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

harvest more than 350 ITAs. The method is simple, inexpensive, requires no special equipment, and eliminates the risk of thermal or sonic injury to the pedicle. In no case to date has an ITA not been used as planned because of carbodissection. The technique allows gentle handling of the pedicle, which almost always has a brisk pulse at the completion of its separation from the chest wall.

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