2-dimensional speckle tracking and
3-dimensional echocardiography.

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STAGED PERCUTANEOUS CORONARY INTERVENTION AND MINIMALLY INVASIVE VALVE SURGERY: RESULTS OF A HYBRID APPROACH TO CONCOMITANT CORONARY AND VALVULAR DISEASE

To the Editor:

We read with great interest the recent publication by Santana and colleagues,¹ entitled "Staged Percutaneous Coronary Intervention and Minimally Invasive Valve Surgery: Results of a Hybrid Approach to Concomitant Coronary and Valvular Disease." A total of 65 patients who underwent staged percutaneous coronary intervention and minimally invasive cardiac surgery (MICS) valve surgery were compared with 51 patients who underwent combined valve/coronary artery bypass grafting. The authors are to be congratulated for their excellent short-term

results in the MICS group, particularly the lack of mortality. However, drawing conclusions between the 2 groups is problematic in that there were 6 different surgeons in the control group versus 1 surgeon in the MICS group. It is well known that mortality and morbidity rates in valve surgery are highly correlated to the caseload of the individual surgeon.² We would question if the 28% versus 1.5% cumulative incidence of adverse short-term outcomes, including death, stroke, and renal failure, in the control versus the experimental group is merely a reflection of this fact.

Another concern is 61 of 65 patients in the hybrid group had percutaneous coronary intervention of the left anterior descending artery before MICS, and only 55.5% of the stents were drug eluting. In view of the the well-documented durability of the left internal thoracic artery to the left anterior descending artery, with both its salutary impact on longevity and major adverse cardiac outcomes, are these patients possibly being shortchanged in the zeal to perform MICS?

We also noted that in the MICS mitral cohort, there was only a 58% repair rate (14/24) despite no mention of rheumatic causes. Other than 6 calcific mitral valves, it seems plausible that the repair rate should have been higher in the hands of such a high-volume surgeon. Again, perhaps the lack of adequate exposure inherent to a "4- to 5-cm" MICS incision compromised the ability to attain a satisfactory repair.

Finally, nowhere in the article was there any mention of atrial fibrillation (AF). It is unlikely that none of these patients would have any preoperative history of such. Ad and colleagues³ recently reported that only 40.6% of patients with a history of AF have it addressed at the time of concomitant heart surgery. Knowing the favorable effects of a concomitant Cox-maze on ejection fraction, stroke, and life span, as well as the fact that this procedure can be applied via a MICS

approach, the authors should reconsider their strategy in those patients with a significant history of AF.

We applaud this and other efforts to make cardiac surgery less invasive. However, suboptimal mitral repair rates and infrequent use of the left internal thoracic artery, as well as overlooking preoperative AF, may significantly compromise long-term patient outcomes.

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ADENOSINE-PROCAINE CARDIOPLEGIA AND ADENOSINE-LIDOCAINE CARDIOPLEGIA: TWO SIDES OF THE SAME COIN?

To the Editor:

Jakobsen and colleagues¹ are to be congratulated for pursuing alternatives to supranormal potassium cardioplegia in cardiac surgery. Since a 15-year moratorium was placed on Melrose's hyperkalemic solution in the late 1950s, investigators in cardioprotection have attempted to find a method

to achieve polarized arrest that reduces (1) Na^+ and Ca^{2+} loading, (2) vasoconstriction, (3) endothelial dysfunction, (4) reperfusion arrhythmias, and (5) contractile stunning.² In the 1990s, adenosine triphosphate-sensitive K^+ channel openers provided initial hope for a method to induce polarized arrest³; however, severe post-arrest arrhythmias thwarted clinical adoption.

The clinical study by Jakobsen and colleagues¹ tested the hypothesis that cold 1.2 mmol/L adenosine crystalloid cardioplegia can achieve polarized arrest and provide superior cardioprotection compared with a 20 mmol/L hyperkalemic solution in low-risk patients. Their study found that adenosine cardioplegia arrested the heart faster, reduced the incidence of atrial fibrillation by more than 50%, and was associated with lower postoperative plasma K^+ levels. There were no differences in postoperative troponin T, creatine kinase-MB, creatinine, hemodynamics, cardiac index, or endothelial activation.¹

Adenosine has been investigated as a potential cardioprotective agent in cardiac surgery during the past several decades. In 1989, Schubert and colleagues⁴ were the first to show that 10 mmol/L of adenosine rapidly arrested the heart. However, persistent atrioventricular block⁴ precluded its further development as a polarizing arresting agent, and it became an adjunct to supranormal potassium.⁵ During the past 20 years, the benefits of adjunctive adenosine (<2 mmol/L) in cardioplegia have been mixed, ranging from modest effects⁵ to no benefits at all.⁶ The protocol reported by Jakobsen and colleagues¹ used 1.2 mmol/L adenosine to achieve polarized arrest. However, this concentration is approximately one tenth that used in earlier studies in which adenosine was the primary arresting agent.⁴ This discrepancy raises an important question of whether adenosine is really the sole arresting agent in Jakobsen's study.

In 2004, Dobson and Jones⁷ reported that adenosine (0.2 mmol/L) and lidocaine (0.5 mmol/L) (AL) in

a modified Krebs–Henseleit solution arrested the heart in a polarized state, with significantly improved functional recoveries compared with St Thomas' solution. Although both lidocaine and procaine are Na^+ fast-channel blockers, lidocaine was chosen in AL cardioplegia because as a class 1B antiarrhythmic, it shortens repolarization time, whereas procaine (class 1A) prolongs repolarization time, which may cause postoperative reanimation irritability. The AL concept of polarized arrest was subsequently confirmed in the canine model of cardiopulmonary bypass.⁸

Therefore, the efficacy of adenosine cardioplegia in the study by Jakobsen and colleagues¹ could be due to its combination with procaine. Whether adenosine acted alone or not is important because future studies testing adenosine as the primary arresting agent will derive their impetus from studies such as the one by Jakobsen and colleagues.¹ Precedence has been set for a polarized alternative using adenosine and local anesthetic (eg, lidocaine), which is the proprietary composition of Hibernation Therapeutics Global, Inc (Wicklow, Ireland) and is trademarked and available as Adenocaine in the United States from Central Admixture Pharmacy Services (Kent, Wash). A recent prospective randomized trial in Verona, Italy, showed superiority of an AL cardioplegic formulation in high-risk patients compared with a 4:1 Buckberg solution⁹; further clinical confirmation for use of AL as a primary arresting formulation that extends the data of Corvera and colleagues⁸ is forthcoming. The good news is that these studies collectively give hope that an alternative to supranormal potassium cardioplegia is on the horizon for the good of low- and high-risk pediatric and adult patients alike.

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Reply to the Editor:

We greatly appreciate the interest by Drs Vinten Johansen and Dobson in our article showing the efficacy and beneficial impact of adenosine instead of hyperkalemia in cold crystalloid cardioplegia. Their comments raise interesting and relevant questions regarding adenosine in cardioplegia