Long-Term Changes in Mitral Valve Area After Successful Mitral Commissurotomy

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SUMMARY We examined the long-term effects of closed instrumental mitral commissurotomy on mitral valve area (MVA) in 18 patients, followed for as long as 14 years after successful operation. Each patient had preoperative and early postoperative cardiac catheterization; a late postoperative determination of MVA was obtained 10–14 years (mean 12.2 years) after commissurotomy. In 17 patients, the MVA was determined by cross-sectional echocardiography and in one patient by repeat cardiac catheterization. Thirteen of 18 patients had no change in MVA between early postoperative study (mean MVA = 2.7 cm²) and late postoperative study (mean MVA = 2.9 cm²). MVA in five patients decreased 0.7–2.2 cm² (mean 1.4 cm²) during the follow-up period. In these five patients, the mean MVA at early postoperative study was 2.7 cm² and at late postoperative study was 1.3 cm² (p < 0.001). At late postoperative evaluation, cardiac symptoms were associated with severity of mitral stenosis but did not predict restenosis. A successful, closed, instrumental mitral commissurotomy can provide substantial long-term improvement in MVA.

RHEUMATIC MITRAL STENOSIS is an acquired form of valvular heart disease characterized by diffuse thickening of the mitral leaflets, fusion of the commissures, and shortening and fusion of the chordae tendineae. These pathologic lesions combine to decrease the size of the mitral valve orifice, thereby restricting the flow of blood into the left ventricle. When the decrease in mitral valve area (MVA) becomes critical, surgical correction of the valvar lesion is frequently required. Surgical improvement in MVA may be accomplished by prosthetic mitral valve replacement, or, in selected cases, by mitral commissurotomy. Mitral commissurotomy appears to be preferable, since the operative morbidity and mortality are less and postoperative complications fewer than those associated with valve replacement.

There is concern, however, that restenosis of the valve may occur after a successful commissurotomy, necessitating a second commissurotomy or a valve replacement. On the basis of clinical and hemodynamic studies, many investigators report that reoperation is often required after a commissurotomy, implying restenosis of the valve.1–7 Clinical studies have relied on changes in symptomatic status to evaluate changes in the status of the mitral valve. Hemodynamic data demonstrate, however, that recurrent symptoms may be due not to restenosis, but to an inadequate operation with residual stenosis or associated cardiac lesions.7 Although serial hemodynamic studies have been undertaken to define the long-term changes in MVA after commissurotomy, they have been limited to either right-heart catheterization data or the study of primarily those patients with recurrent symptoms.6,8 As a result, data concerning the overall, long-term effects of successful mitral commissurotomy on the MVA in patients with and without recurrent symptoms are unavailable. This is important information, since the decision to perform a commissurotomy depends, in part, on the expected long-term results.

We report our findings in a group of patients who had a documented successful commissurotomy, and who were then followed for as long as 14 years to determine the long-term effects of the operation on MVA and the incidence of restenosis.

Methods

To determine the long-term effects of mitral valve commissurotomy on MVA, we studied only patients who had pure mitral stenosis and evidence of a successful mitral commissurotomy. Documentation of the success of a commissurotomy was threefold: 1) a preoperative right and left heart catheterization with calculation of MVA; 2) a closed instrumental or an open mitral commissurotomy; 3) a catheterization performed at least 6 months after surgery that demonstrated an increase in MVA of at least 0.5 cm².

Thirty-five patients originally agreed to undergo these procedures and the hemodynamic studies and cardiac surgery were performed between 1962–1967. During this period, 19 other patients underwent preoperative catheterization and mitral commissurotomy for treatment of pure mitral stenosis, but did not agree to undergo postoperative catheterization.

