Long-Term Clinical and Echocardiographic Follow-Up After Percutaneous Mitral Valvuloplasty With the Inoue Balloon

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***Background*—**The objective of this study was to assess the long-term clinical outcome and valvular changes (area and regurgitation) after percutaneous mitral valvuloplasty (PMV).

***Methods and Results*—**After PMV, 561 patients were followed up for 39 (±23) months and clinical/echocardiographic data obtained yearly. Kaplan-Meier and Cox regression analyses were performed to estimate event-free survival, its predictors, and the relative risks of several patient subgroups. There were several nonexclusive events: 19 (3.3%) cardiac deaths, 55 (9.8%) mitral replacements, 6 (1%) repeated PMVs, 56 (10%) cases of restenosis, and 108 (19%) cases of clinical impairment. Survival free of major events (cardiac death, mitral surgery, repeat PMV, or functional impairment) was 69% at 7 years, ranging from 88% to 40% in different subgroups of patients. Wilkins score was the best preprocedural predictor of mitral opening, but the procedural result (mitral area and regurgitation) was the only independent predictor of major event-free survival. Mitral area loss, though mild [0.13 (±0.21)cm^2], increased with time and was ≥0.3 cm^2 in 12%, 22%, and 27% of patients at 3, 5, and 7 years, respectively. Regurgitation did not progress in 81% of patients, and when it occurred it was usually by 1 grade.

***Conclusions*—**Seven years after PMV, more than two thirds of patients were in good clinical condition and free of any major event. The procedural result was the main determinant of long-term outcome, although a high score had also negative implications. Mitral area decreased progressively over time, whereas regurgitation did not tend to progress. **(*Circulation*. 1999;99:1580-1586.)**

Key Words: mitral valve ■ valvuloplasty ■ follow-up studies ■ restenosis ■ survival

Since its introduction in 1984 by Inoue,[1] percutaneous mitral valvuloplasty (PMV) has emerged as a safe and effective procedure[2–7] for the treatment of symptomatic mitral stenosis with immediate results that may be comparable to those of surgical commissurotomy. Although some series have provided long-term clinical data,[8–15] very little information is available concerning longitudinal changes in mitral valve area (MVA) and regurgitation (MR) long after the procedure. The aim of the present study was (1) to describe and analyze long-term clinical results after PMV and (2) to provide new data on longitudinal changes in MVA and MR, not only in patients with a good procedural result but also in those with poor valvular opening or significant regurgitation.

Methods

Patients

From 1989 to 1995, we performed 620 PMV procedures; in 8 (1.2%) cases no balloon inflation was performed (in 4 because of tamponade), 8 (1.2%) patients required in-hospital mitral surgery, and 3 (0.4%) died. Of the 601 patients discharged after a complete procedure, 40 were lost of follow-up. The remaining 561 (93% of eligible) were indeed followed up for 39 (±23) months (range 6 to 87) and are the subject of this report.

Procedure

PMV was performed with the Inoue balloon. Balloon size was selected according to body surface area (26 mm if <1.5 m^2, 28 mm if 1.5 to 1.7 m^2, and 30 mm if >1.7 m^2), modulated by anatomy (1 to 2 mm smaller in unfavorable cases), and reached after several stepwise inflations. Left ventriculography was performed before and after the last balloon inflation.

Preprocedural and Postprocedural Assessments

Clinical status was determined by New York Heart Association (NYHA) classification. All patients underwent an echo-Doppler study before and 24 hours after PMV. Evaluation included Wilkins scoring,[16] MVA calculation (pressure half-time method[17]), and MR estimation (graded as none, mild, moderate, or severe by color-Doppler semiquantitative method).[18] With the purpose that MVA and MR would be comparable over time, preprocedure, postprocedure, and follow-up data were analyzed on the basis of echo-Doppler studies. If a ≥1 grade discrepancy on MR grading existed between left ventriculography and Doppler, both studies were reviewed, the color-Doppler study (transthoracic and transesophageal) was repeated, and a consensus finally was reached.

