To the Editor:

We read with interest the article by Quintana and colleagues and a letter to the Editor by Buckberg and Athanasuleas 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 Predismissal LVEF,” in the article by Quintana and colleagues. 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 intraaortic 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 predismissal echocardiographic evaluation. This hypothesis strongly strengthens the suspicion of an inadequate intraoperative protection in these groups of patients. 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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REFERENCES

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Reply to the Editor:

We appreciate the interest shown in our work 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 intraaortic 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. 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.

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, 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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References

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