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Reply to the Editor:

We are pleased that Drs Filsoufi and Carpentier enjoyed our article, but they may have misinterpreted our results. We are not aware of other studies of systolic anterior motion (SAM) that include early and late intraoperative echocardiography. Our study also differs from others in that we have included only patients with mitral valve pathologies at risk of SAM development. Some authors do not make this distinction and report an artificially low incidence.[1] The 11% incidence of SAM, which Drs Filsoufi and Carpentier mention, includes all patients with SAM on early intraoperative transesophageal echocardiography. After medical management, the incidence of SAM intraoperatively decreases to 6% and then to 4% by hospital dismissal. Previous investigations that estimate the risk of SAM have reported echocardiographic findings at varying times, and therefore comparisons are difficult.[1-3] But the important point is that the risk of SAM appears low and diminishes with time and ventricular remodeling.

Therefore the question of whether prophylactic measures should be used to "prevent SAM" is a bit more complicated than the correspondents might be thinking. As noted in the article, the risk of development of symptoms related to late SAM using the techniques we describe is extremely low (16/17 patients in New York Heart Association class I, 1 patient lost to follow-up). Also, SAM has been described with all methods of valve repair, including quadrangular resection with a sliding leaflet technique.[4-7]

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Tricuspid valve repair: Indication and type of repair

To the Editor:

We read with great interest the article by Ghanta and colleagues[1] concerning their results with repair of functional tricuspid regurgitation. After the comparison between Kay's annuloplasty versus ring annuloplasty on the tricuspid valve, they concluded that there is not a significant difference in the midterm results. Ghanta and colleagues recommend bicuspidization annuloplasty as an option to ring annuloplasty because it is inexpensive, simple, and reliable.

We do agree with the authors that moderate-to-severe tricuspid regurgitation should be repaired when a concomitant cardiac procedure is performed. At our institution, we are rather liberal in the indication for tricuspid repair. We perform about 50 cases of repair yearly on the tricuspid valve (46–61 per year in the last 3 years) of approximately 1200 total cardiac procedures. Our experiences show that repair of tricuspid regurgitation improves early postoperative outcome and long-term quality of life. Long-term survival after cardiac surgery depends mainly on the postoperative left ventricular performance. Indications for tricuspid valve repair should be decided preoperatively, according to the results of echocardiography. Intraoperative evaluation of tricuspid regurgitation is not reliable enough.

The method of repair on the tricuspid valve is another question. Cohn[2] recommends use of a ring if the tricuspid regurgitation is severe and suture annuloplasty if it is moderate. We have had good experiences with both de Vega annuloplasty and Kay's suture bicuspidization. In our experience (published in 1980), we found that, in about 70% of the cases, tricuspid repair (de Vega or Kay's) provided good results at a mean follow-up of 4 years.[3] Since then, we used mostly de Vega repair with multifilament suture material, and we have had good experiences in several hundred cases. We even used an adjustable half-moon ring for tricuspid annuloplasty in a group of patients with good results.[4] The ring annuloplasty had no better results compared with those of the suture annuloplasty, it is more expensive, and it takes more time and can reduce annular flexibility. That is why we do not use it routinely.

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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 then 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 colleagues[1] detailing their experience with oral sildenafil in intraoperative 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 vasodilation[2]:

1. Cyclic adenosine monophosphate pathway with inhaled prostaglandin I_2
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 (PaO_2) 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 PaO_2 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 PaO_2 in large pulmonary vessels.[2] However, no comprehensive data exist regarding graded response of the pulmonary vasculature to changes in PaO_2 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 Yuan[3] studied the relationship between PaO_2 and pulmonary vascular resistance and demonstrated that there is no further decrease in pulmonary vascular resistance above the PaO_2 of 50 to 60 mm Hg. Therefore, although the PaO_2 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 PaO_2 between the groups should be mentioned to clarify any confounding factors.