All catheterization studies were performed at the Indiana University School of Medicine. Each patient underwent right and retrograde left heart catheterization using standard techniques. Pressures were recorded through fluid-filled catheter systems. The mitral valve gradient was determined by planimetry of the difference between either simultaneous left atrial
(32 patients) or pulmonary capillary wedge pressure (three patients) and left ventricular pressures. Cardiac output was determined in a non-sedated state using the direct Fick method. The MVA was calculated by application of the Gorlin formula for valve orifice size.\textsuperscript{10} We consider a valve orifice size \( \leq 1.5 \text{ cm}^2 \) to be hemodynamically significant. Mitral regurgitation was quantified at preoperative and early postoperative catheterization by left ventricular cineangiography and was graded as none, minimal, mild, moderate or severe.

Of the 35 patients who had preoperative catheterization, mitral commissurotomy and postoperative catheterization, 31 had a documented, successful commissurotomy — one which produced an increase in MVA \( \geq 0.5 \text{ cm}^2 \). The initial evaluation and hemodynamic findings of these patients were presented in an earlier report.\textsuperscript{11} A closed instrumental mitral commissurotomy was performed in 30 of the 31 patients; but in one case an open commissurotomy was necessary. Before surgery, cinefluoroscopy revealed no valvular calcification in any patient, although one was later shown to have moderate calcification of one of the mitral leaflets at the time of surgery.

Of the 31 patients who had a proven successful commissurotomy, 18 returned for further study. Two patients were lost to follow-up and five either refused or could not return for repeat evaluation. Since information concerning long-term change in MVA after commissurotomy was not available in these patients, they were not included in the long-term evaluation. There were six deaths during the follow-up period: in five patients, the cause of death was ascertained from either the autopsy record, the death certificate or the physician's record, but in one patient no such information was available.

Late postoperative studies were done 10-14 years after mitral commissurotomy. Because most patients were asymptomatic at the time of the follow-up study, repeat hemodynamic studies were not felt to be indicated. In order to assess the mitral valve orifice size, therefore, cross-sectional echocardiographic studies of the mitral valve were performed. Repeat cardiac catheterization was performed in three patients. In one, the catheterization data was inadequate to calculate MVA; in another, cross-sectional echocardiography was not yet available to measure MVA; and in the third patient, MVA was measured both by catheterization and cross-sectional echocardiography.

Measuring the size of the mitral valve orifice by cross-sectional echocardiography is a noninvasive method of quantitating mitral stenosis. Studies from four separate laboratories have shown a close correlation between the size of the mitral valve orifice measured at surgery or at catheterization and the size of the mitral valve orifice measured by cross-sectional echocardiography.\textsuperscript{12-18} The MVA measured by cross-sectional echocardiography has generally been within 0.2 cm\(^2\) of the MVA obtained by catheterization or surgical estimates. In a few instances where direct pathologic measurement of MVA was reported, the cross-sectional measurement was identical to the pathologic measurement of the MVA. Thus, measurement of the MVA by cross-sectional echocardiography offers an accurate, noninvasive method of evaluating mitral orifice size.

Cross-sectional examinations were performed using either the mechanical sector scanner developed in conjunction with Fortune-Fry Research Laboratory at the Indiana University School of Medicine, or a commercially available mechanical scanner, EkoSector I (Smith-Kline Instruments), both of which consisted of a modified Ekoline 20A echograph with a pulse repetition rate of approximately 4 kHz. The scanner probe contained a 2.25 MHz transducer which was mechanically driven through a 30° sector at a rate of 30 Hz. This operating mode permitted data recording at a rate of 30 frames/sec (60 fields/sec), which yielded a line density of approximately 100 lines/field.

Cross-sectional studies were recorded on half-inch videotape using a Sanyo VTC-7100 cassette recorder. These images were available for redisplay in a real-time, slow-motion, or a single-frame format. The individual frames were converted to hard copy using a standard Polaroid photographic system.

