Subject Review

Percutaneous Balloon Valvuloplasty

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In the technique of percutaneous balloon valvuloplasty, one or more large balloons are inserted percutaneously and then inflated across a stenotic valve to decrease the degree of obstruction. Currently, the procedure is being performed for patients with pulmonic, mitral, or aortic stenosis. The results vary according to the type of valve and the age of the patient. In patients with pulmonic stenosis, balloon valvuloplasty can be performed safely and the results are excellent. Therefore, at many institutions it is the procedure of choice for the treatment of isolated pulmonic stenosis. In patients with mitral stenosis, the results depend on the morphologic features of the stenotic valve. In patients with highly calcified and fibrotic mitral valve leaflets, the risks of the procedure are increased and the results are suboptimal. In experienced hands, however, balloon valvuloplasty is excellent for patients with a pliable, noncalcified mitral valve or those for whom operation imposes an extremely high risk. The use of balloon valvuloplasty for aortic stenosis has been limited to the frail, elderly patient who either is not a surgical candidate or is at high risk for operation. Although mortality and restenosis rates are high on short-term follow-up, aortic balloon valvuloplasty provides palliation of symptoms in many patients who otherwise would have been unable to undergo any intervention. Long-term follow-up is necessary for determining the ultimate role of balloon valvuloplasty in cardiology.

In recent years, percutaneous balloon valvuloplasty has been added to the armamentarium available for treatment of valvular stenosis. Although surgical treatment has proved effective for relieving the physiologic abnormalities caused by stenotic valves, the risk of an open-heart surgical procedure in certain patients has been an impetus for development of a percutaneous method for relieving valvular stenosis. Percutaneous introduction and inflation of one or more large balloons across a stenotic valve can decrease the degree of obstruction with few complications. The effectiveness of percutaneous balloon valvuloplasty will vary according to the type of valve and the cause of the stenosis. As with any evolving technique, much remains to be learned about this therapy, especially about its long-term efficacy; thus, its ultimate role in interventional cardiology remains to be determined. Initial results, however, have been encouraging in certain groups of patients, and this procedure is being performed with increasing frequency in most large institutions.

In this review, we summarize the results of percutaneous balloon valvuloplasty for stenotic pulmonic, mitral, and aortic valvular lesions in the adult population and present our views of the clinical application of this technique.

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PULMONIC VALVE STENOSIS

Percutaneous balloon valvuloplasty was first attempted by Semb and associates in a newborn in 1979 and then by Kan and colleagues in an 8-year-old patient in 1982. Overall, excellent results have been achieved with pulmonic balloon valvuloplasty, and few complications have ensued. Because of the low risk-to-benefit ratio, pulmonic balloon valvuloplasty is considered the treatment of choice for severe, isolated pulmonic stenosis at many institutions.

Clinical Aspects.—Isolated pulmonic valvular stenosis has been one of the most common types of congenital heart disease in adults. Although most patients have only a mild degree of obstruction, occasionally patients in the fifth or sixth decade of life have profound right-sided failure due to previously unrecognized severe pulmonic stenosis. Diagnosis and treatment of this disorder can be gratifying, in terms of both relief of symptoms and prolongation of life.

The most common cause of isolated pulmonic stenosis is a congenitally malformed valve, typically consisting of a dome-shaped valve with a central opening and fused commissures. Less commonly, the valve is dysplastic and has three leaflets and a narrowed orifice. Secondary causes of pulmonic stenosis are rare but may include rheumatic involvement or carcinoid heart disease. Pulmonic stenosis in adults may be associated with complex congenital anomalies, such as tetralogy of Fallot or transposition of the great arteries. When the pulmonic valvular stenosis is severe and long-standing, secondary infundibular hypertrophy may develop and result in a dynamic right ventricular outflow obstruction.

Clinical symptoms develop in fewer than 25% of patients with pulmonic stenosis because the stenotic valve orifice tends to enlarge with an increase in body surface area. When symptoms do develop, severe stenosis is present and, in adults, is due to fibrosis, thickening, and calcification of the valve.

The symptoms of severe pulmonic stenosis are the result of right ventricular pressure overload. Patients will have shortness of breath, fatigue, and a low-output state as well as increased right-sided pressures that result in edema and ascites. Angina-like pain is frequently present, most likely attributable to the excessive myocardial oxygen demand of a hypertrophied right ventricle. In patients with severe pulmonic stenosis, severe tricuspid regurgitation, intractable right-heart failure, and death will eventually develop.

The diagnosis of pulmonic stenosis in the adult should be suspected when a loud systolic murmur is detected at the left upper sternal border and a right ventricular impulse is easily palpable. The electrocardiogram will demonstrate right atrial or right ventricular hypertrophy (or both), and the chest roentgenogram frequently will show a normal heart size but a prominent pulmonary artery shadow (Fig. 1). The diagnosis can be confirmed by two-dimensional and Doppler echocardiography, which will disclose right ventricular hypertrophy and a high velocity across the pulmonic valve.

In the past, the treatment of symptomatic, severe pulmonic stenosis was operative intervention. Although the topic is controversial, we believe that patients with asymptomatic, severe pulmonic stenosis should also probably undergo intervention to prevent progressive right-heart failure. Surgical valvotomy consists of the use of cardiopulmonary bypass and direct visualization of the pulmonic valve through an incision in the pulmonary artery. The commissures are then incised under direct visualization. Frequently, trivial pulmonary insufficiency results, but this condition is well tolerated. The long-term results of the operation have been excellent, with reduction of hypertrophy and sustained symptomatic relief.

Technique and Mechanism of Percutaneous Pulmonic Balloon Valvuloplasty.—Percutaneous balloon valvuloplasty of the pulmonic valve was initially performed successfully in young children with isolated pulmonic stenosis. By inflating a balloon with a diameter similar to that of the pulmonic annulus, the pulmonic valve gradient was decreased from about 80 mm Hg to 20 mm Hg with no change, or even an increase, in cardiac output. No major complications were reported. These initial re-
ports were soon followed by reports of other series of young patients with similar results (Table 1).13-18 After an initial report of a successful procedure,11 percutaneous pulmonic balloon valvuloplasty was then extended to adults with severe pulmonic stenosis due to calcification.30 This procedure has been lifesaving in the neonatal period when a critical degree of pulmonic stenosis was present.26,31 It has even eliminated the need for systemic-pulmonary shunts in children with complex cyanotic heart disease.19

Balloon valvuloplasty of the pulmonic valve is performed by cannulating the femoral vein and introducing catheters up through the inferior vena cava, across the right atrium and right ventricle, and into the pulmonary artery. After initial measurement of gradients and cardiac output, one or two large balloons are placed across the stenotic pulmonic valve, and several inflations are performed (Fig. 2). During the inflations, especially in the presence of near-systemic right ventricular pressures, severe systemic hypotension frequently occurs but reverses immediately after deflation of the balloons.

