ings is that virtually all valved conduits except for cellular syngrafts resulted in significant sinus thrombus formation. This limitation of the model used could have been addressed in part by using a functional model in which blood flow and leaflet mobility can be demonstrated.[2] The significance of sinus thrombosis is not negligible, and it has been shown to become a nidus for inflammatory cells, potentially affecting neighboring cellular immune responses and any conclusions made from histologic examination.[3,4]

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### *Reply to the Editor:*

We thank Légaré and Ross for their comment on our article describing the in vivo behavior of decellularized rat aortic valve allografts. As they state in their letter, a complication of the model we used is the sinus thrombus formation observed in all decellularized synergrafts and allografts, as well as in the untreated allografts. They speculate that this is due mainly to a lack of leaflet mobility in a nonfunctional implantation model.[1]

To overcome this difficulty Légaré and Ross suggest the use of a functional rat implantation model in which the creation of native aortic valve insufficiency results in a significant diastolic flow reversal in the abdominal aorta, allowing leaflets to move and function.[2] Although we were aware of the existence of this model, we decided not to use it for our study. One reason is that the creation of aortic insufficiency results in progressive congestive heart failure from volume overload, making the model unsuitable for long-term follow-up studies. In addition, a rather high mortality is seen in the model (62.5% survival). We fully agree about the importance of having functional moving leaflets in the bloodstream in studying aortic valve failure; however, we look forward to new data that will encourage us to incorporate the model in our future studies.

We respectfully disagree with the assumption that a nonfunctional model is the main reason for the sinus thrombus formation. Interestingly, in our study none of the untreated (cellular) synergrafts displayed thrombus formation. This is consistent with earlier results in this model by Oei and colleagues,[1] who showed absence of thrombus formation in all synergrafts and even in some allografts. This indicates that loss of cellular structures, resulting in an absent blood-tissue barrier, from the treated synergrafts and allografts and untreated allografts is an essential condition in thrombus formation.

Our study suggests that decellularization reduces the recipient's immune response, resulting in a preservation of the extracellular matrix structure. We therefore believe in the feasibility of acellular allografts as scaffolds for tissue-engineering purposes and are performing seeding experiments at the moment to determine the best cell source.

Regarding the influence of thrombosis on the inflammatory response,[3] seeding of acellular valves with autologous endothelium will result in a new blood-tissue barrier and is expected to protect against thrombus formation. We therefore believe that this model is suitable for further research in tissue engineering of aortic valves.

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## Which is the best surgical strategy for "complete" treatment of ischemic cardiomyopathy?

### *To the Editor:*

We read with great interest the article published in the *Journal* by Maxey and associates[1] concerning surgery for ischemic cardiomyopathy. First of all, Maxey and associates are to be commended for outstanding results in a challenging cohort of patients. The study included 14 patients who underwent mitral valve repair (MVR) associated with coronary artery bypass grafting and left ventricular restoration (LVR), but Maxey and associates[1] did not mention the sequence of the surgical procedures. In a previous article, they reported performing coronary artery bypass grafting first, then MVR, and finally LVR.[2]

Ischemic mitral regurgitation is a functional disorder that is due mainly to changes in the geometric relationships between structurally normal valve components and a severely damaged ventricle. By changing ventricular volume and geometry, LVR may unpredictably result in mitral insufficiency that is absent before surgery[3] or in an amelioration (sometimes disappearance) of mitral regurgitation.[4] It therefore seems more rational to address MVR after completely rebuilding ventricular geometry. In addition, this strategy allows evaluation of valve competence after LVR and a check on leaflet apposition (in Bolling and associates' experience,[5] in addition to the annuloplasty ring 54 of 100 patients required further mitral repairs). Moreover, after LVR with narrower configuration and papillary muscles realignment, the mitral incompetence may disappear, thus making mitral valve repair unnecessary. We could speculate that in the only patient who showed persistent mitral