Immediate and Follow-Up Results of Transluminal Balloon Dilation For Discrete Subaortic Stenosis

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This study presents the findings in 33 patients with discrete subaortic stenosis who were treated by percutaneous balloon dilation and were followed up for 2 months to 6.2 years (mean 34 ± 21 months). The mean age was 13 ± 11 years; 10 (30%) were female and 23 (70%) male. Associated malformations were observed in nine patients (27%). All patients underwent noninvasive studies and cardiac catheterization. The mean valve to membrane distance was 4.5 ± 2 mm/m².

After balloon dilation, the pressure gradient from the left ventricle to the aorta decreased from 68 ± 30 to 20 ± 13 mm Hg (p < 0.00001); there were no significant changes in the degree of aortic regurgitation. A fluttering and widely mobile remaining membrane was clearly visualized after dilation. Better immediate results were obtained in patients with a smaller baseline gradient, a larger aortic anulus and a longer valve to membrane distance.

Serial follow-up echographic studies were available in 30 patients, and 18 hemodynamic reevaluations were performed in 13 patients. However, seven patients who demonstrated restenosis underwent redilation at a mean of 29 ± 17 months after the first dilation. Redilation in six of the seven patients obtained benefits similar to those observed at the first dilation. Only one patient with unsuccessful redilation required surgery. The mean value of the last explored residual gradient (on hemodynamic or Doppler study) in the remaining 32 patients was 21 ± 10 mm Hg. No significant changes were observed in the angiographic evolution of aortic regurgitation.

In conclusion, percutaneous balloon dilation may be a useful complement to surgical therapy in patients with a thin discrete subaortic stenosis. However, close follow-up is needed.

(J Am Coll Cardiol 1991;18:1309–15)

Transluminal balloon dilation has been proposed as a safe alternative for relieving the subvalvular gradient in patients with discrete subaortic stenosis (1). Since the first description, several studies (2–4) have confirmed initial good results in children and young adults. However, factors influencing the degree of pressure relief after balloon dilation are not known, although dilation has been unsuccessful in patients with a fibromuscular ridge (2). Furthermore, little information is available regarding the rate of restenosis and long-term results (5,6). We describe our findings in 33 patients with discrete subaortic stenosis who were treated by percutaneous balloon dilation and were followed up for a mean of 34 ± 21 months.

Methods

Study patients (Table 1). From January 1985 to March 1991, we performed 40 dilation procedures in 33 patients with discrete subaortic stenosis. The indication for balloon dilation always met the following criteria: 1) a thin fixed subaortic membrane <3 mm in width, 2) a hemodynamically detected subaortic chamber, and 3) absence of more than grade II aortic regurgitation. We considered a membrane thin when it was difficult to visualize or, when measurable, it was ≤3 mm thick. No collar-like or tunnel-type subaortic obstructions were attempted.

The patients' age ranged from 2 to 55 years (mean 13 ± 11). Most (n = 24) of the 33 patients were asymptomatic. Six patients reported dyspnea and two angina; two had a syncopal episode. Two patients had associated congenital mitral stenosis and underwent dilation of both lesions during the same procedure (7). Another patient who presented with associated coarctation of aorta also had both lesions dilated at the same procedure (8). All 33 patients underwent baseline noninvasive studies including two-dimensional and Doppler echocardiography on the day before cardiac catheterization. In 31 patients, a fixed subaortic membrane was clearly visualized. Before hospital discharge, the same noninvasive

