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.

We agree with Dobson and colleagues that variations in procedural technique may also be a potential mechanism for variations in study outcomes. Cardioplegia types and techniques, temperature management, and closure of the pericardium may all effect postprocedure RV function. We agree that larger clinical outcome and imaging trials are needed in matched population groups, with careful delineation of the TAVI and surgical techniques and the use of state-of-the-art imaging (such as cardiovascular magnetic resonance imaging) for investigation of myocardial functional and structural effects as well as aortic valve hemodynamics.

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CEREBRAL INFARCTION OCCURS PARTICULARLY IN PATIENTS WITH PORCELAIN AORTA To the Editor:

Porcelain aorta is still an overwhelming hurdle to safely completing aortic valve replacement (AVR).

Recently, Idrees and colleagues¹ reported the outcomes of surgical or transcatheter AVR for patients in whom the operation had been abandoned due to porcelain aorta. Among the 7 surgical cases, the aorta could not be clamped, and the ascending aorta was replaced under deep hypothermic circulatory arrest in 4 patients, whereas the aortic clamp was placed at the less calcified portion in the 3 other patients. None of the patients

FIGURE 1. Double-layered calcification (arrow).

developed complications related to calcification of the aorta, such as embolic stroke, and the authors concluded that surgery for such patients is safe, as in the transcatheter cases.

Although they should be congratulated for their excellent results, we would like to raise a question about their conclusion. Among the 201 patients who underwent AVR for aortic stenosis from 2002–2014 in our institution, aortic cannulation and/or clamping of the aorta was avoided due to severe calcification in 16 patients (8.0%). Cannulation into the axillary artery or the femoral artery or both for arterial return was used in all cases. Aortic clamping was abandoned in 3 cases. AVR was performed after replacement of the ascending aorta in 2 cases and completed under deep hypothermic circulatory arrest with antegrade cerebral perfusion in 1 case, but none of the patients experienced a stroke. In the remaining 13 cases, the ascending aorta was cautiously clamped. A clamping forceps with a curved jaw was applied in 2 cases where there was a higher risk of embolization to avoid injuring the calcified aortic wall. In another case, bilateral axillary arterial perfusion was started, and the left common carotid artery was clamped at the moment of aortic clamping to prevent debris flowing into the cerebral vessels. Among these 16 patients, symptomatic cerebral infarction occurred in 2 patients (12.5%), but asymptomatic infarction was found in 3 other patients on magnetic resonance imaging. We believe that embolic stroke may occur at a higher incidence in patients with a severely calcified aorta and cannot agree with the conclusion drawn by Idrees and colleagues¹ that AVR is safe even in such severely calcified cases. Although stroke was not encountered in the series described by Idrees and colleagues,¹ occult stroke might have been present. Even a small embolus could lead to serious sequelae, and no one can predict or control it.