tioned as well but there was neither infiltration nor postoperative infusion. After the operation we assessed pain control by a visual analog scale at specific time points (extubation and 12, 24, and 36 hours). We also recorded the assisted ventilation time as well as the intravenous administrations of analgesic drugs. Blood gas analysis was performed at extubation and at 6, 12, and 24 postoperative hours. Unlike Dowling and associates' findings,¹ we were unable to demonstrate any effectiveness of this treatment, even though the drug and its dosage were similar to those used in their study. However, the protocol of these authors consisted in a T1-T12 intercostal nerve block, whereas we performed a local soft-tissue infiltration. We therefore may speculate that the efficacy of Dowling and associates' treatment was related more to the intercostal nerve block than to the postoperative continuous infusion, which was common to both studies. An interesting second step of their study would be the comparison between the sole intercostal nerve block and this procedure followed by continuous anesthetic infusion. Regardless, we may conclude that these attempts to reduce postoperative pain are promising and deserve future developments.

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doi:10.1016/j.jtcvs.2004.02.028

Reply to the Editor:

We are responding to the letter of Racigno and Carnelos. As they described, we performed a double-blind randomized controlled trial in which patients undergoing elective bypass surgery received either bilateral intercostal nerve block with ropivacaine or a saline placebo. At wound clo-

sure, two catheters with multiple side openings were inserted percutaneously and placed directly over the sternum. The same agent (ropivacaine or saline solution) was then administered as a continuous regional infusion for 48 hours through an elastomeric pump. The main findings of our study were that patients in the ropivacaine group had improved postoperative pain control and required less narcotic analgesia. Additionally, patients in the ropivacaine group had significantly lower pain scores than did patients who received placebo, and length of stay was also decreased in the ropivacaine group.

Racigno and Carnelos report doing a similar randomized trial in which they were unable to demonstrate any effectiveness of this treatment. In their letter, however, it is not clear how similar the therapeutic approaches truly were. We believe that a key component in our study was the development and clinical use of catheters with multiple side openings, which allow even distributions of the anesthetic agent throughout the operative field. Racigno and Carnelos reported using a small-diameter catheter, but it is not clear whether this catheter was effective in providing a true regional anesthetic. They further suggested that the difference in outcome may have been related to the use in our study of bilateral intercostal nerve block before performing wound closure.

We believe this is unlikely to explain the major differences seen between our two groups for a number of reasons. First, it is widely documented that intercostal nerve blocks, even with long-acting anesthetic agents, do not provide pain control beyond 6 to 8 hours. Second, we assessed pain scores on the second postoperative day to eliminate the effect of this and other confounding variables, such as residual analgesia given at the time of operative therapy. We were able to demonstrate marked decrease in narcotic use on the second postoperative day, when clearly the patient would not be receiving any benefit from the intercostal nerve block.

In addition, since the submission of our article a similar study has been performed that has demonstrated the advantages of a continuous local infusion for pain management after median sternotomy.¹ White and colleagues¹ performed a prospective, randomized, placebo-controlled trial with the same catheter system that was used in our

study. These investigators also showed a significant reduction in pain scores, morphine use, patient dissatisfaction, time to ambulation, and duration of hospital stay.

In our opinion, the results of our study and that of White and colleagues¹ strongly suggest that this approach will result in improved patient comfort and satisfaction, as well as decreased narcotic use after median sternotomy. As noted in both studies, the use of the catheter does not require any physician or nursing intervention after its placement, and there has been no morbidity associated with this approach. We consider this approach an excellent first step to improving the overall approach to pain management for our patients.

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Suggestion for functional model to test effects of decellularization of rat aortic valve allografts on leaflet destruction and extracellular matrix remodeling

To the Editor:

In their article "Decellularization of Rat Aortic Valve Allografts Reduces Leaflet Destruction and Extracellular Matrix Remodeling," Grauss and colleagues¹ elegantly evaluated various methods of decellularization of aortic valve conduits and tested their structural integrity after implantation in a rat model. Their findings suggest that decellularization is possible and may reduce the recipient's immune response to allogeneic tissue, as demonstrated by preservation of the leaflets' structural integrity in decellularized allografts relative to cellular allografts. Despite the lack of immunologic data and the small number of animals, they showed convincing histologic evidence reflecting these findings.

However, one of the major limitations affecting the generalizability of their find-

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 syngrafts 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) syngrafts 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 syngrafts and even in some allografts. This indicates that loss of cellular structures, resulting in an absent blood-tissue barrier, from the treated syngrafts 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 associates¹ 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 associates¹ 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 surgery³ 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