Table 1. Baseline Data in 33 Patients Who Underwent
40 Dilations

Clinical	
Age (yr) (mean; range)	13 ± 11; 2–55
Male patients (no. [%])	23 [70%]
BSA (m²)	1.34 ± 0.5
Associated malformations	
Mitral stenosis	2 (6%)
Coarctation of aorta	1 (3%)
Previous surgery for coarctation	4 (12%)
Previous surgery for PDA	1 (3%)
Previous spontaneous closure of VSD	1 (3%)
Hemodynamic and angiographic	
Peak LV systolic pressure (mm Hg)	176 ± 33
Peak LV to aorta gradient (mm Hg)	68 ± 30
Aortic anulus/BSA (mm/m²)	16 ± 4
LV mass/BSA (mm/m²)	106 ± 60
Mass/volume ratio (g/ml)	0.85 ± 0.3
Valve to membrane distance/BSA (mm/m²)	4.5 ± 2
Technical	
Balloon diameter (mm)	24 ± 6
Anulus/balloon ratio	0.88 ± 0.1
Type of catheter	
Single balloon	16 (40%)
Multiballoon	24 (60%)
Retrograde approach	
With circuit	12 (30%)
Without circuit	28 (70%)
Associated procedures	
Mitral balloon valvuloplasty	2 (6%)
Coarctation angioplasty	1 (3%)
Definitive pacemaker*	1 (3%)

*Previous procedure. BSA = body surface area; LV = left ventricular; PDA = patent ductus arteriosus; VSD = ventricular septal defect.

study was repeated. Table 1 shows baseline clinical and hemodynamic data, as well as technical information.

Technique. Written informed consent was always obtained from the patients or parents. All patients underwent right and left heart diagnostic and therapeutic cardiac catheterization, as previously summarized (1). Diagnostic procedures always included simultaneous pressure measurements, determination of cardiac output (dye-dilution or thermodilution technique) and left ventricular (30° right anterior oblique and long-axis views) and aortic root (lateral projection or 60° left anterior oblique projection) angiograms. After these studies, heparin (100 IU/kg body weight) was administered and a guide wire was placed in the left ventricle through a retrogradely advanced end-orifice catheter in 28 procedures; in the remaining 12 procedures, the guide wire was passed through a venous catheter advanced transseptally to the left atrium, left ventricle and aorta, where the guide wire was snared out of the body through the left femoral artery, as described by Babic et al. (9). A single or multiballoon catheter, ranging in total diameter from 15 to 38 mm, was then retrogradely advanced to the left ventricular cavity over the long guide wire. Balloon size was selected on the basis of the angiographically measured aortic

anulus diameter. This required the balloon diameter to be almost identical to that of the aortic ring or even slightly greater when the pressure relief obtained was inadequate. The balloon was briefly inflated two to six times until the notch disappeared. At the end of the therapeutic phase, the immediate results were evaluated hemodynamically and angiographically under conditions identical to those described in the diagnostic phase.

Follow-up studies. A close clinical follow-up ranging from 2 months to 6.2 years was established for all patients. Doppler echocardiographic studies were available in 30 patients. In addition, we performed 18 hemodynamic reevaluations in 13 patients at a mean follow-up interval of 24 ± 19 months after balloon dilation. Restenosis was defined as the loss of ≥50% of initial gain in the degree of pressure relief, as determined by Doppler echocardiographic studies and confirmed by cardiac catheterization. According to this criterion, seven patients (21%) had restenosis and were treated by redilation a mean of 29 ± 17 months after the first dilation.

Quantitative angiography. Quantitative left ventricular analysis was performed in every angiographic study. Left ventricular volumes, mass and ejection fraction were determined in each condition with use of standard angiographic methods for the single-plane right anterior oblique projection (10). The end-diastolic and end-systolic silhouettes of the left ventricle were drawn, including the valve and subaortic membrane. The diastolic distance between the midpoint of the aortic valve and the center of the membrane was measured (valve to membrane distance); this distance ranged from 1.5 to 12.2 mm/m². The aortic root was also measured. All values were corrected for body surface area. Aortic regurgitation was graded according to the criteria proposed by Sellers et al. (11).

Statistical analysis. Results are expressed as mean values ± SD unless otherwise stated. The paired t test was used to compare two mean values. Differences between proportions were studied by chi-square and Fisher's exact tests as appropriate. Correlations between variables were evaluated by using standard linear regression analyses; correlation coefficients (r) were obtained for each comparison. Event-free probability curves were constructed with use of the Kaplan-Meier method. To identify the factors influencing restenosis, we calculated the 5-year restenosis-free probability in the different groups (Table 2). The log-rank test was used to determine the statistical significance of the differences in cumulative restenosis rate. A p value < 0.05 was considered significant.

