Mitral Regurgitation Following Mitral Balloon Valvotomy

Differing Mechanisms for Severe Versus Mild-to-Moderate Lesions

Mohammed R. Essop, MD; Thomas Wisenbaugh, MD; John Skoularigis, MD; Shirley Middlemost, MD; and Pinhas Sareli, MD

Background. This study was designed to evaluate the incidence and mechanisms of mitral regurgitation following mitral balloon valvotomy (MBV) in 40 consecutive patients with symptomatic tight pliable mitral stenosis.

Methods and Results. Transthoracic echocardiography with color flow mapping was performed before and 24 hours after the procedure. Patients who developed significant mitral regurgitation following MBV also underwent transesophageal echocardiography. The relation between increased mitral regurgitation and both valvular morphology and procedure-related factors was examined. Gorlin mitral valve area increased from 0.81±0.3 to 1.95±0.7 cm² (p<0.001). No patient had more than 2+ mitral regurgitation by angiography and color Doppler prior to MBV. There was a moderate correlation between Doppler and angiographic increase in mitral regurgitation (r=0.73, p<0.0001). By Doppler criteria 33 patients had no (n=6) or mild (n=27) increase in mitral regurgitation (group 1), and seven developed significant new mitral regurgitation (group 2). Baseline clinical, echocardiographic, and procedure-related data for the two groups were similar. Multiple regression analysis did not select any individual valve characteristic (valvular thickening, mobility, calcification, and subvalvular disease), total echocardiographic score, balloon diameter, or ratio of balloon to mitral annular diameter as predictors of mitral regurgitation. All seven patients in group 2 had evidence of major leaflet disruption with a torn anterior or posterior mitral leaflet in six and a ruptured papillary muscle in one. Two of these patients have required mitral valve replacement (6 and 9 months following the procedure), whereas the remainder are significantly symptomatic. By contrast, mitral regurgitation in group 1 either occurred at the site of commissural split (n=20) or was associated with prolapse of the anterior mitral leaflet (n=6).

Conclusions. Thus, severe new mitral regurgitation following MBV is due to noncommissural tearing of the mitral leaflet and confers an adverse long-term prognosis. A mild increase in mitral regurgitation following MBV is frequent and occurs at the site of commissural split or is associated with prolapse of the anterior leaflet. Furthermore, in this study, an increase in mitral regurgitation could not be predicted from any valvular or procedure-related factor.

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The mechanism by which mitral regurgitation develops de novo or is exacerbated following mitral balloon valvotomy (MBV) is not well understood. Although several studies have attempted to correlate valvular and subvalvular morphology1,2 or ratio of effective balloon dilating diameter to mitral annular diameter2 with the development of mitral regurgitation following MBV, no study has examined specifically the mechanism by which mitral regurgitation occurs. In vitro,3,4 autopsy,5 and echocardiographic6 studies have demonstrated that an increase in mitral valve area following MBV is largely due to separation of the mitral leaflets along the natural planes of their commissures. Whether an increase in mitral regurgitation is due to excessive commissural split or disruption of chordal or valvular anatomy, as has been reported in patients undergoing surgical commissurotomy, is unknown.7,8 The

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purpose of this study, therefore, was to determine the frequency, severity, and mechanism by which mitral regurgitation develops following MBV in a homogeneous group of patients with pliable mitral stenosis.

**Methods**

**Patients**

The patient population comprised 40 consecutive patients undergoing MBV for symptomatic tight pliable mitral stenosis. Only patients with an echocardiographic mitral valve score equal to or less than 8 were selected for the study. Informed consent was obtained from all patients. There were 35 women and five men (mean age, 27±8 years; range, 20–44). Nine patients were in New York Heart Association (NYHA) class II, 28 in class III, and three in class IV. Thirty-nine patients were in sinus rhythm and one had atrial fibrillation. Patients with left atrial thrombus or significant coexistent valve lesions were excluded.

**Two-Dimensional Echocardiography**

Each patient underwent a detailed echocardiographic examination performed 24 hours before and after MBV. All studies were performed by the same operator (M.R.E.) using a Hewlett Packard (Sonos 1000) imaging system attached to either 2.5-MHz or 5-MHz transducers to record images on videotape for playback analysis. Mitral valve and subvalvular structures were examined extensively in multiple standard and tilted parasternal long-axis views and apical two- or four-chamber views. An effort was made to maximally visualize the submitral apparatus along its entire length.

