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

dysfunction following SAVR include cardioplegia technique, pericardiotomy (leading to a loss of pericardial support to the RV), hypothermia, and immune-mediated inflammation.³ It is not clear from the methods described by Crouch and colleagues¹ whether there were important differences between the studies in the surgical techniques employed that may account for the differing outcomes. Further, larger studies with more detailed methodology are required to further explore the relationship between SAVR/TAVR and RV function. It is important that the true effect of SAVR and TAVR on RV function is established to inform referral practices.

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RIGHT VENTRICULAR FUNCTION AFTER AORTIC VALVE INTERVENTION: CARDIOVASCULAR MAGNETIC RESONANCE IMAGING IS THE STANDARD

Reply to the Editor:

We thank Dobson and colleagues for their commentary of our recent article.¹ We acknowledge the differences in outcomes regarding right ventricular function between our study and those from Fairbairn and coworkers² and Kempny and associates.³ In the Discussion section of our article, we offered some insights into the potential reasons for these differences and note the review of these by Dobson and colleagues.

Primarily, there are several key differences in study methodology that potentially account for the differences in outcome. Firstly, the timing of postoperative cardiovascular magnetic resonance imaging is significantly different between ours and the studies quoted by Dobson and colleagues. To the best of

our knowledge, our study is the only study to scan patients with cardiovascular magnetic resonance imaging early after the procedure (<14 postoperative days) and thus to have a clear opportunity to look at periprocedural myocardial stunning of both the left and right ventricles. Fairbairn and coworkers² and Kempny and associates³ both looked at right ventricular changes at 6 months, when stunning effects would have dissipated and remodeling changes would predominate. We are currently analyzing our 12-month data to elucidate this later situation in our cohort, and we await the results of this with interest.

Second, a majority of studies (including that of Kempny and associates³) examining periprocedural myocardial function in this setting have used transthoracic echocardiography. As discussed in our article, transthoracic echocardiography has well-documented limitations, especially in the postoperative setting and in the assessment of right ventricular function.

Third, the prospective design and matched patient selection of our study differs from the retrospective and poorly matched euroSCOREs of Kempny and associates³ and the prospective but still poorly matched cohort of Fairbairn and coworkers.² The wide differences in operative risk between surgical aortic valve implantation and transcatheter aortic valve implantation (TAVI) cohorts highlights limitations in the study design that may have contributed to differing outcomes.

In addition, the inclusion of patients undergoing mechanical as well as bioprosthetic valve replacement, the inclusion of those undergoing either concomitant coronary bypass surgery or aortic surgery, and the inclusion of mixed transcatheter prosthesis types are all potential causes of differences in ventricular function after the procedure. The loss to follow-up of 50% of patients in the TAVI cohort in the study by Fairbairn and coworkers² also offers a potential explanation for the observed differences in outcome.

The mechanism of postprocedure paravalvular regurgitation leading to right ventricular strain and dysfunction that we offered was somewhat supported by the correlation between increased paravalvular regurgitation and left atrial volume. We acknowledge there was not a correlation with left atrial volume and right ventricular dysfunction; however, this may have reflected the small cohort size.

We would refute the suggestion that paravalvular regurgitation was not a potential mechanism for the right ventricular dysfunction on the basis of changes in overall regurgitant fraction. First, there was not a significant change in mean regurgitant fraction from before to after procedure in the TAVI group (18% to 16%), which is actually a dramatically poor outcome highlighted by the reduction in the AVR group (14% to 4%). The pressure and volume state of the hypertrophic and “stiff” left ventricle after the procedure is obviously different from the state before the procedure. The introduction of TAVI has essentially created a new pathology of acute aortic regurgitation in stiff, noncompliant left ventricles, which needs further investigation.