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Effect of Abrupt Mitral Regurgitation After Balloon Valvuloplasty on Myocardial Load and Performance

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The concept that mitral regurgitation masks myocardial dysfunction by reducing afterload and augmenting ejection performance has not been well established in humans. The effect of abruptly produced mitral regurgitation on left ventricular loading and performance was therefore evaluated in five patients who developed this complication after an otherwise successful percutaneous balloon mitral valvuloplasty. Mitral valve area by Gorlin formula calculated with forward flow increased from 0.92 ± 0.14 to 2.75 ± 0.82 cm². Mean left atrial pressure did not decrease (19 ± 4 to 19 ± 6 mm Hg). The size of the left atrial *V* wave relative to mean left atrial pressure (peak *V* − mean left atrial pressure) increased from 7 ± 4 to 19 ± 6 mm Hg. Angiographic mitral regurgitation increased from 0+ or 1+ to >3+ in each patient and regurgitant fraction increased from 0.23 ± 0.11 to 0.55 ± 0.09 ($p < 0.01$).

End-diastolic volume increased modestly from 148 ± 15 to 159 ± 15 ml (p = NS). Heart rate increased from 54 ± 5 to 71 ± 8 beats/min ($p < 0.05$), which may have prevented further increases in preload by shortening the filling period. End-systolic stress decreased by 32% from 277 ± 34 to 188 ± 52 kdyn/cm² ($p < 0.01$) as a result of a 25% decrease in end-systolic pressure from 121 ± 8 to 91 ± 7 mm Hg and a 16% decrease in end-systolic volume from 67 ± 13 to 56 ± 8 ml (p = NS). Contractility estimated from the preload-corrected ejection fraction-afterload relation decreased in one of the five patients and did not increase in the others despite an increase in heart rate, possibly as a result of myocardial depression from the balloon procedure itself. Nevertheless, the decrease in end-systolic volume could not be attributed to a net increase in contractility. The result of the changes in loading was an increase in ejection fraction from 0.55 ± 0.05 to 0.65 ± 0.04 ($p < 0.05$).

Thus, abruptly produced mitral regurgitation increases ejection performance by reducing afterload without increasing contractility. This should be taken into consideration when anticipating the results of valve replacement for acute or subacute mitral regurgitation.

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The concept that myocardial dysfunction can be masked by mitral regurgitation has become widely accepted (1) since the demonstration by Urschel et al. (2) that acute mitral regurgitation increased preload, decreased afterload and augmented ejection performance in open chest, anesthetized dogs. However, the extent to which this concept applies to patients with chronic mitral regurgitation has recently been questioned (3–5) and alternative explanations have been offered (6) for the decrease in ejection fraction often observed after mitral valve replacement. The influence of cardioplegia and open heart surgery on left ventricular function further complicates the assessment of loading and performance changes after surgical correction of mitral regurgitation.

Severe mitral regurgitation due to acute rheumatic fever is a major health problem in developing countries. Valve replacement is frequently necessary to treat congestive heart failure when medical therapy fails, not uncommonly when rheumatic fever is still active (7). A recent study (8) showed very little decrease in ejection performance after surgical correction of mitral incompetence in young patients with acute rheumatic valvulitis, casting further doubt on the role of altered loading conditions on ejection performance in mitral regurgitation, even when it is relatively acute.

Because there are few clinical data (9) available to corroborate the experimental data obtained in open chest dogs and there is some doubt about the role of altered loading conditions on left ventricular performance in mitral regurgitation, we examined the effects of acute severe mitral regurgitation on myocardial performance and loading in five patients after balloon mitral valvotomy. Although these data do not necessarily apply to patients with long-standing mitral regurgitation, they may give some indication of the extent to which altered loading conditions affect left ventricular performance in patients with acute or subacute mitral regurgitation who require valve surgery. This clinical model, which allows immediate analysis of acute mitral regurgitation mechanics, has the advantage over previous studies in that the myocardial effects of cardioplegia, anesthesia and interruption of valve-chordal continuity are not complicating factors.