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Follow-Up

Patients were scheduled for clinical and echocardiographic follow-up in a monographic outpatient clinic. The first visit was 6 months after PMV and yearly thereafter. If a patient failed to keep any programmed visit, he or she or his or her physician were encouraged to come back and/or send clinical/Echo-Doppler reports performed elsewhere.

Definitions

Immediate results were defined as "good opening" when post-PMV Doppler MVA was ≥1.5 cm^2 and regurgitation moderate or less; "insufficient opening" when MVA <1.5 cm^2 and regurgitation moderate or less; and "severe regurgitation" (irrespective of post-procedural area). Clinical improvement after PMV was considered as ≥1 NYHA class and class ≤2. Follow-up functional impairment was considered as ≥1 NYHA class and class ≥3. Major cardiac events were cardiac death, surgery, repeat PMV, or functional impairment. Restenosis was defined as loss of ≥50% of initial gain and MVA <1.5 cm^2. Significant area loss was defined as MVA loss ≥0.3 cm^2.

Statistical Analysis

Data are presented as mean (±SD). A value of $P<0.05$ was considered significant. Discrete data were compared by χ^2 analysis and continuous data with the Student's 2-tailed *t* test. The RR for non–time-related variables was determined by simple arithmetic calculations, and the 95% CI was determined on the basis of the normal distribution of the variable. A logistic regression model was adjusted to determine independent predictors of immediate result, and a linear regression model was used to predict MVA loss as a function of time. Event-free survival rate for several single and composite end points was estimated with the use of Kaplan-Meier analysis.[19] When death was noncardiac, data on that patient were censored at the time of death. Survival curves for different patient subgroups were compared with the use of the Breslow exact statistic. With the assumption of proportional hazards, the adjusted RR and CI (95%) was calculated from a Cox regression model.[20] To identify independent predictors of event-free survival, statistically significant variables in the Cox univariate analysis were selected and 2 models constructed, the first based on baseline variables and the second on preprocedural, intraprocedural, and postprocedural variables. Several subgroups were defined according to these predictors, and Kaplan-Meier curves were estimated in each subgroup. Adjusted RR was calculated from the Cox regression model.

Results

Baseline characteristics and initial results are displayed in Table 1. The Inoue balloon diameter was 28.4±2.2 mm, the effective balloon dilation area/body surface area fraction was 3.9±0.2, and patients received a mean of 2.2±1.1 inflations. Global result was summarized as good results in 435 (78%), insufficient opening in 101 (18%), and severe regurgitation in 25 (4%). Patients with good results were younger (50 vs 55 years, $P<0.05$) and had a lower score (7.3 vs 8.6, $P<0.05$) than those with insufficient opening. By contrast, patients with severe regurgitation were similar in age (49 vs 50 years, NS) and score (7.4 vs 7.3, NS) than those with good results, but they had an smaller pre-PMV MVA (0.84 vs 1.0 cm^2, $P<0.05$). RR of severe MR in subgroups of patients defined according to baseline MVA and score is shown in Table 2.

Follow-Up Events

Follow-up events were: (1) Death: 29 patients died, 10 (1.7%) of noncardiac and 19 (3.3%) of cardiac causes (4 after mitral replacement, 1 of prosthetic endocarditis, 3 of persistent severe pulmonary hypertension after successful PMV, and 11 of heart failure; (2)Mitral replacement: 55 (9.8%) patients underwent surgery indicated by poor result in 13, MR in 21, a combination of both in 14, and restenosis in 7; (3) Repeat PMV: 6 procedures in 5 patients; (4)Restenosis: 56 (10%) patients met restenosis definition, but only 30 were in class ≥3; (5) Functional impairment occurred in 108 patients; 60 underwent invasive procedures, and 48 did not, 11 because clinical condition was thought to be secondary to severe pulmonary hypertension and 37 because of surgical high risk; 10 of these 48 patients died. Event-free survival curves for several end points are presented in Figure 1.