Initially, the maximal diameter of the balloons used was equal to the size of the pulmonic valve annulus.7,12 Subsequently, it was shown that the use of balloons with a diameter 20 to 30% larger than the annulus could be effective and safe.16,18,21,32 Because the largest balloon size is 25 mm in diameter, large patients frequently will require the use of two balloons.16,18,21 An “effective” valve orifice can be calculated from these two balloons by using the formula of Yeager33 (Table 2). A dual-balloon technique may allow the right ventricle to vent between the two balloons; thus, the incidence of severe hypotension is reduced.35,36

The mechanism of pulmonic balloon valvuloplasty has been studied by direct visualization of the pulmonic valve after a procedure.15,37-39 In most cases, cusp tearing or commissural splitting will occur, depending on the anatomy of the pulmonic valve. Occasionally, avulsion of the cusp from the annulus may occur.15,38 Such cusp avulsion is associated with infundibular hypertrophy that may cause retraction of the balloon during inflation.38 If the balloons are oversized, especially in adults with less compliant arteries, the pulmonic valve annulus may be disrupted, with resultant intimal and medial injury and intramural formation of hematoma.
Table 1.—Summary of Reported Results of Percutaneous Pulmonic Balloon Valvuloplasty

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>No. of patients</th>
<th>Age range</th>
<th>Before</th>
<th>After</th>
<th>Value</th>
<th>Time (mo)</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lababidi &amp; Wu</td>
<td>1983</td>
<td>23</td>
<td>10 mo-19 yr</td>
<td>113</td>
<td>32</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Lababidi et al</td>
<td>1984</td>
<td>18</td>
<td>11 mo-19 yr</td>
<td>81</td>
<td>23</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Rocchini et al</td>
<td>1984</td>
<td>7</td>
<td>1-9 yr</td>
<td>90</td>
<td>38</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Kan et al</td>
<td>1984</td>
<td>20</td>
<td>3 mo-50 yr</td>
<td>68</td>
<td>23</td>
<td>22</td>
<td>2-20</td>
<td>9</td>
</tr>
<tr>
<td>Walls et al</td>
<td>1984</td>
<td>39</td>
<td>10 mo-19 yr</td>
<td>69</td>
<td>21</td>
<td>23</td>
<td>5-16</td>
<td>11</td>
</tr>
<tr>
<td>Ali Khan et al</td>
<td>1986</td>
<td>32</td>
<td>6 mo-12 yr</td>
<td>99</td>
<td>23</td>
<td>16</td>
<td>6</td>
<td>14</td>
</tr>
<tr>
<td>Rao</td>
<td>1986</td>
<td>26</td>
<td>4 mo-20 yr</td>
<td>98</td>
<td>33</td>
<td>21</td>
<td>6-15</td>
<td>10</td>
</tr>
<tr>
<td>Radtke et al</td>
<td>1986</td>
<td>27</td>
<td>6 days-19 yr</td>
<td>65</td>
<td>16</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>*Rey et al</td>
<td>1988</td>
<td>51</td>
<td>...</td>
<td>73</td>
<td>22</td>
<td>22</td>
<td>1-17</td>
<td>...</td>
</tr>
<tr>
<td>*Mullins et al</td>
<td>1988</td>
<td>63</td>
<td>3 mo-76 yr</td>
<td>64</td>
<td>22</td>
<td>20†</td>
<td>6-30</td>
<td>30</td>
</tr>
<tr>
<td>*Rao et al</td>
<td>1988</td>
<td>41</td>
<td>7 days-20 yr</td>
<td>91</td>
<td>30</td>
<td>33</td>
<td>6-34</td>
<td>29</td>
</tr>
</tbody>
</table>

*Most recent comprehensive series.
†Doppler gradient (all others are catheterization gradients).

**Early Results of Percutaneous Pulmonic Balloon Valvuloplasty.**—After balloon valvuloplasty, a dramatic decrease in gradient and an improvement in cardiac output frequently occur (Fig. 3). A residual gradient from the right ventricle to the pulmonary artery of 25 to 40 mm Hg is often present (Table 1). In some cases of long-standing, severe pulmonic stenosis and resultant right ventricular hypertrophy, the relief of the pulmonic valve obstruction unmasks the presence of the infundibular obstruction. Thus, a significant gradient may still be present even though the pulmonic valve stenosis has been relieved. In this situation, the infundibular stenosis must be treated with aggressive fluid management and β-

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Fig. 2. Fluoroscopic images of 23-mm balloon placed across a stenotic pulmonic valve. A, Initial inflation demonstrates “waist” that appears in region of stenosis. B, Full inflation of balloon is evident after relief of stenosis. (From Cooke and associates.)
Table 2.—Effective Orifice Diameter
With Use of Two Balloons for
Percutaneous Valvuloplasty

<table>
<thead>
<tr>
<th>Diameter (mm)</th>
<th>Balloon 1</th>
<th>Balloon 2</th>
<th>Effective diameter (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>12.0</td>
<td>15.0</td>
<td></td>
<td>22.1</td>
</tr>
<tr>
<td>12.0</td>
<td>20.0</td>
<td></td>
<td>26.5</td>
</tr>
<tr>
<td>15.0</td>
<td>15.0</td>
<td></td>
<td>24.6</td>
</tr>
<tr>
<td>15.0</td>
<td>18.0</td>
<td></td>
<td>27.1</td>
</tr>
</tbody>
</table>

From Nishimura and associates. By permission of The American Heart Association.

Overall, pulmonic balloon valvuloplasty has been safe and associated with few complications. Pulmonary insufficiency does not seem to occur to a degree that would cause hemodynamic problems. Residual infundibular stenosis may be effectively treated with fluids and β-adrenergic blockade. Isolated reports have described complete heart block, disruption of the annulus, and vascular complications. In more than 250 cases of pulmonic balloon valvuloplasty reported in the literature, however, no mortality or serious irremediable complication has occurred.

Follow-Up.—Numerous studies have reported the intermediate-term follow-up of patients after percutaneous pulmonic balloon valvuloplasty. At 1 to 3 years of follow-up, no restenosis has been evident. In several studies, the gradient across the pulmonic valve was decreased at follow-up in comparison with immediately after the procedure—most likely

Fig. 3. Simultaneous tracings of right ventricular (RV) and pulmonary artery (PA) pressures before (left) and after (right) valvuloplasty, demonstrating substantial reduction in gradient. Simultaneous Doppler velocities across the pulmonic valve are also shown. (Illustration courtesy of Dr. James B. Seward, Division of Cardiovascular Diseases and Internal Medicine, Mayo Clinic.)
because of regression of the infundibular hypertrophy. Symptomatic improvement has been sustained with improvement in exercise tolerance. Although long-term studies are needed for substantiation of the full efficacy of this technique, pulmonic balloon valvuloplasty may be the procedure of choice in a patient with isolated, severe pulmonic valve stenosis. Currently, balloon valvuloplasty of the pulmonic valve is the only procedure that has been approved by the US Food and Drug Administration for clinical use.

