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The surgical replacement of diseased heart valves is based on the premise that the prosthesis chosen to replace a stenotic or insufficient valve will have a beneficial impact on the patient’s heart function, taking into account the risks of surgery and the recognized complications of prosthetic heart valves. It is well established that mitral valve replacement (MVR) is associated with higher short- and long-term mortality than mitral valve repair.1-2 Although the exact mechanism for this discrepancy is unknown, it increases the importance of repairing valves whenever possible and, when MVR is necessary, intervening to prevent prosthesis- and/or patient-related factors that may lead to less favorable postoperative outcomes.

A growing literature has identified aortic valve patient-prosthesis mismatch (PPM) after aortic valve replacement. Studies have suggested that aortic PPM may be accompanied by lower rates and absolute degree of regression of left ventricular hypertrophy. Some authors have linked incomplete regression of left ventricular hypertrophy to more postoperative cardiac events and worse long-term survival.3-7 This linkage is controversial, however; other authors suggest that PPM is of less importance after aortic valve replacement and indeed that valve size may not matter.8 Many fewer data are available regarding PPM after MVR.

In this issue of Circulation, Magne et al9 investigate the impact of PPM on survival after MVR. The authors hypothesize that the effective orifice area (EOA) of prosthetic valves is often small relative to the patient’s size, thus causing a mismatch between valve EOA and flow across the valve. As a result, seemingly normally functioning mitral prostheses often have relatively high transvalvular gradients. These gradients mimic those found in patients with mitral stenosis. In addition to delaying the regression of left atrial and pulmonary artery hypertension, these high gradients and the resulting high left atrial pressures may lead to atrial dilation and subsequent atrial fibrillation. This same group recently published a study demonstrating that mitral valve PPM, defined as an indexed EOA of ≤1.2 cm²/m², is a frequent occurrence after MVR, occurring in up to 70% of patients, and is associated with persistent pulmonary hypertension.10 Given the deleterious effects of pulmonary hypertension on right heart function and the associated risks of morbidity and mortality, the authors further hypothesized that PPM after MVR might increase the risk of mortality.

The report from Magne and colleagues is a single-institution study of 929 patients undergoing MVR over a 20-year period. Patients requiring other valve surgery were excluded, but those requiring coronary bypass grafting and those with previous coronary bypass grafting and/or mitral valve surgery were not excluded. Patients were divided into 3 groups based on projected EOA, indexed for body surface area. Indexed EOA >1.2 cm²/m² was defined as not clinically significant, between 0.9 and 1.2 cm²/m² as moderate PPM, and ≤0.9 cm²/m² as severe PPM. In this study, the projected indexed EOA was derived from the published normal in vivo EOA values for each valve model and size implanted in this patient cohort. Three valves used in this study do not have published EOA values. For these cases, the authors used EOA values determined by echocardiography performed 1 year after operation. With the above criteria, of the 929 patients in this cohort, 204 (22%) had nonsignificant PPM, 644 (69%) had moderate PPM, and 81 (9%) had severe PPM.

The end point for this study was survival from the time of MVR. With a mean follow-up of 6.3±4.5 years, in multivariate analysis, patients with severe PPM had worse survival after MVR at 6 and 12 years than patients with moderate PPM (P=0.027) and patients with nonsignificant PPM (P=0.002). A suggestion existed that patients with moderate PPM also had worse survival at 6 and 12 years than those with nonsignificant PPM; however, this effect did not persist in multivariate analysis. The presence of severe PPM proved to be an independent predictor of mortality after MVR in the series as a whole and in the subset of patients undergoing isolated MVR (ie, those with no significant coronary artery disease).

The authors investigated geometric orifice area as well as labeled prosthesis size in this patient cohort, and found that there was no association of either of these parameters with higher mortality. They conclude that the indexed EOA is the only valid metric suitable to describe PPM after MVR.