TABLE 1. Baseline, Postprocedural, and Last Follow-Up Clinical and Echocardiographic Data

	Pre-PMV	Post-PMV	Last Follow-Up, at 39 (±23) Months
Age, y	53 (13)		
Female sex	462 (82%)		
Sinus rhythm	240 (43%)		
Previous commissurotomy	70 (12%)		
Wilkins score			
Flexibility	1.62 (0.62)		
Thickening	2.17 (0.61)		
Subvalvular	2.14 (0.61)		
Calcification	1.63 (0.75)		
Mitral area, cm^2	0.98 (0.22)	1.79 (0.40)	1.68 (0.38)
Mitral regurgitation			
None	327 (58%)	197 (35%)	171 (30%)
Mild	220 (39%)	255 (45.5%)	268 (48%)
Moderate	14 (3%)	84 (15%)	83 (15%)
Severe	0	25 (4.5%)	39 (7%)
Clinical improvement		510 (91%)	387 (69%)
NYHA class			
I	2 (0.3%)	381 (68%)	265 (47%)
II	285 (51%)	152 (27%)	123 (22%)
III	255 (46%)	21 (4%)	145 (26%)
IV	19 (3%)	7 (1%)	28 (5%)

Quantitative data are expressed as mean (SD).

Echocardiographic and Clinical Data During Follow-Up

Echocardiographic and clinical data during follow-up are displayed in Table 1.

MVA Loss During Follow-Up

For analysis of MVA loss, only patients with follow-up ≥1 year (n=480) were included. Because Doppler MVA deter-

TABLE 2. Preprocedural Area and Incidence of Severe Mitral Regurgitation

	Preprocedural Mitral Area, cm^2		
	≤0.79	0.80–0.99	≥1.0
No. of patients	68 (12%)	188 (33%)	305 (55%)
Severe regurgitation, %	8 (11.7%)	9 (4.8%)	8 (2.6%)
Relative risk (95% CI)	4.5 (2.9–7.1)	1.8 (1.2–3.9)	1

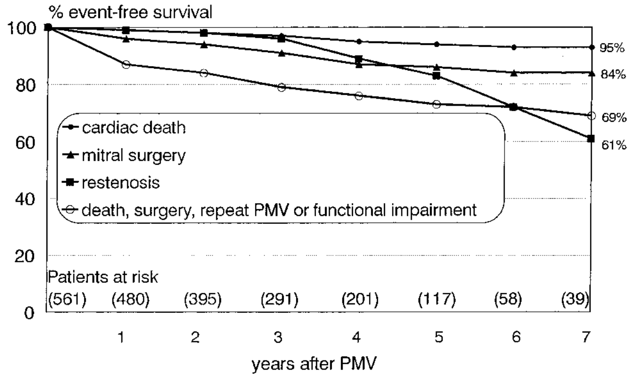

Figure 1. Event-free survival curves for several end points after PMV.

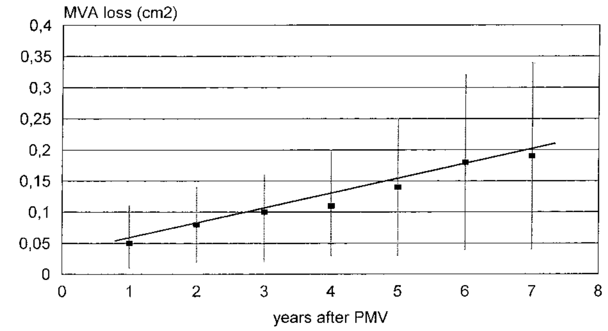

Figure 2. MVA loss as a function of time in patients followed up for >1 year (n=480).

mined early after PMV is not accurate enough because of acute changes in left atrial compliance, we considered the Doppler MVA determined at 6 months (when hemodynamic conditions are already stable) as baseline. MVA loss was variable, with a mean of 0.13 (±0.21) cm^2, and was remarkably similar in all score subgroups (Table 3). It was clearly time related (Figure 2) and consequently, the rate of patients with significant MVA loss (≥0.3 cm^2) increased from 12% to 22% and 27% at 3, 5, and 7 years, respectively. None of the baseline characteristics were able to predict area loss, being that postprocedural MVA (larger area, larger loss, P=0.020) and follow-up duration (P=0.001) were the only independent predictors.