MITRAL VALVE STENOSIS

Mitral balloon valvuloplasty, first introduced in 1984 by Inoue and associates, has evolved into an effective technique for the treatment of severe mitral stenosis in selected patients. Technically, it is a more difficult procedure than either pulmonic or aortic balloon valvuloplasty. The preoperative assessment of patients is particularly important for determining the potential results and complications of the procedure. If long-term results prove to be as effective as results obtained from surgical commissurotomy, mitral balloon valvuloplasty may become the procedure of choice for selected patients with severe, symptomatic mitral stenosis.

Clinical Aspects.—Isolated mitral stenosis in adults is almost always attributable to rheumatic heart disease. This disorder results in several physiologic processes, including the following: (1) fusion of commissures, (2) conversion of mitral leaflets into stiff and rigid membranes, and (3) fusion and shortening of the subchordal apparatus. The stenosis of the mitral valve may be caused by one or more of these abnormalities, with commissural fusion present in 75% of cases.

In a patient with mitral stenosis, the symptoms are due mainly to the increased left atrial pressure that results from the obstruction across the mitral valve. This pressure is reflected back into the pulmonary circulation and causes dyspnea, paroxysmal nocturnal dyspnea, and orthopnea. Atrial fibrillation may result from pressure overload in the left atrium, causing acute exacerbation of symptoms due to a rapid ventricular response. Left atrial thrombi are frequently present, especially in the presence of atrial fibrillation, and systemic emboli can be a devastating complication. Mitral stenosis is a disease of plateaus, with a 10- to 30-year latent period between the onset of rheumatic fever and the development of limiting symptoms. Once a patient with severe mitral stenosis begins to have limiting, debilitating symptoms, however, the downhill progression is rapid.

The diagnosis of mitral stenosis is based on the auscultatory findings of an increased intensity of the first heart sound and an opening snap and diastolic rumble. The severity of the stenosis may be determined by the interval between the second heart sound and the opening snap. As the stenosis progresses, a higher left atrial-to-left ventricular gradient results in a shorter second heart sound-to-opening snap interval. As calcification of the mitral valve increases, the intensity of both the first heart sound and the opening snap diminishes. Patients with severe, long-standing mitral stenosis will have a right ventricular lift and an increase in the intensity of the pulmonic component of the second heart sound as a result of the secondary pulmonary hypertension.

An electrocardiogram will frequently demonstrate left atrial enlargement (if the patient is in normal sinus rhythm) or atrial fibrillation. Right ventricular hypertrophy with right-axis deviation is present if pulmonary hypertension exists. The chest roentgenogram shows straightening of the left heart border, signifying left atrial enlargement (Fig. 4).

Two-dimensional echocardiography and Doppler echocardiography are most commonly used for diagnosis and quantitation of mitral stenosis. The typical “doming” deformity of the anterior leaflet of the mitral valve, with diastolic doming and restricted opening evident on two-dimensional echocardiography, is diagnostic of mitral stenosis (Fig. 5). The severity of mitral stenosis can be determined by using planimetry on the two-dimensional short-axis view as well as Doppler assessment of the mean gradient and diastolic half-time, from which the mitral valve area can be calculated. Coexistent
conditions, such as mitral regurgitation, aortic and tricuspid valve disease, left atrial thrombus, and pulmonary hypertension, can also be assessed.

Critical to the pre-valvuloplasty assessment of a patient with mitral stenosis is information about the degree and type of pathologic involvement of the mitral valve apparatus. In a patient with a pliable, noncalcified valve and no subvalvular involvement, excellent hemodynamic results can be obtained from either surgical commissurotomy or balloon valvuloplasty. If severe calcification, fibrosis, and subvalvular fusion are present, however, the possibility of a successful balloon valvuloplasty is considerably diminished. An echocardiography score, recently derived by using two-dimensional echocardiography, allows semiquantitation of this involvement (Table 3). This score is based on four variables: (1) leaflet mobility, (2) leaflet thickening, (3) degree of subvalvular involvement, and (4) degree of calcification. Each of these factors is assigned a score from 1 to 4, with 4 being the greatest involvement, and the scores are then totaled. Patients with a score of less than 8 are usually excellent candidates for balloon valvuloplasty, and those with a score of more than 10 have a higher possibility of having complications and a lower success rate.

In the past, the treatment of severe, symptomatic mitral stenosis has been surgical. When leaflets were pliable and noncalcified, closed commissurotomy was the initial procedure of choice; a transseptal finger fracture or transventricular dilator was used to split the commissures. With the advent of cardiopulmonary bypass, however, open commissurotomy under direct visualization became the preferred therapy. With open commissurotomy, atrial thrombi can be removed, and the commissures can be incised; if necessary, the fused chordae or papillary muscles can be separated. In the presence of calcified, rigid valves with subvalvular fusion or significant mitral regurgitation (or both), surgical commissurotomy seldom results in adequate hemodynamics, and replacement with a mitral valve prosthesis is performed.

Although a successful mitral valve commissurotomy produces excellent improvement in hemodynamics and relief of symptoms, clinical deterioration does occur over time because of restenosis. About 50% of patients will require repeat procedures by the end of 10 years, and the incidence of restenosis increases dramati-
Fig. 5. Diastolic still frame of two-dimensional echocardiogram in long-axis view, demonstrating typical "hockey stick" deformity of anterior leaflet and immobile posterior leaflet, which are diagnostic of mitral stenosis. $A = $ anterior; $AV = $ aortic valve; $I = $ inferior; $LA = $ left atrium; $LV = $ left ventricle; $P = $ posterior; $PW = $ posterior wall; $RV = $ right ventricle; $S = $ superior; $VS = $ ventricular septum.

Historically after 12 to 15 years. Repeat mitral commissurotomy is possible in some patients, but because of extensive calcification and fibrosis, mitral valve replacement is more frequently necessary for patients in whom restenosis develops.

