Mechanisms and predictors of transient left ventricular dysfunction early after successful percutaneous balloon mitral valvuloplasty

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Mitral stenosis; Percutaneous mitral valvuloplasty; Ventricular function.

Background. **The immediate effects of balloon mitral valvuloplasty (BMV) on left ventricular (LV) function in patients with mitral stenosis are still controversial. The aim of this study was to investigate the mechanisms and potential clinical, echocardiographic and hemodynamic predictors of transient LV dysfunction occurring in patients with mitral stenosis early after successful percutaneous BMV.**

Methods. **Sixty patients without residual mitral regurgitation were divided into two groups according to the changes in the left atrial (LA) pressure 15 min after successful BMV: 18 patients (group A) did not present with any reduction in LA pressure, and underwent nitroglycerin administration (0.4 mg, sublingually). The remaining 42 patients (group B) presented with a decrease in LA pressure.**

Results. **At baseline, both the mitral valve gradient and area assessed at echocardiography and during cardiac catheterization were similar in groups A and B. Group A patients presented with, however, higher LV early- and end-diastolic pressures and peak V waves during cardiac catheterization both prior to and 15 min after BMV than group B patients (all p values < 0.05). In group A, nitroglycerin administration was associated with a decrease in LV end-diastolic pressure (p = 0.049), LA pressure (p < 0.001), and peak V wave (p < 0.001) that was still persistent 30 min after its administration, reaching values similar to those observed in group B early after BMV. At multivariate analysis, the only independent predictors of LV dysfunction early after BMV were found to be LV early- (p = 0.015) and end-diastolic (p = 0.023) pressures at baseline and the Wilkins' score (p = 0.004).**

Conclusions. **After successful BMV a transient lack of LV adaptation to the increased LV preload resulting in a persistently elevated LA pressure is predicted by higher baseline LV diastolic filling pressures and higher Wilkins' scores. It is promptly and steadily reversed by nitroglycerin administration through a transient LV unloading, thus allowing a correct hemodynamic evaluation of the immediate results of the procedure.**

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Introduction

The immediate effects of percutaneous balloon mitral valvuloplasty (BMV) on left ventricular (LV) function in patients with mitral stenosis are still controversial. Some previous studies reported an early increase in the LV stroke volume after successful BMV thus suggesting a preserved LV diastolic function[1-4], but other studies did not[2,5-9]. An impaired LV diastolic function is an important feature of mitral valve stenosis. It has been related to: a) chamber atrophy due to unloading; b) myocardial fibrosis following rheumatic myocarditis; c) internal restrictions due to the tethering of the thickened mitral valve apparatus; d) abnormal right ventricular and LV interactions due to right ventricular pressure overload; or e) a combination of these mechanisms[4,10]. The lack of LV adaptation in response to the increase in the LV preload after BMV has been related to a larger end-diastolic volume and to a reduced ejection fraction at baseline[2]. In this setting, the abrupt increase in the LV preload after BMV translates in an increase in the LV end-diastolic pressure without an increase in the LV end-diastolic volume; this, in turn, causes an augmentation (or a lack of reduction) of the mean left atrial (LA) pressure and a peak V wave of the LA pressure.

Thus, the purpose of this study was to investigate the prevalence, mechanisms and potential clinical, echocardiographic and hemodynamic predictors of transient LV dysfunction early after successful BMV. The effects of vasodilation with nitroglycerin on the hemodynamics of LV dysfunction after BMV were also investigated.

Methods

Patients. From January 1998 to December 2002, 180 patients with mitral stenosis underwent BMV by the previously described[11] transseptal approach. Briefly, BMV was performed using an Inoue balloon 26-30 mm catheter. Sequential inflations were performed at increasing balloon diameters until the balloon waist disappeared. Seventy-two patients were excluded from this study because they were in atrial fibrillation. Forty-five patients were also excluded because they had more than trivial mitral regurgitation (30 patients) or owing to associated aortic valve or coronary artery diseases (15 patients). Finally, 3 other patients were excluded from the study after the procedure because they had residual mitral regurgitation ≥ grade 2 at left ventriculography. Thus, the study population consisted of 60 patients.

