caused by intraoperative factors can be often detected between 1 and 3 days after surgery. Thereafter, the AKI is more likely to be attributable to postoperative factors, such as hemodynamic instability, anemia, transfusion, and so on. Westenbrink and coworkers⁵ have found that postoperative anemia is common after CABG, frequently persists for months, and is associated with impaired postoperative outcomes. We therefore emphasize that efforts to avoid the modifiable risk factors for AKI should be continued in the postoperative period to prevent new AKI or aggravation of existing kidney injury by postoperative factors, especially for patients who are at high risk for AKI.

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Reply to the Editor:

We thank Xue and colleagues for their interest in our publication, in which we showed the potential modifiable risk factors of acute kidney injury (AKI) after coronary artery bypass grafting.¹

The focus of this study was on studying the effects of 3 known modifiable risk factors, the lowest hematocrit during cardiopulmonary bypass (CPB), preoperative anemia, and intraoperative red blood cell transfusion, on postoperative AKI in our Asian patient population with smaller average body sized. As such, we did not look in this study specifically at the effects of intraoperative hemodynamic changes and their potentiation of hemodilutional anemia to result in postoperative AKI. The intraoperative systolic blood pressure was typically maintained within a tight range of 100 to 130 mm Hg, however, it is likely that a greater decrease in systolic blood pressure would be present in hypertensive patients. We also showed a history of hypertension to be an independent risk factor for AKI,¹ albeit an unmodifiable factor.

We are cognizant that liberal pre-CPB crystalloid infusion may result in hemodilution, especially for patients with smaller body size. Although the pre-CPB crystalloid infusion volume was not recorded, it was standard practice to give no more than 1000 mL of crystalloid before CPB.

The influences on AKI of perioperative medications, such as diuretics, inotropes, statins and antiarrhythmics, were studied. Of these, only loop diuretics were significantly associated with AKI in the univariate analysis presented in our article.¹ None of the medications were independently associated with AKI in the multivariate analysis.

In our study, postoperative creatinine measurements were done routinely in the intensive care unit in accordance

with institutional protocol. Continuous creatinine measurements were not done. The peak serum creatinine was the highest creatinine value obtained within the first 3 postoperative days and was used to calculate against the preoperative serum creatinine to determine whether a patient had postoperative AKI. AKI was defined according to the Acute Kidney Injury Network (AKIN) stage 1 criteria in this study.

To compare postoperative AKI incidence with other study cohorts, we had to use a uniform and standard classification of AKI. Numerous articles have used either the RIFLE (Risk, Injury, Failure, Loss of function, and End-stage renal disease) or AKIN criteria to define AKI. We chose the AKIN criteria because they account for smaller changes in serum creatinine, which is shown to be associated with adverse outcomes such as mortality.² Moreover, the AKIN criteria use a shorter time frame of 48 hours to assess renal function than the RIFLE criteria, which has a 7-day window period for AKI diagnosis.³

We found that not only was postoperative anemia associated with postoperative AKI, it was independently associated with the persistence of AKI up until hospital discharge (unpublished data). We therefore agree that strategies to improve postoperative anemia can reduce the persistence of AKI and prevent the subsequent development of chronic kidney disease in cardiac surgical patients.

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IS EARLIER AORTIC VALVE SURGERY IN REPAIR CANDIDATES JUSTIFIED?

To the Editor:

I read with great interest the report by Sharma and colleagues¹ about their experience with aortic valve (AV) repair and congratulate them on their honest presentation of the long-term data. I believe, however, that the interpretation of some aspects needs additional comments and questions. The authors stated that survival after AV repair in their population was excellent and, with 91%, 80%, and 58% survival at 5, 10, and 15 years, respectively, was indistinguishable from the healthy population. Such a survival of the healthy population with a median age of 54 years seems to be rather low, and this impression could be intensified when looking at the US Life Tables,² which revealed corresponding survival rates of 96%,

90%, and 83% for a 54-year-old population containing 76% men. The authors found (not unexpectedly, because this aspect has already been revealed after AV replacement³) that myocardial impairment (MI) was a significant predictor of mortality. They correctly noted that this is barely avoidable, because the late presentation of symptoms in some patients with aortic insufficiency does not lead to referral for surgery until the development of MI. They suggested, however, earlier surgery for patients with suitable repair anatomy. In this context, 2 questions arise: how reliable is the assessment of AV repair suitability; and are patients with MI proper candidates for AV repair? I believe AV replacement should be preferred for patients with considerable MI because such patients need a fast and safe surgery without the risk of residual or recurrent insufficiency, let alone any intraoperative revisions. The remaining aortic insufficiency, which can never be ruled out after repair, can even be considered as a potential factor that can worsen long-term survival in this special subgroup. However, the authors did not provide the rate of postoperative aortic insufficiency—neither in the entire nor in this special cohort. Thus, I am wondering whether the rate of reoperation alone would be adequate enough for assessment of clinical and, especially, functional results. Furthermore, the number of patients operated at Mayo Clinic, with an average of about 12 annually, does not indicate that a considerable share of aortic valve pathologic entities could be scheduled for the repair. By coincidence, we submitted an abstract for the same American Association for Thoracic Surgery meeting that included 137 repairs performed during a 1-year period, but it was not well received by the reviewers.

It seems that AV repairs are still limited to single centers or, even, surgeons; hence, we can hardly speak about the "expanding relevance of

AV repair." The repair rate, as presented in our abstract, was 40% for all AV surgery patients younger than 70 years or, after excluding patients with contraindications (eg, MI), even greater. Even if the suitability of repair could be assessed with a probability of about 80%, the final decision for AV repair was always performed during surgery. Thus, I am convinced that AV repair is a valuable option; however, the repair candidates must fulfill the same indication criteria as for AV replacement,³ because the need for replacement can never be ruled out.

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COX MAZE III PROCEDURE: THE BEST ALTERNATIVE IN SURGERY FOR ATRIAL FIBRILLATION

To the Editor:

We read with particular attention the article by Stulak and colleagues.¹ After close scrutiny of this report one cannot escape the conclusion that standard cut-and-sew Cox maze III procedure, as first described by Cox and associates,² remains the criterion standard choice for the surgical treatment of any type of atrial fibrillation (AF). Of special interest is the entity of mitral valve (MV) disease with concomitant long-standing persistent