

**Residual pulmonary artery hypertension after mitral valve replacement: Size matters!**

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## EDITORIAL COMMENT

# Residual Pulmonary Artery Hypertension After Mitral Valve Replacement Size Matters!\*

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It has been previously observed that the rate and extent of regression of preoperative pulmonary artery hypertension is highly variable after mitral valve surgery and that residual pulmonary artery hypertension may be a risk factor for poor outcomes after surgery. In this issue of the *Journal*, Li et al. (1) hypothesized that valve prosthesis-patient mismatch (PPM) might be an important factor in preventing the regression of pulmonary artery hypertension after mitral valve replacement (MVR). They retrospectively analyzed data from 56 patients who had previously undergone MVR and who were evaluated by Doppler echocardiography at varying time intervals postoperatively. Patients with prosthetic valve dysfunction and significant aortic valve disease were excluded, but no mention was made of the status of left ventricular function pre- or postoperatively in the study patients. Patient-prosthesis mismatch was defined as in-

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dexed effective orifice area (EOA)  $\leq 1.2 \text{ cm}^2/\text{m}^2$  and pulmonary artery hypertension as systolic pulmonary artery pressure  $>40 \text{ mm Hg}$ . Many different types of prosthetic valves (overall 84% mechanical, 16% tissue) were utilized, and prosthetic valve selection criteria were not defined. The authors found that average postoperative systolic pulmonary artery pressure was significantly higher in patients with PPM compared to patients with no PPM ( $46 \pm 8 \text{ mm Hg}$  vs.  $34 \pm 8 \text{ mm Hg}$ ;  $p < 0.001$ ), and that the prevalence of persistent pulmonary artery hypertension after MVR was greater in patients with PPM than in those with no PPM (68% vs. 19%). The indexed EOA was by far the strongest predictor of systolic pulmonary artery pressure. They concluded that PPM can best be prevented by implanting the largest possible prosthesis with the greatest EOA and recommended that a "simple" prospective strategy of utilizing the largest bileaflet mechanical mitral valve prosthesis

might be most effective in avoiding PPM and residual postoperative pulmonary artery hypertension.

A relatively large body of information has been developed regarding PPM after aortic valve replacement. Various studies have suggested that aortic PPM resulting in a residual transprosthetic gradient might be associated with slower and less regression of left ventricular hypertrophy postoperatively. Some studies have indicated that incomplete regression of left ventricular hypertrophy is related to more postoperative cardiac events and poorer long-term survival (2) while others have suggested that it is of less importance (3,4). Much less data has accumulated regarding PPM after MVR. The original patient reported by Rahimtoola and Murphy (5) may in fact not represent mitral PPM at all.

While the findings in this study are interesting, there are significant limitations, some of which are acknowledged by the authors. The study is retrospective, and while the study patients are "consecutive" in regards to *evaluation* of patients presenting to the clinic for follow-up, it did not represent a consecutive contemporary series of patients undergoing MVR. The patients were evaluated at intervals ranging from 8 to 102 months postoperatively, and, in fact, there was a significant correlation between the time to follow-up and postoperative pulmonary artery pressure by univariate analysis. The study would be stronger if follow-up intervals (time of evaluation) were similar in all patients because pulmonary artery hypertension does not always regress immediately after operation. Despite the known correlation between pulmonary artery pressure obtained by Doppler echocardiography and that obtained by catheterization, it would have been nice to have the Doppler data confirmed by catheterization (at least in some patients) and pulmonary vascular resistance measured because increased pulmonary vascular resistance may be an independent cause of residual pulmonary artery hypertension despite satisfactory relief of mitral stenosis or mitral regurgitation by surgery.

Several things about this study stand out immediately, or at least to the surgeon. One is the large number (52%) of relatively small prostheses (size 25 and 27) that were utilized. It should not be too surprising, therefore, that a significant number of patients in the series had residual pulmonary artery hypertension given the fact that the majority had small prosthetic valves utilized. While many different types of prosthetic valves were utilized in this study, it is well known that size 25 and 27 prostheses may be associated with residual gradients due to relatively small EOA. When prosthetic valves of this size are utilized in adult patients, especially those with  $>1.8 \text{ m}^2$  body surface area, one is, in effect, replacing a stenotic mitral valve with a stenotic prosthetic valve, and residual pulmonary artery hypertension should be expected rather than surprising. In our most recent 100 consecutive adult patients undergoing MVR (all types of prostheses), only 35% required mitral valve prostheses of size 27 or less compared to the 52% in

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this series. In a recent follow-up of all patients undergoing MVR with *only* St. Jude Medical prostheses (used in 67% of the patients in the Li et al. [1] study), only 16% were size 25 or 27 (6). The manuscript does not provide selection criteria for valve type nor does it explain why such small valves were utilized in so many patients. This does, however, probably explain the unexpectedly high rate of residual pulmonary hypertension, and, accordingly, their recommendation to utilize the largest possible mitral valve prosthesis (within reason) is appropriate.

A second surprising finding is that the chordal apparatus was preserved in only 41% of the patients in this series. Unless an extremely high percentage of the patients had underlying rheumatic disease, one would expect the percentage of chordal preservation (either total or partial) to approach 100% given the knowledge that failure to do so may be a cause of postoperative left ventricular dysfunction. In fact, no real mention is made of either pre- or postoperative left ventricular function, and left ventricular dysfunction of any type could play a role in residual pulmonary artery hypertension.

The final thing that stands out is the very high rate of utilization of mechanical prostheses (84%), especially in a patient population with a mean age of 65 years. Currently, the trend is toward a greater utilization of tissue valves as opposed to mechanical valves, and in our most recent 100 MVRs, only 28% received mechanical prostheses (compared to 84% in this series). In addition, the authors go on to recommend that PPM and residual pulmonary artery hypertension might be prevented by utilization of the largest bileaflet mechanical valve prosthesis in all patients because of the superior hemodynamics associated with these valves. In their series, there was no difference in the rate of utilization of mechanical valves in patients with PPM (88%) versus those without PPM (83%). Because 84% of the patients in the entire series received mechanical valves, only an additional 16% could have possibly benefitted from their recommendation, and far more than that had residual pulmonary hypertension. Mechanical valves require long-term anticoagulation and are clearly contraindicated in patients with bleeding disorders, in most elderly patients, and women of childbearing age, as well as in some other patients. Thus, the authors' recommendations for more widespread utilization of mechanical bileaflet valves are simply not an option for all patients despite possible hemodynamic advantages.

The authors have made an important observation that residual pulmonary hypertension after MVR is possibly

related to PPM. However, they have not shown that this relatively mild degree of residual pulmonary hypertension has a significant effect on long-term survival or functional capacity. Nevertheless, all cardiothoracic surgeons should be aware of the implications of their observation and should attempt to implant the largest prosthesis that fits comfortably at the time of MVR. However, no attempt should be made to "oversize" the valve, as this could lead to potential complications.

Perhaps, most importantly, the authors' observation indirectly reinforces the superiority of mitral valve *repair* over MVR with any size or type of prosthesis because of the well-documented improved hemodynamics, decreased rate of thromboembolism, and superiority of long-term outcomes associated with mitral valve repair (7,8). Mitral valve repair as opposed to replacement in as many patients as possible would perhaps be the most important recommendation of this study in order to avoid PPM and residual pulmonary artery hypertension. In order to document this hypothesis, it would be interesting to repeat the study in a series of patients who have undergone mitral valve repair (as opposed to replacement) in order to compare the rates of residual pulmonary hypertension.

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