Study protocol. The study design is shown in figure 1. All patients underwent left ventriculography immediately after BMV, in order to exclude those with residual mitral regurgitation ≥ grade 2. Then, patients could be divided into two groups according to the changes in the LA pressure 15 min after BMV: 18 patients (group A, 14 males, 4 females, aged 28 to 79 years, mean 58 years) did not present with any reduction in the mean LA pressure, and underwent nitroglycerin administration (0.4 mg, sublingually). The remaining 42 patients (group B, 31 males, 11 females, aged 31 to 68 years, mean 53 years) presented with a decrease in the LA pressure and served as controls (Table I). Thus, the two groups were representative of patients with (group A) or without (group B) transient LV dysfunction early after successful BMV.

Hemodynamic measurements. Hemodynamic measurements were assessed through heparin-filled catheters connected directly to strain-gauge manometers (Baxter Healthcare Corporation, Uden, The Netherlands) before and 15 min after BMV. In group A, hemodynamic parameters were also assessed 5 and 30 min after nitroglycerin administration. Hemodynamic measurements included LV early- and end-diastolic pressures, LV end-systolic pressure, mean LA pressure, peak V wave of the LA pressure, mean transmitral pressure gradient, mitral valve area, pulmonary arterial systolic pressure, and heart rate.

Echocardiographic measurements. Echocardiography was performed 1 day before and 1 day after BMV, using a Hewlett-Packard series 5500 echocardiograph equipped with a 2.0-2.5 MHz transducer. The functional mitral valve area (with the pressure half-time method)[12,13], LA diameter, LV end-diastolic and end-systolic indexed volumes, LV ejection fraction, the ratio of the early and atrial filling velocities (E/A ratio), the Wilkins' score[14] and the mean transmitral pressure gradient were measured in each patient by averaging ≥ 10 cardiac cycles. All patients also underwent transesophageal echocardiography 1 day before BMV in order to rule out LA or appendage thrombosis.

Statistical analysis. Two-factor repeated measures analysis of variance with one-factor repeated measures

