Balloon dilatation of calcific aortic stenosis in elderly patients: postmortem, intraoperative, and percutaneous valvuloplasty studies

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ABSTRACT To assess the safety and efficacy of percutaneous balloon valvuloplasty in calcific aortic stenosis, balloon dilatation of critically stenosed, calcified aortic valves was performed in five postmortem hearts, in five patients intraoperatively before aortic valve replacement, and in two elderly patients percutaneously at the time of diagnostic catheterization. The etiology of aortic stenosis in the 12 cases was rheumatic in two, congenital bicuspid calcific stenosis in one, and senile calcific degenerative stenosis in the remaining nine. Prevalvuloplasty examination in the 10 postmortem and intraoperative cases revealed rigid valve leaflets with commissural fusion in three valves and extensive nodular calcification in seven. Subsequent balloon dilatation with 15 to 18 mm valvuloplasty balloons resulted in decreased cusp rigidity and increased mobility of valve leaflets in all cases, without evidence of tearing of valve leaflets, disruption of the valvular ring, or liberation of calcific or valvular debris. In the three valve specimens with commissural fusion, balloon dilatation resulted in partial or complete separation of leaflets along fused commissures. In two cases with extensive nodular calcification, balloon dilatation resulted in a fracture of a calcified leaflet that was evident on both gross and radiologic examination. After postmortem and intraoperative studies, percutaneous catheter valvuloplasty was performed at the time of diagnostic catheterization in two elderly patients (93- and 85-year-old women) with long-standing calcific aortic stenosis. Balloon dilatation with 12 to 18 mm balloons resulted in significant decreases in aortic gradients and significant increases in cardiac index and aortic valve area in both patients. Percutaneous valvuloplasty in both patients resulted in a mild increase in aortic insufficiency and no evidence of embolic phenomena. It is concluded that balloon valvuloplasty is possible in elderly patients with calcific aortic stenosis and can result in significant improvement in ventricular and valvular function without the production of life-threatening complications. The mechanism of valvular improvement may be secondary to separation of fused commissures, fracture of extensive nodular calcification, and/or stretching of rigid leaflets.


PERCUTANEOUS transluminal balloon angioplasty is a technique that has been safely applied as treatment for peripheral vascular1, 2 and coronary artery disease, 3 hypoplastic and stenotic pulmonary arteries, 4 aortic coarctation, 5 venous obstruction, 6 pulmonic stenosis, 7 and mitral and congenital aortic stenosis in children and young adults. 8 Particularly, our laboratory and others have reported the success of percutaneous balloon valvuloplasty in elderly patients with calcific mitral 10, 11 and aortic stenosis. As a palliative procedure in elderly patients with critical valvular stenosis in whom valve replacement is deferred because of high operative risk, percutaneous balloon valvuloplasty potentially offers a viable, nonsurgical approach that may be life-saving.

Although the short-term hemodynamic benefits of balloon aortic valvuloplasty have been described, 12 the anatomic correlates of successful valvular dilatation are largely unknown. Possible explanations for success include separation of fused commissures, fracture of calcified leaflets, or stretching of the anulus and leaflet structure. An understanding of the pathophysi-
ology of valvular dilatation is critical in assessing the risks and potential complications of the procedure. In adults, the presumed potential complications of balloon valvuloplasty include embolization of calcific debris, disruption of the valve ring, acute valvular regurgitation, and valvular restenosis.

To date, no studies have been published describing in vitro or intraoperative aortic valvuloplasty in calcified, stenotic aortic valves. Accordingly, we studied the effects of balloon dilatation performed in five postmortem hearts as well as intraoperatively in five patients with critical aortic stenosis immediately before aortic valve replacement. After our postmortem and intraoperative studies, percutaneous balloon valvuloplasty was attempted at the time of cardiac catheterization in two elderly patients with long-standing calcific aortic stenosis who refused surgical intervention. The objectives of these studies were (1) to describe the impact of balloon dilatation on commissural fusion, leaflet mobility, nodular calcification, and valve ring integrity, and (2) to assess the potential effect of balloon dilatation on embolization and aortic regurgitation in the intact patient.

