

## Letters to the Editor

valve reconstruction. For example, Bacha and colleagues<sup>2</sup> described an important incidence of late failure because of calcification in an aortic valve repair with autologous pericardium.

We were surprised by the finding of our study that *shorter* pretreatment duration was associated with greater calcification, suggesting that longer but nevertheless clinically feasible pretreatment times should be used. In the future, we are interested in exploring the efficacy of the many anti-calcification treatments that have been applied to xenograft valves in the hope that they might be effective in reducing the degree of calcification of autologous pericardium in the pediatric setting.

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### THE CARDIOPULMONARY BYPASS MODEL IN RATS

#### To the Editor:

We read with great interest the study for the establishment of a cardiopulmonary bypass (CPB) model in rats titled "Rat model of cardiopulmonary bypass for deep hypothermic circulatory arrest" by Waterbury and colleagues,<sup>1</sup> showing that they had successfully developed a rat CPB model with deep hypothermia circulatory arrest for the research of postoperative neurocognitive function. The authors mentioned

that their model had several innovative elements, including peripheral cannulation, instead of thoracotomy or sternotomy, the lack of a need for tracheal intubation with mechanical ventilation, and so on.

However, some confusion was aroused by these innovations. First, we wonder whether peripheral cannulation, instead of thoracotomy or sternotomy, should be truly called innovative. Logically, to investigate postoperative cognitive function in rats undergoing cardiac surgery or CPB, the experimental rats must be alive after the induced surgical trauma for the subsequent cognitive assessment. Therefore, to date, for all those CPB models in rats that need to survive, thoracotomy or sternotomy is avoided. However, there is an exception. In the research of perioperative myocardium injury in rats using the CPB model from Günzinger and colleagues,<sup>2</sup> cardioplegic arrest was achieved through cardioplegia injection by direct aortic clamping. To achieve access to the ascending aortic artery for cardioplegia injection, sternotomy was performed, and cannulation and ligation of the bilateral carotid arteries were also performed; thus, theoretically, the survival of the experimental rats was close to impossible.

Second, the authors stressed that the rats undergoing CPB were not intubated and not ventilated, which is fully beyond our knowledge. Without tracheal intubation and mechanical ventilation, how would it be possible to maintain proper anesthesia, because we also know that too deep an anesthesia will compromise the respiration and hemodynamics. However, the rats might move under a light anesthesia. In addition, without supportive ventilation, coming off CPB will undoubtedly be very challenging for the rats buffeted by the sluggish circulation during the rewarming period. More importantly for the CPB model in the research by Waterbury and colleagues,<sup>1</sup> the CPB flow was incomplete, which was about 70% of

normal cardiac output, according to their report. Thus, how to oxygenate the other 30% of the blood? Under such circumstances, desaturation would gradually occur, which was evidenced by the severely reduced pH and base excess in their Table 1 for the physiologically relevant parameters of rats undergoing CPB with deep hypothermia circulatory arrest.

Third, we noted that no vein channel was established for medicine administration. When coming off CPB, the rats might need some inotropes; however, at that time, all the cannulas used for CPB have been extracted and the corresponding vessels ligated.

Finally, bilaterally, the femoral arteries were cannulated and also would have been ligated after surgery, which would produce some difficulties for the motor function of those experimental animals, although the experiment was performed for neurologic evaluation after surgery.

In the Duke Anesthesiology CPB laboratory, the CPB model in rats was instituted at a flow rate of 160 to 180 mL/kg/min, the normal cardiac output of rats. All the experimental rats were also intubated and ventilated for the purposes of both anesthesia and oxygenation. Additionally, thoracotomy or sternotomy was avoided to ensure the survival of the rats. Also, the superficial caudal epigastric artery and vein, instead of the femoral artery and vein, were cannulated for continuous measurement of the mean arterial pressure and for intravenous drug administration to ensure better survival.<sup>3,4</sup>

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### Reply to the Editor:

I thank Ma and colleagues for their comments on our article, "Rat Model of Cardiopulmonary Bypass for Deep Hypothermic Circulatory Arrest."<sup>1</sup> Ma and colleagues mention that peripheral cannulation alone is not necessarily innovative. I agree, and I would clarify that what we deemed innovative was the combination of: vacuum-assisted venous drainage, a small prime volume membrane oxygenator, nearly normal high-flow delivery rates, peripheral cannulation, ultrasonic flow meters, and active cooling and rewarming with a heat exchanger. In combination, this is a sophisticated cardiopulmonary bypass model for the rat.

These animals can be kept alive beyond bypass in this model without the need for endotracheal positive-pressure ventilation. The flow rates achieved on bypass of 100 to 120 mL/(kg · min) are adequate for rats during hypothermia. Although I agree that ventilation may be easier for survival surgery, it is not a requisite. Its main use in our model is to provide positive pressure to help reexpand the lungs. We have successfully weaned these animals with gradual

warming and an oxygen-rich environment without endotracheal intubation.

We routinely administer inotropic medications, as we do in human patients, when separating from cardiopulmonary bypass as a response to low blood pressure and acidemia; these parameters are routinely monitored by means of i-Stat (Abbott Laboratories, Abbott Park, Ill) and indwelling arterial lines in real time during the procedure. I agree that a venous line is useful, and we routinely infuse through the right atrial venous cannula when we are keeping our animals alive. We administer protamine and remove the femoral cannula when animals are being kept alive after bypass, with gentle hemostasis at the exit sites of the catheters. With these modifications, we have established a successful clinically relevant model of cardiopulmonary bypass and deep hypothermic circulatory arrest in the rat.

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### SOME ISSUES ABOUT THE DECISION-MAKING PROCESS FOR TRICUSPID VALVE REPAIR To the Editor:

Congratulations to Roshanali and colleagues<sup>1</sup> for their valuable study, "Echocardiographic Approach to the Decision-Making Process for Tricuspid Valve Repair," which appeared

in the June 2010 issue. Functional tricuspid regurgitation develops secondary to the annular dilation as a result of enlargement of the right ventricular cavity, which in turn is related to the increase in pulmonary arterial pressure secondary to left ventricular pathology. This physiopathology does not coincide with the view provided by Roshanali and colleagues' original Figure 1,<sup>1</sup> because elongation of the leaflets and chordae is seen in regurgitation. The absence of coaptation depth among the data given in their original Table 1 supports our claim.<sup>1</sup>

The criterion for pericardial patch use is unclear, although preoperative tricuspid annular diameter is given in their original Table 1.<sup>1</sup> The ring size and the method of reduction of annular diameter are unclear as well. We think that—at least in this study—the correlation between the degree of postoperative tricuspid regurgitation and pulmonary arterial pressure should be investigated.

The number of patients in this study is adequate for such statistical analysis. Reevaluation of the data therefore could yield important results.

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