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Methods

Study subjects. Among 60 patients who underwent percutaneous balloon valvuloplasty at Baragwanath Hospital and the University of Kentucky Medical Center between October 1988 and February 1990, severe (>3+) mitral regurgitation was a complication in 8. In five of these patients, we obtained good quality left ventricular cineangiograms simultaneously with left ventricular micromanometer pressure recordings during normal sinus rhythm, and these five patients form our study group. None of these five patients required urgent surgery, but two required mitral valve replacement between 1 and 2 months after valvotomy because of New York Heart Association class III or IV heart failure. One had an anterior mitral leaflet tear and the other a posterior leaflet tear. The other three patients were managed medically. Ages ranged from 25 to 38 years. All five patients were being treated with furosemide and atenolol at the time of balloon valvuloplasty. Two-dimensional echocardiograms were performed before valvotomy on Hewlett-Packard Sonos 500 or Sonos 1000 equipment. In all five patients, valve pliability was considered to be excellent with regard to anterior leaflet mobility and leaflet thickness, and none had more than a small amount of leaflet calcification or evidence of severe subvalvular disease. All patients gave informed consent to the protocol approved by the respective institutions.

Catheterization procedure. Patients were premedicated with phenergan (25 mg intramuscularly) and meperidine (50 mg intramuscularly). Right heart catheterization was performed through a femoral vein with a thermodilution flotation catheter. Left heart catheterization was performed retrograde through a femoral artery using an 8F micromanometer catheter with a pigtail configuration. A transseptal approach to the mitral valve was used with two balloons inserted over two guide wires whose tips were positioned either in the apex of the left ventricle or in the descending aorta. In Patients 1 and 2, 20 and 25 mm balloons were used in tandem, and in Patients 3 to 5, two 20 mm Mansfield balloons were used.

After right and left heart hemodynamics were measured, sodium meglumine ioxaglate (Hexabrix), 40 to 50 ml was injected into the left ventricle during biplane cineangiography (30° right anterior oblique and 60° left anterior oblique views); left ventricular pressure was recorded from the micromanometer catheter at 100 mm/s paper speed during left ventricular cineangiography. Precise synchronization between pressure and cine frame was achievable with a cine frame marker, which records a mark for each film exposure (50 or 60/s) simultaneously with the pressure recording and exposes every 100th frame with a diode simultaneously with an accentuated mark on the pressure recording. After balloon valvuloplasty, hemodynamic variables were again recorded and left ventricular cineangiography repeated using the same injection protocol, image intensifier and table positions as for the baseline study with simultaneous left ventricular micromanometer recordings.

Analysis of catheterization data. Left ventricular silhouettes for each frame of the first well opacified beat of each left ventricular cineangiogram not preceded by an ectopic beat were digitized using a hand-held cursor. Left ventricular wall thickness was measured at the mid-third of the anterior wall in the right anterior oblique view for the end-diastolic frame. Correction factors for ventricular measurements were derived from the grids positioned at the center of the ventricle. Left ventricular volume was computed with use of the area-length method and a regression equation (10). Because the silhouette borders in the left anterior oblique view were sometimes unclear over the spine and diaphragm and segmental dyssynergy was absent, volumes were computed from the single plane right oblique view. Left ventricular mass was computed by using wall thickness measured at end-diastole (11). Left ventricular pressure for the corresponding cardiac cycle was digitized with use of the mid-portion of the QRS complex as a reference point for end-diastole.

Mitral regurgitation was defined on angiography as 1+ if regurgitation of contrast medium was only faint and insufficient to define the atrial silhouette, 2+ if there was just enough contrast to define the atrial silhouette but less contrast than in the ventricle, 3+ if there was dense opacification of the atrium nearly equal to that of the ventricle after the third beat of the cine frame and 4+ if there was complete opacification of the atrium equal to that of the ventricle within the first three beats. In three patients, regurgitation was graded as 3 to 4+ (equal systolic opacification of the atrium and ventricle within the first three beats, but some clearing in diastole).

Left ventricular volume and pressure were plotted by computer frame by frame. We (12) have previously shown the intraobserver and interobserver variability of our volume measurements to be approximately 5%. To make the volume-time (V/T) waveform analysis as objective as possible, the data were fit to a 9th or 10th order polynomial equation with use of a Marquardt curve-fitting program. Differentiation of this equation at each frame with respect to time (dV/dT) yields instantaneous flow rate. End-diastolic volume was located as the maximal volume and end-systolic volume as the minimal volume where the equation dV/dT = 0. The angiographic (total) stroke volume was computed as the difference between end-diastolic volume and end-systolic volume. Forward stroke volume was computed from thermodilution cardiac output and heart rate. Although mixed venous and arterial oxygen saturations were also obtained to enable calculation of Fick cardiac output (using an assumed oxygen consumption), thermodilution output was used because no patient had evidence on two-dimensional echocardiography or physical examination of more than mild tricuspid regurgitation. None of the patients had a >1.4:1 atrial shunt as assessed by the vena caval and pulmonary artery saturations.