Results

Immediate results. After balloon dilation, the left ventricular to aortic gradient decreased significantly from 68 ± 30 to 20 ± 13 mm Hg (p < 0.00001) (Fig. 1); in 3 patients (9%), the gradient disappeared completely and in 11 (33%), the immediate residual gradient was <10 mm Hg. The mean percent

Table 2. Factors Influencing Immediate and Long-Term Results in 33 Patients (40 procedures)

	Immediate Residual Gradient (mm Hg)	5-Year Restenosis-Free Probability (%)*
Age (yr)		
>13 (n = 15)	23 ± 15 } $p = NS$	100 } $p < 0.05$
<13 (n = 25)	19 ± 12	38 ± 16
Peak gradient (mm Hg)		
<50 (n = 11)	13 ± 8 } $p < 0.01$	66 ± 27 } $p = NS$
>50 (n = 29)	23 ± 13	39 ± 19
Anulus/BSA (mm/m²)		
<20 (n = 32)	22 ± 13 } $p < 0.01$	33 ± 18 } $p = NS$
>20 (n = 8)	11 ± 7	85 ± 13
V-M distance/BSA (mm/m²)		
<4.5 (n = 19)	25 ± 11 } $p < 0.01$	50 ± 23 } $p = NS$
>4.5 (n = 19)	14 ± 11	28 ± 23
Anulus/balloon ratio		
<1 (n = 32)	21 ± 12 } $p = NS$	48 ± 22 } $p = NS$
>1 (n = 8)	15 ± 14	44 ± 22

*By Kaplan-Meier method, expressed as % ± SE. BSA = body surface area; V-M = valve to membrane.

gradient reduction was $69 \pm 20\%$. Angiographically, there were no significant changes in left ventricular volumes or ejection fraction. No significant changes were observed in the degree of aortic regurgitation; in two patients,

Figure 1. Simultaneous pressure recordings from a patient before (BASAL) and after (POST) treatment. In both conditions, a pullback from the left ventricular outflow chamber to the ascending aorta was recorded.
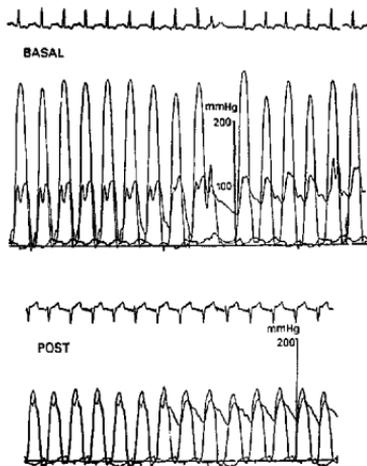
regurgitation disappeared immediately after dilation. In 30 patients, a fluttering and widely mobile remaining structure was clearly visualized on angiography in the outflow tract after dilation (Fig. 2). This finding was also observed on two-dimensional echocardiography (1). Improvements in aortic valve opening were frequently noted. Factors influencing the degree of pressure relief are shown in Table 2. The immediate residual gradient was smaller in patients with a lesser baseline gradient, larger anulus diameter and longer valve to membrane distance. There was a significant inverse correlation between the immediate residual gradient and the valve to membrane distance ($r = -0.40$; $p < 0.05$).

Complications and outcome. One 8-year old patient who had combined mitral stenosis developed severe mitral regurgitation after balloon mitral valvuloplasty. He required an operation that was performed 7 days later. Operative findings revealed a rupture of the anterior leaflet; the valve was replaced by a mechanical prosthesis (21 mm) and the patient did well. The remaining 32 patients did not have major complications at the time of the 39 therapeutic procedures. Mitral regurgitation of any degree was never induced by balloon dilation of the membrane. Three patients had a decreased femoral pulse at the puncture site without ischemic compromise; four patients had blood loss during the procedure and required blood transfusion. All patients without major complications during the 39 procedures were discharged within 2 to 6 days after a new noninvasive evaluation was performed.

