Operative Risk Stratification and Predictors for Long-Term Outcome in Low-Gradient Aortic Stenosis

To the Editor:

We would like to congratulate Monin et al1 for their important multicenter study concerning risk stratification for valve replacement in low-gradient aortic stenosis using dobutamine echocardiography. It is possible that the lack of correlation in their study between the presence of previous myocardial infarction (MI) and perioperative mortality (a correlation that we did find in our study2) occurred because of a difference in our patient populations (the average ejection fraction of their patients was 31%, and that of our patients was 21%). In our patient population, 90% of the perioperative deaths occurred in those with a previous MI (mortality was 45% in those with prior MI, and only 3% in those without, despite very poor left ventricular (LV) function and relatively low aortic gradients). Apparently when LV damage is permanent (scar) and severe, replacing the aortic valve, with or without revascularization, does not result in recovery of LV function.

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Response

We appreciate the interest of Drs Tunick and Kronzon regarding our recent article in Circulation.1 They raise the issue of the lack of correlation between previous myocardial infarction (MI) and perioperative mortality in our study. This is in contrast to their own series, in which previous infarction was the only independent predictor for operative mortality.2 In the series of Powell et al,2 36% of patients had previous MI, mean left ventricular (LV) ejection fraction (LVEF) was 0.22, and 9 patients (16%) had severe mitral regurgitation (MR) that required surgery in 6 cases. In contrast, in our series,1 among 95 operated patients, only 20% had previous MI, mean LVEF was 0.30, and patients with significant MR were excluded. Thus, we agree that the higher prevalence of previous MI, with lower LVEF and (for some patients) severe MR in the series of Powell et al,2 might explain the different predictors for operative mortality found in the 2 studies. Furthermore, the possible interaction between LV contractile reserve and previous MI might also explain, in part, the predictive value of this latter parameter in the study by Powell et al.2 Our results show that among 19 patients with previous MI, 4 died within 30 days (21%), 3 of whom had no contractile reserve on dobutamine hemodynamics. Conversely, among the 76 patients without MI, there were 9 perioperative deaths (12%), 7 of whom were without reserve. We agree that a large scar from previous MI prevents postoperative recovery of LV function; furthermore, LV contractile reserve is unlikely to be present in such patients. Thus, a lack of contractile reserve and previous MI may be related in some cases, but not all, considering that extensive myocardial fibrosis due to longstanding hypertrophy may also explain the lack of reserve in some other patients. This possible interaction between contractile reserve and previous MI might have been found in the population studied by Powell et al2 if the issue of LV contractile reserve had been addressed in this study.

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