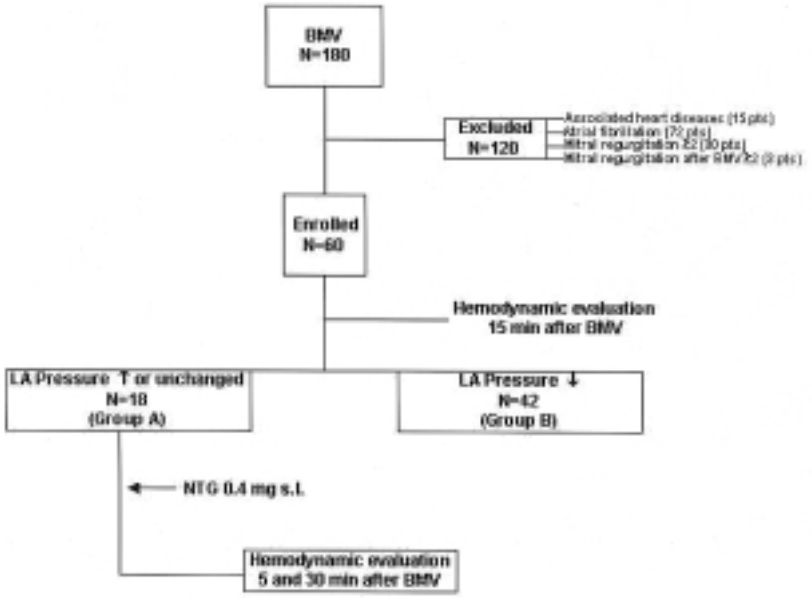

Figure 1. *Diagrammatic representation of the study protocol. Of 180 consecutive patients with mitral stenosis undergoing percutaneous balloon mitral valvuloplasty (BMV), 120 were excluded from this study because of atrial fibrillation (n = 72), mitral regurgitation ≥ grade 2 (n = 30), associated aortic valve or coronary artery diseases (n = 15) or residual mitral regurgitation ≥ grade 2 at left ventriculography performed after the procedure (n = 3). The remaining 60 patients were included in the study and could be divided into two groups according to the changes of the mean left atrial (LA) pressure 15 min after BMV: 18 patients (group A) did not exhibit any reduction in LA pressure, and underwent nitroglycerin (NTG) administration. The remaining 42 patients (group B) exhibited a decrease in LA pressure and served as controls.*

Table I. Hemodynamic parameters in the two groups.

Variables	Group A				Group B		p	
	Before BMV	15 min after BMV	5 min after NTG	30 min after NTG	Before BMV	After BMV	A vs B Before	A vs B After
LV early-diastolic pressure (mmHg)	1.9 ± 2.8	1.3 ± 3.7	0.7 ± 4.3	0.8 ± 2.2	0.05 ± 0.8	0.05 ± 0.9	0.001	0.07
LV end-diastolic pressure (mmHg)	14.8 ± 3.4	19.6 ± 7.2*	9.9 ± 6.1**	9.2 ± 3.5**	10.5 ± 3.1	10.1 ± 3.3	0.001	0.38
LV end-systolic pressure (mmHg)	139 ± 18	138 ± 16	130 ± 16§	136 ± 19	141 ± 18	140 ± 17	0.66	0.41
Mean left atrial pressure (mmHg)	27.8 ± 5.5	30.6 ± 8.4*	15.0 ± 6.2**	13.0 ± 1.0**	23.2 ± 8.0	13.1 ± 6.2*	0.03	0.95
Peak V wave pressure (mmHg)	41.4 ± 7.6	50.8 ± 13.1*	19.9 ± 6.4**	19.1 ± 6.1**	32.8 ± 9.9	20.0 ± 8.3*	0.002	0.66
Heart rate (b/min)	79 ± 12	81 ± 20	79 ± 15	79 ± 8	79 ± 9	81 ± 9	0.91	0.30
Mean transmitral pressure gradient (mmHg)	17.7 ± 5.6	17.6 ± 7.4	8.4 ± 3.3**	5.5 ± 1.6**	17.1 ± 4.4	6.2 ± 3.3*	0.66	0.38
Mitral valve area (cm^2)	1.0 ± 0.2	–	–	1.8 ± 0.4*	1.0 ± 0.2	2.0 ± 0.3*	0.44	0.17
Pulmonary arterial systolic pressure (mmHg)	55 ± 18	–	–	38 ± 12*	53 ± 15	35 ± 11*	0.57	0.31

BMV = balloon mitral valvuloplasty; LV = left ventricular; NTG = nitroglycerin. For the comparisons between groups after BMV, the hemodynamic parameters in group A refer to those obtained 30 min after NTG administration. * $p < 0.05$ vs values before BMV; ** $p < 0.05$ vs values before BMV and 15 min after BMV; § $p < 0.05$ vs values before BMV, 15 and 30 min after BMV.

was used to compare the hemodynamic and echocardiographic changes following BMV in the two groups of patients. When significant differences were detected, pairwise comparisons were made using Scheffè F test.

Comparisons of the remaining continuous or discrete variables between the two groups were performed using an unpaired Student's t-test or a χ^2 test, respectively. Multivariate independent predictors of LV dysfunction early after successful BMV were evaluated by stepwise logistic regression. All clinical, hemodynamic and echocardiographic variables were tested, and a final multivariate model was constructed by backward deletion of the least significant variables. Data are expressed as mean ± SD. A p value of < 0.05 was considered statistically significant.