Technique and Mechanism of Percutaneous Mitral Balloon Valvuloplasty.—Percutaneous mitral balloon valvuloplasty was first described by Inoue and associates in 1984; they successfully performed this procedure on five of six adults by using a newly developed "pillow-shaped" balloon placed across the mitral valve through a transseptal approach. The mean gradient across the valve was decreased from 14 mm Hg to 6 mm Hg without complications. In 1985, Lock and colleagues described a series of eight children and young adults, all of whom had a successful percutaneous mitral balloon valvuloplasty; the mitral valve area index was increased from 0.73 cm$^2$/m$^2$ to 1.34 cm$^2$/m$^2$. This article was soon followed by reports of successful procedures with use of different techniques in adults by Babic and co-workers from Yugoslavia and Al Zaibag and associates from Saudi Arabia.

In the United States, the initial procedures were performed only in elderly patients who had severely calcified valves and were high operative risks. With experience and short-term follow-up, however, the indications have shifted toward younger patients with less heavily calcified mitral valve leaflets.

The procedure of percutaneous mitral balloon valvuloplasty has several variations. At our institution, we use a transseptal approach through the right femoral vein (Fig. 6). This approach necessitates the use of a specially designed Mullins catheter to create a puncture in the area of the fossa ovalis, through which the guidewires are introduced into the left atrium and then subsequently across the mitral valve into the left ventricle. The ends of the guidewires are left curled in the apex of the left ventricle. After introduction of the guidewires, an 8-mm balloon or long dilator is advanced across the atrial septum to enlarge the opening so that the large balloons can be introduced into the left atrium. Then one or more larger balloons are passed along the guidewires across the mitral valve, and several inflations are performed.

Variations of this method have included use of a retrograde approach by which the guidewires are snared and brought out through the femoral artery; thus, the balloon or balloons can be introduced through the femoral arterial approach. Other approaches include the antegrade route with long transseptal sheaths through which the balloons are placed and placement of the guidewires from the left ventricle across the aortic valve into the aorta, to decrease the possibility of perforation. During inflation of the balloons, severe reduction in antegrade flow results in hypotension and ventricular ectopic beats. This hemodynamic deterioration necessitates inflations of
very short duration (generally less than 10 seconds). Usually, however, the hemodynamics improve immediately after deflation of the balloons. In the presence of severe pulmonary hypertension, acute right ventricular failure may occur because of the increase in afterload in an already failing right ventricle.

Commisural splitting is the mechanism by which mitral balloon valvuloplasty relieves stenosis, similar to open commissurotomy. This result has been substantiated by direct visualization of the effect of balloon inflation, either intraoperatively or on pathologic specimens. Two-dimensional echocardiograms have shown an increase in the angle of the anterior or the lateral commissure (or both). Fracturing of nodular calcifications within the leaflets results in increased valve mobility.

**Early Results and Complications of Percutaneous Mitral Balloon Valvuloplasty.** — The early results of percutaneous mitral balloon valvuloplasty, which have been published in numerous reports, are comparable to the results achieved by open commissurotomy. The mean gradient across the mitral valve usually decreases from 16 to 20 mm Hg before valvuloplasty to 3 to 9 mm Hg after the procedure. Frequently, cardiac output increases so that the mitral valve area is improved from about 1 cm² to more than 2 cm². Figures 7 and 8 illustrate the initial results published from several institutions in the United States. The pulmonary artery pressure and pulmonary vascular resistance decrease gradually during the 24 hours after the procedure.

Although the overall results of this procedure seem encouraging, the outcome of mitral balloon valvuloplasty depends on the underlying disease of the mitral valve. In the presence of a mobile, noncalcified mitral valve with no subvalvular fusion, the results are excellent; mitral valve areas of more than 2 cm² and mean gradients of less than 5 mm Hg are achieved. In the presence of calcification and fibrosis of the valve in conjunction with restricted mobility and subvalvular fusion, however, the results are suboptimal and the risks are higher (Fig. 9). Consequently, the echocardiography score index was developed, to represent the degree of leaflet mobility, leaflet thickening, subvalvular

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**Table 3.—Mitral Valve Echocardiography Score Based on Morphologic Features**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mobility</strong></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Highly mobile valve with only leaflet tips restricted</td>
</tr>
<tr>
<td>2</td>
<td>Normal mobility of leaflet midportion and base</td>
</tr>
<tr>
<td>3</td>
<td>Valve continues to move forward in diastole, mainly from the base</td>
</tr>
<tr>
<td>4</td>
<td>No or minimal forward movement of leaflets in diastole</td>
</tr>
<tr>
<td></td>
<td><strong>Leaflet thickening</strong></td>
</tr>
<tr>
<td>1</td>
<td>Leaflets nearly normal in thickness (4-5 mm)</td>
</tr>
<tr>
<td>2</td>
<td>Midportion of leaflets normal; marked thickening of margins (5-8 mm)</td>
</tr>
<tr>
<td>3</td>
<td>Thickening extending through entire leaflet (5-8 mm)</td>
</tr>
<tr>
<td>4</td>
<td>Marked thickening of all leaflet tissue (&gt;8-10 mm)</td>
</tr>
<tr>
<td></td>
<td><strong>Subvalvular thickening</strong></td>
</tr>
<tr>
<td>1</td>
<td>Minimal thickening just below mitral leaflets</td>
</tr>
<tr>
<td>2</td>
<td>Thickening of chordal structures extending up to one-third of the chordal length</td>
</tr>
<tr>
<td>3</td>
<td>Thickening extending to distal third of the chords</td>
</tr>
<tr>
<td>4</td>
<td>Extensive thickening and shortening of all chordal structures extending down to papillary muscles</td>
</tr>
<tr>
<td></td>
<td><strong>Calcification</strong></td>
</tr>
<tr>
<td>1</td>
<td>Single area of increased echo brightness</td>
</tr>
<tr>
<td>2</td>
<td>Scattered areas of brightness confined to leaflet margins</td>
</tr>
<tr>
<td>3</td>
<td>Brightness extending into midportion of leaflets</td>
</tr>
<tr>
<td>4</td>
<td>Extensive brightness throughout much of the leaflet tissue</td>
</tr>
</tbody>
</table>

Modified from Abascal and associates. By permission of The American College of Cardiology.
Fig. 6. Diagram of method used for percutaneous mitral balloon valvuloplasty. A, Modified four-chamber view demonstrates stenotic mitral valve. Left ventricular and left atrial pressures are shown, indicating a significant diastolic mitral valve gradient. Mullins sheath is introduced into femoral vein and advanced to superior position in right atrium. Under fluoroscopic guidance, catheter is withdrawn inferiorly until region of the fossa ovalis is reached. A small, sharp needle is then advanced through the sheath to puncture the fossa ovalis and obtain access to left atrium. B, After placement of guidewires across fossa ovalis into left atrium and left ventricle, a small (8-mm) balloon is placed across atrial septum and inflated. This allows the passage of larger balloons into left atrium. C, One or more large balloons are then passed along the guidewires across mitral valve, and several inflations are performed. During balloon inflation, patient will have severe systemic hypotension because of obstruction caused by the balloon. D, Final results of effect of balloon valvuloplasty, demonstrating splitting of commissures and subsequent decrease in mitral valve gradient.