Regurgitant fraction was computed from the equation (total stroke volume − forward stroke volume)/(total stroke volume). Hemodynamic variables were measured as the average of three beats.

Ventricular wall stress was computed with the equation of Mirsky, and end-systolic stress was determined by defining the upper left corner of the stress/volume loop where the stress/volume ratio reaches its maximal value, as previously described (5).

As an estimate of ventricular contractility, we determined the ejection fraction calculated from a common preload of 50 kdyn/cm^2 of diastolic wall stress and compared the relation between preload-corrected ejection fraction and end-systolic stress with that of previously studied normal control subjects (5).

Statistics. Data are reported as mean values ± SD. A commercially available statistics program was used to perform paired *t* tests to detect differences between baseline and postvalvotomy variables. A p value <0.05 was considered significant.

Results

Hemodynamics. Micromanometer recordings of left atrial and left ventricular pressure before and immediately after mitral regurgitation abruptly developed after balloon valvotomy are shown in Figure 1. A large *V* wave was observed in each patient (range 30 to 56 mm Hg), indicating the hemodynamic severity of the regurgitation. The size of the *V* wave relative to the mean left atrial pressure for each patient is shown in Table 1. Despite the severity of mitral regurgitation, cardiac output did not decrease because of relief of severe mitral stenosis and a threefold increase in valve area. This increase in valve area prevented an increase in mean left atrial pressure despite an increase in the *V* wave.

Angiographic data. Mitral valve area calculated from mitral (angiographic) flow was increased substantially after balloon valvuloplasty (Table 1). Before valvuloplasty, none of the patients had >1+ angiographic mitral regurgitation; after valvuloplasty, angiographic mitral regurgitation was graded as 4+ in two patients and 3 to 4+ in the other three. Regurgitant fraction increased to 0.55 after valvuloplasty.

Wall stress data. The angiographic and wall stress data are shown in Table 2. Left ventricular ejection fraction increased by 18% with the development of mitral regurgitation. This was associated with a 7% increase in end-diastolic volume (p = NS). End-diastolic volume failed to increase by >5 ml or actually decreased in three of the five patients, and in two of these there was a marked increase in heart rate (24 and 33 beats/min, respectively), suggesting that a decrease in filling time as a result of tachycardia might have prevented the expected increase in preload with the abrupt development of mitral regurgitation. Reduced afterload was a more significant factor in augmenting ejection performance because of a 32% decrease in end-systolic stress and a 16% decrease in end-systolic volume. This was associated with a

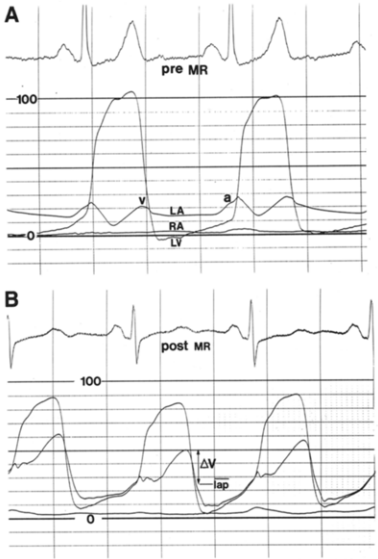


Figure 1. Patient 4. Left ventricular (LV), left atrial (LA) and right atrial (RA) pressure all on a scale of 0 to 100 mm Hg before (pre) (**panel A**) and after (post) (**panel B**) the development of mitral regurgitation (MR) produced by balloon valvuloplasty. The mean left atrial pressure (lap) is indicated by the **horizontal line in panel B**. The size of the left atrial *V* wave (ΔV) is indicated by the **arrow**. a = *A* wave.

25% decrease in end-systolic pressure and a 25% decrease in forward stroke volume.

The stress-volume loop in Figure 2, (derived from Patient 2) is representative of the average change in afterload exhibited by the five patients with acute mitral regurgitation. Note that although peak systolic stress is similar both before and after mitral regurgitation, stress decreases rapidly throughout systole to a much lower end-systolic stress and a correspondingly smaller end-systolic volume after mitral regurgitation compared with the control state. The time course of wall stress during the cardiac cycle before and after abrupt mitral regurgitation is shown in Figure 3.

