BETTER TO AVOID DISASTER THAN RESCUE DEFEAT—VENTRICULAR DYSFUNCTION AFTER DELAYED MITRAL VALVE REPAIR

Reply to the Editor:

We appreciate the interest of Buckberg and Athanasuleas in our recent article on ventricular function in patients with American College of Cardiology and American Heart Association stage C1 disease undergoing early mitral valve repair for degenerative mitral regurgitation (MR) with “normal” ejection fraction ($\geq 60\%$) according to class IIa indications.¹ The scientific understanding of myocardial protection has, historically, benefited from the work of Buckberg. Specifically, for patients with MR the improvement in operative survival² reaffirms the progress made in the safe conduct of cardiac surgery. The vexing issue of postoperative paradoxical septal motion to which he refers may indeed warrant further investigation, and we applaud the efforts of Buckberg and colleagues in this regard.

There are, however, several, perhaps unintended, misrepresentations in the letter that warrant clarification. First, Buckberg and Athanasuleas have incorrectly cited crossclamp time as 60 minutes, whereas it was in fact listed in Table 1 of our article as 43 minutes overall and 42 minutes in the dysfunction group. This number reflects median cardioplegia time for the entire series, including both open and robotic mitral valve repair operations, and for all mitral valve prolapse categories (from single scallop posterior leaflet to complex multiscallop bileaflet prolapse). Moreover, the procedures were performed by an array of surgeons in our practice over time, which further diminishes the likelihood that cardioplegia strategy was influential. Second, postoperative left ventricular dysfunction may result not only from potential intraoperative ischemia but also from unrecognized preoperative left ventricular

impairment, as noted in our report. Several additional studies have also demonstrated a link between preoperative ejection (1) fraction and (2) end-systolic dimensions and the postoperative (1) echocardiographic and (2) clinical outcomes.^{3–7} Third, recent data reaffirm that preoperative indices of LV size and function are predictive not only of postoperative events but also of poor outcome under medical management of patients with severe degenerative MR,^{7,8} demonstrating that the specter of left ventricular dysfunction does not necessarily require cardiac surgery to manifest in this patient population. In the vast majority of individuals with organic MR and left ventricular dysfunction, these left ventricular alterations in fact predate surgery but may be difficult to detect because of the particular hemodynamics associated with chronic severe MR. Recent data also show that although decreased ejection fraction ($<60\%$) is of great concern in those with MR under all forms of clinical management,^{5,8} an ejection fraction of at least 60% is not entirely reassuring, with a mortality under medical management that does not decline with increasing ejection fraction.⁸ Fourth, as shown in our study,¹ postoperative left ventricular dysfunction is specifically associated with markers of delayed operation and is independently predictive of excess postoperative mortality, despite expeditious intraoperative course and successful mitral valve repair. Fifth, the observed postrepair diminution in ejection fraction and its prognostic impact have not been heretofore identified in any valve subgroup in our practice to date, further reaffirming the efficacy of the myocardial protection used. Finally, in reference to the changes in left ventricular function associated with percutaneous therapy in patients with inoperable MR who undergo merely palliative diminution of regurgitant volume burden with currently existing percutaneous devices⁹ this vastly

different from early mitral valve patients undergoing contemporary surgical operations. Surgical mitral valve repair is designed to definitively address the regurgitant lesion with, at most, trivial to mild regurgitation as the intended aim and expected standard.

In summary, although we applaud the efforts of Buckberg and colleagues to improve myocardial protection, it is imperative to realize that the dangers associated with untreated severe MR extend beyond the particular electrolyte formulations used during the procedure. The false reassurance associated with apparently “normal” left ventricular function endangers patients when clinicians fail to provide options for safe and effective MR correction. Efforts should thus be directed toward promoting early repair by experienced heart teams capable of providing the best possible outcomes to patients with severe organic MR.¹⁰

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SIZE MATTERS

To the Editor:

I congratulate Oki and colleagues,¹ who clearly demonstrated the benefit of combined single-scope endobronchial ultrasound (transbronchial; EBUS) and endoscopic ultrasound (transesophageal; EUS) staging relative to EBUS alone (sensitivity for mediastinal nodes, 73% vs 52%). Since the Assessment of Surgical Staging Versus Endosonography (ASTER) trial,² guidelines have stated that mediastinal nodal tissue staging should start with endosonography.³ Whether optimal endosonographic staging can be achieved by EBUS alone or whether the EBUS plus EUS combination is needed, however, is subject to debate. Although it is obvious that 2 tests increase the rate of detected nodal metastases relative to a single procedure, the routine addition of EUS performed with the EBUS scope is under discussion, with a recent study reporting little sensitivity benefit

(increase from 82% to 85%).⁴ An important message of the study of Oki and colleagues¹ is that the sensitivity of EBUS alone in small nodes seems substantially lower than the sensitivity of EBUS reported in a recent guideline (89%).³ It should be acknowledged that various of the initial EBUS studies reported outcomes of selected patients with either enlarged or fludeoxyglucose-avid nodes. These selected patients differ from consecutive patients in daily practice, who often have subcentimeter nodes, as in the study by Oki and colleagues.¹ The sensitivity of EBUS in routine practice will thus most likely be lower than initially reported, especially when dealing with patients with small nodes.

In my experience, these subcentimeter nodes in the often affected stations 4L (paratracheal left) and 7 (subcarinal) can be very elegantly sampled by EUS-B. Oki and colleagues¹ clearly demonstrated that EUS-B has an added value to EBUS alone even if the same node had been sampled by EBUS before, particularly in the case of station 7. This is an important take-home message: EUS-B not only has an added value in evaluating nodes located out of reach of EBUS but also in evaluating those within reach of EBUS that have been punctured before. An explanation for this finding might be the far easier sampling by the esophageal route because of the absence of cartilage rings and cough.

With regard to the performance of EUS-B, however, I wonder why all the patients in the study of Oki and colleagues¹ were turned from a supine to a left lateral position when switching from EBUS to EUS-B. The latter position is the classic approach for conventional EUS, but in my experience it is not necessary for an adequate mediastinal evaluation. Simply repositioning the EBUS scope from the trachea into the esophagus in single elegant move—without turning the patient—further

simplifies the combined EBUS and EUS-B procedure.

The study by Oki and colleagues¹ underscores the importance of the EUS-B approach, showing a large diagnostic benefit by adding just 10 minutes of procedure time. It is now time for EUS-B implementation, with the mental switch for pulmonologists and surgeons to enter the esophagus probably being the largest barrier.⁵

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TWO TECHNIQUES ARE BETTER THAN ONE

Reply to the Editor:

We thank Dr Annema for his insightful comments regarding our study¹ evaluating the roles of combined endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA) and transesophageal endoscopic ultrasound-guided fine-needle aspiration (EUS-FNA) with a single EBUS