MR During Follow-Up

MR did not change significantly during follow-up for the whole population, and if severe, it remained unchanged. When regurgitation was not severe, it increased by 1 grade in 114 (20%) and decreased in 77 (14%), usually from mild to moderate and vice versa. In 14 (3%) patients there was a well-documented ≥2 grade increase in MR severity, usually progressive over time; surprisingly, 8 patients had a 2-grade decrease in MR.

Predictors of Event-Free Survival

Several variables (age, sex, cardiac rhythm, previous commissurotomy, score, balloon size, and postprocedural MVA and MR) were tested as potential predictors of event-free survival by continuous univariate Cox analysis, and significant variables were categorized (Table 4). On multivariate analysis and considering only baseline variables, score (P=0.0021) was found to have an independent value. When preprocedural, intraprocedural, and postprocedural variables were included in the multivariate analysis, only post-PMV MVA (P=0.0000) and MR (P=0.0000) were independent predictors. Several subgroups of patients were established according to independent predictors (MVA and MR) and the best preprocedural predictor (score) of event-free survival (Figure 3). A single cut-off point was established: 1.5 cm^2 for MVA and 8 for score. In the case of MR, given that Kaplan-Meier curves for mild and moderate MR tended to converge after 3 years (Figure 4), subgroups were none/mild/moderate and severe MR.

To explore the impact of score in major event-free survival, curves for 4 score ranges were constructed (Figure 5). Each score component was also individually analyzed (1 to 2 vs 3 to 4). Kaplan-Meier curves were not different in the case of flexibility and subvalvular components of the score, whereas a high score for thickening (74% vs 68% at 7 years, P=0.020) and calcification (71% vs 54% at 7 years, P=0.001) had a significant impact on event-free survival.

TABLE 3. Mitral Area (in cm^2) in 4 Subgroups of Patients Defined by Score

	4–6	7–8	9–10	11–13
No. (%)	125 (26%)	241 (50%)	81 (17%)	34 (7%)
Baseline	1.12 (0.19)	0.99 (0.21)	0.91 (0.15)	0.85 (0.17)
6 Months	2.13 (0.29)	1.85 (0.26)	1.61 (0.24)	1.46 (0.22)
Last follow-up	1.89 (0.27)	1.62 (0.25)	1.43 (0.24)	1.28 (0.24)
Gain	1.01 (0.23)	0.86 (0.22)	0.70 (0.24)	0.61 (0.21)
Loss	0.24 (0.24)	0.23 (0.26)	0.18 (0.21)	0.18 (0.19)

All patients were followed up for ≥1 year (n=480).

Predictors of Restenosis

Because restenosis was not considered a major event, we estimated the restenosis-free survival rate (Figure 1). Potential predictors were tested, but only score (P=0.0002) was found to have an independent value. Since postprocedural MVA was not a predictor of restenosis as a continuous variable, several cut-off points were explored, and an MVA ≥1.8 cm^2 was found to be a negative predictor of restenosis (P=0.0009) better than score ≤8 (P=0.0018).

Discussion

Our report involves PMV in a population with a majority of middle-age women with pliable valves, although patients with a less favorable profile were also present. This series of Inoue balloon procedures is large and homogeneous, and follow-up was universal regardless of procedural result. The information provided on longitudinal changes in MVA and regurgitation is new and may contribute to our current understanding of post-PMV mitral disease progression.

