could result in deleterious hemodynamic changes secondary to the added hemodynamic burden on the patient’s right ventricle. Of note, however, pulmonary vascular resistance in all patients with atrial septal defects did improve progressively. With further refinement in mitral valvuloplasty equipment, particularly in the deflated profile of the valvuloplasty balloon, the occurrence of atrial septal defects will probably disappear.

The progressive increase in mitral valve area that occurred in one patient in the study group (patient 9) raises an important issue regarding the calculation of mitral valve area by the Gorlin equation in the presence of severe pulmonary hypertension. Previous studies have suggested that multiple factors can lead to discrepancies in the calculated versus the anatomic mitral valve area,37 including changes in orifice area associated with increasing cardiac output, the concept of “valve inertia” (some of the kinetic energy supplied by the opening pressure is absorbed in moving the stenotic valve itself), and the fact that the Gorlin constant may not be a constant in low flow states. In the case of mitral stenosis with elevated pulmonary vascular resistance, the calculation of mitral valve area by the Gorlin equation is done with total flow across “two stenoses” — the stenotic mitral valve and the pulmonary vasculature. Balloon mitral valvuloplasty increases the calculated and actual mitral valve area (the “first stenosis”), and has been shown to produce improvement in pulmonary hypertension. Given that total flow may increase further with resolution of pulmonary hypertension, there may be a further increase in the calculated mitral valve area. Of note, this has been documented previously in two recent studies of patients undergoing mitral valvuloplasty.28,38 This indicates that in some patients the mitral valve area calculated by the Gorlin equation immediately after valvuloplasty may improve after the resolution of pulmonary hypertension.

Conclusion

The present study shows that balloon mitral valvuloplasty in patients with calcific mitral stenosis and severe pulmonary hypertension has an immediate beneficial effect on pulmonary hypertension and that in most patients substantial further regression of pulmonary vascular disease can be expected. Mitral restenosis may be associated with a return of pulmonary hypertension to predilatation values. The variable improvement in mitral valve area and a significant rate of valvular restenosis in this subgroup of patients suggests a role for balloon mitral valvuloplasty as a preparative technique to improve pulmonary hypertension before surgical intervention.

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**KEY WORDS** • pulmonary hypertension • mitral stenosis
Progressive improvement in pulmonary vascular resistance after percutaneous mitral valvuloplasty.

Circulation. 1989;79:1061-1067
doi: 10.1161/01.CIR.79.5.1061

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