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*

Angeborenen Herzkrankheiten, a book and systematic review of embryology that included congenital cardiac anomalies. One of the sections was entitled, "Incomplete Division and Unilateral Transformation of a Primary Truncus. Persistence of the Truncus Arteriosus—Presence of Only One Single Major Arterial Vessel."³ In this section, he used descriptors for the common arterial trunk similar to those presented in the article by Russell and colleagues.¹ This simplified approach was discussed by Calder and associates⁴ and emphasized by Jacobs,⁵ as indicated in Russell and colleagues' article,¹ but had initially been presented almost a century before. Vierordt's original classification was also discussed on page 1250 of the seminal work by Collette and Edwards.⁶ Vierordt³ reviewed 40 cases (including some of Rokitansky's original preparations) and proposed 3 categories: (1) "partial persistence of an embryonic truncus arteriosus: one major vessel from which arises an aorta and pulmonary artery"; (2) "a single major vessel arising from the heart of the character of an aorta with more or less typical branching of the same, lacking a pulmonary artery as such, and the pulmonary vessels arise from the main arterial vessel"; and (3) "a single vessel of the character of a pulmonary artery." It is also ironic that Vierordt³ states before presenting his 3 categories, "It would be highly desirable that there would be agreement in the nomenclature." He pointed out how useful this would be for better definition of these types of lesions. It seems that we have come full circle in the description and categorization of the common arterial trunk and have rediscovered the original classification of these lesions and a plea for common nomenclature. We would like to recognize the initial classification and echo the plea of Vierordt³ by encouraging the adoption of this simple categorization presented recently by Russell and colleagues¹ and realized by others before them.³⁻⁶

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Reply to the Editor:

We appreciate the letter from Erickson and Opitz in support of our categorization of common arterial trunk as either aortic or pulmonary dominant.¹ We are intrigued that they have brought to our attention the work of Vierordt² published more than 100 years ago. Although his theme was similar, there are important differences that we believe deserve emphasis.

As we understood Vierordt's categorization,² he distinguished between a common arterial trunk, a solitary aorta, and a solitary pulmonary trunk. Although this is similar to our concept, our suggestion is that common arterial trunk itself, according to its pattern of branching, is best

subdivided on the basis of aortic or pulmonary dominance. The semantics are important. Our emphasis on this particular categorization is related to the accepted definition of common arterial trunk: a solitary trunk that exits the heart through a common ventriculoarterial junction and supplies directly the systemic, pulmonary, and coronary arterial pathways. A solitary aorta, such as exists in tetralogy with pulmonary atresia and major aortopulmonary collateral arteries (historically and erroneously labeled "pseudotruncus"), does not fit this definition. A solitary pulmonary artery, such as seen in hypoplastic left heart syndrome with aortic atresia, also does not fit this definition. Thus our interpretation of Vierordt's analysis² is somewhat different than that of Erickson and Opitz.

We support Erickson and Opitz in their acknowledgement of the works of Vierordt,² Calder and associates,³ Van Praagh and Van Praagh,⁴ Jacobs,⁵ and Collette and Edwards⁶ in arriving at what we consider to be the optimal approach to categorization of common arterial trunk.

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