Reply to the Editor:

We thank Professor Anderson for his interest in our recent article.¹ First of all, we must apologize for having missed the article by Sutton, Ho, and Anderson² in our literature survey, and consequently for not having discussed it, while preparing our article. Should the wrong impression have been evoked that we deliberately omitted the article, we can assert that this is not our style.

The article by Sutton, Ho, and Anderson² is certainly interesting and deservedly emphasizes the role and build of the interleaflet triangles. Indeed, these authors deserve credit for mentioning "a very thin layer of elastic tissue on the luminal surface" of the interleaflet triangles. What surprised us most, however, during our investigations is that this elastic tissue appears to be isolated from the other areas of elastic tissue. Although we have to consider that a more refined technique may show the elastic tissue to be continuous, we do think it is interesting from a developmental point of view. Likewise, as Professor Anderson suggests, the conus tendon is intriguing and has our attention in our morphogenetic studies.

With respect to the subpulmonary infundibulum, we are well aware that the subpulmonary infundibulum is not made of the ventricular septum proper. We (M.M.B. and A.C.G-de G.) have investigated this issue extensively and also clarified its morphogenesis.³⁻⁶ We are somewhat surprised by Professor Anderson's comment, as he was the one who invited us, some years ago, to write a review on the morphologic and embryologic correlations of the outflow tract.³ The most important message from the latter article was that because of the asymmetric configuration of the level of the arterial orifices, already present in early embryonic hearts, the separation process results in the formation of the posterior wall of the pulmonary infundibulum, "rather than a septum between both outflow tracts." Figs. 1, 3, and 4 of that article emphasize that this (muscular) part of the pulmonary infundibulum is adjoining the ascending aorta, from which it is separated "by loose fibrous tissue and can thus not be called an 'actual' outlet septum." In the discussion of that article, we stated that our findings were in agreement with the observations of Anderson, Becker, and Tynan,⁷ who said that "it is not a septal structure at all." It appears that modesty with "self-quotation" does not always pay. In our recent paper¹ on the pulmonary and aortic roots, we did not repeat this discussion because we assume that surgeons performing the Ross procedure do know these morphologic details. When mentioning that part of the pulmonary anulus inserts into "the septal part of the right ventricular myocardium," we used this designation to distinguish its location which, strictly speaking, is the myocardium immediately above the ventricular septum proper. To avoid misunderstanding, we will consider a more strict formulation in the future.

There remains, however, one issue to be discussed. We do not agree with Professor Anderson's statement that the autograft in a Ross procedure "does not possess a septal component." Most of the autograft is taken out of the freestanding myocardium in a plane approximately perpendicular to the subpulmonary myocardial surface. The most critical part of dissecting the autograft, however, concerns the portion that is scooped out in a plane

between slightly oblique and parallel to the surface on the right ventricular side of the curved anterior part of the ventricular septum, thereby avoiding the septal branch of the left coronary artery. Should the section be made perpendicular to this part of the septum, the chances of damaging the coronary branch would be increased and the lumen of the left ventricle anterior to the subaortic area would be opened. Thus this part of the myocardium is not "freestanding" but an "actual" part of the ventricular septum. Unfortunately, the illustration accompanying Professor Anderson's letter does not provide a proper view of this area. Morphogenetically, we consider this part of the septum to be the most cranial part of the primary septum formed by the primary fold.

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Cerebral oxygenation during cardiopulmonary bypass in children

To the Editor:

We read with great interest the study of Kurth and coworkers¹ on cerebral oxygenation during cardiopulmonary bypass in children. Our understanding of this very important subject has been improved by the introduction

of near-infrared devices. In Kurth's study, cerebral saturation was obtained during cardiac operations with various degrees of pump flow, hematocrit values, and temperatures. The purpose of the study was to determine the factors influencing oxygen extraction of the brain during cardiopulmonary bypass, which others have also studied experimentally and clinically.²⁻⁵ Kurth and associates concluded that hypothermia is the factor that decreases cerebral oxygen extraction, with hematocrit value and pump flow as cofactors. We believe that this statement is debatable and unproved by this study.

