NEW DEAIRING TECHNIQUE: QUESTIONS AND CAVEATS To the Editor:

Al-Rashidi and colleagues¹ provide important information with regard to a new technique for deairing in cardiac surgery; however a number of questions need to be answered before adoption of their proposed new technique.

First, their technique of deairing the heart by apical venting and active suctioning on the root may potentially entrain air into the aortic root,² because the active suctioning causes a negative pressure in the aorta—causing it to collapse, as they mention in their Methods section. Opening the root to the atmosphere is safer, because the positive pressure in the aortic root forces air outward instead of sucking it inward.

Second, concerns of the effect of carbon dioxide insufflation on arterial blood gases can be negated by its use at the end of the procedure before deairing, because it is not needed until the crossclamp has been removed. This allows normal oxygenator gas flow rates. Altering the arterial carbon dioxide levels may be detrimental, which is the whole basis of the pH stat and alpha techniques for acid–base balance.

Third, allowing both lungs to collapse causes pulmonary vasoconstriction, meaning that the lungs will only derive oxygen from the bronchial arteries. Because bronchial arteries are highly variable in their number, size, and flow and are frequently blocked

by atherosclerosis in elderly patients,³ pulmonary ischemia may become an important issue, causing postoperative pulmonary dysfunction and adding to the problem of postoperative atelectasis. A study involving only 20 patients is statistically underpowered to evaluate this potential deleterious side effect or to make any conclusions other than that the technique is possible.

Fourth, transesophageal echocardiographically guided deairing through the left ventricular apex is frequently ineffective for residual bubbles in the left ventricle, and passive root venting is more efficient. In addition, air emboli in the left atrium and ventricle are easier to detect with transesophageal echocardiography, but in practice the aortic root is clinically the most important anatomic compartment with regard to deairing.

Finally, 10 minutes of suction on the aortic root to deair will undoubtedly cause blood component damage and extend cardiopulmonary bypass. These effects need to be balanced against a transesophageal echocardiographic or transcranial Doppler statistical finding with no clinical correlation.

Al-Rashidi and colleagues need to address these points in a study with a clinically significant number of patients undergoing more homogeneous operative interventions before their findings can be adopted.

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chial arteries: an analysis with 64-detector-row computed tomographic angiography. *J Comput Assist Tomogr.* 2011;35:253-9.

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Reply to the Editor:

I thank Poullis for his valuable comments and questions with regard to our article in this journal.¹ I will address his questions one by one in the same order that they were posed.

I agree with Poullis that active suction of the aortic root may entrain air in the aortic root if a left ventricular vent is used simultaneously. We therefore recommended in the text that the left ventricular vent should be occluded while the aortic root is on active suction. Opening the aortic root to atmosphere is safe, but it is not as effective as active suction, especially if the aortic root has been replaced with a vascular prosthesis (unpublished data). It is likely that air emboli get entrained in the crimp of the vascular prosthesis and get detached first when the aortic root gets fully distended with an adequate systemic arterial blood pressure. In an earlier study and in the evolution of our deairing technique, we let the aortic root deair spontaneously by open exposure to the ambient atmosphere and found that the number of microembolic signals recorded by transcranial Doppler was significantly higher after removal of the aortic crossclamp and before the cardiac ejection had started, suggesting entrained air emboli in the aortic root and ascending aorta.² These microembolic signals were significantly reduced with active suction of the aortic root.¹

I believe that it is worthwhile finding out in a prospective randomized study whether the use of carbon dioxide at the end of the open surgery on the left side of the heart is as effective as when the gas is used from the beginning of the surgery. I am concerned, however, about the air that escapes into the left atrium and

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pulmonary veins after the left side of the heart is exposed to the ambient atmosphere. We now know from continual transesophageal echocardiographic monitoring that these entrained air emboli come from pulmonary veins and that they get flushed out first when the entire calculated cardiac output gets diverted through the lungs. Our study showed that despite the early insufflation of carbon dioxide before exposing the left side of the heart to the ambient atmosphere, we recorded microembolic signals on transcranial Doppler in these patients for as long as 25 minutes after the release of the aortic crossclamp, suggesting that air did get into the pulmonary veins despite all the precautions described in our Methods section.¹

I fully agree with Poullis that there is always a risk of pulmonary parenchymal damage during cardiopulmonary bypass (CPB) as a result of pulmonary ischemia secondary to lack of pulmonary arterial flow. This may and does happen with or without collapse after complete CPB and despite patent bronchial arterial supply. In pig experimental studies performed in our laboratory, an 18-hour interruption in pulmonary arterial blood flow in noncollapsed lungs at normothermia produced fatal pulmonary parenchymal damage in all 6 experimental animals.³ During this ischemic period, all these animals were provided with dead space ventilation with 20% inspired oxygen fraction to prevent atelectasis. In another experimental study, 25% of the calculated cardiac output was diverted through the lungs at normothermia for 18 hours and the lungs ventilated to generate normal blood gas levels in the pulmonary venous blood.⁴ All 6 animals included in that study survived after termination of the CPB, and the lungs were able to provide adequate ventilation in the next 6 hours, when experiments were terminated electively. Histopathologic

examination, however, revealed all lungs to have patchy bilateral parenchymal damage.

There are few data available in the English-language medical literature discussing in a systematic manner the deleterious effects of CPB combined with induced bilateral pulmonary collapse on postoperative pulmonary function (subtle and clinically overt) and the effects of core cooling, regional cooling, pulmonary ventilation, and other strategies on these effects. Hypothermia remains the mainstay for lung protection in clinical lung transplantation, and the donor lungs (mostly from older donors) are preserved at present in a collapsed or quasicollapsed state for protracted periods of cold and warm ischemia. In our earlier study, 37 consecutive patients underwent Ross operations under moderate hypothermia for aortic valve disease.⁵ Bilateral pulmonary collapse was induced in all patients in this study to facilitate effective deairing. The median aortic occlusion and CPB times in that series were relatively long, 2.5 and 3 hours, respectively. The postoperative median time on the ventilator was, however, 6 hours (9 hours, 3rd quartile), and the median stay in the intensive care unit was 1 day, figures exactly similar to those in our more recent study.¹ Our most recent study is, however, definitely not powered highly enough to show lack of inferiority of the Lund deairing technique relative to carbon dioxide deairing technique with respect to postoperative pulmonary function.

Poullis states, "Transesophageal echocardiographically guided deairing through the left ventricular apex is frequently ineffective for residual bubbles in the left ventricle, and passive root venting is more efficient." This statement lacks scientific evidence, and I therefore refrain from commenting on this point.

Poullis's statement that "10 minutes of suction on the aortic root to deair will undoubtedly cause

blood component damage and extend cardiopulmonary bypass" is not in conformation with either of the deairing techniques discussed in our article, and I therefore refrain from commenting on this point as well.

I and my coauthors appreciate Poullis's in-depth study of our article and thank him once again for his valuable questions and comments.

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CATEGORIZATION OF COMMON ARTERIAL TRUNK To the Editor:

After reading "A Simplified Categorization for Common Arterial Trunk" by Russell and colleagues,¹ we thought it important to direct readers to one of the original descriptions and categorization of these hearts. At the end of the 19th century, Professor Hermann Vierordt² (1853–1943) from the University of Tübingen published *Die*