Results

There were no significant differences between the two groups with regard to age (58 ± 14 vs 53 ± 12 years, p = 0.12), body mass index (23.1 ± 3.8 vs 24.4 ± 3.9 kg/m^2, p = 0.23), NYHA functional class III or IV (72 vs 60%, p = 0.35), and prevalence of hypertension (17 vs 19%, p = 0.82).

Hemodynamic findings. *Hemodynamic changes after balloon mitral valvuloplasty in group A.* The mean LA pressure did not decrease after BMV in any of the patients; indeed, it increased from 27.8 ± 5.5 to 30.6 ± 8.4 mmHg (p = 0.03). The increase ranged from 0 to 22 mmHg. Yet, it decreased 5 and 30 min after nitroglycerin (15.0 ± 6.2 mmHg and, respectively, 13.0 ± 1.0 mmHg, $p < 0.001$ vs baseline and 15 min after BMV). The LV end-diastolic pressure also increased 15 min after BMV from 14.8 ± 3.4 to 19.6 ± 7.2 mmHg (p = 0.05), but it decreased 5 and 30 min after nitroglycerin (9.9 ± 6.1 mmHg and, respectively, 9.2 ± 3.5 mmHg, $p < 0.05$ vs baseline and 15 min after BMV). Similarly, the peak V wave increased 15 min after BMV from 41.4 ± 7.6 to 50.8 ± 13.1 mmHg (p = 0.002), but it significantly decreased 5 and 30 min after nitroglycerin (19.9 ± 6.4 mmHg and, respectively, 19.1 ± 6.1 mmHg, $p < 0.001$ vs baseline and 15 min after BMV). As a consequence of the LA and LV end-diastolic pressure changes, the transmitral pressure gradient did not decrease 15 min after BMV (17.6 ± 7.4 mmHg, p = 0.9 vs baseline), but it significantly decreased 5 and 30 min after nitroglycerin (8.4 ± 3.3 mmHg and, respectively, 5.5 ± 1.6 mmHg, $p < 0.001$ vs baseline and 15 min after BMV). The LV end-systolic pressure did not change 15 min after BMV (139 ± 18 vs 138 ± 16 mmHg, p = 0.11); it significantly decreased 5 min after nitroglycerin (130 ± 16 mmHg, $p < 0.001$), but it returned to a near-baseline level 30 min after nitroglycerin (136 ± 19 mmHg, p = 0.36). The LV early-diastolic pressure

and heart rate did not significantly change throughout the study (Table I).

The mitral valve area increased from 1.0 ± 0.2 to 1.8 ± 0.4 cm^2 ($p < 0.001$) at the end of BMV (30 min after nitroglycerin), with a concomitant decrease in the pulmonary arterial systolic pressure ($p < 0.001$) (Table I).

Hemodynamic changes after balloon mitral valvuloplasty in group B. The mean LA pressure decreased after BMV in all patients (from 23.2 ± 8.0 to 13.1 ± 6.2 mmHg, $p < 0.001$, range -2 to -17 mmHg). The LV early- and end-diastolic pressures, LV end-systolic pressure and heart rate did not significantly change after BMV (Table I).

The mitral valve area increased from 1.0 ± 0.2 to 2.0 ± 0.3 cm^2 ($p < 0.001$) at the end of the procedure, with a concomitant decrease in the peak V wave ($p < 0.001$), transmitral pressure gradient ($p < 0.001$), and pulmonary arterial systolic pressure ($p < 0.001$) (Table I).

Comparisons between groups. Despite a similar transmitral pressure gradient and mitral valve area before BMV, the LV early- and end-diastolic pressures, LA pressure and peak V wave were significantly higher in group A than in group B (Table I). The hemodynamic parameters after BMV (in group A they refer to those obtained 30 min after nitroglycerin administration) were similar in the two groups (Table I; Fig. 2).