Cross-sectional examinations were performed with the patient in either a supine or a 30° left lateral position with the head elevated approximately 30°. The transducer was first aligned parallel to the long axis of the left ventricle to identify the location of the mitral valve orifice. The mitral valve orifice was then placed in the center of the 30° scan and the probe was rotated 90° to visualize directly a short axis configuration of the mitral valve orifice. Once the mitral valve orifice was located, the probe was angled slightly superiorly and inferiorly along the cone of the mitral valve to record the smallest mitral valve orifice. The mitral valve orifice was then measured by direct planimetry of the internal margins of the echoes from this structure. Figure 1 is a photograph of the cross-sectional image seen at the level of the mitral valve orifice.

The assessment of the level of systems and functional classification was determined by review of the hospital records from the time of initial contact and then by interview at the time of the follow-up examination. The criteria of the New York Heart Association were used for functional classification.\textsuperscript{18} Fixed analysis of the data was performed using the \( t \) test and standard table of probability.

Results

Late postoperative determination of MVA was obtained in 18 patients followed 10-14 years (mean 12.2 years) after documented successful mitral commissurotomy. The data for preoperative, early postoperative, and late postoperative MVA, degree of mitral regurgitation, and functional classification are listed in table 1. The data illustrating the change in MVA for each patient are displayed in figure 2. In patient 15, the late postoperative MVA was determined by cardiac catheterization alone. In the remain-
ing 17 patients, MVA was determined by cross-sectional echocardiography and one (patient 18) also had late postoperative catheterization.

At late postoperative examination, 13 of 18 patients had no change in MVA compared with early postoperative catheterization. At late follow-up examination the mean MVA for the 13 patients was 2.9 cm² (range 2.0-4.4 cm²). At early postoperative catheterization, these same 13 patients had a mean MVA of 2.8 cm² (range 1.9-4.9 cm²).

Five patients had significant decreases in MVA (range 0.7-1.8 cm²) during the 10-14-year follow-up period. The mean MVA at early postoperative study for these five patients was 2.7 cm² (2.0-3.2 cm²), compared with a mean late postoperative MVA of 1.3 cm² (range 0.7-1.7 cm², p < 0.001).

Patients 6 and 7 had very large MVAs at both early and late postoperative evaluations. The apparent differences between MVAs at the two evaluations probably reflects difficulties in precisely determining large MVAs by catheterization and echocardiographic methods. The variation between these two methods of measuring large MVAs cannot be predicted. However, it is clear that both patients had minimal or no mitral stenosis at early and late postoperative evaluations.

The initial severity of mitral stenosis and the effectiveness of mitral commissurotomy may influence long-term prognosis. These factors were evaluated by comparing preoperative MVA, early postoperative MVA, and change in MVA for the two subgroups of patients. The mean preoperative MVA for patients without late progression of their disease was 1 cm², compared with a mean preoperative value of 0.8 cm² for patients with late restenosis. At early postoperative evaluation the 13 patients with no late progression of disease had a mean MVA of 2.8 cm², compared with 2.7 cm² for patients with late restenosis. Likewise, the mean increase in MVA after commissurotomy was not different between these subgroups of patients, averaging 1.8 cm² in those without late progression and 1.9 cm² in those with late restenosis. The preoperative and early postoperative MVAs are similar for these two subgroups of patients, indicating that severity of mitral stenosis at entry to the study and effectiveness of mitral commissurotomy were similar. Therefore, these subgroups appeared to be matched for two factors likely to affect long-term prognosis.

In an attempt to identify other factors that might influence long-term prognosis, we compared

![Figure 1](http://circ.ahajournals.org/)

**FIGURE 1.** Short-axis, cross-sectional echogram of a mildly stenotic mitral valve (patient 13). The mitral valve area is 2.2 cm². RV = right ventricle; IVS = interventricular septum; MVO = mitral valve orifice; PW = posterior wall of left ventricle.