thickening, and calcification. Patients who have a score of less than 8 have excellent results after mitral valvuloplasty. Patients who have echocardiography scores of more than 10 have suboptimal results and are at higher risk for complications.49

The complications of mitral valve dilation are listed in Table 4. The transseptal puncture necessitated by the procedure has associated risks. The needle used to perforate the fossa ovalis can penetrate the aortic root and cause dissection or the free atrial wall and cause a hemopericardium and cardiac tamponade. Residual atrial septal defects may be created by the transseptal passage of the balloons but are usually not hemodynamically significant (Qp/Qs ratios of less than 1.5).62-64,70 An occasional large, symptomatic left-to-right shunt

Fig. 7. Results of percutaneous mitral balloon valvuloplasty. Acute decrease in mean mitral valve gradient in several series is shown. Mean gradient is calculated from simultaneous pressure measurements in left atrium and left ventricle. Mass Gen = Massachusetts General; USC = University of Southern California.

Fig. 8. Results of percutaneous mitral balloon valvuloplasty. Acute increase in mitral valve area (MVA) in several series is shown. Mitral valve area is calculated by the Gorlin equation. Mass Gen = Massachusetts General; USC = University of Southern California.

may occur, most likely because of unintentional inflation of one of the larger balloons across the region of the fossa ovalis during dilation of the mitral valve. The smaller atrial septal defects are usually not detected at follow-up because the left atrial pressure has been decreased.

Because atrial thrombus may be present in a patient with mitral stenosis, balloon valvuloplasty can result in systemic embolization. In several large series, the incidence of cerebrovascular events was 4%, Therefore, at our institution transesophageal echocardiography is performed before each procedure to exclude the presence of visible thrombi in either the left atrial cavity or the left atrial appendage. Optimally, the patient should receive anticoagulation therapy for 3 months before the procedure, for treatment of potential small thrombi that may not be detected by transesophageal echocardiography.

Mitral regurgitation may increase after a mitral balloon valvuloplasty. The mitral regurgitation will increase by one grade in about 30 to 40% of patients and by two grades in about 10%. A few patients (approximately 3%) will have severe mitral regurgitation. These patients usually have heavily calcified, rigid leaflets with severe commissural fusion, and balloon inflation results in tearing of the leaflets rather than separation of the commissures. This complication may necessitate urgent surgical intervention. Another potential complication, especially in elderly patients with thin ventricular walls, is perforation and subsequent cardiac tamponade.

At our institution, use of the Inoue balloon has recently been initiated. Because of its “pillow-shaped” configuration, inflations are performed in a much more stable position than with conventional balloons. Preliminary reports suggest that the use of this balloon may result in a lower incidence of cerebrovascular events, atrial septal defects, or severe mitral regurgitation.

Follow-Up.—Follow-up studies of patients who have undergone percutaneous mitral balloon valvuloplasty have been limited. Overall, notable functional improvement ensues;
most patients have improvement from New York Heart Association functional class III or IV before valvuloplasty to class I or II at 3-month follow-up.\textsuperscript{70} Objectively, a 50% improvement in exercise capacity is noted at 3 months.\textsuperscript{70}

Short-term follow-up (9 to 13 months) has recently been reported in two series, one of 97 patients\textsuperscript{75} and one of 100 patients.\textsuperscript{76} Patients with pliable, noncalcified valves have had a 5 to 7% recurrence of severe symptoms\textsuperscript{75,76} but no significant stenosis.\textsuperscript{73,76} In patients with nonpliable, heavily calcified valves, the incidence of recurrent symptoms has been 30 to 40%\textsuperscript{75,76} and the restenosis rate has been 42%.\textsuperscript{76}

**Recommendations.**—It is essential to determine the long-term prognosis for patients undergoing balloon valvuloplasty before final recommendations can be made for its use in the treatment of mitral stenosis. Nonetheless, from the available preliminary data, this technique can be performed at a very low risk and with excellent results in patients with pliable, noncalcified valves. We believe that the recent use of the Inoue balloon will further decrease the incidence of complications. If long-term follow-up demonstrates an incidence of restenosis comparable to that after open commissurotomy, percutaneous mitral balloon valvuloplasty may become the procedure of choice for this subgroup. In the patients with more heavily calcified valves and subvalvular fusion, the results are less optimal and the complication rate is higher. These same patients, however, are also at somewhat higher risk for an open-heart surgical procedure. If other factors are involved that would impose a substantially increased operative risk, such as coexistent medical disease or severe left ven-

![Graph](image-url)

Fig. 9. Plot of final mitral valve area (MVA) in square centimeters versus the echocardiography score in initial group of Mayo patients who underwent percutaneous mitral balloon valvuloplasty. Those patients with a score of less than 8 had excellent results, whereas patients with a score of more than 10 had suboptimal results and complications. *Open circles* = complications (death or severe mitral regurgitation).

<table>
<thead>
<tr>
<th>Table 4.—Reported Complications of Mitral Valvuloplasty*</th>
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</thead>
<tbody>
<tr>
<td><strong>Complication</strong></td>
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<tr>
<td></td>
</tr>
<tr>
<td>Cerebrovascular</td>
</tr>
<tr>
<td>accident</td>
</tr>
<tr>
<td>Cardiac tamponade</td>
</tr>
<tr>
<td>Death</td>
</tr>
<tr>
<td>Severe mitral</td>
</tr>
<tr>
<td>regurgitation</td>
</tr>
<tr>
<td>Prolonged</td>
</tr>
<tr>
<td>hypotension</td>
</tr>
<tr>
<td>Local vascular</td>
</tr>
<tr>
<td>complication</td>
</tr>
<tr>
<td>Atrial septal</td>
</tr>
<tr>
<td>defect</td>
</tr>
</tbody>
</table>

* NHLBI = National Heart, Lung, and Blood Institute.
tricular dysfunction, valvuloplasty may be a reasonable option in these older patients with calcified valves.

Currently, we attempt to limit the use of this technique to those patients who are relatively ideal candidates for closed commissurotomy. Elderly patients with associated diseases may also be considered, especially if operative intervention poses extremely high risks.

**AORTIC VALVE**

Percutaneous aortic balloon valvuloplasty was first performed by Lababidi and colleagues in young children with congenital aortic stenosis. In contrast to pulmonic and mitral balloon valvuloplasty, this procedure resulted in only a modest decrease in gradient across the aortic valve. Other investigators have found the same suboptimal results in neonates, infants, and children. In 1986, however, considerable enthusiasm was generated when Cribier and associates reported that this technique produced dramatic relief of symptoms in three elderly patients with calcific aortic valve stenosis who were at high operative risk. Subsequent follow-up of this technique has shown that percutaneous aortic balloon valvuloplasty is not the treatment of choice for most patients with calcific aortic stenosis because relief of obstruction is incomplete and the restenosis rate is high. Nonetheless, it remains a therapeutic option for certain subsets of patients.