Mitral valve and subvalvular morphology was graded semiquantitatively as outlined by Wilkins et al.9 Scores of 0–4 were assigned to represent the severity of leaflet immobility, valve thickening and calcification, and subvalvular thickening with higher scores representing more morphological abnormality. The scores from all individual features were summed in each patient to obtain a total echocardiographic score.

Mitral annular diameter was measured in the apical four-chamber view in late diastole at the time of maximal mitral opening as the distance between the points of reflection of the septal and mural endocardium onto the anterior and posterior mitral leaflets, respectively. The normal for healthy adult volunteers in our laboratory is 28±2 mm.

Separation of the commissures was identified when new echocardiographic dropout in the region of one or both commissures was observed following MBV. Diag-

**Table 1. Baseline Clinical, Echocardiographic, and Hemodynamic Data Before and After Balloon Mitral Valvotomy**

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n=33)</th>
<th>Group 2 (n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-MBV</td>
<td>Post-MBV</td>
</tr>
<tr>
<td>Age (years)</td>
<td>27±8</td>
<td>27±7</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.6±0.2</td>
<td>1.6±0.1</td>
</tr>
<tr>
<td>NYHA FC</td>
<td>2.8±0.6</td>
<td>2.8±0.4</td>
</tr>
<tr>
<td>Echo score</td>
<td>6.2±1.2</td>
<td>6.6±1.2</td>
</tr>
<tr>
<td>Thickness</td>
<td>1.6±0.5</td>
<td>1.8±0.5</td>
</tr>
<tr>
<td>Mobility</td>
<td>1.4±0.5</td>
<td>1.5±0.5</td>
</tr>
<tr>
<td>Calcification</td>
<td>1.6±0.5</td>
<td>1.5±0.5</td>
</tr>
<tr>
<td>Submitral disease</td>
<td>1.7±0.5</td>
<td>1.7±0.5</td>
</tr>
<tr>
<td>MAD (mm)</td>
<td>37±3</td>
<td>36±4</td>
</tr>
<tr>
<td>EBD (mm)</td>
<td>36±3</td>
<td>37±3</td>
</tr>
<tr>
<td>EBD:MAD</td>
<td>0.9±0.1</td>
<td>1.0±0.1</td>
</tr>
<tr>
<td>Mean LAP (mm Hg)</td>
<td>21±7</td>
<td>11±4§</td>
</tr>
<tr>
<td>LA v (mm Hg)</td>
<td>29±11</td>
<td>15±7§</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>7±3</td>
<td>8±2</td>
</tr>
<tr>
<td>MVG (mm Hg)</td>
<td>11±3</td>
<td>4±3§</td>
</tr>
<tr>
<td>COTd (L/min)</td>
<td>3.5±0.8</td>
<td>4.1±0.8§</td>
</tr>
<tr>
<td>Gorlin (cm²)*</td>
<td>0.8±0.2</td>
<td>1.9±0.8§</td>
</tr>
<tr>
<td>2D MVA (cm²)+</td>
<td>0.8±0.2</td>
<td>2.2±0.4§</td>
</tr>
<tr>
<td>Doppler MVA (cm²)‡</td>
<td>0.7±0.2§</td>
<td>2.0±0.5</td>
</tr>
</tbody>
</table>

Pre-MBV, Pre-mitral balloon valvotomy; Post-MBV, post-mitral balloon valvotomy; BSA, body surface area; NYHA FC, New York Heart Association functional class; MAD, mitral annular diameter; EBD, effective balloon diameter; LAP, left atrial pressure; LA v, left atrial peak v wave; LVEDP, left ventricular end-diastolic pressure; MVG, mitral valve gradient; COTd, cardiac output by thermodilution.

*Mitral valve area calculated by Gorlin formula.
†Mitral valve area calculated by two-dimensional (2D) echocardiography.
‡Mitral valve area calculated by Doppler pressure half-time.
§Indicates p<0.05 for prevalvotomy vs. postvalvotomy.
||Indicates p<0.05 for group 2 postvalvotomy results vs. group 1 postvalvotomy.
nosis of commissure related mitral regurgitation was based on a clearly definable jet of mitral regurgitation originating at the commissure. The presence of mitral valve prolapse was determined using previously published criteria. A transesophageal examination was performed in all patients prior to MBV and following MBV in those who developed significant mitral regurgitation using a single-plane 5-MHz probe.

