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Reply to the Editor:

I greatly appreciate Dr Bassano and his colleagues' interest in our article.¹ I read the results of their clinical data with interest as well. They found a steep increase in the rate of aortic expansion at an aortic diameter of 50 mm with strong statistical significance even in the analysis on a relatively small number of patients. This result is quite different from that of our previous study in which no significant correlation was found between preoperative aortic diameter and the aortic expansion rate after aortic valve replacement (AVR).¹ Perhaps these discordant results may be attributable to differences in the patient cohorts, but I believe they need to look back at their data to see whether any other plausible factors exist as confounders (eg, connective tissue disorder) behind their correlation.

There have been mixed conclusions in the literature on whether to replace a moderately dilated aorta or not during AVR, and controversies are ongoing.²⁻⁴ Our previous study showed that AVR alone seemed reasonable in a moderately dilated ascending aorta based on a low rate of clinical events and aortic aneurysm formation as well as the limited rate of aortic expansion, but the study was not meant to be conclusive; its results were believed to be provocative of further studies to define a more reasonable indication for concomitant replacement of the aorta. Safety in concomitant aortic surgery is another issue to be addressed. Although there was no operative mortality among 70 patients who underwent aorta replacement in our series, I don't agree that it can be performed "without significant

additional risks." Evaluations on tens of patients are not enough to determine the risk of a procedure, and I believe that any additional procedure always carries a certain level of risk. What is important is how great the risk is. Current risk calculating systems for cardiovascular surgery, EuroSCORE II for instance, also consider this "additional procedure" as the factor that increases early mortality calculated in a logistic function. Because a dilated aorta is a common finding during AVR, the recommendations we make may affect a huge number of patients, even if the increased risk of concomitant aorta surgery is small. Thus, risk-benefit assessments on concomitant aorta surgery should be made through large-scale studies such as multiinstitutional registry-based analyses.

We found that the rate of aortic expansion was -5.6 ± 8.0 mm/y in aortic stenosis, -2.6 ± 5.2 mm/y in mixed stenoregurgitation, and -1.4 ± 4.5 mm/y in aortic regurgitation; the differences were statistically marginal as shown in our previous study ($P = .083$ using the Kruskal-Wallis test).¹ When we compared the expansion rates only between aortic stenosis and aortic regurgitation, the difference was statistically significant ($P = .003$ using the Mann-Whitney U test), in concordance with Dr Bassano and colleagues' assumption. This is an unexpected finding in my view, and Dr Bassano's hypothesis may explain the mechanism behind this phenomenon, but other plausible mechanisms can be speculated as well. Further studies are also needed to address this issue, and I believe the study population should be extended to those with all sizes of aorta rather than confining it to a certain range to reach an appropriate conclusion on this.

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Reply to the Editor:

We appreciate much the interest in our report.¹ The issues raised by this letter can be summarized as follows: (1) what was the etiology of the tricuspid regurgitation (TR); (2) what was the pulmonary artery pressure (PAP) level; and (3) whether TR associated with a left-sided cardiac lesion will exhibit a poorer prognosis?

With regard to the etiology of TR, 8 patients had a history of blunt chest trauma, all of whom showed leaflet prolapse. For these patients, the etiology would probably fit into "traumatic" but that might not be confirmative. Another 12 patients had leaflet prolapse; however, a history of chest trauma was not evident. This could have been, in part, because some patients might not recall the traumatic event that happened long ago. Although the etiology, in these cases, could have been degenerative or traumatic, it would be better for it to remain as "unknown." Regarding rheumatic involvement, the presence

of leaflet thickening might best represent rheumatic disease, which was seen in 4 patients. Another 27 patients had no leaflet pathologic features (only annular dilatation with or without tethering), and it could be defined as “idiopathic” or “functional.” As such, specifying the etiology in every case would have been very difficult or perhaps impossible in some cases. Therefore, we believe that describing the pathologic features (leaflet abnormality, annular dilatation, tethering), rather than specifying the etiology, was a more reasonable method.

The PAP can be estimated by the levels of the TR pressure gradient, which were 27.5 ± 9.0 mm Hg and 22.6 ± 7.7 mm Hg for those undergoing tricuspid valve repair and replacement, respectively, as described in our Table 2.¹ Generally, systolic PAP has been estimated by this value, with 5 mm Hg added for the estimated right atrial pressure.² Given that patients with severe TR tend to have a higher right atrial pressure, the addition of ≤ 15 to 20 mm Hg might be needed in some cases. Nevertheless, the estimated PAP was still within normal range, as expected, because the patients did not have any left-sided cardiac lesions.

Finally, regarding the influence of PAP on the long-term outcomes in patients with severe TR, we have tried to address this issue by analyzing patients with severe TR of all etiologies and the results now available from a published study.³ In that study, 449 patients were included. Of these patients, 60 (13.4%) had a history of left-sided valve surgery and 359 (80.0%) had undergone concomitant left-sided valve surgery. On multivariate analysis, survival was affected by advanced heart failure symptoms, comorbidities, end-organ dysfunction, and laboratory abnormalities, but not by the type of surgery, causes of TR, or PAP level. It is well known that

the preoperative PAP level is associated with late outcomes after left-sided valve surgery; however, we also know that the surgical outcomes of primary isolated TR (with normal PAP) have also been poor, which has been more related to right ventricular dysfunction. Therefore, we have assumed that the effects of “higher PAP” in patients with secondary TR and “normal PAP” in those with primary TR have been counterbalanced in the long-term outcomes. However, we also believe this issue should be addressed in additional studies with more sophisticated designs.

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EXTRACORPOREAL MEMBRANE OXYGENATION RESUSCITATION IN ADULT PATIENTS WITH REFRACTORY SEPTIC SHOCK

To the Editor:

We read the interesting report by Huang and colleagues,¹ in which they described their experience using extracorporeal membrane oxygenation (ECMO) for adults with refractory septic shock. The investigators

conclude that ECMO should not be regarded as a rescue modality in patients with refractory septic shock, as highlighted by impaired hemodynamics and progression of inflammation-mediated organ failure.

The findings of profound shock and multiple organ dysfunction among these patients supported by ECMO, despite appropriate antibiotics and high-dose inotropes, redirect our concern about a profound inflammatory response, which possibly affects their pharmacokinetics. Recently, Angus and Van der Poll² inferred that proinflammatory reactions lead to collateral tissue damage in severe sepsis, and impaired tissue oxygenation plays a vital role in organ dysfunction. Moreover, it is well known that venoarterial ECMO provides oxygenation and hemodynamic stability, although it does not filter the uncontrolled, circulating, acute inflammatory mediators that worsen tissue damage.

Currently, extracorporeal blood purification therapies have been proposed to limit these circulating inflammatory mediators,³ but they do not yet seem to be used in mainstream clinical practice because of their practical and theoretic limitations. In our experience,⁴ a combination of extracorporeal life support and plasma purification by resin adsorption has been shown to play an important role in the removal of proinflammatory cytokines in the early phase of septic shock, thereby preventing further hemodynamic deterioration and reducing the risk of multiple organ dysfunction. In this ex vivo study, the extracorporeal blood flow through the resin adsorption filter was set at a flow rate of 300 mL/min to achieve higher single-pass clearance and quantities of acute inflammatory mediators. The improvements in hemodynamics were reflected in a decrease in the mean pulmonary artery and central venous pressure, and a concurrent increase in systemic vascular resistance was observed in