From a surgeon’s perspective, this study exhibits a number of somewhat perplexing features. First, it is important to note that nearly 50% of patients in this series had mitral stenosis caused by rheumatic or calcific disease.
This is certainly not the current experience with patients requiring MVR. Patients with rheumatic and calcific mitral stenosis, however, often do have a small annulus and other features such as severe calcification that make the implantation of an adequately sized prosthesis difficult. Furthermore, 50% of the patients in this series had mitral regurgitation and received MVR. This is also not representative of the current practice of mitral valve surgery; the majority of these patients would currently undergo mitral valve repair. In addition, the distribution of valve types and sizes is of note. Eighty-one percent of patients in this series received mechanical prostheses. Because mechanical prostheses often have better hemodynamics compared with tissue valves of a similar size, it is concerning that, despite a high preponderance of mechanical valve implantation in this series, such a high incidence of PPM still existed. It is also of interest that 47% of patients received a prosthesis that was ≤27 mm, and 15.5% received a prosthesis that was ≥25 mm. Therefore, it should not be surprising, given the fact that nearly 50% of patients in this study received valves that were ≤27 mm, that there should be a high residual gradient after surgery. Finally, compared with patients with nonsignificant PPM, patients with moderate and especially those with severe PPM had longer aortic cross-clamp times (P = 0.001). Plus, patients with higher degrees of PPM had significantly more concomitant coronary bypass graft procedures performed. The combination of longer cross-clamp times and more complex procedures such as coronary revascularization somewhat clouds the picture with respect to elucidating the impact of PPM on postoperative survival in this patient series.

Prior studies have demonstrated that PPM after aortic valve replacement is associated with worse hemodynamics, less regression of left ventricular hypertrophy, more cardiac events, and higher mortality rates. PPM after MVR was first described by Rahimtoola and Murphy11 in 1981. This is the first study to demonstrate that, indeed, severe PPM is associated with worse survival after MVR. Furthermore, even moderate PPM demonstrates a strong trend to worse survival compared with nonsignificant PPM after MVR. Considering that moderate PPM was found in 69% of patients in this series and severe PPM in 9%, the overall importance of this observation is obvious.

This study has limitations. It is retrospective in nature and subject to the vagaries of retrospective observational studies. The large number of mechanical valves used and the relatively small proportion of valves repaired in this series are of concern. The preponderance of small prostheses in this report, namely ≤27 mm, is of concern and doubtless contributes to the high incidence of PPM observed. The vast majority of patients (78%) in the study had chordal preservation, as is now the widespread practice, and no significant difference existed between groups in this regard. As with PPM after aortic valve replacement, the importance of this phenomenon after MVR is controversial. Ruel et al12 published a report examining the predictors of heart failure after MVR. They found that postoperative heart failure is predicted by preoperative New York Heart Association class and left ventricular grade, both of which have long been recognized as primary determinants of outcome after MVR. Atrial fibrillation, coronary artery disease, and other factors, such as smoking, persistent tricuspid regurgitation, and redo status, also were associated with a higher incidence of heart failure. However, prosthesis size and elevated transprosthesis gradient, which are surrogates for PPM, were not predictive of freedom from heart failure after MVR.13 The discrepancy between this study and the report by Magne et al9 suggests that the mechanism for poor survival after MVR is multifactorial and incorporates features other than just the EOA of the particular valve that is implanted.

As in the situation with PPM in the aortic position, Magne et al9 recommend prospective strategies to attempt to avoid PPM in the mitral position. An obvious approach would be to increase the prevalence of mitral valve repair in this population. With currently established and proven, durable techniques, repair would largely eliminate PPM in this cohort of patients. If replacement is necessary, strategies to avoid PPM are definitely warranted. One should attempt to implant a prosthesis with as large an EOA as possible for a given annulus size, within reason. Oversizing the prosthesis in MVR is also fraught with difficulties and potentially serious complications such as disruption of the atrioventricular groove. The information contained in this study should serve as a stimulus to the development of better-performing mitral replacement prostheses. It should also serve as a stimulus to redouble the educational efforts necessary to increase the prevalence of mitral valve repair whenever possible. Finally, some things in surgery remain obvious. A relatively small, inactive, elderly individual can get by with a small prosthesis in either the aortic or mitral position. A larger, younger, more active individual requires measures to implant a significantly larger prosthesis. Further studies are indicated to allow a better understanding of the phenomenon of PPM after MVR. However, this noteworthy study points out that we, as cardiac surgeons, should approach mitral valve surgery with the following goals in mind: (1) increase the prevalence of mitral valve repair in appropriate candidates, (2) calculate the indexed EOA for each patient requiring MVR and use strategies to avoid PPM whenever feasible, and (3) increase awareness that we potentially compromise patient survival by creating significant PPM at the time of MVR.

Disclosures

None.

References


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