**Doppler Color Flow Mapping for Assessment of Severity and Mechanism of Mitral Regurgitation**

Doppler color flow examination was performed with multiple orthogonal parasternal and apical views. Values for red blood cell velocities were superimposed on the two-dimensional image in 16–32 shades of either red or blue, representing flow toward or away from the transducer, respectively. Green color was added to areas demonstrating variance in the Doppler signal, serving as an indicator for turbulent flow. Each examination was performed with use of the shallowest depth and narrowest sector angle capable of encompassing the entire jet area in order to maximize the frame rate. Care was taken to use for each examination an optimal gain setting, which was defined as the maximal gain level possible without introducing signals outside of flow areas or onto tissue from an adjoining chamber.

Severity of mitral regurgitation was determined by expressing the ratio of maximal jet area to left atrial area in the same view. Recordings containing the largest regurgitant jet area were identified and measured by tracing the outer border of the largest clearly definable flow. The planimetered area of disturbed flow included central varianced and aliased signals as well as immediately contiguous nonturbulent velocities that were moving in the same direction as the jet. A regurgitant jet–to–left atrial area ratio of

5–25% was graded as +1, 26–50% as +2, 51–75% as +3, and 76–100% as +4.

**Mitral Balloon Valvotomy**

Mitral balloon valvotomy was performed using a standard antegrade double-balloon technique as described by Al-Zaibag et al. Effective balloon dilating diameter was expressed as the sum of the individual diameters of the two balloons. Simultaneous left atrial and left ventricular pressures using micromanometer catheters and thermodilution cardiac outputs were recorded to compute mitral valve area before and after MBV. Gated left ventricular cineangiography was performed under identical conditions before and after MBV in 32 of the 40 patients. Mitral regurgitation was graded from +1 to +4 as previously described.

**Data Analysis**

Mitral valve morphology score, Doppler, and angiographic mitral regurgitation before and after balloon valvotomy were assessed separately and independently by two observers. Using linear regression analysis, correlation between the two observers for the mitral valve morphology score (r=0.93, p<0.001), Doppler (r=0.91, p<0.001), and angiographic (r=0.95, p<0.001) mitral regurgitation was excellent. An increase in mitral regurgitation grade was calculated as the arithmetic difference between the grade before and after MBV. Patients were divided into two groups according to the color Doppler increase in mitral regurgitation following MBV. Group 1 comprised those individuals with an increase in mitral regurgitation grade less than two and group 2 those with an increase equal to or more than two. Measurements before and after MBV were compared by the Student's paired t test and comparisons between group 1 and group 2 using the unpaired
Results

Overall Results

MBV was uneventful in all but two patients. One patient had a transient hypotensive episode with ischemic changes on the electrocardiogram suggesting a coronary embolus and the other a cerebral embolus resulting in a transient ischemic attack. An atrial septal defect with a shunt greater than 1.5:1 was detected on oximetry in four patients. No left ventricular perforation or death occurred during this study.

Acute Hemodynamic Results

Despite a reduction in diastolic mitral gradient in both groups, mean left atrial pressure in group 2 failed to drop because of a significant increase in the left atrial V wave (Table 1). This was accompanied by an increase in left ventricular end-diastolic pressure, persistent pulmonary hypertension, and a significant drop in forward thermodilution cardiac output. Underestimation of true mitral valve flow resulted in a lower Gorlin mitral valve area compared with planimetered or Doppler mitral valve area.

Frequency and Severity of Mitral Regurgitation Following Balloon Valvotomy

Left ventricular cineangiograms of suitable quality for analysis were available in 32 patients. Regurgitant grade remained the same in 13, increased by one grade in nine, two grades in five, three grades in three, and four grades in two patients (Figure 1). By Doppler color flow imaging, regurgitant grade remained unchanged in seven, increased by one grade in 26, two grades in one, three grades in five, and four grades in one patient (Figure 2). There was a modest correlation between angiographic and Doppler mitral regurgitation before MBV (r=0.52, p=0.002), after MBV (r=0.82, p<0.001), and the increment in mitral regurgitation (r=0.73, p<0.001).