The authors used alpha-stat management during the operation, meaning that they did not correct blood gas values for temperature. Therefore other important operative parameters in the study, especially pH and carbon dioxide tension (P_{CO_2}), were by no means constant, as stated by the authors. These changes are simply neglected. In a recent study, we⁴ also showed a similar correlation between oxygenated hemoglobin in brain tissue measured by near-infrared spectroscopy and jugular bulb oxygen saturation on the one hand and the temperature during cardiac surgery on the other hand. However, this relation became insignificant if controlled for pH and P_{CO_2} . In fact, pH and P_{CO_2} were the only significant parameters influencing oxygen saturation in the jugular bulb and oxygenated hemoglobin in brain tissue. Other clinical and experimental studies have confirmed the importance of pH and P_{CO_2} management for cerebral oxygenation and postoperative brain function.⁶

Kurth and associates suggest that a decrease in temperature might increase cerebral oxygenation during cardiac operations. This statement should be accepted cautiously. In fact, using multivariate analysis, we found that hypothermia had a slight but significant *adverse* effect on the redox status of cytochrome aa3, which could be explained by the shift of the hemoglobin binding curve with hypothermia. Furthermore, the redox status of cytochrome aa3 was the only intraoperative parameter correlated with postoperative neuropsychologic dysfunction.⁵ Therefore one might argue that decreasing blood temperature during a period of high cerebral oxygen demand is not beneficial but rather detrimental.

In conclusion, we appreciate the honest attempt of the authors to increase our knowledge of cerebral oxygenation during cardiac surgery. However, the conclusions of this study may be misleading, because important variables influencing cerebral oxygenation were not controlled. In addition, as Jonas⁷ stressed in his commentary, brain tissue saturation measured by near-infrared spectroscopy does not reflect the true oxygen demand of the brain during hypothermia.

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Reply to the Editor:

We appreciate the interest of Nollert and colleagues in our article "Cerebral Oxygenation During Cardiopulmonary Bypass in Children."¹ In their letter, several comments were directed toward the conclusions in our paper and the physiology of tissue oxygen transport, to which we are happy to respond.

The purpose of our study was to determine the effect of temperature, pump flow, and hematocrit value on cerebral oxygen saturation (ScO_2) as measured by near-infrared spectroscopy (NIRS) in children. As background, we had observed distinct changes in ScO_2 in children during deep hypothermic cardiopulmonary bypass (CPB) and circulatory arrest.^{2,3} These changes included an increase in ScO_2 during CPB cooling, a curvilinear decrease during circulatory arrest, and a return to preoperative levels during CPB reperfusion and rewarming. We wished to determine which factors influenced ScO_2 during CPB cooling because children with neurologic sequelae did not experience the typical increase in ScO_2 .³ In our study, we concluded that brain hypothermia was the main factor increasing ScO_2 during CPB cooling, although a certain CPB pump flow and hematocrit value were required to effect this increase.¹

Nollert and colleagues propose an alternative conclusion that is inconsistent with the data. They correctly point out that arterial carbon dioxide tension (P_{CO_2}) influences cortical oxygen extraction during CPB cooling.⁴ They also correctly point out that we had used alpha-stat management during CPB and that temperature-corrected arterial P_{CO_2} would have decreased during hypothermic CPB. However, they incorrectly conclude that this decrease in P_{CO_2} would have increased ScO_2 during CPB cooling, because hypocapnia decreases ScO_2 , just as hypocapnia decreases cerebral blood flow and increases cortical oxygen extraction. Thus hypothermia is associated with an increased ScO_2 , in contradistinction to the reduction that would be predicted on the basis of diminished P_{CO_2} .

Nollert and associates also misstated our conclusions