of 35 (29%) in the pH-stat group and 7 of 35 (20%) in the alpha-stat group. Further analysis of this subgroup of patients showed that 7 of the 10 pH-stat patients (70%) had cognitive dysfunction in two or more tests (which was the same as for the whole group at this level) whereas only 1 of the 7 alpha-stat patients (14%) had cognitive dysfunction at the same level (compared with 46% for the whole group). This discrepancy further emphasizes the advantage bestowed by the use of alpha-stat acid-base management of CPB on reducing cognitive dysfunction compared with pH-stat management.

Professor Murkin further points out that there appears to be a considerable difference in the ages of the patients between the two studies. His statement is based on the upper limit of the confidence interval for our patient population being less than the mean value for his population. Further analysis does not support this contention, however. We concede that the use of 95% confidence intervals for mean values of parameters such as age, CPB duration, and cross-clamp duration may not be ideal. In Table III of Murkin's study, it is not stated whether the mean values are given with standard deviation or standard error; we assume it to be standard deviation because this was used in Table V. On this basis, the mean age ($\pm$ standard deviation) of our patients was 56.9 ± 5.7 years for the alpha-stat group and 57.6 ± 7.8 years for the pH-stat group. These ages would not appear to be significantly younger than those in Murkin's study. In addition, our aortic crossclamp durations were 39.7 ± 10.8 minutes and 40.3 ± 14.7 minutes and CPB durations were 75.1 ± 17.3 minutes and 83.4 ± 38.7 minutes for the alpha-stat and pH-stat groups, respectively. Consequently, although the aortic crossclamp durations were similar, the mean CPB durations of our patients do appear to have been shorter.

Professor Murkin professes to be puzzled with our finding that "there was a significantly greater reduction in CMRO₂ [cerebral metabolic rate for oxygen] in the pH-stat group during hypothermia." We admit that it is not immediately obvious from Fig. 3 that these values are significantly different. However, the *Results* section states that CMRO₂ decreases by 49% and by 63% of the prebypass value in the alpha-stat and pH-stat groups, respectively. The actual values were 0.561 and 0.432 ml oxygen $\cdot$ 100 gm⁻¹ $\cdot$ min⁻¹, respectively, and the *p* value for the difference between these values was 0.0064. Thus we stand by our statement that the value of CMRO₂ for the pH-stat group of patients, during the hypothermic phase of CPB, was reduced significantly more than in the alpha-stat group.

Professor Murkin also comments on the apparently significantly higher values of cerebral blood flow (CBF) and CMRO₂ obtained in our study compared with those of a previous study³ conducted by his group. He points out that the difference observed between these studies could be accounted for by the differences in anesthetics administered during the operative procedure. We agree that anesthetic agents are among the factors that can influence cerebral hemodynamics. We also consider that the depth of anesthesia is important and would respectfully point out that we acknowledged such in the *Discussion* of our article, citing Professor Murkin's study³ as an example of where this might be the case. However, it is the changes in CBF and CMRO₂ that are of interest; the initial baseline values are, essentially,

irrelevant (so long as they fit within the accepted normal values). In addition, we should point out that, in our study, continuous on-line arterial blood monitoring was practiced, which enabled precise control of acid-base status in both the alpha-stat and pH-stat groups. This precise control was not practiced by Murkin and colleagues.

We thank Professor Murkin for his overall congratulations regarding our study, which we readily reciprocate. Now that there are two relatively large trials demonstrating the benefits of alpha-stat acid-base management during CPB on reducing cerebral dysfunction in patients having CABG alone, we hope that other centers that currently do not use the alpha-stat management regimen during CPB may be persuaded of the benefits.

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12/8/78753

Median sternotomy for prolonged resuscitation in neonatal tetralogy of Fallot with absent pulmonary valve

To the Editor:

In a recent brief communication, van Son and Mohr¹ discussed specific preoperative strategies designed to stabilize the condition of neonates who have tetralogy of Fallot with absent pulmonary valve syndrome.

We recently encountered a neonate with tetralogy of Fallot and absent pulmonary valve who failed to respond to the "usual" preoperative maneuvers designed specifically for the associated bronchial compression and severe respiratory distress. Preoperative nursing in the prone position and median sternotomy may be advantageous, as previously described by Heinemann and Hanley² and van Son and Mohr.¹ However, depending on the associated anatomic lesions, these selected maneuvers may be useless, as described below.

A female neonate (2.6 kg) was intubated 2 hours after birth because of cyanosis and respiratory insufficiency and was transferred to our institution. Because of the patient's instability a limited transthoracic echocardiogram was

performed, demonstrating tetralogy of Fallot with absent pulmonary valve. A massively dilated right pulmonary artery (15 mm) and an enlarged left pulmonary artery (8 mm) were identified. Additional anomalies were a right aortic arch and an atrial septal defect.

Even after the administration of paralytics it was difficult to obtain satisfactory oxygen saturation. Importantly, the carbon dioxide tensions remained near normal. The baby was supported by continuous-mode ventilation with 100% oxygen. Prone positioning was attempted without significant improvement. Within the next 24 hours, increasingly large amounts of inotropic support were required. The patient remained critically ill with bradycardia and severe cyanosis. Repair of the cardiac malformation was considered urgent for survival and was not delayed.

Cardiopulmonary bypass was established and the lesion was repaired under low flow with moderate hypothermia. Exploration of the right atrium revealed a large inferior sinus venosus defect (posterior defect) extending deep into the inferior vena cava. An autologous pericardial patch closure was performed in addition to ventricular septal defect closure and pulmonary arterial reduction (Fig. 1). The combination of early takeoff of the massively enlarged right pulmonary artery and an anomalous left anterior descending coronary artery originating from the right coronary artery (crossing the pulmonary anulus) made insertion of a valved conduit impossible. Therefore right ventricular outflow tract reconstruction was accomplished with creation of a monocusp outflow patch. The patient was weaned successfully from cardiopulmonary bypass with an oxygen saturation of 100% on an inspired oxygen fraction of 40%.

Decreased oxygen saturation combined with a near normal carbon dioxide tension in this infant cannot be explained by airway obstruction alone. Oxygen saturations did not improve with either prone positioning before the operation or median sternotomy in the operating room. The hypoxemia can now be explained by disadvantageous streaming at the atrial level. The high pulmonary resistance immediately after birth, augmented by impaired diastolic filling of the right ventricle as a result of free pulmonary valve insufficiency, right ventricular dilation, and decreased ventricular compliance, all increased the degree of atrial admixture.³ We agree that surgical correction of this defect should not be delayed, for sometimes when one hears hoof steps it really is a zebra!

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Fig. 1. Anterior view of the intraoperative repair. The right atrium has been opened. The large inferior sinus venosus defect (posterior defect) extending deep into the inferior vena cava has been closed with an autologous pericardial patch. Note the aneurysmal enlargement of the pulmonary arteries and the anomalous origin of the left anterior descending artery.

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Carbodissection of the internal thoracic artery pedicle

To the Editor:

Blake and associates¹ demonstrated that 40% of internal thoracic arteries (ITAs) harvested by electrocautery were damaged or failed to respond to contractile stimuli. Lesser degrees of damage were documented with the use of an ultrasonic aspirator.

In 1988, I² described carbodissection of the ITA pedicle to separate the pedicle from the chest wall and facilitate exposure of its branches in an atraumatic manner. A 5 L flow of carbon dioxide is introduced anterior to the ITA pedicle through an 8F pacemaker lead introducer. This rapidly dissects the pedicle from its fascial investments. Gentle downward pressure with the electrocautery spatula exposes the branches, which are then clipped.

My colleagues and I have now used this technique to