LETTERS TO THE EDITOR

Predictors of Left Ventricular Dysfunction Following Mitral Valve Repair for Mitral Regurgitation

We read the study by Matsumura et al. (1) and the accompanying editorial by Wisenbaugh (2) with great interest. Unfortunately, in developing countries, rheumatic heart disease remains the major cause for severe mitral regurgitation. Furthermore, many patients are seen for the first time when adverse echocardiographic characteristics such as an ejection fraction <60% or an end-systolic diameter >45 mm are already present. The results of mitral valve replacement under these circumstances have been clearly documented to be poor (3), and an often-asked question is whether mitral valve repair—although more difficult with rheumatic disease compared to degenerative disease—would not be preferable in these patients.

Although not stated clearly in the report or editorial, it would appear that the echocardiographic predictors of a poor outcome following mitral valve repair are numerically similar if not identical to those previously defined for mitral valve replacement. This is disappointing because given the well-known benefits of valve repair in terms of preservation of chordal-ventricular continuity, one would have anticipated that postoperative left ventricular (LV) function could be guaranteed at lower preoperative ejection fractions or higher end-systolic diameters.

Also of great interest, but not commented upon, is the fact that in a significant number of patients, postoperative ejection fraction was higher compared to the preoperative value (Fig. 1 in Matsumura et al. [1]). Although preservation of ejection-phase indices of LV function have been well documented with mitral valve repair, the apparent increase in ejection fraction noted by Matsumura et al. (1) is a less known phenomenon, especially as the greatest impact of any mitral valve surgery is an immediate and significant decline in the preload. A postoperative reduction in afterload has been documented in some studies of mitral valve repair (4,5) and is supported in the current study by a reduction in the end-systolic diameter. However, postoperative reduction in afterload in the studies by Lessana et al. (4) and Bonchek et al. (5) did not result in an increase in ejection fraction but was sufficient only to prevent a significant decline in the ejection fraction. A depressed preoperative contractile state, which may have improved with removal of chronic volume overload, is also unlikely as the majority of patients with improvement in ejection fraction had normal ejection fractions to begin with (6).

Finally, it would be of interest to know what proportion of patients had moderate mitral regurgitation, as most guidelines require that mitral regurgitation be considered significant before contemplating operative intervention.

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REFERENCES


REPLY

We appreciate the comments by Dr. Essop regarding our recent report on left ventricular (LV) dysfunction after mitral valve repair (1).

First, as Essop points out, echocardiographic predictors of a poor outcome in our study might be similar to those previously reported for mitral valve replacement. However, to compare mitral valve repair with mitral valve replacement, we must interpret our findings carefully because our study was not designed so. Some previous data suggest that LV function is preserved better after mitral valve repair than after mitral valve replacement (2,3). Further studies are needed to compare their echocardiographic predictors of a poor outcome. And even if echocardiographic predictors are similar, we believe that mitral valve repair is the procedure of choice that brings the better quality of life whenever a complete repair is expected.

Second, we must admit that only two factors, the preoperative ejection fraction and the LV end-systolic diameter, cannot fully explain which patients will have the better postoperative ejection fraction and which patients will not. Our results showed that the LV ejection fraction decreased significantly after mitral valve repair as a whole, but that in some patients the postoperative ejection fraction can be higher than the preoperative one. In the study by Lessana et al. (4) that Essop refers to, 26 patients underwent catheterization 2 to 60 months after mitral valvuloplasty (average 14 months). Mean ejection fraction decreased from 0.58 ± 0.11 to 0.52 ± 0.11. Ejection fraction in individual patients rose in 8 and fell in 18. In the other study by Bonchek et al. (5), 10 patients were studied with catheterization one week postoperatively. Mean ejection fraction was unchanged (0.66 ± 0.1 vs. 0.62 ± 0.1). Ejection fraction in individual patients rose in three, was unchanged in one, and fell in six. Although their shorter follow-up periods and our use of recently developed surgical techniques may preclude simple comparison between their results and ours, our results are at least not inconsistent with their results (5).

The improved ejection fraction in some patients can be considered to reflect the increased LV contractility as stated in our report. Stading et al. (6) showed that contractile function is impaired in some patients with long-term mitral regurgitation and a normal