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Reply to the Editor:

We thank Drs Szentkiralyi, Péterffy, and Galajda for their thoughtful comments about suture techniques for tricuspid valve repair. We believe, as they do, that every case of moderate tricuspid regurgitation should be repaired. The options are numerous, but we believe that the suture annuloplasty technique we described in this article will be very successful in these cases. The implication of their letter is that a suture annuloplasty likewise would be equal for very severe cases. We have tended to use a rigid ring for very severe cases with obvious clinical manifestation of massive tricuspid insufficiency.

The suture bicuspidization technique described can be performed in less than 10 minutes. This technique is effective, reduces the expense of operations, and certainly should stimulate the use of a simple repair techniques in every patient with moderate to moderately severe tricuspid regurgitation.

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Intraoperative oral sildenafil for management of pulmonary hypertension: A stepping stone to the future

To the Editor:

I read with great interest the recent article by Dr Shim and colleagues¹ detailing their experience with oral sildenafil in intraop-

erative pulmonary hypertension (N = 53). Although they controlled for confounders such as anesthetic depth, hypercarbia, and serum pH, it is not clear whether they adequately controlled for hyperoxia, a potent pulmonary vasodilator.²

In their Methods section, they specify that all patients' lungs were ventilated with 60% oxygen with standardized mechanical ventilation, but they fail to report what range of blood oxygen tensions were obtained and whether they varied significantly between study groups. Thus, a major confounder is not adequately accounted for in this otherwise interesting paper.

A second limitation with the implementation of the study findings into one's practice is that oral sildenafil is not as convenient in the intraoperative period as is an inhalational or intravenous formulation.^{1,2} Because these formulations of sildenafil exist, this study paves the way for their investigation in the management of intraoperative pulmonary hypertension, a common scenario in patients undergoing cardiac surgery.

It would be no surprise that these formulations of sildenafil are demonstrated to be clinically effective pulmonary vasodilators. Of far more clinical importance would be the clinical trials looking at synergistic combinations of selective pulmonary vasodilators. There are at least 4 different pathways that can be manipulated for synergistic selective pulmonary vasodilation²:

1. Cyclic adenosine monophosphate pathway with inhaled prostaglandin I₂
2. Cyclic guanosine monophosphate pathway with inhaled nitric oxide
3. Phosphodiesterase 5 inhibition with sildenafil
4. Endothelin blockade with drugs such as bosentan

The future is in synergistic approaches to the management of pulmonary hypertension, just as we have seen in the management of systemic hypertension over the past 30 years.

I congratulate Dr Shim and colleagues on a thought-provoking study. I look forward to their feedback about these intraoperative considerations.

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Reply to the Editor:

We appreciate the constructive comments by Dr Augoustides regarding our study.¹ He raised the issue of whether hyperoxia, which is a potent pulmonary vasodilator, was adequately controlled between the control and sildenafil groups in our study. The arterial oxygen tension (PaO₂) levels were 271 ± 89 mm Hg versus 254 ± 108 mm Hg (P = .602) before the first hemodynamic measurement and 276 ± 48 mm Hg versus 235 ± 55 mm Hg (P = .03) immediately after the last measurement with 60% oxygen in the control and sildenafil groups, respectively. There were no significant changes in the PaO₂ values within each group.

Hypoxic pulmonary vasoconstriction has been well studied for decades, with the stimulus identified as both mixed venous and alveolar oxygen tension in small arteries and systemic PaO₂ in large pulmonary vessels.² However, no comprehensive data exist regarding graded response of the pulmonary vasculature to changes in PaO₂ above 120 mm Hg (hyperoxia), especially in patients with long-standing valvular heart disease with concomitant pulmonary hypertension, as in our study. In an animal study, Rudolph and Yuan³ studied the relationship between PaO₂ and pulmonary vascular resistance and demonstrated that there is no further decrease in pulmonary vascular resistance above the PaO₂ of 50 to 60 mm Hg. Therefore, although the PaO₂ was lower in the sildenafil group after the last measurement, this should not have any further clinically significant effects on the pulmonary vascular resistance.

We agree with Augoustides that proper control of PaO₂ between the groups should be mentioned to clarify any confounding factors.