Contractile performance. Contractile performance was evaluated by examining the relation between preload-corrected ejection fraction and end-systolic stress. In four of the five patients, mitral regurgitation was associated with a leftward and upward shift to a lower end-systolic stress and

Table 1. Hemodynamic Data Before (Pre) and After (Post) Mitral Regurgitation in Five Patients

Patient No.	EDP (mm Hg)		LAP (mm Hg)		ΔV Wave (mm Hg)		MVA (cm²)		HR (beats/min)		CO (liters/min)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	9	20	20	28	5	28	0.88	1.91	61	67	3.00	3.21
2	19	23	21	18	14	18	0.79	2.18	47	71	3.26	3.52
3	9	2	22	11	3	15	1.08	2.59	52	85	3.96	3.54
4	8	25	11	18	4	12	1.04	3.42	52	67	3.23	3.71
5	24	32	20	18	8	20	0.80	3.97	56	67	3.13	3.52
Mean	14	20	19	19	7	19	0.92	2.75	54	71	3.32	3.28
±SD	7	11	4	6	4	6*	0..	0.82*	5	8*	0.37	0.34

*$p < 0.05$ compared with values before mitral regurgitation. CO = thermodilution cardiac output; ΔV Wave = magnitude of the *V* wave above mean left atrial pressure; EDP = end-diastolic pressure; HR = heart rate; LAP = left atrial pressure; MR = mitral regurgitation; MVA = mitral valve area.

higher preload-corrected ejection fraction in keeping with the normal relation between afterload and performance (Fig. 4). In one patient, preload-corrected ejection fraction decreased despite a decrease in end-systolic stress with mitral regurgitation, indicating a decrease in contractility. These data indicate that an increase in contractility immediately after balloon valvuloplasty could not account for the lower end-systolic volume.

Discussion

To our knowledge, this is the first confirmation in humans that acute mitral regurgitation "unloads" the left ventricle and augments ejection performance without increasing contractility. The mechanism for this augmented ejection performance, which was observed in each of our five patients, can be explained by an analysis of ejection mechanics.

Preload. Preload was not a major determinant of augmented ejection performance. A substantial increase in end-diastolic volume occurred with abrupt mitral regurgitation only in Patients 1 and 4, whereas end-diastolic volume increased very little or actually decreased in the other three. In Patients 2 and 3, an increase in heart rate by 24 and 33 beats/min, respectively, may have limited diastolic filling and an increase in preload that would have occurred in the absence of tachycardia. Thus, augmented preload may not always contribute to augmented ejection performance in acute mitral regurgitation, particularly when increases in heart rate limit diastolic filling or, perhaps, when preload reserve is already limited by increased intravascular volume or reduced left ventricular compliance.

Afterload. In contrast, afterload was affected in a more consistent fashion with the development of acute mitral regurgitation. In all patients except Patient 4, the increase in ejection fraction with mitral regurgitation was associated with "unloading" during systole to a smaller end-systolic volume. The primary mechanism for reduced afterload with acute mitral regurgitation appears to be a decrease in forward stroke volume and a parallel decrease in left ventricular end-systolic pressure.

Contractile function. Although difficult to assess, it seems that ejection performance-afterload relations are currently the most meaningful way to assess contractile function (5,13). These relations are based on the observation that ejection performance is inversely related to afterload. If contractility increases simultaneously with a decrease in afterload, this should be reflected as an upward shift away from the normal ejection fraction-afterload relation. In none of our patients was an increase in contractility manifested as an upward shift of the normal relation between preload-corrected ejection fraction and end-systolic stress, and in one patient (Patient 4), there was movement to a lower

Table 2. Angiographic and Wall Stress Data Before (Pre) and After (Post) Mitral Regurgitation in Five Patients

Patient No.	EDV (ml)		ESV (ml)		EF		RF		ESS (kdyn/cm²)		ESP (mm Hg)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	142	172	66	59	0.54	0.66	0.35	0.58	330	262	126	100
2	138	143	57	42	0.59	0.71	0.14	0.51	247	154	114	88
3	174	164	89	61	0.49	0.63	0.10	0.60	287	126	130	80
4	147	172	57	63	0.61	0.63	0.31	0.63	266	200	110	89
5	141	142	66	56	0.53	0.61	0.25	0.41	273	196	126	96
Mean	148	159	67	56	0.55	0.65	0.23	0.55	277	188	121	91
±SD	15	15	13	8	0.05	0.04*	0.11	0.09*	34	52*	8	7*

*$p < 0.05$ compared with before mitral regurgitation. EDV = left ventricular end-diastolic volume; EF = ejection fraction; ESP = left ventricular end-systolic pressure; ESS = left ventricular end-systolic wall stress; ESV = left ventricular end-systolic volume; RF = regurgitant fraction.