Follow-up. One 55-year old patient died of noncardiac causes 1 year after dilation. The remaining 32 patients are alive 34 ± 21 months later; 30 are asymptomatic, and 2 still have mild dyspnea; no syncope or palpitation was recorded. None developed endocarditis or other complications. Reste-
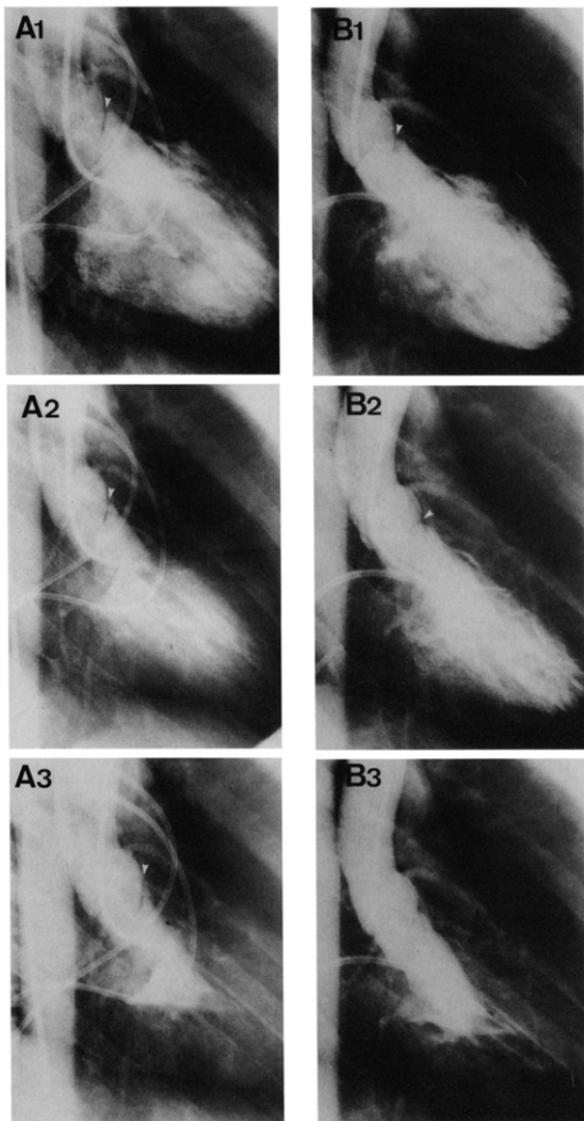
Figure 2. Angiographic sequences of left ventricular contraction before ($A_{1,2,3}$) and after ($B_{1,2,3}$) treatment (1 = end-diastole; 2 = mid-systole; 3 = end-systole). Note the motion of the remaining membrane after balloon dilation (**white arrowhead**).
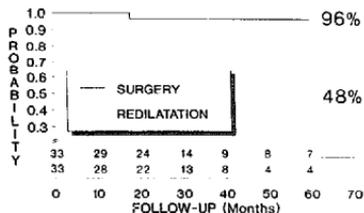
Figure 3. Kaplan-Meier event-free curves in our 33 patients. The number of patients who still remain under observation without surgery (upper row) or without redilation (lower row) is expressed on the x axis.

nosis was suspected clinically in seven patients because of changing murmur intensity: in all seven, the increase in residual gradient was confirmed by Doppler echocardiography and cardiac catheterization. The increased mobility of the remaining membrane when observed after dilation persisted at follow-up in patients who did not exhibit restenosis, but it was not identified when restenosis was clinically detected. No progression of left ventricular hypertrophy was detected. No relevant changes in the degree of aortic regurgitation were observed on continuous wave Doppler color echocardiography at late follow-up.

We performed 18 hemodynamic reevaluations in 13 patients; 7 of these studies included repeat dilation procedures. In our seven patients with restenosis, the anatomic features of the membrane reproduced those observed at the first dilation. The only significant factor influencing restenosis rate was age (Table 2). All patients who presented with restenosis were <13 years old, whereas none >13 years old developed restenosis. Redilation was performed in seven patients with restenosis at a mean of 29 ± 17 months after the first dilation. It was considered successful in six and unsuccessful in one whose gradient did not change; this patient was surgically treated 2 months later. In the six patients, the degree of pressure relief obtained (61 ± 19%) was similar to that observed on the first dilation.