Immediate Results

Procedural result was summarized as good in 78% of cases, 18% had insufficient opening, and 5.7% (4.5% in these series plus 1.2% who required in-hospital surgery), had severe MR.

TABLE 4. Predictors of Event-Free Survival: Univariate Analysis

	Patients	Events	1 y	3 y	5 y	7 y	*P*	RR (95% CI)
Sex								
Male	111	13	98	95	84	67	0.1213	1
Female	450	60	96	87	80	77		1.22 (0.67–2.22)
Age, y								
<50	278	32	96	91	83	78	0.2514	1
50–65	200	27	97	90	82	73		1.25 (0.75–2.09)
>65	83	14	96	80	73	72		1.82 (0.97–3.47)
Previous comm								
No	496	57	97	91	83	76	0.0001	1
Yes	65	16	94	80	72	67		2.4 (1.44–3.99)
Sinus rhythm	240	25	97	92	85	75	0.0513	1
Fibrillation	321	48	95	86	80	66		1.56 (0.94–2.53)
MVA pre-PMV								
<1 cm^2	259	39	95	88	80	71	0.1910	2.56 (0.82–5.93)
≥1 cm^2	302	34	97	91	83	70		1
Score								
≤8	426	44	96	91	85	83	0.0255	1
9–10	103	19	97	88	72	67		1.86 (1.12–3.08)
≥11	32	10	97	75	57	57		3.51 (1.49–8.27)
Balloon size								
<28 mm	135	21	93	81	80	80	0.0063	1.66 (0.95–2.41)
≥28 mm	426	52	97	91	82	75		1
Post-PMV MVA								
<1.5 cm^2	105	30	90	74	66	61	0.0000	4.21 (2.33–7.59)
1.5–1.7 cm^2	98	18	98	83	73	63		2.43 (1.29–4.56)
>1.7 cm^2	358	25	98	96	89	81		1
Post-PMV MR								
≤Mild	452	44	98	93	85	76	0.0000	1
Moderate	84	17	90	80	73	73		2.04 (1.15–3.59)
Severe	25	12	75	57	45	45		6.12 (3.17–11.80)

Comm indicates surgical commissurotomy; MR, mitral regurgitation; MVA, mitral valve area; and PMV, percutaneous mitral valvuloplasty.

Clinical improvement was the rule for patients with good results, frequent in those with insufficient opening, and still possible in some with severe regurgitation. As reported by others,[3,5–10] mitral anatomy was the best predictor of mitral opening, though a good result could also be obtained in cases with high score.

Some increase in regurgitation was frequent, but severe MR was a rather unexpected complication[21–24] of PMV, although Padial et al[25] have recently reported that dissimilarities in valve thickening, commissural calcification, and severe subvalvular involvement were associated with a higher

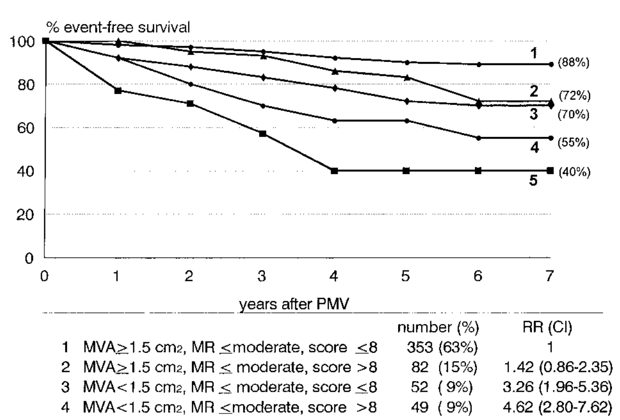

Figure 3. Kaplan-Meier curves for any major event in 5 different subgroups of procedural results.