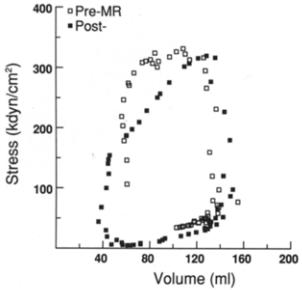


Figure 2. Patient 2. Stress-volume loops plotted frame by frame before (Pre) (**open squares**) and after (Post-) (**closed squares**) the development of mitral regurgitation (MR) produced by balloon valvuloplasty.

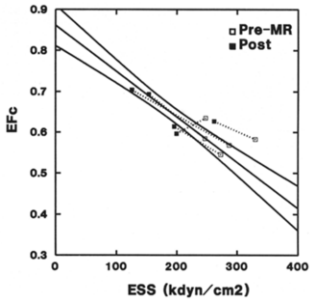
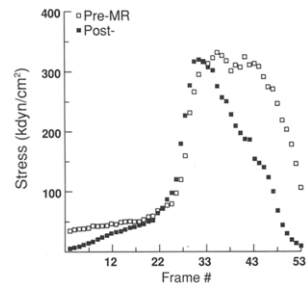


Figure 4. Ejection fraction corrected for preload (EFc) plotted against end-systolic stress (ESS) before (Pre-) (**open squares**) and after (Post) (**solid squares**) mitral regurgitation (MR) produced by balloon valvuloplasty in the five patients. The regression line and 95% confidence intervals of the regression line are for a control group (see Methods). Values before and after the development of mitral regurgitation are connected by **dotted lines**.

position away from the normal curve (Fig. 4). As a result of the mild decrease in contractility, this patient demonstrated no decrease in end-systolic volume with abrupt mitral regurgitation. Thus, a decrease in contractile function in one patient and the lack of any observable change in contractile function in the others make it unlikely that decreases in end-systolic volume and increases in ejection fraction were attributable to augmented contractility.

The failure to detect an increase in contractile function despite an increase in heart rate suggestive of increased sympathetic tone is not easy to explain. One possible explanation is that there was a simultaneous depression of the myocardium due to the hemodynamic effects and possibly the mechanical effects of balloon inflation during valvotomy.

Figure 3. Patient 2. Ventricular wall stress plotted at 17 ms frame intervals before (Pre) (**open squares**) and after (Post) (**closed squares**) mitral regurgitation (MR) produced by balloon valvuloplasty.



This possibility has been proposed (14) as an explanation for more severe mitral regurgitation immediately after balloon valvuloplasty than is observed at later follow-up study. Thus, the tendency of sympathetic tone to increase contractility may have been offset by the opposite influence of balloon inflation on contractility, with a net result of no or little change in relation between preload-corrected ejection fraction and end-systolic stress. Another contributing factor may have been a withdrawal in vagal tone at rest.

Comparison with chronic mitral regurgitation. The alterations in loading and performance observed in our patients with acute mitral regurgitation and in short-term canine studies (2,15) of acute mitral regurgitation may not persist as mitral regurgitation becomes chronic. In fact, a comparison of characteristics between our patients and those who have chronic mitral regurgitation reveals some notable differences. Preload is more consistently augmented in patients (5,16,17) and animals (18) with chronic mitral regurgitation than in our study of acute mitral regurgitation. Afterload is not reduced and may be increased compared with that in normal subjects as mitral regurgitation becomes chronic (4,5,16,17) possibly because of long-term adaptive changes in the myocardium or peripheral circulation. However, the exact mechanism by which afterload increases during the transition from acute to chronic mitral regurgitation has not been fully explained. The results of Carabello et al. (18) in dogs studied during this transition from acute to chronic mitral regurgitation suggest that a decrease in the thickness/radius ratio may cause wall stress to increase. Conversely, a comparison of subacute and chronic mitral regurgitation in humans by Kontos et al. (19) suggests that systolic pressure

