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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<http://dx.doi.org/10.1016/j.jtcvs.2013.12.036>

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

the study group with significant levels of adsorption of the measured cytokines. Further decrease in interleukin-6 and tumor necrosis factor α levels were observed even after stopping the adsorption process, possibly as a result of the compensatory antiinflammatory response syndrome.⁵

Therefore, we can assume that early resolution of hemodynamics might significantly influence the survival outcome. Thus, adjuvant plasma purification therapy during ECMO support should be considered to adsorb unregulated quantities of acute inflammatory mediators. Nonetheless, further large-scale studies are necessary to establish whether a

combination of extracorporeal life support and plasma purification by resin adsorption may play an important role in preventing further hemodynamic deterioration and reducing the risk of multiorgan failure in septic shock.

We are grateful to Huang and colleagues¹ for sharing their experience and knowledge in this excellent study and for giving an important insight into such a controversial issue.

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