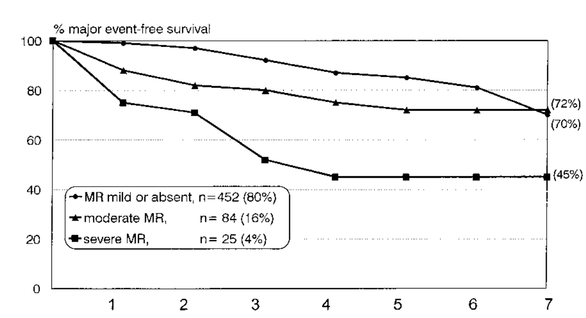

Figure 4. Kaplan-Meier curves for any major event in patients with none/mild, moderate, and severe MR.

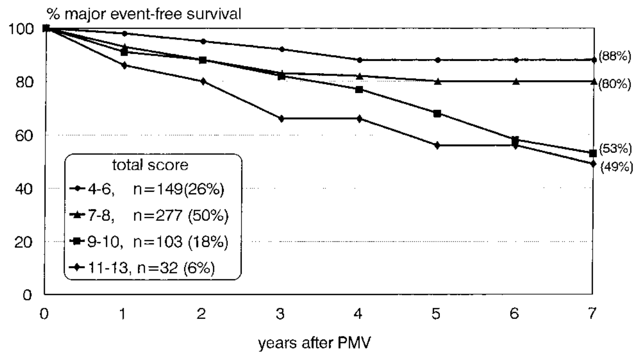

Figure 5. Kaplan-Meier curves for any major event in 4 subgroups of patients defined according to echocardiographic score.

risk. We found that a very tight stenosis was also a risk factor, particularly when associated with a low score. Changes in procedural indication (earlier interventions while MVA is still >1.0 cm^2) and performance (smaller balloons in patients at high risk) might contribute to a rate reduction of severe MR rate.

Progression of MR

Severe MR after PMV (usually caused by a leaflet tear[23]) persists unchanged during follow-up, and 60% of patients required mitral replacement. When MR was mild or moderate (originated at the split commissure[26]), it did not tend to progress in the population as a whole, although it appeared to increase by 1 grade in 20% and decrease in 14% of patients. We believe that many 1-grade variations in MR severity might be caused by interobserver variability of the method rather than in the regurgitation in itself because 1-grade variations either way were frequently observed. Interestingly, long-term outcome tended to be similar to that of patients with nonsignificant MR Clinical implications of truly stable, moderate regurgitation requires further investigation in long-term studies.

Longitudinal Changes in MVA

Disease progression in rheumatic mitral disease may be the result of low-grade subclinical rheumatic process and/or abnormal turbulences generated by the already deformed valve.[27] Both mechanisms might contribute to further commissural fusion, thickening, and calcification of valvular and subvalvular structures both in natural and previously commissurotomized valves. In a study of native mitral valves, Sagie et al[27] reported an MVA loss of 0.09 cm^2/y and found that patients with aortic insufficiency experienced a more accelerated area loss probably related to the stress caused by the aortic regurgitation jet. In another study, Gordon et al[28] reported a similar pace of MVA loss (0.09 cm^2/y) that accelerated in valves with a high score. In a post-PMV series, Chen et al[12] reported an MVA decrease of 0.2 cm^2 at 5 years and Treviño et al[10] reported 0.25 cm^2 at 3 years. In our series, although scattering was wide, MVA decreased 0.2 cm^2 at 7 years (smaller than that reported in untreated valves) and was not influenced by score; nevertheless, because patients with a high score obtained smaller areas, they might have been operated on before a large MVA loss had occurred.