Figure 3 shows the observed event-free probability curves after balloon dilation; 52% of patients may require redilation at 5 years of follow-up, but only 4% may require surgical resection. Figure 4 shows the evolution of the catheterization- or Doppler-derived residual gradient observed at late follow-up study; that in the group of seven patients who had restenosis is shown separately. The last explored mean peak residual gradient (on hemodynamic or Doppler study) was 21 ± 10 mm Hg in 32 patients. No significant changes were observed in left ventricular volumes or ejection fraction at follow-up. The mass/volume ratio in reevaluated patients was not significantly different (0.9 ± 0.4 g/ml) from that observed during baseline conditions. Figure 5 shows the
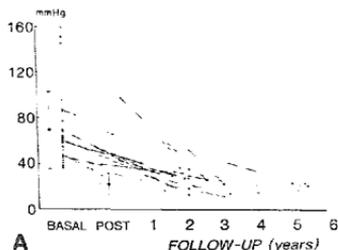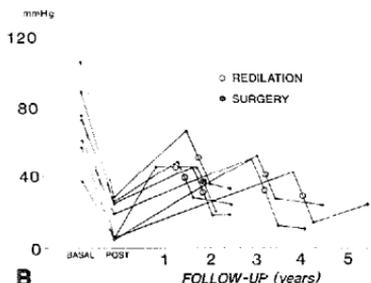

Figure 4. Evolution of peak residual hemodynamic or Doppler gradient in 26 patients who did not have restenosis (22 of them were followed up for >1 year) (A) and 7 patients who had restenosis requiring redilation in 6 and surgery in 1 (B). Abbreviations as in Figure 1.

angiographic evolution of aortic regurgitation; no significant changes were observed at follow-up. Most reevaluated patients had no or a mild degree of aortic regurgitation as compared with that observed immediately after balloon dilation.

Figure 5. Angiographic evolution of the degree of aortic regurgitation in 33 patients as evaluated by the criteria of Sellers et al. (11). Abbreviations as in Figure 1.
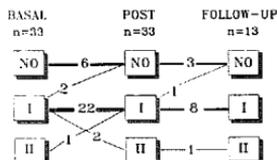
Discussion

Discrete subaortic stenosis. Under the term discrete subaortic stenosis lies a spectrum of disease related to fixed and localized subaortic structures. As differentiated from idiopathic hypertrophic subaortic stenosis, three types of discrete subaortic stenosis can be distinguished (12–14). However, the most common clinical presentation (85%) is that of the membranous type (13) and our study focuses on this form of the disease.

The natural history of discrete subaortic stenosis has been described (15,16). Serial hemodynamic studies (13,17) have shown that the gradient usually increases over time in patients who are not operated on. Aortic regurgitation, infectious endocarditis and extended muscular obstruction can complicate the natural course. For these reasons, discrete subaortic stenosis is considered to be a potentially progressive disease.

Indications for treatment. Surgery is an effective and safe treatment in reducing the left ventricular to aortic gradient. Indications for operation have included a pressure gradient >50 mm Hg, electrocardiographic signs of left ventricular strain or symptoms such as dyspnea, angina or syncope (18,19). However, the progressive nature of the disease led others (13–15,20,21) to recommend surgical treatment in patients with mild subaortic obstruction. Precise recommendations for operation based on gradient level are not available because the postoperative gradient in some reported series (13,20) is higher than the initial gradient in the group with mild subaortic obstruction (14,15).

Postoperative follow-up. Although the pressure relief after surgical treatment persists in most patients, several reports (13,15,17,20,22) have shown an increase in the residual gradient in some patients at the time of late postoperative catheterization. Long-term follow-up studies (1 to 17 years) have shown a mean peak residual gradient of 20 to 30 mm Hg in most series (14,15,20,21,23). Aortic regurgitation can be arrested but not reversed by surgical resection (14,20,21). Some investigators (20,23) noted that aortic regurgitation may develop even after surgical relief of discrete subaortic stenosis, which is more frequent in patients with a high preoperative pressure gradient and seems related to a longer time since operation (23). Thus, over time, a large proportion of surgically treated patients may become candidates for additional surgical procedures because of residual or recurrent stenosis or progressive deterioration of aortic valve function.

