strategies other than pharmacologic treatment, or the use of other drugs of the same class with predominantly analgesic effects not affecting brain receptors (eg, morphine derivatives) may induce the surgeon to refrain from administering a life-threatening drug cocktail that may be responsible for triggering or worsening delirium.

Francesco Pollari, MD
Giuseppe Santarpino, MD

Theodor Fischlein, MD
Department of Cardiac Surgery
Klinikum Nürnberg
Nuremberg, Germany

References

1. Arenson BG, MacDonald LA, Grocott HP, Hiebert BM, Arora RC. Effect of intensive care unit environment on in-hospital delirium after cardiac surgery. *J Thorac Cardiovasc Surg.* 2013;146:172-8.
2. Inouye SK. Delirium in older persons. *N Engl J Med.* 2006;354:1157-65. Erratum in: *N Engl J Med.* 2006;354:1655.
3. Santarpino G, Fasol R, Sirch J, Ackermann B, Pfeiffer S, Fischlein T. Impact of bispectral index monitoring on postoperative delirium in patients undergoing aortic surgery. *HSR Proc Intensive Care Cardiovasc Anesth.* 2011;3:47-58.
4. Martin BJ, Buth KJ, Arora RC, Baskett RJ. Delirium: a cause for concern beyond the immediate postoperative period. *Ann Thorac Surg.* 2012;93:1114-20.
5. Zeller A, Crestani F, Camenisch I, Iwasato T, Itoharu S, Fritschy JM, et al. Cortical glutamatergic neurons mediate the motor sedative action of diazepam. *Mol Pharmacol.* 2008;73:282-91.

<http://dx.doi.org/10.1016/j.jtcvs.2013.09.079>

Reply to the Editor:

We are appreciative and in agreement with the point of view that Pollari and colleagues have expressed regarding our study outlining some of the risk factors for postoperative delirium after cardiac surgery. One of the messages inherent in our observational study is that certain potentially modifiable factors can, if appropriately commented on the study by Santarpino and colleagues examining the effect of cerebral monitoring as a method of identifying patients who might have a greater incidence of delirium. Although it is exciting that the use of

a cerebral monitor might represent a clinically available biomarker, the data supporting the use of processed electroencephalographic monitoring (eg, the bispectral index and near-infrared spectroscopy cerebral oximetry) are quite sparse. Importantly, their report appears to have confused near-infrared spectroscopy and processed electroencephalographic monitoring. Thus, although we are appreciative of their interest in our report, their points regarding near-infrared spectroscopy would be much better supported by a study that had not used a processed electroencephalography (bispectral index) to estimate the cerebral saturation. Finally, we do agree with the authors that a focus on preventative measures is a necessary direction of future research in addressing delirium in the postoperative cardiac surgery patient.

Benjamin G. Arenson, BSc^a
Lindsey A. MacDonald, BSc^b
Hilary P. Grocott, MD, FRCPC^a
Brett M. Hiebert, MSc^c
Rakesh C. Arora, MD, PhD, FRCSC^d

^aDepartment of Anesthesia and
Perioperative Medicine
^bUniversity of Manitoba Faculty of
Medicine

Winnipeg, Manitoba, Canada
^cCardiac Sciences Program
Winnipeg Regional Health Authority
Winnipeg, Manitoba, Canada

^dDepartments of Anesthesia and
Perioperative Medicine and Surgery
University of Manitoba Faculty of
Medicine
Winnipeg, Manitoba, Canada

<http://dx.doi.org/10.1016/j.jtcvs.2013.11.059>

THE EFFECTS OF BICUSPID AORTA

To the Editor:

Congratulations to Katayama and colleagues¹ for this study showing that tricuspid valves are superior in

the aortic position. The diameter of the aorta increases 10% in systole relative to diastole. Tricuspid aortic leaflets do not resist this increase, but bicuspid aortic leaflets do resist because of their position. Bicuspid aortic leaflets thus undergo trauma during systole. In addition, tricuspid aortic leaflets get closer to the aortic wall during systole than do bicuspid ones. This causes bicuspid aortic leaflets to be exposed to more friction and shear stress. Twisting of the ascending aorta laterally and dorsally and localization of the coaptation line of bicuspid leaflets, however, were ignored in this simulation. The differences in the characteristics will influence the flow patterns. Configuration of the aortic valve and ascending aorta should therefore be considered in the simulation.

The noncoronary wall has the highest dp/dt in the ascending aorta. Therefore, in people with normal (tricuspid) aortic valves, the longest free edge is that of the noncoronary leaflet. Ninety-five percent of aortic dissections start from the wall of the noncoronary cusp. One of the most important factors affecting the surgical success of repair of bicuspid aorta is the localization of the commissure on the annulus. The localization of the commissures at the anteroposterior position on the annulus would increase surgical success. In this position, the noncoronary wall of the aorta would be less influenced by the dp/dt ratio. We think that the anatomic positions of the commissures on the annulus should be taken into consideration in this study.

Habib Cakir, MD
Mert Kestelli
Ismail Yurekli, MD
Koksal Donmez, MD
Department of Cardiovascular
Surgery

Katip Celebi University Izmir Atatürk
Training and Research Hospital
Izmir, Turkey

Reference

1. Katayama S, Umetani N, Hisada T, Sugiura S. Bicuspid aortic valves undergo excessive strain during opening: a simulation study. *J Thorac Cardiovasc Surg.* 2013;145:1570-6.

<http://dx.doi.org/10.1016/j.jtcvs.2013.08.088>

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.

Joon Bum Kim, MD, PhD
Department of Thoracic and
Cardiovascular Surgery
Asan Medical Center

University of Ulsan College of
Medicine
Seoul, Korea

References

1. Lee SH, Kim JB, Kim DH, Jung SH, Choo SJ, Chung CH, et al. Management of dilated ascending aorta during aortic valve replacement: valve replacement alone versus aorta wrapping versus aorta replacement. *J Thorac Cardiovasc Surg.* 2013;146:802-9.
2. Borger MA, Preston M, Ivanov J, Fedak PW, Davierwala P, Armstrong S, et al. Should the ascending aorta be replaced more frequently in patients with bicuspid aortic valve disease? *J Thorac Cardiovasc Surg.* 2004;128:677-83.
3. McKellar SH, Michelena HI, Li Z, Schaff HV, Sundt TM III. Long-term risk of aortic events following aortic valve replacement in patients with bicuspid aortic valves. *Am J Cardiol.* 2010;106:1626-33.
4. Girdauskas E, Disha K, Raisin HH, Secknus MA, Borger MA, Kuntze T. Risk of late aortic events after an isolated aortic valve replacement for bicuspid aortic valve stenosis with concomitant ascending aortic dilation. *Eur J Cardiothorac Surg.* 2012;42:832-7.

<http://dx.doi.org/10.1016/j.jtcvs.2013.12.035>

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