Methods

Postmortem valvuloplasty. All patients undergoing postmortem examinations who had evidence of aortic stenosis were considered for study. Valves were examined by a pathologist to determine the number of cusps, the presence of commissural fusion, and the degree of leaflet distortion, calcification, and immobility. When possible, roentgenograms of the valve were taken to assess the degree and distribution of calcification. The aortic valves were then dilated with a 15 or 18 mm valvuloplasty balloon (Medi-Tech, Watertown, MA) with maximum balloon inflation to 3 atm for 30 sec. After dilatation, valves were examined grossly and radiographically for evidence of commissural separation, leaflet fracture, valve ring distortion, calcific debris, and changes in leaflet mobility.

Intraoperative valvuloplasty. All patients undergoing aortic valve replacement for critical aortic stenosis were considered for study. All patients gave informed consent for intraoperative valvuloplasty according to a protocol approved by the Beth Israel Hospital Human Studies Committee. After institution of cardiopulmonary bypass, cross-clamping of the aorta, and opening of the aorta in preparation for aortic valve replacement, a detailed inspection of the valve was performed as described above. Next, an 18 mm balloon catheter was advanced retrograde through the aortic orifice and a 30 sec dilatation was performed. After valve dilatation, detailed inspection of the valve was repeated. In those cases in which the valve specimen was not too fragmented after excision, further gross and radiographic studies were performed as described above.

Percutaneous valvuloplasty. Percutaneous valvuloplasty was performed in two patients at the time of diagnostic catheterization. Both patients gave informed consent after being informed of the risks and potential complications according to a protocol accepted by the Beth Israel Human Studies Committee. The first patient was a 93-year-old white woman with long-standing, calcific aortic stenosis and a 4 year history of angina pectoris and severe biventricular congestive heart failure. The second patient was an 86-year-old white woman with a 7 year history of angina and intermittent syncope. Both patients were evaluated by a cardiac surgeon but refused surgical intervention.

Each patient was brought to the catheterization laboratory and the right femoral artery and femoral vein were instrumented with No. 8F Hemokit sheaths after the administration of a local anesthetic. Systemic anticoagulation was accomplished with 5000 U of heparin after placement of the femoral arterial sheath. Right heart catheterization was accomplished with a No. 7F balloon flotation catheter advanced from the right groin. A No. 7F pigtail catheter was placed in the ascending aorta and a supravalvular aortogram was obtained. Subsequently, the aortic valve was crossed retrograde with a 0.038 straight guidewire and the pigtail catheter was advanced into the left ventricle. After placement of left and right heart catheters, measurements were made of systemic arterial and left ventricular pressures as well as left ventricular and pulmonary arterial saturations. Oxygen consumption was measured with a metabolic rate meter (MRM-2, Waters Instruments).

Next, a 300 cm 0.038 exchange wire was advanced through the pigtail catheter into the left ventricle and the pigtail catheter was removed. A 12 mm balloon valvuloplasty catheter was then advanced over the guidewire into the left ventricle. With the balloon positioned at the aortic valve, three inflations were made with hand injections of a saline/Angiovist (meglumine diatrizoate) mixture. In the first patient, a waist in the balloon was seen to disappear and the valvuloplasty catheter was then deflated and withdrawn. In the second patient, no waist was seen and the 12 mm balloon valvuloplasty catheter was subsequently replaced, first with a 15 mm valvuloplasty catheter and finally with an 18 mm valvuloplasty catheter. After three inflations of the 18 mm balloon, a waist was also noted to disappear and the valvuloplasty catheter was deflated and withdrawn. The maximum time of balloon inflation was 12 to 15 sec. After balloon dilatation with the 12 mm balloon in the first patient and with the 18 mm balloon in the second patient, the pigtail catheter was exchanged over the guidewire and placed into the left ventricle and repeat measurements were made of pressures and cardiac output. A pullback of the left ventricular catheter was then performed, followed by a repeat supravalvular aortogram. An additional 5000 U of heparin was administered immediately after valvuloplasty.