Echocardiographic findings. Before BMV the LA diameter, Wilkins' score, E/A ratio and LV indexed volumes were significantly greater in group A than in group B. The remaining parameters were similar in the two groups before BMV (Table II).

The mitral valve area significantly increased in both groups after BMV, with a concomitant decrease in the transmitral pressure gradient and LA diameter (all p values < 0.05), without any differences between groups (Table II). The LV ejection fraction significantly increased in both groups after BMV ($p < 0.05$), due to an increase (although not statistically significant) in the LV end-diastolic volume index, whereas the E/A ratio significantly decreased in group A only ($p < 0.05$) (Table II). Of note, the LV indexed volumes and E/A ratio were still greater in group A ($p < 0.05$) even after BMV.

Independent predictors of left ventricular dysfunction. The LV early- ($p = 0.015$) and end-diastolic ($p = 0.023$) pressures at baseline and the Wilkins' score ($p = 0.004$) were the only statistically significant independent predictors of LV dysfunction after successful BMV.

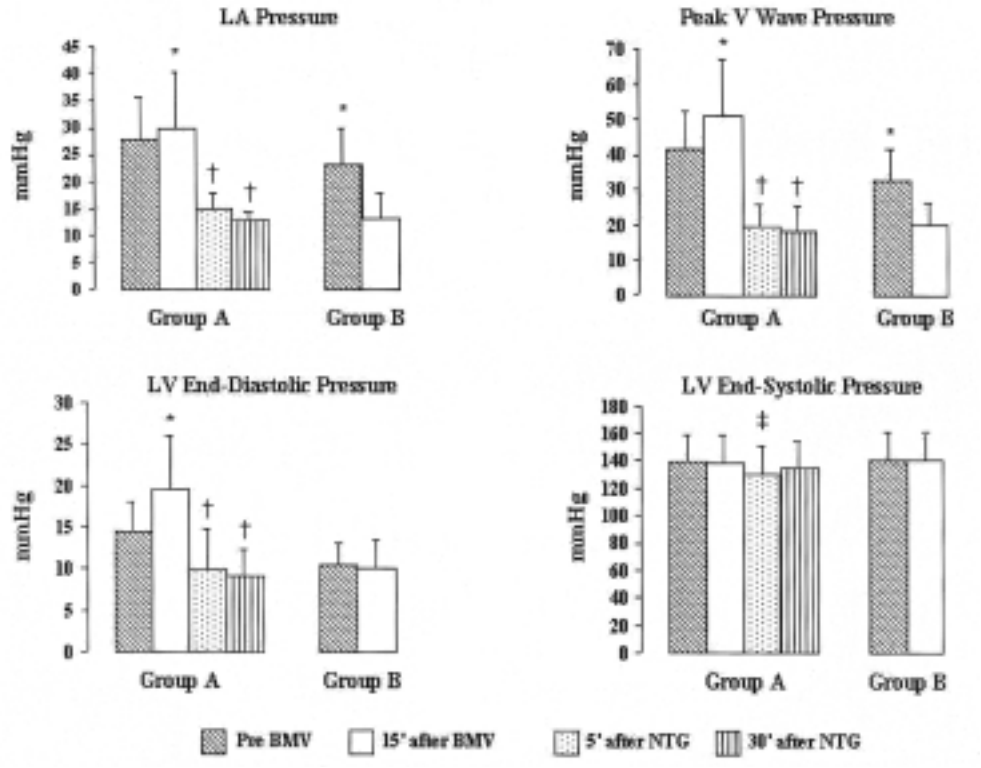

Figure 2. *Changes in the mean left atrial (LA) pressure, peak V wave pressure and left ventricular (LV) end-diastolic and end-systolic pressures following percutaneous balloon mitral valvuloplasty (BMV) in the two groups of patients. Data are presented as mean ± SD. * $p < 0.05$ vs values before BMV; † $p < 0.05$ vs values before BMV and 15 min after BMV; ‡ $p < 0.05$ vs values before BMV, 15 and 30 min after BMV. Of note, despite a similar transmitral pressure gradient and mitral valve area before BMV, the LV end-diastolic pressure, LA pressure and peak V wave were significantly higher in group A than in group B (all p values < 0.05). After BMV, the hemodynamic parameters (in group A they refer to those obtained 30 min after nitroglycerin [NTG] administration) were similar in the two groups.*

Table II. Echocardiographic parameters in the two groups.