**Clinical Aspects.**—Clues about the cause of aortic valvular stenosis can be obtained from the age at which the symptoms manifest. In children and young adults, congenital malformations of the aortic valve are the most common cause of left ventricular outflow obstruction. Either rheumatic involvement or calcification of a bicuspid valve is the most common cause of severe valvular aortic stenosis in patients in the fifth or sixth decade of life.

In the past decade, however, the pattern of patients with symptomatic, severe aortic stenosis has been evolving. The majority of such patients now are in the seventh or eighth decade of life. The mechanism of stenosis in these patients is senile degeneration of a tricuspid valve with large, calcific deposits at the base of the cusps. Commissural fusion is absent, and the stenosis is produced because these calcific deposits prevent the cusps from opening during systole.

The main symptoms of aortic stenosis are angina, dyspnea, and syncope. These symptoms are related to both the obstruction to outflow and the severe left ventricular hypertrophy that develops in response to the pressure overload. The left ventricular hypertrophy and high intracavitary pressures produce an increase in myocardial oxygen demand that, with reduced oxygen supply from compressive effects, results in angina. Dyspnea occurs from the increased left ventricular diastolic pressure because of the abnormal diastolic compliance from the hypertrophy. The syncope may be due to arrhythmias or to a fixed cardiac output and vasodilatation with exertion.

The physical findings in severe aortic stenosis consist of a delayed carotid upstroke, a sustained left ventricular impulse, and a systolic ejection murmur. It is not the intensity of the murmur but rather the duration and timing that indicate the severity of the stenosis. The aortic component of the second heart sound should be diminished or absent because of calcification of the valve. The electrocardiogram will show evidence of left ventricular hypertrophy. Left ventricular predominance and a prominent ascending aorta will be evident on the chest roentgenogram (Fig. 10).

In elderly patients, the classic physical findings may not always be present. Therefore, two-dimensional and Doppler echocardiograms should be used to confirm the diagnosis. Two-dimensional imaging will demonstrate the presence of a calcified, immobile aortic valve and the secondary left ventricular hypertrophy. Doppler echocardiography accurately measures the aortic valve gradient and aortic valve area, thus, the severity of the obstruction can be quantitated.

Although patients with severe aortic stenosis can remain asymptomatic for many years, the prognosis is poor once symptoms occur. The mean duration of survival after the onset of
angina and syncope is 2 to 3 years, whereas with congestive heart failure, it is 1 to 2 years. Therefore, it has been recommended that patients with severe aortic stenosis who become symptomatic undergo aortic valve operation, an approach that produces substantial clinical and hemodynamic improvement as well as prolongation of life.

Aortic valve replacement can be performed at a relatively low risk in young, otherwise healthy patients and has thus become the treatment of choice for severe, symptomatic aortic stenosis. Because of the aging population, however, many of the patients with severe aortic stenosis in the past decade have been elderly and have had multiple coexistent medical problems, factors that substantially increase the surgical risk. In patients older than 70 years of age who underwent aortic valve replacement, the operative mortality in a large recent series was 16%—more than quadruple that in younger patients. In patients older than 80 years of age, the early mortality rate has been 31%. Many of these elderly patients have numerous other medical problems, such as chronic renal failure, severe chronic obstructive pulmonary disease, or a malignant lesion, that would prohibit an operation. Finally, many elderly patients will refuse operation when faced with such a high mortality rate and long recovery time.

Recently, it has been recognized that the natural history of severe, symptomatic aortic stenosis in this subgroup of elderly patients who are not candidates for operation is dismal, with 1-, 2-, and 3-year survival rates of 57%, 42%, and 25%, respectively. Therefore, the initial report by Cribier and co-workers demonstrating a symptomatic improvement in three elderly patients who underwent aortic balloon valvuloplasty was received with high hopes for salvaging these patients.

**Technique and Mechanism of Percutaneous Aortic Balloon Valvuloplasty.**—At our institution, percutaneous aortic balloon valvuloplasty is performed from the femoral artery route (Fig. 11). In contrast to valvuloplasty of the pulmonic and mitral valves, this technique necessitates placement of large balloons through an arterial site and thus creates a greater possibility of femoral artery complications. This drawback led to the development of other techniques to avoid the vascular trauma, such as an ante-
Fig. 11. Diagram of method of percutaneous aortic balloon valvuloplasty with use of dual-balloon technique. 

A, Right anterior oblique view demonstrates heavily calcified aortic valve. Two guidewires have been introduced through both femoral arteries, placed across stenotic valve, and stabilized in apex. Left ventricular and aortic pressures are shown at right. 

B, During balloon inflation, systolic pressure decreases and degree of obstruction increases substantially. 

C, Final result of balloon valvuloplasty, with modest reduction in gradient.

Grade transseptal approach\textsuperscript{93} or a dual-balloon technique.\textsuperscript{3,35,36,94} With the availability of new low-profile catheters, which allow larger balloons to be introduced through smaller sheaths, the single-balloon technique has again become the procedure of choice at many institutions.

After diagnostic catheterization to measure baseline pressures and cardiac output, long exchange wires are placed retrogradely from the femoral artery across the aortic valve and stabilized in the left ventricular apex. A balloon (or balloons) is then threaded over the wires and placed across the aortic valve. Several inflations are performed, after which aortic valve gradients and areas are measured. If the gradient has not decreased by 50\% or is more than 40 mm Hg, a larger balloon (or balloons) is then introduced across the aortic valve and inflated several times. The optimal balloon size needed to obtain the best result without complication varies from patient to patient. Because of results of in vivo studies,\textsuperscript{95} we do not use balloons larger than 120\% of the annulus size measured by echocardiography. Most commonly, a 20- or 23-mm balloon is used.

During balloon inflation, systemic blood pressure frequently decreases modestly, but this effect is well tolerated.\textsuperscript{92,96,97} In patients with pronounced resting left ventricular dysfunction, however, hemodynamic compromise is severe during balloon inflation; thus, the inflation must be limited to 5 to 10 seconds.\textsuperscript{92,96,97} The balloon inflation produces an appreciable increase in afterload on the left ventricle, as substantiated by significant prolongation of ejection time during inflation.\textsuperscript{97} The left ventricular systolic pressure may increase, remain the same, or decrease, depending on the ventricular response to this increase in afterload. During balloon inflation, coronary perfusion is substantially decreased, as measured by coronary sinus flow and coronary sinus metabolites, but it returns to normal immediately after balloon deflation.\textsuperscript{98} This combination of factors results in an appreciable depression of ventricular function during balloon inflation.