Restenosis is an ambiguous term that includes a mixture of poor result, inaccuracies in MVA determination, very early (within days) area loss, true restenosis, and disease progression. Its definition can be made on a clinical basis or in terms of mitral area, absolute area loss, % of area loss, or loss of gain. With all these confounding factors, it is not surprising that restenosis rate after PMV has ranged from 3% to 70%[9,10] at 1 to 3 years, and restenosis definition in itself may play a major role in restenosis rate. When restenosis is defined as a 50% loss of area gain, patients with a poor initial result meet restenosis criteria with only a mild area loss. This fact explains the apparent contradictory finding that patients with a high score had a smaller mitral area loss but a higher restenosis rate that those with a lower score. On the other hand, very early restenosis (within weeks), caused by a mixture of annular and/or valvular recoil and methodological aspects of MVA calculation, should be differentiate from late, true restenosis. Our data support the fact that restenosis is rare within 3 years of the procedure in patients with a good mitral opening, but it increased over time, reaching 39% at 7 years, although not always associated with clinical impairment. Whether restenosis rate at 3 to 10 years after PMV is similar to that reported after surgical commissurotomy remains unclear, but differences in baseline characteristics might justify a different pace of progression in valvular disease.

Event-Free Survival

Patients with good result have a good prognosis. An unsatisfactory late outcome might be due to a mixture of poor result, regurgitation, restenosis, pulmonary hypertension, polyvalvular disease, or left ventricular dysfunction. Cardiac death during follow-up was due to heart failure in nonoperated patients and to postoperative death in surgical ones. Mitral replacement was required by 15% of patients at 7 years, and its indication was more frequently a poor result rather than progression of mitral disease. Repeated PMV was an option in selected patients with mitral restenosis, but otherwise mitral replacement would be recommended. Long-term event-free survival has been reported by others investigators. Cohen et al[11] found a 51% event-free survival at 6 years in 146 patients (mean age 59 years, score 7.7). Pavlides et al[14] reported an event-free survival rate of 85% at 3 years in 128 patients (mean age 60 years, score 8.6). Dean et al[15] from the National Heart, Lung, and Blood Institute registry (736 patients, mean age 54 years) described an event-free survival of 60% at 4 years, and Jung et al[13] (528 patients with successful procedures, mean age 46 years) described a rate of 76% at 5 years. In our series, the event-free survival was 69% at 7 years and confirms the long-term beneficial effect of PMV. It is important to emphasize that patients with good results and low score (63% of the population undergoing PMV in our center) had a very high probability (88%) of being event free at 7 years. On the other hand, those with less favorable baseline characteristics or poorer results had an event-free survival rate between 55% and 72%, provided no severe MR had occurred. In such cases, 60% of patients will require mitral replacement within 3 years. That simple postprocedural assignment into 3 subgroups with good, intermediate, or poor prognosis may be useful.

Comparison With Surgical Series

Immediate results appear to be very similar[29–32] to closed surgical commissurotomy as reported by Arora et al[29] in a prospective randomized study of very young (mean age 19 years) patients and Raghaba et al[30] in older patients. The only long-term though relatively small (30 patients in each group) randomized study comparing surgical closed, open, and percutaneous commissurotomy has been recently published by Farhat et al[33] in a young population with pliable, noncalcified valves. The 7-year results were better for open and percutaneous procedures than for closed commissurotomy as assessed by a higher event-free survival (93%, 90%, and 50%, respectively), a better mitral area (1.8, 1.8, and 1.3 cm^2 and a lower restenosis rate (6%, 6%, and 37%).

In long-term surgical series,[34–38] Hickey et al[34] reported on 103 patients with closed commissurotomy (mean age 38 years) a mitral replacement rate of 22% at 10 and 53% at 20 years; Rhial et al[35] reported on 267 patients (mean age 43 years) a replacement rate of 43% and 76% at 10 and 20 years. In our series and others,[39] even if patients have a poorer baseline clinical profile (many not even have been candidates for commissurotomy should open heart surgery be performed), mitral replacement rate was not worse than that of surgical series at least by the seventh year. Nevertheless, longer follow-up periods are needed for making such a conclusion.

Conclusions

PMV is a safe and effective procedure for patients with mitral stenosis. Patients with a good score obtain better initial and long-term results, but those with a less favorable profile may still have sustained hemodynamic and symptomatic relief. The decrease in MVA, although mild in most patients, was progressive over time, whereas regurgitation tend to be stable.

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