Results

Postmortem and intraoperative valvuloplasty. Postmortem or intraoperative balloon dilatation was performed in 10 patients. There were five women and five men, with an average age of 78 years (range 67 to 90). Eight of the 10 patients had undergone prior cardiac catheterization, with confirmed critical aortic stenosis and an average valve area of 0.7 cm² (range 0.4 to 0.8). The etiology of aortic stenosis and hence the pathologic anatomy differed in these patients; postinflammatory stenosis (presumed rheumatic) was found in two, congenital bicuspid calcific stenosis in one, and senile calcific stenosis in the remainder.

Gross examination of the valves before dilatation revealed severe cusp rigidity in all patients. Commisural fusion was present in only three patients. The patient with congenital bicuspid aortic stenosis had the typical congenitally fused commissure or rudimentary raphe between the right and left coronary cusps; the
leaflet fracture on both gross and radiographic examination (figures 1 and 2). In the remaining five patients, dilatation resulted in visible displacement and stretching of rigid valve cusps.

In no valve undergoing balloon dilatation was there evidence of tearing of valve cusps, liberation of calcific or other valvular debris as a potential source of emboli, or disruption of the valve ring anulus. Clinical characteristics and findings before and after valvuloplasty are summarized in tables 1 and 2.

Percutaneous valvuloplasty. Cardiac catheterization and percutaneous aortic valvuloplasty in our first patient (a 93-year-old woman with severe congestive heart failure) was carried out on December 28, 1985. Prevalvuloplasty hemodynamic evaluation revealed a heavily calcified aortic valve with a peak-to-peak gradient of 66 mm Hg, a Fick cardiac index of 2.1 liters/min/m², an aortic valve area of 0.4 cm², and trace aortic regurgitation. Balloon dilatation resulted in a reduced gradient of 32 mm Hg, an increased cardiac index of 2.4 liters/min/m², an increased aortic valve area of 0.6 cm², and 1+ aortic regurgitation (figure 3). M mode echocardiography demonstrated increased mobility of aortic leaflets, whereas carotid pulse tracings demonstrated an improvement in time to one-half carotid upstroke (80 to 60 msec). Serial radionuclide ventriculography demonstrated left ventricular ejection fractions of 23% 1 day before valvuloplasty, 40% 2 days after valvuloplasty, and 58% 6 weeks after valvuloplasty. Clinically, the patient improved strikingly, with decreased symptoms of orthopnea, dyspnea on exertion, and weakness.

Valvuloplasty in the second patient (an 86-year-old woman with angina and syncope) was carried out on January 15, 1986, and resulted in a reduction of peak-to-peak aortic gradient from 44 to 31 mm Hg, an increase in cardiac index from 2.2 to 2.7 liters/min/m², an increase in aortic valve area from 0.5 to 0.7 cm², and an increase in aortic regurgitation from trace to 1+. Serial radionuclide ventriculography demonstrated a mild increase in left ventricular ejection fraction from 42% 1 day before valvuloplasty to 46% 2 days after valvuloplasty. The patient continued to experience angina in the week after the procedure. Notably, coronary angiography at the time of the valvuloplasty demonstrated severe three-vessel coronary artery disease, including left main involvement. After considerable discussion, the patient elected to undergo coronary bypass surgery and aortic valve replacement. At the time of surgery, gross inspection of the dilated aortic valve showed freely mobile leaflets with no evidence of commissural fusion (figure 4). The

commissures between the two functional bicuspid leaflets showed mild fusion at their periphery. Two additional patients with postinflammatory (presumed rheumatic) aortic stenosis had more extensive commissural fusion (one with fusion of one commissure and one with fusion of all three commissures). Seven patients had extensive nodular calcification of all aortic valve leaflets.

