of Thoracic Surgeons recently started detailed data collection on devices; the information is incomplete, however, and it will take a long time to achieve enough data and statistical power to detect small differences in valve outcomes. In the interim, we should consider collection of explants and evaluation of autopsy reports to get more information on causes of death and to provide key information for decision making for regulators, patients, and surgeons.

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SURGEONS AND DEVICES IN AORTIC VALVE REPLACEMENT

Reply to the Editor:

Although critical to our understanding of clinical medicine, retrospective study—even with the most refined statistical analysis—is fraught with well-known limitations. Moreover, the limited ability of administrative data to answer clinical questions is legendary. The medical literature is replete with scientifically sound hypotheses supported by robust anecdotal and retrospective data that failed to withstand the scrutiny of prospective randomized evaluation. While our medical colleagues are still trying to sort out the dramatic failures of hormone therapy in postmenopausal women,¹ our own cardiac surgical community is absorbing the failure of statins to affect the progression of aortic valve disease,² and, more recently, the failure of a restrictive blood transfusion strategy to improve

clinical outcomes.³ It is certainly possible that the evolution of devices has rendered earlier prospective, randomized control trials of mechanical versus prosthetic valves irrelevant. However, if it is the valve (or the requisite technical or medical aspects of valve selection) that is specifically driving the observed difference in mortality, then the difference should be reflected specifically in the valve-relatedness of the mortalities observed. In the absence of any such signal, the distinction between valve selection and quality of care will await more convincing data. However, with the emerging success and increasing application of transcatheter aortic valve replacement, the question may well remain unanswered.

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EPIDURALS, INTRAOPERATIVE FLUID ADMINISTRATION, AND HYPOTENSION

To the Editor:

I enjoyed the recent article, “Effect of the amount of intraoperative fluid administration on postoperative pulmonary complications following anatomic lung resections” by Arslantas and colleagues¹ in the January issue of *Journal of Thoracic and Cardiovascular Surgery*.

Our group agrees that all patients undergoing anatomic pulmonary resection should have limited fluid administration because it decreases the number of postoperative pulmonary complications. We have seen substantially greater amounts of fluid administration in patients who receive epidural catheters for pain control, and in recent years have decreased our use of epidural catheters for this reason. Our impression is that the reduced fluid administration as well as the reduced incidence of intraoperative and postoperative hypotension decreases postoperative complication rates.

The study by Arslantas and colleagues¹ noted that epidural use was not significantly different between patients

with and without pulmonary complications. Was there any difference in the fluid administration between those receiving epidural catheters and those not receiving epidurals?

A follow-up question is whether there was any difference in intraoperative/postoperative hypotension in patients receiving epidural catheters. Finally, it did not appear that intraoperative hypotension was listed as a risk factor for postoperative pulmonary complications. Did intraoperative hypotension have any effect on pulmonary complications?

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EPIDURALS, INTRAOPERATIVE FLUID ADMINISTRATION, AND HYPOTENSION

Reply to the Editor:

We thank Baciewicz for his comments and questions on our article.¹

When anatomic lung resections are performed through a posterolateral thoracotomy, thoracic epidural analgesia is a preferred method in the most centers, and its use has been associated with lower pulmonary morbidity in previous studies.²

In our cohort, patients who had epidural analgesia ($n = 72$) and who did not ($n = 67$) had received almost the same amount of crystalloid and colloid infusion (crystalloid infusion of 1663 ± 980 mL and colloid infusion of 250 ± 351 mL vs crystalloid infusion of 1646 ± 1013 mL and colloid infusion of 250 ± 422 mL, respectively) (Figure 1). Thoracic epidural catheters were placed preoperatively. After administration of a test dose (3 mL of 1.5% lidocaine with epinephrine [1:200,000]), a 5-mL bolus injection of 2.5% bupivacaine and fentanyl (1 μ g/mL) was administered, and this was followed by a continuous infusion of 5 mL/h. Postoperatively, patient-controlled analgesia was applied. We did not observe any adverse effects as a result of thoracic epidural catheter placement.

We attribute this outcome to 2 main reasons: (1) The local anesthetic dose in our study was relatively low, and thus it did not lead to high levels of sympathectomy to affect hemodynamics. (2) Vasopressors were started immediately without allowing any intraoperative hypotension.

FIGURE 1. Intraoperative infusion rate greater than 6 mL/kg/h was associated with postoperative pulmonary complications. Reprinted from Arslantas MK, Kara HV, Tuncer BB, Yildizeli B, Yuksel M, Bostanci K, et al. Effect of the amount of intraoperative fluid administration on postoperative pulmonary complications following anatomic lung resections. *J Thorac Cardiovasc Surg.* 2015;149:314-20, 321.e1.

We also believe that intraoperative hypotension is a possible cause of postoperative organ dysfunction, including pulmonary complications. Regarding our data, however, we cannot give a strong statement about the association between intraoperative hypotension and postoperative pulmonary complications, because our patients were not allowed to become hypotensive intraoperatively.

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