Left ventricular mass regression after aortic valve replacement: Does size really matter?

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Since the initial conceptual description of prosthesis-patient mismatch by Rahimtoola¹ in 1978, many studies have been published that have substantiated or called into question its clinical relevance among patients undergoing aortic valve replacement. Most of these studies have narrowly focused on seeking correlations among transprosthetic gradients, indexed effective orifice area, left ventricular (LV) mass regression, and postoperative survival. Conclusions have been inconsistent,²⁻⁶ suggesting that the determinants of survival after aortic valve replacement may be more complex than simple replacement of a stenotic native valve with a prosthetic that is often just less stenotic. With this notion as a backdrop, Helder and colleagues' broader analysis of a wider array of factors that might affect LV mass regression prompts provocative questions pertaining to the relative impact of aortic valve replacement on survival, and the significant effects adjunctive therapeutics might exert.

The authors quite reasonably analyzed each factor in the context of its effect on LV mass regression based on the well-established premise that LV hypertrophy (LVH) negatively affects postoperative survival after aortic valve replacement.⁷⁻⁹ Of most interest in this study were the findings that treatment with beta-blockers or calcium-channel blockers at discharge was independently associated with an increased probability of complete LV mass regression, whereas prosthesis-patient mismatch was not. These findings, coupled with the observation that there was no difference in 5-year survival rates or New York Heart Association functional status among patients with complete versus incomplete LV mass regression, suggest that the absolute extent of LVH reduction after aortic valve replacement may not affect survival rates or symptomatic status among patients undergoing aortic valve replacement as much as a physiologically meaningful and sustained reduction in ventricular afterload and wall stress afforded by the

operation and the institution of beta-blockade and/or calcium-channel blockers. The authors' concomitant analyses of LV mass regression as a dichotomous (ie, complete vs incomplete) and continuous variable may have uncovered this more relevant clinical conclusion.

Certainly, the benefits of adjunctive medical therapies to cardiac operations have precedent, with the established positive impact of beta-blockade, statins, and aspirin on survival after coronary artery bypass grafting,¹⁰ and anti-impulse therapy after thoracic aortic aneurysm repair.¹¹ It would be interesting to determine whether adjunctive medical therapies after aortic valve replacement mitigate, or even eliminate, any negative impact of prosthesis-patient mismatch on LV remodeling.

The validity of this argument is somewhat muted, however, by the authors' finding of no differences in mean systolic pressure at follow-up in patients who had complete versus incomplete LV mass regression. Furthermore, the lack of information on compliance, duration, and dosing of beta- and calcium-channel blockers prescribed upon discharge render the true impact of these adjunctive therapies unclear. It is curious that prosthesis-patient mismatch did not affect LV mass regression or survival in this study, as such an effect has been observed by others.^{3,6} However, Ruel and colleagues,⁵ and Mohty and colleagues,⁴ similarly noted that prosthesis-patient mismatch did not have an impact on LV mass regression, freedom from heart failure symptoms or death, or overall late survival among patients with normal preoperative LV function; it seems that most of the patients in this study had good LV function. Furthermore, purported improved hemodynamics of the Trifecta valve, compared with other stented bioprostheses used in previous studies, resulted in relatively few patients with severe prosthesis-patient mismatch in this study cohort.

The authors rightly noted the inherent limitations of this multi-institutional study, which was further handicapped by limited longitudinal follow-up, owing to the fact that the Trifecta valve is a relatively new product. Nevertheless, the conclusions of this study raise interesting questions pertaining to the relative contributions of systemic hypertension, transvalvular gradients, and effective orifice area to LV mass regression in patients undergoing aortic valve replacement. Further studies with more-rigid postoperative beta- and calcium-channel-blocker compliance, and protocolized amelioration of postoperative systemic hypertension in patients undergoing aortic valve replacement, would help clarify the impact of these adjunctive medical therapies on survival and regression of LVH. Furthermore,

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computational fluid-dynamic analyses comparing various stented bioprosthetic valves might further refine our understanding of the effects of various stent designs on LV energetics. Certainly, such understanding would seem to be of increasing import, given the newer aortic bioprosthesis designs and the advent of transcatheter aortic valve replacement and valve-in-valve strategies.

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