Variables	Before BMV		After BMV	
	Group A	Group B	Group A	Group B
Mean transmitral pressure gradient (mmHg)	10.2 ± 3.7	9.0 ± 2.8	4.5 ± 2.3§	3.8 ± 2.1§
Functional mitral valve area (cm^2)	1.0 ± 0.2	1.1 ± 0.3	1.9 ± 0.3§	2.0 ± 0.2§
E/A ratio	1.8 ± 0.7*	1.0 ± 0.2	1.4 ± 0.4*§	0.9 ± 0.2
LV end-diastolic volume index (ml/m^2)	75 ± 12**	62 ± 20	81 ± 20**	66 ± 23
LV end-systolic volume index (ml/m^2)	31 ± 7**	25 ± 11	30 ± 7**	24 ± 10
Ejection fraction (%)	58 ± 6	60 ± 8	61 ± 6§	63 ± 7§
Septal wall thickness (mm)	9.6 ± 1.2	9.8 ± 1.6	9.5 ± 1.5	9.7 ± 1.6
Posterior wall thickness (mm)	9.3 ± 1.5	9.7 ± 1.4	9.2 ± 1.4	9.5 ± 1.5
Left atrial diameter (mm)	51 ± 10**	47 ± 3	47 ± 7§	45 ± 4§
Wilkins' score	10 ± 2**	8 ± 3	–	–
Subcomponents				
Thickness	2.7 ± 0.6	2.2 ± 0.7	–	–
Calcium	1.7 ± 0.8	1.2 ± 1.0	–	–
Mobility	2.9 ± 0.8*	1.9 ± 0.6	–	–
Subvalvular	2.7 ± 0.7	2.3 ± 0.8	–	–

BMV = balloon mitral valvuloplasty; LV = left ventricular. * $p < 0.001$ vs group B; ** $p < 0.05$ vs group B; § $p < 0.05$ vs values before BMV.

Discussion

This study demonstrates that early after successful BMV a transient lack of LV adaptation to the increased LV preload occurs in about one third of patients with mitral stenosis. It is predicted by higher baseline LV diastolic filling pressures and higher Wilkins' scores and is likely to be due to a less effective exploitation of the Frank-Starling mechanism. Notably, it may be promptly reversed by nitroglycerin administration through a transient unloading of the left ventricle, thus allowing a correct hemodynamic evaluation of the immediate results of the procedure.

Effect of balloon mitral valvuloplasty on left ventricular function. Previous studies indicated that, after BMV, most patients with mitral stenosis exhibit an increase in the LV stroke volume thus suggesting a preserved diastolic function[1-4]. It has been suggested that the mobilization of the mitral valve apparatus may have a major role in the immediate improvement in LV diastolic stiffness[4]. Thus, the increase in mitral valve area together with an immediate adaptation of the left ventricle to the different loading conditions cause a fall in the LA pressure and in the peak V wave with a consequent reduction or disappearance of the transmitral pressure gradient, as was the case in our control group. This implies that, in these patients, LV filling shifts from suction to the Frank-Starling mechanism immediately after successful BMV. Yet, in agreement with previous observations[2,5-9], we found that in a sizeable proportion of patients the abrupt increase in LV preload translates in an increase in LV filling and LA pressures still persistent 15 min after BMV. It is likely that in these patients, LV diastolic filling after BMV does not immediately shift from suction to the Frank-Starling mechanism because the LV end-diastolic volume fails to increase. In this setting, a transient nitroglycerin-induced LV unloading could have allowed the left ventricle to quickly adapt to the increased preload. It may also be speculated that the transient persistence of LV suction is responsible for the high initial deceleration velocity of the E wave commonly observed at Doppler echocardiography performed immediately after successful BMV[15].