Mechanism of Mitral Regurgitation

Echo Doppler examination clarified the mechanism of mitral regurgitation in all patients with increased mitral regurgitation following MBV. Dis-
ruption of valvular or of the subvalvular apparatus was identified in all patients in group 2. A tear of the posterior mitral leaflet was seen in five patients (Figure 3), one patient was found to have a torn anterior mitral leaflet (Figure 4) and one an avulsed anterolateral papillary muscle (Figure 5). Confirmation of the mechanism was obtained at surgery in two patients and by transesophageal echocardiography in
all seven patients. In contrast, mild mitral regurgitation was seen to occur at the sites of one or both commissures that had been split in 20 patients (Figures 6 and 7) or in association with mild prolapse of the anterior mitral leaflet in six patients (Figure 8).

**Long-term Follow-up**

Group 1 patients had an immediate and sustained improvement in symptoms at a mean follow-up of 14 months. By contrast, group 2 patients remained symptomatic at the time of discharge and showed progressive deterioration in functional capacity thereafter. Although no patient required immediate surgery, two patients in this group have been subjected to mitral valve replacement, a third has refused surgery, and a fourth patient is awaiting surgery.

**Predictors of Mitral Regurgitation**

Baseline valve morphological characteristics were similar for both groups of patients (Table 1). Similarly, predilatation and postdilatation valve area, degree of mitral regurgitation prior to balloon valvotomy, effective balloon diameter, ratio of effective balloon diameter to mitral annular diameter, and ratio of effective balloon dilating area to body surface area were similar in both patient groups. Correlation analysis failed to demonstrate any relation between the aforementioned characteristics and the development of mitral regurgitation.

**Discussion**

There are four major findings of this study. 1) Increase in severity of mitral regurgitation following MBV is frequent (33 of 40, 83%) but is significant in only a small number of patients (seven of 40, 17%). 2) The mechanisms for mild versus significant mitral regurgitation as defined by transthoracic and transesophageal echocardiography are different. 3) Mitral regurgitation following MBV in patients with pliable mitral stenosis cannot be predicted by any of the currently available criteria. 4) Although the development of significant new mitral regurgitation carries an unfavorable long-term prognosis as defined by necessity for mitral valve surgery (four of seven, 57%), it does not necessarily result in severe adverse hemodynamics requiring immediate surgery.

The results of this study emphasize the significance of mitral regurgitation as the major factor contributing to morbidity following MBV. The reported incidence of significant mitral regurgitation following MBV varies from 0% to 19%. Heterogeneity in study populations, valvular morphology, balloon valvotomy technique, and size of balloons used made comparisons difficult. The subjects included in this study were a relatively homogeneous group of young patients with tight pliable mitral stenosis who previously would have been ideal candidates for a closed mitral commissurotomy. Despite rigid selection criteria we have found an 83% incidence of an increase in regurgitant grade and a 17% (seven of 40) incidence of significant mitral regurgitation (Doppler increase of two grades or more). By conventional angiographic criteria these incidences were 59% (19 of 32) and 31% (10 of 32), respectively. Although Doppler and angiographic mitral regurgitation correlated well, there was a consistent trend for angiography to overestimate the degree of mitral regurgitation. When studying patients with pure mitral regurgitation, Spain et al. found a good correlation between Doppler and angiography. Because angiographic assessment of mitral regurgitation is dependent on an accumulation of contrast in the left atrium over several cardiac cycles with Doppler evaluation visualizing instantaneous regurgitation, we have hypothesized that residual mitral stenosis would enhance contrast accumulation in this group of patients and therefore account for the difference. Alternatively, it is possible that color Doppler underestimated the magnitude of mitral regurgitation by the Coanda effect. Furthermore, evaluation of mitral regurgitation by the two techniques was not performed simultaneously. Differences in loading conditions at the time of examination might have been a further factor.