![Figure 2](http://circ.ahajournals.org/)

**FIGURE 2.** Graphic display of the serial changes in mitral valve area (MVA) in 18 patients. All patients had an increase in MVA at postoperative study, and in most, the MVA did not change at late follow-up examination. Pre-op = preoperative catheterization; Early postop = early postoperative catheterization; late postop = late postoperative study.
Table 1. Clinical, Hemodynamic and Cross-Sectional Echocardiographic Data in 18 Patients with Long-Term Follow-up After Mitral Commissurotomy

| Patient | Age at entry (yrs) | Sex | MVA* pre-op (cm²) | MVA* early post-op (cm²) | MVA† late post-op (cm²) | Length of follow-up (yrs) | NYHA functional class Pre-op | Early post-op | Late post-op |
|---------|--------------------|-----|-------------------|--------------------------|-------------------------|--------------------------|-----------------------------|----------------|-------------|-------------|
| 1       | 40                 | F    | 0.6               | 0                        | 2.7                     | 0                        | 3.2                         | 12             | II          | I           |
| 2       | 32                 | F    | 0.9 MIN           | 1.9 MIN                  | 2.3                     | 13                       | II                          | II             | II          |
| 3       | 36                 | F    | 0.5              | 1.9                      | 2.2                     | 12                       | II                          | I              | I           |
| 4       | 39                 | F    | 1.4             | 3.2                      | 3.1                     | 13                       | III                         | I              | I           |
| 5       | 31                 | F    | 0.9              | 2.2                      | 2.0                     | 14                       | III                         | I              | II          |
| 6       | 36                 | M    | 1.4              | 4.9 MIN                  | 4.4                     | 11                       | II                          | I              | I           |
| 7       | 16                 | M    | 0.9              | 3.8 MIN                  | 4.4                     | 12                       | III                         | I              | I           |
| 8       | 35                 | F    | 0.9              | 2.4                      | 2.9                     | 13                       | III                         | I              | I           |
| 9       | 28                 | F    | 1.1              | 2.0                      | 2.1                     | 11                       | III                         | I              | I           |
| 10      | 34                 | F    | 1.3 MIN          | 2.8                      | 2.9                     | 13                       | II                          | I              | I           |
| 11      | 27                 | F    | 1.0              | 3.4 MIN                  | 3.1                     | 11                       | III                         | I              | I           |
| 12      | 47                 | F    | 0.9              | 2.4 MIN                  | 2.6                     | 12                       | III                         | II             | I           |
| 13      | 53                 | F    | 0.8              | 2.2 MIN                  | 2.2                     | 12                       | III                         | II             | I           |
| 14      | 38                 | F    | 0.6              | 2.0 MIN                  | 1.3†                    | 11                       | III                         | II             | I           |
| 15      | 30                 | F    | 1.1              | 2.7                      | 1.7*                    | 11                       | III                         | I              | III         |
| 16      | 40                 | F    | 0.8 MIN          | 3.2 MILD                 | 1.7                     | 13                       | III                         | II             | I           |
| 17      | 28                 | F    | 0.9              | 2.8                      | 1.2                     | 12                       | III                         | I              | II          |
| 18      | 35                 | F    | 0.8              | 2.9 MIN                  | 0.7† (0.5)*            | 13                       | III                         | I              | III         |

*Mitral valve area calculated from cardiac catheterization data and Gorlin formula.10
†Mitral valve area measured by cross-sectional echocardiogram.

Abbreviations: MR = mitral regurgitation; 0 = none; MIN = Minimal; MVA = mitral valve area; NYHA = New York Heart Association.

hemodynamic parameters of the two subgroups at the time of initial catheterization. There were no significant differences in cardiac index, systolic pulmonary artery pressure, mean pulmonary artery pressure or pulmonary vascular resistance between these patients, the age and sex of patients in these two subgroups were similar.

