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Reply to the Editor:

We appreciate the response by Stöllberger, Schneider, and Finsterer to our work¹ and thank them for their valuable comments.

As they mentioned, this series in dogs was too small to confirm the long-term safety and tissue response to the atrial exclusion device (AED). However, the main purpose of this series was to assess the feasibility of the device implant during a beating heart procedure, and thus we did not include a control group or an atrial fibrillation (AF) animal model, which would have been helpful to evaluate the safety and efficacy of this device.

Because of the main goal of this study series, we also did not analyze biochemistry levels such as serum natriuretic peptide, troponin, and creatine kinase, nor did we obtain hemodynamic data derived from cardiac catheterization with the exception of left atrial (LA) pressure, heart rate, and systemic arterial pressure. However, we performed echocardiographic evaluations at baseline, after AED implant, and at follow-up. The pulmonary venous flow, transmitral flow, and tissue Doppler imaging of the mitral annular motion, which provide additional information regarding atrial and ventricular filling and function,^{2,3} were evaluated to assess the influence of left atrial appendage (LAA) exclusion on LA function. The Doppler pulmonary venous flow data suggested that LAA exclusion may affect LA reservoir function without affecting left ventricular diastolic function. After we obtain echocardiographic data from our next series of animals, we intend to report more detailed echocardiographic evaluation results in the near future.

With respect to the effect of device implant on flow through the left circumflex artery, although specific evaluations including coronary angiography and left circumflex flow measurement with a flow probe were not performed in this series, no macroscopic findings of myocardial infarction at explant surgery or gross examination were found.

As also mentioned, LAA exclusion may cause adverse effects due to lack of serum natriuretic peptide. In this series, there were no clinical signs of heart failure, including general fatigue or significant changes in the weights and heart rates of any dog, throughout the study.

To date, there have been no reports showing clear evidence of a role of LAA exclusion on stroke prevention for patients with non-valvular AF. However, considering that LA thrombi in more than 90% of cases of non-valvular AF are located in the LAA⁴ and surgical LAA ligation or excision does not appear to have clinically important deleterious effects according to extensive study on the Maze procedure by Cox and associates,⁵ LAA exclusion should be considered one of the most important therapeutic options, especially for patients with AF who are not eligible for any anticoagulation therapy.

We agree with the potential concerns related to hemodynamic and neurohumoral consequences after LAA exclusion. In preparation for a clinical AED application, further evaluations using both a control and AF animal model will definitely be required to address the following points: (1) competency of LAA exclusion with various LAA sizes, (2) long-term stability of the AED, (3) potential LA thrombus formation due to the device implant in an AF model, and (4) neurohumoral effects of LAA exclusion. We hope these further evaluations will provide us with clearer information that elucidates the exact role of LAA exclusion.

We continue to work on this subject and look forward to publishing more information on the AED, the implantation procedure for LAA exclusion, and its effects on physiologic function.

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Is it really the number of clamps that is responsible for worse postbypass neurological outcomes?

To the Editor:

I read with interest the article by Dr Hammon and colleagues¹ comparing varying levels of aortic manipulation in bypass surgery with regard to postoperative neurobehavioral outcomes. Even as the results are in some concurrence with certain previous trials in this direction, I do believe that this report requires careful review before its recommendations can be accepted.

First, the multiple aortic clamping group had greater age, incidence of hyperlipidemia, and aortic fibrillation than the single aortic clamping group, 3 independent predictors of postoperative stroke.² Second, the authors were unable to demonstrate any significantly worse result in the 2 groups, apart from the neuropsychologic deficit at 6-month follow-up. Recent prospective neuropsychologic testing indicates that these late neurocognitive deficits are likely to be caused by established risk factors for cerebral vasculopathy not having anything to do with the bypass procedure itself.³

It is therefore arguable that the mere reduction in the number of clamps would have a significant effect on patient neurologic profile after bypass surgery, considering that there exists already conflicting evidence, with no additional benefit of reduction in clamp number being recorded previously.⁴ Consensus in the literature has

generally been that off-pump surgery results in better neurocognitive outcomes than on-pump procedures. Apart from the well-documented role of cardiopulmonary bypass in emboli generation, this finding might be explained by the interesting fact that it is actually the act of aortic cannulation rather than the application of the clamp itself that is likely to generate the greatest number of emboli.⁵ This might explain the better results in the off-pump group and the comparable results in the 2 on-pump groups with different degree of clamp use in this trial, obviating the little additional benefit of reduction in clamp number.

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Reply to the Editor:

The authors of our article thank Omer Ashraf for his comments relating to our published article. We thank him for his careful reading and analysis.

Ashraf makes the comment that it was invalid to compare the multiple clamping group with the single clamping group and the off-pump coronary artery bypass group

because the group is significantly older. As we stated in the "Discussion" section, we have performed additional analyses on patients in all 3 groups who were older than the age of 65 years and found that the multiple clamping group still had a statistically greater number of neurocognitive deficits at 3 and 6 months, thus invalidating this criticism. There was no significant difference between the incidence of hyperlipidemia or atrial fibrillation in all 3 groups.

Relating to late neuropsychologic testing, as mentioned by Ashraf, in our experience deficits that occur after 6 months often appear in a new domain and thus might represent a new brain lesion, usually related to spontaneous atherosclerotic emboli.

Our analysis of the literature would suggest that the question of better neurological or neuropsychologic outcomes with OPCAB surgery has evidenced no clear difference, as did this study. Evidence from our institution would also suggest that the more applications of aortic clamps, cannulas, or proximal anastomoses, the greater number of emboli. The temperature at which those emboli reach the brain is also important in determining outcome.

We thank Ashraf for his comments and appreciate the time spent on the article.

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Redo coronary bypass grafting: Role of arterial grafts and time interval To the Editor:

I read with interest the article by Sabik and colleagues wherein they present their experience with reoperations in bypass surgery.¹ Although the authors have conducted an extensive temporal analysis, some important concerns remain in reference to the implications drawn from this report.

An important factor left unaddressed in this study was the mean time for reoperation. The time between primary and secondary bypass grafting is a determinant not just of reoperation outcome but also of the risk factors and patient characteristics deeming reoperative therapy.² Therefore it

is important to keep in consideration that the risk factors for reoperation shown in this report may not have uniform applicability and may differ for patients undergoing their second bypass at varying times from their first surgery. The patency of arterial and venous bypass conduits similarly is likely to have individual alterations at different points in time from the first operation, illustrating the efficacy of arterial grafts in reducing reoperation rate only beyond a certain time interval from the primary procedure.

The authors in this report, in cognizance with certain trials in the past, advocate extensive use of arterial revascularization to decrease reoperation rate on basis of their findings. However, some amount of conflict prevails on this issue, with a number of studies advocating no additional benefit of using more than a single arterial graft.^{3,4} In fact the reason that authors were unable to document high reoperation rates among patients with multiple arterial grafts may be owing to the notion that such patients are more likely to undergo percutaneous angioplasty intervention rather than a second bypass procedure. It is arguable, thus, that broad use of arterial grafting would prevent future myocardial ischemia in these patients. Even if a small extra degree of reoperative risk was conferred by avoiding use of more than 1 arterial graft, there remains little overall clinical justification for overuse of multiple arterial grafts, bringing into view one of Dr Sabik's own recent reports that reoperation procedures pose little extra patient hazards today.⁵

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