It is worth noting that the hemodynamic changes observed early after BMV in group A did not allow a correct evaluation of the immediate results of the procedure because of a paradoxically persistent transmitral pressure gradient and/or an even higher than baseline peak V wave mimicking acute mitral valve insufficiency. Low-dose nitroglycerin, which is known to improve LV compliance mainly through a reduction of LV preload[16], was able to reverse such an acute LV dysfunction, thus allowing a correct hemodynamic evaluation of the immediate results of the procedure, which were confirmed at echocardiography performed 24 hours after BMV. Of note, the nitroglycerin-induced reductions in the LV end-diastolic pressure, LA pressure and peak V wave were still persistent 30 min after its administration, when the LV end-systolic pressure had already returned to the baseline level. Thus, the beneficial effect of nitroglycerin was not due to a reduction in the systemic arterial pressure (i.e., LV afterload), but rather to an improvement in the LV compliance through a transient unloading of the left ventricle and, perhaps, to direct enhancement of myocardial relaxation[17]. Of note, the favorable response to nitroglycerin was detected in all patients of group A.

Predictors of left ventricular dysfunction after balloon mitral valvuloplasty. Despite the similar clinical characteristics, transmitral pressure gradient and mitral

valve area before BMV, patients in group A presented with higher LV early- and end-diastolic pressures, LA pressures and peak V waves. Furthermore, they also presented with a greater baseline Wilkins' score, E/A ratio, LA diameter and LV end-diastolic and end-systolic volumes at echocardiography. These findings confirm and expand those obtained in a smaller patient population by Yasuda et al.[2], who found a persistent increase of the LV end-diastolic pressure after successful BMV in those patients with larger LV end-diastolic volumes and a reduced LV ejection fraction at baseline. However, at multivariate analysis we found that the only independent predictors of LV dysfunction after successful BMV are the baseline LV diastolic filling pressures and the Wilkins' score. In these patients, higher LV diastolic filling pressures probably reflect a greater reduction in LV compliance; the latter, indeed, is also dependent on a more severe alteration of the mitral valve apparatus, as suggested by the higher Wilkins' score.

Study limitations. There are certain potential limitations of this study. First, we used fluid-filled catheters instead of a catheter-tip micromanometer to measure the intracardiac pressures. However, this problem is minimized when the primary endpoint is the evaluation of intra-patient pressure changes, as was the case in our study. Second, a pressure-volume loop analysis would have provided a more accurate evaluation of LV function. Nonetheless, we assessed several hemodynamic and echocardiographic parameters that consistently showed a lack of LV adaptation to the increased preload after BMV in patients with a more compromised baseline LV diastolic function. Finally, the exclusion of patients with atrial fibrillation and mitral regurgitation ≥ grade 2, which was mandatory for the most accurate evaluation of hemodynamic tracings and pathophysiological interpretation of the obtained results, resulted in the selection of a population with a high prevalence of male patients, in whom mitral stenosis and LV diastolic dysfunction are less frequent. Thus, our findings should be referred to patients with pure mitral stenosis with sinus rhythm but not to all patients with mitral valve disease undergoing BMV.

In conclusion, this study demonstrates that about one third of patients with mitral stenosis undergoing successful BMV exhibit a transient lack of LV adaptation to the increased LV preload. This is predicted by the baseline LV diastolic function and is likely to be due to a less effective exploitation of the Frank-Starling mechanism. Finally, it is promptly reversed by nitroglycerin administration through a transient unloading of the left ventricle, thus allowing a correct hemodynamic evaluation of the immediate results of the procedure.

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