Since recurrence of cardiac symptoms is a clinical tool for measuring success of commissurotomy and need for further catheterization, changes in functional classifications during the follow-up period were evaluated. Six patients (2, 3, 5, 15, 17 and 18) reported an increase in cardiac symptoms following commissurotomy. Three of these six (15, 17 and 18) had decreased MVAs. The other three with recurring symptoms had no decrease in MVA during the follow-up period.

Examination of all patients with functional limitation at the time of late postoperative evaluation revealed that they constituted a subgroup of patients with suboptimal results from commissurotomy. At late postoperative evaluation, 10 patients were New York Heart Association functional class II or III. Five patients (14–18) had restenosis of the mitral valve, but five (patients 2, 3, 5, 12 and 13) had no evidence of restenosis. These five patients without restenosis had late postoperative MVAs ranging from 2.0–2.6 cm² (mean 2.3 cm²) compared with the other patients without restenosis, but free of symptoms, who had MVAs ranging from 2.1–4.4 cm² (mean 3.3 cm²) (p < 0.02). Thus, although deterioration in functional class was not a good indication of restenosis, the presence of cardiac symptoms at late postoperative evaluation identified a group of patients with restenosis as well as a group who appeared to have suboptimal postoperative results.

The six deaths in the follow-up period occurred 2–11 years after commissurotomy. Preoperative and postoperative catheterization data for MVA and mitral regurgitation for these patients are given in table 2. Mean preoperative MVA was 1.1 cm² (range 0.9–1.4 cm²) and mean postoperative MVA was 2.9 cm² (range 1.9–5.5 cm²). Nonsurvivors had MVAs similar to those of survivors, indicating that initial severity of stenosis and early operative success were not different in the nonsurvivors. Autopsy data were available for three patients (1, 3, and 4) but comments about mitral valve morphology were made in only one case (patient 4), where the orifice was described to admit one finger of the examiner, suggesting at least moderate stenosis.

Patients 4 and 5 had an increase in severity of mitral regurgitation after commissurotomy, and both died of bacterial endocarditis within 2 years of surgery. A complication of the operative procedure may have contributed to the early death of these two patients.

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Discussion

Rheumatic mitral stenosis presents anatomic and hemodynamic impediments to cardiac function by producing obstruction of the mitral valve orifice. Mitral commissurotomy may relieve the obstruction; however, the long-term prognosis is unknown. Since the size of the mitral valve orifice determines the amount of mitral stenosis, serial measurements of the size of the mitral orifice would be an important method of determining the long-term prognosis of mitral stenosis. This report describes changes in MVA in 18 patients studied 10–14 years after successful mitral commissurotomy. Each patient had hemodynamically significant mitral stenosis and underwent closed, instrumental mitral commissurotomy. Each patient had a documented, postoperative increase in MVA > 0.5 cm². In the 10–14 years after commissurotomy, 13 patients had no decrease in MVA, while five patients had evidence of restenosis of the mitral valve. The long-term course could not be predicted by differences in preoperative or postoperative MVA, preoperative hemodynamics, or the age of the patients. No patient reported symptoms of recurrent rheumatic activity so this did not appear to be a factor leading to restenosis.

The classic, long-term studies of Ellis and Harken and other clinical studies demonstrated that most patients with mitral stenosis obtain symptomatic relief after commissurotomy. They also reported that a significant number of patients required reoperation. This raised the question whether the need for reoperation represented restenosis of the valve or the late result of an inadequate commissurotomy. Further information was provided when Higgs et al. reported that recurrent symptoms after commissurotomy often resulted from residual stenosis from an inadequate commissurotomy, mitral regurgitation produced at the operation, or associated valvular and cardiac abnormalities. The present study was designed to discount the influence of residual stenosis on long-term prognosis after commissurotomy. In order to achieve this goal, each patient was required to have documentation of a successful operation as judged by early postoperative catheterization. We obtained data in this manner to accommodate long-term follow-up of actual changes in MVA and not clinical symptoms alone.