Balloon dilation. Our findings show that percutaneous balloon dilation is an effective and safe method for reducing subaortic obstruction in patients with discrete subaortic stenosis. Two possible mechanisms may explain the pressure relief obtained after balloon dilation. One is stretching of the stenotic orifice (3) and the other is tearing of the fibrous tissue (2). Our observations suggest that the second mechanism is operative in most patients. After dilation, the fixed subaortic structure becomes widely mobile and fluttering in accordance with blood flow (Fig. 2). Better immediate results were observed in patients with a lower baseline gradient, larger aortic anulus and a membrane farther away from the valve (Table 2). Of interest, the anulus size may similarly influence the result in surgically treated patients. The fibrous tissue can involve the aortic valve anulus, an effect that probably contributes toward limiting anulus size (13,22,24,25). Indeed, recent studies (26) have shown retardation of growth of the aortic root among patients with discrete subaortic stenosis as compared with a control group. In other instances, the membrane can be more remote in the outflow tract (13,22,27), probably representing a more localized and nonadherent fibrous structure that could be more widely torn by balloon inflation.

Follow-up after balloon dilation. Subsequent anatomic and physiopathologic changes that can occur with a broken and widely mobile membrane in the outflow tract remain unknown. However, at a mean follow-up time of 34 ± 21 months, none of our patients had infectious endocarditis, extended muscular obstruction or significant progression of aortic regurgitation. Actuarial analysis suggests that in 48% of patients, the obtained pressure relief after dilation may persist at 5 years, but restenosis may also develop and its nature is uncertain. In our seven patients with restenosis, the echocardiographic and angiographic features of the membrane reproduced those observed at the first attempted dilation, a finding that suggests that recurrence of obstruction may result from regrowth of fibrous tissue. The persistent turbulence caused by residual stenosis may provoke recurrence of fixed subaortic obstruction. In fact, mobility of the membrane could not be observed when restenosis was detected. Restenosis seems to be influenced by patient age and by time after dilation. In our study, the mean patient age was 13 years and we selected that average as a cutoff point for comparison. To date, no patient >13 years of age has had restenosis, the follow-up time being similar in younger and older patients. Explanations for this significant influence remain speculative, but although further confirmation is needed, it is possible that recurrence after balloon dilation is more infrequent after puberty.

Redilation can also be attempted safely and in six of our seven patients, it brought about benefits in the degree of pressure relief similar to those observed after the first dilation. After 14 ± 3 months, all six patients with successful redilation remain with a degree of pressure relief (last explored residual gradient 22 ± 7 mm Hg) similar to that observed immediately after redilation. The persistent pressure relief obtained in most patients after balloon dilation could favorably influence the evolution of aortic regurgitation. In fact, most angiographically reevaluated patients had no or only a mild degree of aortic regurgitation. Similar findings were observed by Doppler echocardiographic analysis. Thus, balloon dilation does not seem to unfavorably affect valve competence. However, because we know that aortic regurgitation may develop even after surgical resec-

tion (20,23), further evaluation after balloon dilation is needed.

Conclusions. Because discrete subaortic stenosis is a progressive disease, we believe that during the course of the disease, percutaneous balloon dilation may represent a complementary treatment to surgery in patients with a thin subaortic membrane. The initial rupture of the membrane is a safe procedure that persistently relieves the left ventricular pressure overload and can be repeated if restenosis develops. The long-term efficacy of a second dilation is still not known, but mid-term evaluations in a lesser number of patients reveal a persistent residual gradient decrease. Thus, close follow-up is needed because >50% of patients may have a recurrence over a period of 5 years. This possibility seems to decrease significantly in older patients. If gradient reduction somehow prevents further complications, the procedure may be useful in delaying or even avoiding the need for early operation in most patients as assessed by a 5-year study. However, given the progressive nature of the disease, a larger series studied over longer periods of time is also needed to further determine the long-term influence of this treatment on the course of the disease.

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