During balloon inflation, severe aortic regurgitation\textsuperscript{92,97} and a decrease in left ventricular...
Both of these effects result in a pronounced increase in left ventricular end-diastolic pressure, sometimes exceeding aortic diastolic pressure. Patients with preexisting mitral regurgitation have a considerable increase in the degree of regurgitation resulting in an extreme elevation of pulmonary pressures.

Efforts to prevent hemodynamic compromise during inflation have led to the development of a trefoil balloon, in which three smaller balloons are placed parallel along a single catheter. This arrangement allows blood flow to continue while the balloon is inflated. The dual-balloon approach has been reported to cause less hemodynamic deterioration because venting is possible at the junction of the two balloons. Nevertheless, a three-lumen, single-balloon catheter with two ports for inflation and deflation of the balloon has been the most effective because this design allows rapid inflation and deflation of a single, large balloon.

Because elderly patients undergoing aortic balloon valvuloplasty frequently have other cardiac diseases, "combined procedures" have been performed in the cardiac catheterization laboratory and proved successful. In patients with symptomatic coronary artery disease, percutaneous transluminal coronary angioplasty has been done in conjunction with aortic balloon valvuloplasty. In patients with multivalvular disease and symptomatic aortic and mitral stenoses, percutaneous mitral and aortic balloon valvuloplasties have been done during the same procedure.

The mechanism by which percutaneous aortic balloon valvuloplasty relieves left ventricular outflow obstruction is multifactorial. In some patients, the mechanism is splitting of the fused commissures. In most patients with senile aortic stenosis, however, direct intraoperative visualization of the aortic valve shows little, if any, change in the appearance of the valve or valve orifice. In most patients, the mechanism of balloon valvuloplasty is fracture of calcific plates or calcified nodules. This result allows a mild increase in the mobility of the valve without actually affecting the structure of the valve.

**Early Results and Complications of Percutaneous Aortic Balloon Valvuloplasty.**—Hemodynamic assessment of the early results of percutaneous aortic balloon valvuloplasty has demonstrated a modest reduction in aortic valve gradient and an improvement in aortic valve area in elderly adults. On the basis of several reported series, the aortic valve gradient (both peak-to-peak and mean) decreased from 70 mm Hg to 36 mm Hg, with an improvement in valve area from 0.6 cm² to 0.9 cm² (Table 5). The final valve area and gradient depend primarily on the initial hemodynamics present before the procedure. When the aortic stenosis is critical and the valve area is less than 0.4 cm², the final area will be approximately 0.6 cm²; although this result is an improvement, severe aortic stenosis is still present. Patients who initially have an aortic valve area of 0.7 cm² or larger may have resultant valve areas of more than 1.2 cm² (only mild aortic stenosis).

This modest improvement in hemodynamics results in dramatic alleviation of symptoms. Investigators believe that a "critical" level of left ventricular outflow obstruction is related to development of symptoms. Because of the curvilinear relationship of gradient to valve area, a small increase in valve area may be sufficient to alter the loading conditions of the heart, improve hemodynamics, and alleviate symptoms. This result is particularly dramatic in patients who undergo emergency aortic balloon valvuloplasty while in cardiogenic shock—frequently, the clinical status improves notably in conjunction with an immediate increase in blood pressure and cardiac output.

Studies with use of radionuclide angiography have demonstrated an overall decrease in left ventricular end-diastolic index and a decline in stroke volume ratio at 48 hours after the procedure. Patients with depressed initial left ventricular function may have a substantial increase in ejection fraction. Left ventricular function gradually improves during the first 24 to 48 hours after the procedure—perhaps be-
cause of the transient depression of ventricular function caused by balloon inflation.\textsuperscript{111}

Major complications can occur with percutaneous aortic balloon valvuloplasty; in part, they are attributed to the critically ill nature of the patients undergoing the procedure (Table 6). A high incidence of complications involves the femoral artery, approaching 10 to 15\% in some series.\textsuperscript{79,80} Because of the stiff catheters used, perforation of the left ventricle and subsequent cardiac tamponade have occurred. Both of these complications should diminish with newer developments in catheters.

Systemic embolization has been substantiated in patients undergoing aortic balloon valvuloplasty.\textsuperscript{106,112} These emboli usually cause acute, highly focal, small neurologic deficits, such as homonymous hemianopia and vertebro-basilar insufficiency, which are suggestive of small calcific emboli. Prospective evaluation by computed tomography and funduscopic examination has demonstrated new areas of low attenuation and hemorrhagic infarcts.\textsuperscript{112}

Aortic regurgitation has not been a major problem after aortic balloon valvuloplasty.\textsuperscript{34,79,92} About 10 to 15\% of patients will have new or increased aortic regurgitation, but it is hemodynamically significant in less than 2\%.\textsuperscript{79,86,92} Rupture of the annulus, aortic wall tears, and leaflet disruption are uncommon but have been reported and are thought to be due to use of a balloon larger than the aortic annulus.\textsuperscript{95,103,113-115} These complications may lead to severe aortic regurgitation, congestive heart failure, aortic dissection, and death. In patients with severe congestive heart failure and appreciably depressed left ventricular function, inflation of the balloon may result in electromechanical dissociation and death.

**Follow-Up.**—Follow-up of patients who have undergone percutaneous aortic balloon valvuloplasty has been critical for evaluating the role of this procedure in cardiology. Initial reports described the dramatic relief of symptoms in critically ill patients during limited follow-up.\textsuperscript{6,92,107} As the follow-up period has been extended, however, a high mortality has been found as a result of the severe underlying disease in these critically ill, elderly patients.\textsuperscript{79,80,109} Restenosis will occur in most patients (approaching 50 to 60\%) by 6 months (Fig. 12).\textsuperscript{34,79} A substantial proportion of patients, however, continue to remain minimally symptomatic, despite the occurrence of restenosis (Fig. 13).\textsuperscript{34,79} Even with restenosis, the degree of obstruction may not always reach the "critical" level for the onset of symptoms. Depending on the population studied, about 50 to 60\% of patients will remain free of symptoms at 6 months to 1 year.\textsuperscript{34,79,80} If a patient has recurrence of severe