Table 3. Comparison of Preload, Afterload, Contractility and Ejection Fraction in Acute Versus Chronic Mitral Regurgitation

MR	Preload	Afterload	Contractility	EF
Acute	+ or nc	– –	nc	+ +
Chronic	+	+ or nc	nc or –	nc or –

Preload is estimated as end-diastolic volume; afterload is estimated as end-systolic stress. EF = ejection fraction; MR = mitral regurgitation; nc = no change; + = mild increase; + + = marked increase; – = mild decrease; – – = marked decrease.

may gradually increase with chronic mitral regurgitation, a factor that might account for an increase in wall stress. Contractility is commonly (17,18) though not always (5) depressed in chronic severe mitral regurgitation, but it was generally preserved in our patients and in dogs with acute mitral regurgitation as reported by Carabello et al. (18). These differences in loading and contractility between acute and chronic mitral regurgitation may explain why ejection performance is augmented in acute but not chronic mitral regurgitation. A summary of the characteristics in chronic versus acute mitral regurgitation is shown in Table 3.

Clinical implications. Alterations in loading observed in patients with chronic and acute mitral regurgitation are thought to have a major impact on surgical results. It has been generally assumed that "unloading" of the left ventricle with chronic mitral regurgitation explains a decrease in ejection fraction that occurs after corrective surgery because of an increase in afterload. Although there may be an abrupt increase in loading immediately after replacement of an incompetent mitral valve (9), end-systolic volume is generally unchanged or even decreases after recovery from mitral valve replacement (20–22). In contrast, if ventricular afterload was increased by correcting chronic mitral regurgitation, one would expect an increase in end-systolic volume in accordance with the end-systolic pressure-volume relation. On the basis of these data, it seems more likely that the decrease in ejection fraction after mitral valve replacement for chronic mitral regurgitation is largely due to reduced preload, although in some cases, it may be partly a consequence of impairing the chordal-ventricular continuity by removing the native valve (6). Also, cardioplegic arrest may have some influence on myocardial performance and loading, particularly in the immediate postoperative state (9). However, after acute mitral regurgitation is corrected, one would anticipate an increase in afterload due to an increase in end-systolic pressure, which would result in an increase in end-systolic volume. To our knowledge, there are no data available to support this assumption. This question is relevant to the management of patients needing urgent mitral valve replacement for acute problems such as active rheumatic valvulitis, endocarditis or ischemic papillary muscle damage.

Although ejection fraction before valvuloplasty was ≤0.60 in four of our five patients, contractile function as assessed by the relation between preload-corrected ejection fraction and end-systolic stress was not depressed compared with that in normal subjects. This observation supports the concept that abnormal loading conditions (that is, increased afterload) may in part account for depressed ejection performance in rheumatic mitral stenosis, as has been determined by other investigations (23).

Limitations. Several limitations should be addressed. The finding of a mean regurgitant fraction of 0.23 before balloon valvuloplasty suggests that either 1) we underestimated the angiographic grade of mitral regurgitation before valvuloplasty (1+ or no mitral regurgitation in all five patients); 2) our angiographic flow calculations slightly overestimated the thermodilution flows (and forward output) even in the absence of mitral regurgitation; or 3) small changes in heart rate between the time of the thermodilution measurements and the left ventricular cineangiogram caused small changes in output that affected the calculation of regurgitant fraction. Another confounding variable in the measurement of cardiac output after balloon valvuloplasty is that there may have been a shunt too small to detect by oximetry, which would have caused overestimation of forward flow and underestimation of postvalvuloplasty regurgitant fraction. Another limitation is that there was mild residual mitral stenosis in our patients, which may have contributed to the lack of increase in end-diastolic volume observed in three of the patients, although the valve area was >2 cm^2 in all three of these patients. The effect of beta-adrenergic blocking drugs on contractility and heart rate in our patients is difficult to assess. An increase in contractility with acute mitral regurgitation might have occurred without beta-blockade. Conversely, it is likely that some of the beta-blockade effect may have dissipated by the end of the catheterization procedure as suggested by the observed increase in heart rate, although some withdrawal of vagal tone might also have contributed to the increased heart rate. Finally, as with all currently available indexes, the preload-corrected ejection fraction-afterload relation has limitations as a measure of contractility. For example, pericardial restraint may influence the assessment of preload, as previously discussed (5). However, the data support the concept that acute mitral regurgitation "unloads" the left ventricle and augments ejection performance without—by currently available methods—increasing contractility.

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