Splitting of fused commissures has been documented to be the mechanism by which mitral valve area increases following MBV. This same mechanism appears to be responsible for the observed exacerbation in mitral regurgitation following successful MBV. Two-dimensional echocardiography
FIGURE 6. Mild mitral regurgitation following mitral balloon valvotomy due to unilateral commissural split. Upper panel: Parasternal short-axis view at mitral valve level shows echo dropout in the region of the lateral commissure. Lower panel: Color Doppler shows mitral regurgitant jet spurting from lateral commissure. SLC, split lateral commissure.
FIGURE 7. Mild mitral regurgitation following mitral balloon valvotomy due to bilateral commissural split. Upper panel: Parasternal short-axis view at mitral valve level shows echo dropout in the region of both commissures. Lower panel: Color Doppler shows two jets of mitral regurgitation originating at the medial and lateral commissures. LA, left atrium; RA, right atrium; LVOT, left ventricular outflow tract.

with localization of regurgitant jet origin by color Doppler flow mapping demonstrated that post-MBV mitral regurgitation occurs at the site of a successful commissural split. The accuracy of color flow information on jet origin and direction in localizing the site of mitral regurgitation has been recently con-
Commissural mitral regurgitation (Figures 6 and 7) is usually mild, is of no clinical significance, and represents the most frequent mechanism by which a mild increase in mitral regurgitation occurs (20 of 26, 77%). The mechanism of mild increase in mitral regurgitation in a smaller group of patients (six of 26, 23%) was prolapse of the anterior mitral leaflet (Figure 8). In these patients anterior mitral leaflet prolapse is most likely due to the combination of annular dilatation (Table 1), mild leaflet retraction, and absence of significant subvalvular disease with no chordal shortening.

In contrast, severe mitral regurgitation following MBV is almost always due to noncommissural tearing of the anterior or posterior mitral leaflet with disruption of the valve mechanism (Figures 3–5). These findings are similar to those reported by Nobuyoshi et al\(^\text{17}\) following MBV and Gross et al\(^\text{18}\) following open mitral commissurotomy. Using an in vitro model, Reifart et al\(^\text{19}\) found a 13% incidence of leaflet laceration with more advanced morphological changes at the commissures in valves that tended to rupture. They postulated therefore that the leaflets themselves were less resistant to rupture than the commissures. The excised valves that they examined, however, were fibrotic and calcific.

Other than a single study,\(^\text{2}\) no other series including our own has been able to demonstrate a correlation between valvular morphology and the development of mitral regurgitation. The rather high incidence of mitral regurgitation that we have demonstrated in a series of patients notable for its young age and low echocardiographic score has led us to speculate whether excessively pliable valves might not actually constitute a risk factor for leaflet rupture and abrupt mitral regurgitation. This has important implications in areas characterized by a high prevalence of mitral stenosis where MBV has potentially its widest application. Data on the effect of balloon diameter on mitral regurgitation are conflicting. Two studies have shown no correlation of mitral regurgitation with either absolute effective balloon dilating area\(^1\) or ratio of balloon dilating area to body surface area.\(^2\) In contrast, two studies have demonstrated an adverse correlation between high balloon dilating area to body surface area ratio and production of mitral regurgitation.\(^3,20\) In order to rationalize selection of balloon size, Chen et al\(^2\) investigated the relation between the ratio of effective balloon dilating diameter to mitral annular diameter and the incidence in mitral regurgitation following MBV. A ratio of more than 1.1 was found by them to carry a 70% risk of abrupt mitral regurgitation as opposed to an 18% risk if the ratio was less than 1.1. We have not been able to demonstrate a relation between any morphological characteristic, balloon diameter, or ratio of balloon to mitral annular diameter and the development of abrupt mitral regurgitation.

Prognosis in patients who develop mitral regurgitation following surgical commissurotomy\(^18\) or MBV\(^17\) is poor with failure of improvement in symptoms, persistent adverse hemodynamics, and ultimate necessity for mitral valve replacement. Although none of our patients required emergency mitral valve replacement, two of the seven patients with severe mitral regurgitation after MBV have thus far been submitted to surgery 6 months and 9 months, respectively, following the initial procedure. Surgical confirmation of the echocardiographic findings was obtained in both patients.

**Conclusion**

From this study we conclude that 1) mitral regurgitation is a relatively frequent complication of MBV in young patients with pliable mitral stenosis; 2) the mechanism of mild increase in mitral regurgitation grade is commissural split with probable inadequate leaflet coaptation at this site, or less frequently, prolapse of the anterior mitral leaflet; and 3) severe mitral regurgitation is an unpredictable event and is usually due to leaflet rupture.

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**Figure 8.** Parasternal long-axis view in a patient with mild mitral regurgitation following mitral balloon valvoplasty due to prolapse of the anterior mitral leaflet (arrow).
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