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>No. of patients</th>
<th>Gradient (mm Hg)</th>
<th>Valve area (cm(^2))</th>
</tr>
</thead>
<tbody>
<tr>
<td>\textsuperscript{t}Lababidi et al\textsuperscript{a}</td>
<td>1984</td>
<td>23</td>
<td>Before: 113</td>
<td>After: 32</td>
</tr>
<tr>
<td>\textsuperscript{t}Walls et al\textsuperscript{b}</td>
<td>1984</td>
<td>27</td>
<td>Before: 125</td>
<td>After: 35</td>
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<tr>
<td>\textsuperscript{t}Cribier et al\textsuperscript{c}</td>
<td>1987</td>
<td>92</td>
<td>Before: 77</td>
<td>After: 39</td>
</tr>
<tr>
<td>McKay et al\textsuperscript{d}</td>
<td>1987</td>
<td>32</td>
<td>Before: 57</td>
<td>After: 30</td>
</tr>
<tr>
<td>\textsuperscript{t}Schneider et al\textsuperscript{e}</td>
<td>1987</td>
<td>9</td>
<td>Before: 47</td>
<td>After: 32</td>
</tr>
<tr>
<td>\textsuperscript{t}Isner al\textsuperscript{f}</td>
<td>1987</td>
<td>6</td>
<td>Before: 62</td>
<td>After: 26</td>
</tr>
<tr>
<td>\textsuperscript{t}Dorros et al\textsuperscript{g}</td>
<td>1988</td>
<td>10</td>
<td>Before: 79</td>
<td>After: 36</td>
</tr>
<tr>
<td>\textsuperscript{t}Safian et al\textsuperscript{h}</td>
<td>1988</td>
<td>170</td>
<td>Before: 71</td>
<td>After: 36</td>
</tr>
<tr>
<td>Litvack et al\textsuperscript{i}</td>
<td>1988</td>
<td>25</td>
<td>Before: 66</td>
<td>After: 40</td>
</tr>
<tr>
<td>\textsuperscript{t}Nishimura et al\textsuperscript{j}</td>
<td>1988</td>
<td>55</td>
<td>Before: 48</td>
<td>After: 33</td>
</tr>
</tbody>
</table>

\*NA = not available.
\*Children and young adults.
\*Most recent comprehensive series.
Table 6.—Reported Complications of Percutaneous Aortic Balloon Valvuloplasty

<table>
<thead>
<tr>
<th>Complication</th>
<th>Safian et al&lt;sup&gt;9&lt;/sup&gt; (N = 170)</th>
<th>Cribier &amp; Letac&lt;sup&gt;109&lt;/sup&gt; (N = 218)</th>
<th>Nishimura et al&lt;sup&gt;34&lt;/sup&gt; (N = 55)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>Vascular&lt;sup&gt;*&lt;/sup&gt;</td>
<td>17</td>
<td>10.0</td>
<td>9</td>
</tr>
<tr>
<td>Perforation</td>
<td>3</td>
<td>1.8</td>
<td>3</td>
</tr>
<tr>
<td>TIA, CVA&lt;sup&gt;†&lt;/sup&gt;</td>
<td>...</td>
<td>...</td>
<td>3</td>
</tr>
<tr>
<td>Death&lt;sup&gt;‡&lt;/sup&gt;</td>
<td>6</td>
<td>3.5</td>
<td>10</td>
</tr>
<tr>
<td>Severe aortic regurgitation</td>
<td>2</td>
<td>1.2</td>
<td>...</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>1</td>
<td>0.6</td>
<td>1</td>
</tr>
</tbody>
</table>

<sup>*</sup>Necessitating surgical treatment or resulting in severe ischemia.
<sup>†</sup>Transient ischemic attack or cerebrovascular accident.
<sup>‡</sup>In-hospital mortality.

Symptoms, balloon valvuloplasty can be repeated with good symptomatic results.<sup>79,80</sup>

**Recommendations.**—Because of the modest improvement in hemodynamics, the high rate of recurrence of stenosis, and the high mortality, percutaneous aortic balloon valvuloplasty should be considered a palliative procedure and should not be recommended for patients who are surgical candidates.<sup>2,3,4,79,80</sup> Nonetheless, there will be patients who are not candidates for aortic valve operation because of other medical problems, such as disseminated carcinoma or malignant hematologic lesions. Moreover, other patients are at high surgical risk because of coexistent severe left ventricular dysfunction, chronic renal failure, or chronic obstructive pulmonary disease. Finally, some elderly, critically ill patients absolutely refuse surgical treatment. In these instances, if the patient and the physician are willing to accept the risks of valvuloplasty in order to provide palliation of symptoms, percutaneous aortic balloon valvuloplasty is a reasonable alternative to aortic valve operation.

Fig. 12. Results of serial Doppler echocardiographic studies, demonstrating high incidence of restenosis at 6 months after aortic valvuloplasty. A, Mean aortic valve gradient measured before, 24 to 36 hours after, and at follow-up (FU). B, Aortic valve area (AVA) measured before, 24 to 36 hours after, and at follow-up. (From Nishimura and associates<sup>34</sup> By permission of The American Heart Association.)
Interest in percutaneous aortic balloon valvuloplasty for several other subsets of patients has been increasing. One such group consists of elderly patients with severe aortic stenosis who must undergo a noncardiac operation. Because the risk of noncardiac surgical procedures is increased in patients with severe aortic stenosis, even a modest improvement in hemodynamics achieved by balloon valvuloplasty may be sufficient to allow the noncardiac operation to be performed safely. Another candidate is the patient who has cardiogenic shock from aortic stenosis. The risk of an emergency surgical procedure is extremely high in these patients, and balloon valvuloplasty can sometimes result in dramatic alleviation of symptoms and improvement in hemodynamics. In such patients, percutaneous aortic balloon valvuloplasty has been successfully performed to allow stabilization and preparation for later surgical treatment if the patient is then deemed an operative candidate. A third category is patients with a low-output low-gradient state in whom it is unclear whether severe myocardial dysfunction or severe aortic stenosis is the primary problem. If percutaneous aortic balloon valvuloplasty produces substantial improvement in left ventricular function, these patients most likely have severe aortic stenosis and would then become candidates for aortic valve operation. A fourth category may be the adolescents and young adults who have noncalcified congenital aortic stenosis. Improvement in gradients by aortic balloon valvuloplasty may be sufficient to delay aortic valve replacement for a period of years.

CONCLUSION
The new and evolving technique of percutaneous balloon valvuloplasty deserves careful application in certain subsets of patients. The best results have been obtained in patients with congenital pulmonic stenosis, especially if they are young. Good results, comparable to those obtained by operation, can be achieved in adults with isolated mitral stenosis if the echocardiographic score index, reflecting the extent of valve deformity, is low. Currently, use of balloon valvuloplasty for severe aortic stenosis is best limited to the frail, elderly patient who is a poor surgical candidate or has an associated life-limiting noncardiac disease. Further investigation is needed for long-term follow-up analysis of the results of all these procedures, as is currently being done by several national multicenter registries.

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