After dilatation, the aortic valve orifice increased grossly in dimension in all patients with increased leaflet mobility. However, the mechanism by which this occurred varied. In the patient with congenital bicuspid aortic stenosis, there was separation of the two major commissures but no effect on the rudimentary commissure. In the two patients with commissural fusion secondary to postinflammatory aortic stenosis, there was either partial or complete separation of fused commissures along the commissural lines. In two additional patients without commissural fusion but with extensive nodular calcification, there was evidence of

FIGURE 1. A, Postmortem specimen after valvuloplasty in an 82-year-old woman with calcific aortic stenosis (aortic valve area 0.5 cm²), showing leaflet fracture (arrow) on gross examination. B, The fracture was also evident on radiographic examination of the postmortem specimen.
FIGURE 2. Postmortem specimen in an 85-year-old man (aortic valve area 0.4 cm²) undergoing valvuloplasty with an 18 mm balloon (outlined by stippled line). A, With partial balloon inflation, a fracture through a calcified leaflet has been produced and is indicated by an arrow. B, With complete balloon inflation, the fracture was noted to enlarge. C, After removal of the balloon, the fracture is still apparent.

### TABLE 1
Summary of clinical characteristics and findings before and after valvuloplasty in postmortem specimens

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Etiology</th>
<th>Leaflet number</th>
<th>Commisural fusion</th>
<th>Nodular calcification</th>
<th>Cusp rigidity</th>
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### Discussion

Both patients tolerated valvuloplasty well without any evidence of embolic phenomena either at the time of balloon dilatation or after the procedure.

The natural history of critical aortic stenosis treated medically is poor, with at least 50% of patients dying within 2 years after the appearance of left ventricular failure. As a result, the accepted therapy of symptomatic aortic stenosis in the adult patient is aortic valve replacement. In general, aortic valve replacement can be performed with an operative mortality of 5% to 10%. Moreover, surgery has clearly been shown to be beneficial in terms of the relief of symptoms, the reversal of functional deficits, the regression of left ventricular dilatation and hypertrophy, and an improvement in longevity. Present estimates suggest that nearly 20,000 aortic valve replacements are performed per year, with at least 60% being performed for aortic stenosis.

R = rheumatic; S = senile degenerative calcific.
TABLE 2
Summary of clinical characteristics and findings before and after valvuloplasty in intraoperative specimens

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Etiology</th>
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Prevalvuloplasty

Postvalvuloplasty

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<tr>
<th>Patient</th>
<th>Commisural separation</th>
<th>Leaflet fracture</th>
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<th>Embolic debris</th>
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<th>Torn ring</th>
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R = rheumatic; B = congenital bicuspid calcific; S = senile degenerative calcific.

In spite of the success of surgical aortic valve replacement, the risks of surgery are clearly high in certain subgroups of patients. For example, when surgery is performed in patients with frank left ventricular failure or depressed ejection fraction, the operative risk is higher, and the mortality ranges from 10% to 25% depending on the skill of the surgical team and the severity of the left ventricular dysfunction. Additional factors such as associated coronary artery disease, advanced age, renal insufficiency, and chronic pulmonary disease place certain patients with critical aortic stenosis in a category of 40% to 50% surgical mortality risk.

Given the overall incidence of critical aortic stenosis in patients who are considered to be at extremely high surgical risk, the possibility of percutaneous balloon valvuloplasty has been considered. The use of percutaneous balloon valvuloplasty has been well established in children and young adults with a variety of congenital and acquired cardiovascular disorders. The application of this technique in elderly patients has been limited primarily by fears of embolization of calcific debris and abrupt valvular regurgitation. However, recent clinical studies have described successful application of percutaneous balloon valvuloplasty to elderly patients with calcific mitral and aortic stenosis. These studies clearly document the short-term hemodynamic improvement after balloon valvuloplasty, but the pathophysiologic basis for this improvement is largely unknown.

Postmortem and intraoperative findings. Earlier studies of the pathology of aortic stenosis have demonstrated that calcification of a congenital bicuspid valve, postinflammatory fibrocalcific disease, and degenerative
calcification account for over 90% of cases of adult aortic stenosis. Degenerative aortic stenosis is the most common cause of aortic stenosis in patients over the age of 70 and is usually associated with extensive leaflet calcification and varying degrees of cusp rigidity. Commissural fusion is not a feature of degenerative aortic stenosis but is one of the hallmarks of post-inflammatory (including rheumatic) aortic stenosis.

