LEFT VENTRICULAR FUNCTION AFTER MITRAL SURGERY: TIME TO FOCUS ON INTRAOPERATIVE MANAGEMENT?

To the Editor:

We read with interest the article by Quintana and colleagues¹ and a letter to the Editor by Buckberg and Athanasuleas² about this article published in the *Journal* in December 2014 and March 2015, respectively. We have some observations for the authors.

We focused our attention on Table 2, “Comparison of Postoperative Clinical Course Stratified by Early Predissmissal LVEF,” in the article by Quintana and colleagues.¹ In the footnotes of the table, it is clear that the data are presented as median (interquartile range) or n (%), but looking carefully at the line “Need for inotropic support,” we found 2 mistakes: If 136 patients in the first group of 1391 (ejection fraction [EF] ≥ 50) required inotropic support, they do not represent 28% but 9.77%. The same applies to the second group (EF ≤ 50) of 50 patients of 314. They do not represent 37.3% but 15.9%. This error is reported in the results: “As expected, patients with early dysfunction required more frequently postoperative inotropic support (28% vs 37.3%, $P = .04$),” but in the light of the wrong percentage calculation, the P value may be incorrect.

The second observation is related to the need of intra-aortic balloon pump (IABP). The authors report the same incidence of IABP use in the 2 groups. This event could be explained assuming that patients in the first group (EF $> 50\%$) have had a transitory left ventricular dysfunction (stunning) requiring IABP support, which quickly resolved without leaving any sign at predissmissal echocardiographic evaluation. This hypothesis strongly strengthens the suspicion of an inadequate intraoperative protection in these groups of patients.² Finally, the authors give no definition for “Need for inotropic support,” especially regarding the duration (>6 hours, >12 hours, >24 hours).

These data support the need to expand our research not only in the preoperative evaluation of patients with mitral

insufficiency but also in their intraoperative management with the objective to limit as much as possible the intraoperative damage related to cardiac arrest.

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CHANGES IN LEFT VENTRICULAR VOLUME AND DECLINE IN EJECTION FRACTION AFTER DEGENERATIVE MITRAL VALVE REPAIR: THE MATH MAKES SENSE

Reply to the Editor:

We appreciate the interest shown in our work¹ by the authors from Ancona. In regard to their first point, we confirm that the numbers presented in our article are correct. Documentation of the use of inotropic support was available in only 619 patients because of changes in recording methodology over time. Among these individuals, 433 received inotropic support and 186 did not. The presented percentages, P values, and conclusions in the article are all accurate. Further, the frequency of intra-aortic balloon counterpulsation use was similar between groups with and without postoperative dysfunction, as documented.

The suggestion that a decrease in ejection fraction relates to the adequacy of myocardial protection reflects a commonly held misunderstanding regarding changes in ventricular volumes and left ventricular function immediately after correction of valvular regurgitation.² Consider, for instance, a minimally symptomatic patient with an ejection fraction of 65% and severe mitral valve regurgitation. If one assumes that the regurgitant volume is 80 mL and that the patient has normal preoperative cardiac output with a forward stroke volume of 70 mL, the preoperative left ventricular end-diastolic volume would necessarily be

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approximately 230 mL. We know that end-diastolic volume changes little immediately after mitral valve repair. Thus, if Capestro and associates speculate that ideal myocardial protection should result in a “normal” ejection fraction, then a patient with severe mitral valve regurgitation undergoing repair would have a forward stroke volume of 150 mL after correction, producing an unphysiologic supranormal cardiac output (>10 L/min), which of course, does not occur. The typical early physiologic adjustment to having a large end-diastolic volume after elimination of mitral regurgitation is a compensatory increase in end-systolic volume to maintain a normal or only slightly greater than normal stroke volume, translating into a decline in ejection fraction. In most patients, the decrease in ejection fraction early after correction of valvular regurgitation thus should be thought of as a compensatory mechanism and not a reflection of myocardial injury. This is discussed in detail in the clinical study by Ashikhmina and colleagues.³

Reverse remodeling of the ventricle subsequently occurs over time after successful correction of severe degenerative mitral regurgitation. As end-diastolic volume regresses, ejection fraction increases, but the extent of recovery is highly variable, often incomplete,⁴ and difficult to accurately predict. As documented in our article, persistent left ventricular dysfunction may occur even in patients who have a “normal” ejection fraction before mitral valve repair. To minimize the occurrence of this event and optimize outcomes of operation, valve repair should be undertaken before the development of excessive ventricular enlargement and pulmonary hypertension.

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AORTIC VALVE REPLACEMENT AND THE RIGHT VENTRICLE—THE PLOT THICKENS

To the Editor:

We read with interest the recent article by Crouch and colleagues¹ describing the early effects of surgical and transcatheter aortic valve replacement on right ventricular (RV) function. The authors describe a fall in right ventricular ejection fraction (RVEF) assessed using cardiovascular magnetic resonance imaging—the gold standard technique for RV assessment—early following transcatheter aortic valve replacement (TAVR), but a relative preservation of RVEF following surgical aortic valve replacement (SAVR). We welcome the addition of further insight into RV reverse remodeling following TAVR and SAVR given the importance of RV function in prognosis following valve replacement.

The RV response to aortic valve replacement has not been well described and observations are limited to a small number of studies. In a similarly sized study published by our group,² patients undergoing SAVR and TAVR had similar baseline RV size and ejection fraction. Our 6-month postprocedure cardiovascular magnetic resonance imaging study² found that there was a trend toward an increase in RVEF in the TAVR group and a significant reduction in RVEF in the SAVR group. The cardiopulmonary bypass time and crossclamp times were longer in our study than that of Crouch and colleagues,¹ which may have accounted for the different findings.

An echocardiography study by Kempny and colleagues³ supports the findings of our group. Those researchers studied 123 patients undergoing TAVR and SAVR and found a significant deterioration in longitudinal and radial RV function in those undergoing SAVR. There was no correlation between decline in RVEF and aortic crossclamp or cardiopulmonary bypass time. No deterioration of any parameter of RV function was seen following TAVR. Crouch and colleagues¹ suggest that aortic regurgitation may be the mechanism for the deterioration in RVEF observed in the TAVR group. Although this is feasible, the postprocedure level of aortic regurgitation was still overall reduced compared with the preprocedure scan. Furthermore, the lack of an association in the study by Crouch and colleagues¹ between left atrial size (a surrogate marker for elevated left atrial pressure) and RVEF seems to make this mechanism less plausible. The patients described in their article had 3 to 4 bouts of rapid pacing, which is greater than that performed at our institution. The hypotension associated with the rapid pacing may induce a global myocardial injury with a resultant impairment of RV function. Mechanisms that may be implicated in RV