A potential hindrance to serial measurement of MVA after commissurotomy has been the need to repeat cardiac catheterization in asymptomatic patients. The recent development of noninvasive methods of evaluating mitral valve orifice size overcomes this limitation. Cross-sectional echocardiography allows two-dimensional, direct visualization of the mitral valve orifice in real-time. Direct measurement of MVA by this method correlates well with the estimate of mitral orifice size by cardiac catheterization. Clinical experience in this laboratory with more than 75 patients indicates an excellent correlation between the hemodynamic and the cross-sectional echocardiographic estimates of MVA. Furthermore, in three cases the cross-sectional echocardiographic measurement of MVA was identical to the measurement of MVA at pathologic examination. Thus, clinical experience — our own and that reported by three independent laboratories — indicates that cross-sectional echocardiography provides a reliable measure of MVA.

Closed, instrumental commissurotomy using a transventricular mitral valve dilator was done in 17 of 18 patients; one patient had open commissurotomy. Since techniques in cardiopulmonary bypass have improved, open commissurotomy currently appears to be the operation of choice and closed commissurotomy is less frequently done. However, a study using pre- and postoperative hemodynamic evaluations indicates that closed, instrumental commissurotomy produces a significant increase in postoperative MVA in carefully selected patients. In this study, the early postoperative results of commissurotomy were
assessed by determining MVA at postoperative cardiac catheterization.

Eighteen patients were reexamined 10–14 years after successful commissurotomy. The MVA had not changed in 13, and had decreased in five — a 28% incidence of restenosis. This finding agrees with data from a similar study by Lyons et al. who found restenosis in three of 12 patients 5 years after documented successful commissurotomy. The present study extended the follow-up period from 5 to 14 years and found a similar rate of restenosis, suggesting that it may occur in the early years after commissurotomy. However, further documentation of serial changes in MVA are required to support this hypothesis. Although both studies deal with small groups of patients, they agree that in a population of patients who receive a successful commissurotomy, many will not show progressive restenosis over a long-term period.

Evaluation of cardiac symptoms remains an important clinical tool for evaluating the results of mitral commissurotomy. Improvement of symptoms occurs in nearly all patients who undergo commissurotomy. Subsequent deterioration appears to be multifactorial, with residual mitral stenosis as an important factor. In this study, the effect of residual stenosis was minimized by including only patients with a documented increase in MVA. At late postoperative examination, 10 patients had some degree of cardiac symptoms. These 10 patients each had a late postoperative MVA < 2.6 cm². Evidence of restenosis was present in five and the remaining five had no restenosis, although they appeared to have suboptimal symptomatic relief. Since late postoperative cardiac catheterization was not performed, other valvular abnormalities or the presence of coronary artery disease could not be systematically evaluated. In the five patients without restenosis who had recurrent or residual symptoms, the MVA was 2.0–2.6 cm², suggesting that factors other than mitral stenosis itself may have contributed to the functional limitation.

Late postoperative information was not obtained in the entire group of 31 patients with documented successful commissurotomy because six patients died and seven patients either refused evaluation or could not be located. Long-term status could not be determined in the seven patients without follow-up. In the six who died, at least one had evidence of restenosis of the mitral valve. Because the follow-up information is incomplete, a true incidence of restenosis cannot be reliably calculated from this study. Both the small number of patients and the incomplete long-term follow-up illustrate the difficulty in obtaining information on long-term changes in MVA. Despite the limitation, however, it is evident that no decrease in MVA was present in 13 of 18 patients with complete late postoperative data. If we assume that all 31 patients were at risk of restenosis, then 13 of 31 (42%) had no change in MVA over a 14-year follow-up.

Successful mitral commissurotomy may alter both the clinical and anatomic course of an otherwise progressive disease. In properly selected patients, commissurotomy will result in a significantly increased mitral valve orifice size. Although restenosis will occur in some, many patients will maintain clinical improvement and have no change in MVA during long-term follow-up after mitral valve commissurotomy.

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