indicates lactate acidosis, will affect the blood lactate concentration, especially in very little infants.² This study has not analyzed the use of banked blood in 2 groups statistically. Therefore, is it more appropriate to limit the inclusion objects to a more narrow age span?

Second, crystal solutions would be used in the extracorporeal circulation. The crystal solutions in use now include Ringer's lactate and Ringer's acetate. Lactate has 2 isoforms: L-lactate and D-lactate. L-lactate is the products of anaerobic glycolysis in humans and has been used as a marker of cellular hypoxia and tissue malperfusion.³ It would be converted to sodium bicarbonate by tricarboxylic acid cycle. However, D-lactate is mostly exogenous from Ringer's lactate solution. Its clearance is much slower than L-lactate, mainly depending on the liver function.³ Massive exogenous D-lactate Ringer's solution infusion would cause iatrogenic hyperlactacidemia in those infants with immature liver function. This phenomenon would not occur when Ringer's acetate is used in CPB. However, this study has not mentioned the kinds and dosage of the crystal solution.

Ronghua Zhou, MD
Jin Liu, MD, PhD
Department of Anesthesiology
West China Hospital
Sichuan University
Chengdu, Sichuan, China

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COMMENT ON EDITORIAL BY LAPEYRE AND COLLEAGUES To the Editor:

The editorial by Lapeyre and colleagues,¹ "Prosthetic heart valves: Difficult to make something simple," emphasizes the need for more durable tissue valves for patients younger than 65 years, especially juveniles and young adults in emerging countries. To address that need, our group has worked on the concept of a non-immunogenic autologous tissue valve for the last 25 years. The initial clinical application was a stent-mounted valve made in the operating room at the time of valve replacement.² More recently, a totally stentless aortic valve made with disposable instruments at the time of valve replacement has been developed and used clinically.³ Follow-up of the first 11 patients implanted with the stentless autologous pericardial valve in a prospective pilot study from February 2003 to May 2005 at the Harefield Hospital by Dreyfus can be seen, before publication, on the webcast of the 2010 American Association for Thoracic Surgery annual meeting in Toronto.⁴ The first patient in the series was a 42-year-old man with a heavily calcified bicuspid aortic valve, a gradient of 131 mm Hg, and an ascending aortic aneurysm. He has normal valve function with no evidence of calcification 7.2 years post-aortic valve reconstruction. The last patient in the series was a 22-year-old woman with congenital aortic stenosis and a calcified bicuspid aortic valve with a gradient of 71 mm Hg. She has normal valve function with no evidence of calcification 5 years after aortic outflow enlargement and aortic valve reconstruction. These results suggest that lightly tanned autologous pericardium may be a suitable biomaterial for fabricating a more durable tissue heart valve for younger patients. The use of autologous tissue and disposable instruments for the valve reconstruction

has the potential for being a cost-effective solution to the need for a tissue heart valve in emerging countries.

It is largely forgotten that the first tissue heart valves were made of autologous tissue, by Åke Senning in the early 1960s. He did freehand reconstruction of aortic valves with the patient's own fascia lata. Senning explained presciently why he used autologous tissue: "Having worked with organ transplantation, I felt the allografts or homografts would be slowly destroyed by immunologic factors. In the same year, I began replacing the aortic valve with autogenous fascia lata, which was taken sterile at the time of operation, eliminating the problems of sterilization, preservation, immunologic reactions, and thromboembolism."⁵ He used untreated autologous fascia lata, and although 18 of 90 patients survived 12 to 20 years with their reconstructed aortic valves, 13 with "no or minimal dysfunction," most of the valves became insufficient because of thickening and shrinkage of the tissue. Others, notably Björk⁶ and Edwards,⁷ confirmed poor results with untreated fascia lata and pericardium, and the use of autologous tissue fell out of favor. On revisiting the concept, we discovered serendipitously that a brief immersion of autologous pericardium in dilute buffered glutaraldehyde solution, light tanning,² eliminated the thickening and shrinkage that had been observed by Senning, Björk, and Edwards. The tissue had a healthy, pristine appearance after valve implantation for 5 months in juvenile sheep, a known severe test for calcific degeneration. Love⁸ summarized the early history of autologous tissue heart valves in a monograph on the subject.

The valve industry has shown little interest in an autologous tissue heart valve that might prove to be more durable and cost a fraction of currently available bioprostheses.

Jack W. Love, MD, DPhil
 CardioMend LLC
 Santa Barbara, Calif

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PSEUDOANEURYSM OF ASCENDING AORTA COMBINED WITH AORTIC VALVE INFECTIVE ENDOCARDITIS AFTER CARDIAC OPERATIONS FOR CONGENITAL VENTRICULAR SEPTAL DEFECT

To the Editor:

We read with great interest the article of Emaminia and colleagues¹ and congratulate them for their successful repair of an iatrogenic pseudoaneurysm of the ascending aorta. Their strategy to safely reopen the sternum is to drain the blood into the oxygenator reservoir for 1 to 2 minutes to have an empty, relatively collapsed heart and aorta without establishment of femorofemoral bypass. We want to know how

FIGURE 1. Preoperative and postoperative computed tomography scans of patients. Defect of anterior wall of ascending aorta (dotted line); pseudoaneurysm arising from the right anterior wall of ascending aorta (single arrow); repaired aorta with aortic valvular prosthetic replacement (double arrow).

much blood should be drained and how to maintain hemodynamics during the procedures. Fortunately, the pseudoaneurysm was located at the back of ascending aorta. Otherwise, how would the hemodynamics problem be dealt with if the reentry of the sternum was not uneventful?

Ascending aortic pseudoaneurysms are found on rare occasions, and direct surgical repair is the only choice, but this carries a high risk of fatal hemorrhage resulting from rupture of pseudoaneurysm at the time of sternal reentry. From the limited case reports in the literature,²⁻⁵ the authors concluded that median sternotomy with the use of femorofemoral bypass and hypothermic circulatory arrest is the strategy of choice.

We recently surgically managed a case of ascending aortic pseudoaneurysm combined with aortic valve infective endocarditis after congenital ventricular septal defect. An 11-year-old boy was admitted to our hospital 2 months later with intermittently high fever accompanying stridor and dizziness experienced for approximately 1 month. Echocardiography showed masses in the prolapsed right coronary cusp with severe aortic regurgitation. *Staphylococcus aureus* was cultured from the blood. A contrast computed tomography scan showed a defect in the right anterior

wall of the ascending aorta 1.5 cm above the aortic valve, from which a pseudoaneurysm was arising (Figure 1). Intravenous antibiotics according to in vitro susceptibility testing were continued for 2 more weeks, bacteremia was controlled, and operation was performed. The chest was opened through the previous scar. After institution of femorofemoral bypass, the patient was cooled to 32°C and the heart was relatively emptied. A sternotomy was then performed. Adhesions over the ascending aortic pseudoaneurysm and right ventricle were released uneventfully. The aorta was clamped at 28°C and opened through the pseudoaneurysm, which was approximately 8 × 7 × 5 cm. The 2.5 × 2.5-cm defect was at the previous aortotomy site. Cardioplegic solution was administered directly, and the aortic native valve was replaced by a 21-mm St Jude mechanical prosthesis (St Jude Medical Inc, St Paul, Minn). The aortic defect was closed using a 5 × 3-cm polytetrafluoroethylene patch with 4-0 Prolene suture. The patient was weaned uneventfully from cardiopulmonary bypass. The postoperative course was uncomplicated. No special treatment was given except for continuing intravenous antibiotics for another 4 weeks and oral warfarin. The cause of the pseudoaneurysm appeared to