In our study of elderly patients, the most common cause of aortic stenosis was degenerative calcification with cuspid rigidity, which is in agreement with prior studies. Commissural fusion was present in one patient with a congenital bicuspid valve and in two patients with postinflammatory aortic stenosis. Successful valvuloplasty was related to separation of fused commissures in three patients, to fracture of valvular nodular calcification in two patients, and to apparent plastic deformation of rigid valve cusps in the remaining five. Leaflet tearing, fragmentation of valve leaflets, and valve ring disruption were not apparent in any patient.

These findings have several important implications regarding the morphologic basis for success, potential complications, and long-term outlook. Although embolization of calcific debris remains a major concern, particularly with the fracture of nodular calcification that was seen in two patients, we did not observe loosening or dislodgment of calcific tissue to suggest that embolization will be a likely occurrence during balloon valvuloplasty. Similarly, gross disruption of leaflets, tearing of leaflets from the aortic ring, or disruption of the ring itself did not occur in any patient, suggesting that acute deterioration secondary to massive aortic insufficiency will not be common. Although commissural fusion was successfully separated with balloon dilation, the relatively low incidence of this finding in elderly patients suggests that separation of commissures will be a major mechanism of successful valve dilatation in only some of the patients in this age group. Even with successful immediate results, failure to modify the underlying causes of degenerative calcification will likely lead to restenosis, although the rate at which this will occur cannot be estimated.

**Percutaneous valvuloplasty.** Cribier et al. recently reported successful percutaneous aortic valve dilatation in three elderly patients with critical aortic stenosis. Our study complements their report and adds additional morphologic information. Although these authors noted no change in the amount of aortic regurgitation after valvuloplasty, there was a small but significant increase in aortic insufficiency in both of our patients. This suggests that balloon dilatation should be limited to patients with mild or nonexistent valvular regurgitation. A marked increase in aortic insufficiency, particularly in the setting of left ventricular hypertrophy, could presumably lead to acute pulmonary edema. Second, in the three patients described by Cribier et al. and in our two patients, the range of balloon sizes that was needed to effect dilatation varied from 8 to 18 mm. This variation in balloon sizes could possibly be secondary to the differences in pathophysiology of balloon dilatation noted above, with smaller balloon sizes resulting in effective dilatation in the presence of commissural fusion and heavy nodular calcification and with larger balloon sizes needed for less calcified valves without commissural fusion. Nevertheless, the range of balloon sizes that has been noted in this small series suggests that successful percutaneous valvuloplasty will probably necessitate serial balloon dilatations. Third, the marked reversal of left ventricular dysfunction that occurred in our first patient, with left ventricular ejection fraction increasing from 20% to 58% over a period of 6 weeks, suggests that small increases in aortic valve area may lead to major improvements in global ventricular function. Finally, the lack of successful palliation of angina in our second patient suggests that aortic valve dilatation may be of limited value in the presence of severe coronary artery disease.

**Conclusions.** The results of this study suggest that patients with significant aortic stenosis who are not considered surgical candidates and in whom medical therapy is failing may undergo aortic valvuloplasty as a palliative procedure. Patients with moderate or severe preexisting aortic insufficiency should not be considered as candidates for balloon valvuloplasty because of the potential for increasing aortic regurgitation. Similarly, patients with coexisting severe coronary artery disease who primarily are symptomatic from angina may also not be candidates because of the potential for continued ongoing chest pain. The mechanism of successful dilatation is varied and includes separation of fused commissures, fracture of nodular calcification, or plastic deformation of cusps. In the limited experience to date, liberation of calcific or other valvular debris and the production of massive aortic insufficiency do not appear to be significant risks.

**Addendum**

Since this article was submitted, we have done percutaneous balloon valvuloplasty in an additional seven elderly patients with similar hemodynamic results and no evidence of systemic embolization or substantial increase in aortic regurgitation.

**References**

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