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
ejection function, and that impaired contractile function may not be irreversible in all these patients. As Essop mentions, Wisenbaugh (7) reported that, among patients with mitral regurgitation, 10 of 14 with ejection fraction <0.60 and 4 of 13 with ejection fraction >0.60 had muscular dysfunction and that others did not. His results and ours clearly show that LV dysfunction cannot be predicted with ejection fraction alone. It may be important to us to acknowledge that ejection fraction decreases after surgery in general, but that in some patients it can increase owing to improved contractility.

Finally, among 171 patients in our analysis of LV dysfunction, 65 patients had moderate mitral regurgitation, and 106 patients had severe mitral regurgitation.

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REFERENCES


Composite Confusion

In their retrospective post hoc analysis of the CADILLAC study, Dr. Cox et al. (1) state that the one-year composite adverse event rate (death, reinfarction, disabling stroke, or target vessel revascularization [TVR]) was greater after optimal percutaneous transluminal coronary angioplasty (PTCA) than routine stenting (21.9% vs. 13.8%, p = 0.001) and that this was driven largely by increased rates of ischemic TVR (19.1% vs. 9.1%, p = 0.001). As a result, they conclude that early and late outcomes can be further improved with routine stent implantation.

The conclusion seems excessive given results glossed over as simply nonsignificant. Perhaps what the investigators might have stated was that the difference in the composite outcome was driven entirely by the least clinically relevant and softest outcome event, namely TVR. Indeed, as shown in Table 3 of their study, the risks of death, recurrent myocardial infarction (MI), and disabling stroke were all higher (but not statistically significantly so) with stenting at 30 days. Moreover, at one year the relative increase in the risk of death with stenting was 31.3% and the absolute increase was 1.0%; this is about the same as the benefit of tissue plasminogen activator over streptokinase and far larger than any putative benefit of glycoprotein Ilb/Ilia inhibitors in acute coronary syndromes. I cannot help but believe that had the estimates for the individual outcome events been reversed (that is, favoring stenting) readers would have seen phrases such as “strong trends” or “lack of statistical power” as an explanation. This worrisome increase in clinically important outcome events, also seen in other studies of stents in MI, should not be dismissed as just play of chance or as not significant. I would have thought it might merit a line or two in the discussion.

Could the investigators please provide readers with the differences between stent and optimal PTCA groups for: 1) death and disabling stroke, and 2) death, disabling stroke, and recurrent MI at 30 days and at 1 year?

Finally, should we not discard this silly notion of composite endpoints that equate a death and a disabling stroke as equivalent to a recurrent revascularization procedure? Is that how our patients view these events? Interpretive difficulties are sure to arise in problematic reports, such as the study by Cox et al. (1), where elements of the composite go in opposite directions (the treatment reduces TVR, but death, disabling stroke, and recurrent MI may be increased) (2). Careful wordsmithing often gives the illusion that all elements of the composite end point are favorably affected (3).

In the absence of a consensus-weighting scheme for elements of a composite, perhaps we need a hierarchical nomenclature for composites that make the results more transparent, particularly in the published abstract. For example, one could state that the composite was significantly lower among patients randomized to stenting (21.9% vs. 13.8%, p = 0.001, death [inc-ns], disabling stroke [inc-ns], re-MI [nd-ns], TVR [dec-sig]). All elements of the composite are reported, and they are ranked starting with death followed by those of lesser clinical importance. Those elements deemed to be hard and objective are capitalized, whereas those that are more subjective or clinician-driven are given in lowercase. It is specified whether the point estimate is in keeping with an increase (inc), decrease (dec), or no difference (nd); and whether there is conventional statistical significance (sig) or not (ns).