the sober judgment by Menasché² in his commentary. I am reminded of Robert Hutchison's litany, recounted here, "From inability to let well alone; from too much zeal for the new and contempt for what is old; from putting knowledge before wisdom, science before art, and cleverness before common sense; from treating patients as cases, and for making the cure of the disease more grievous than the endurance of the same, Good Lord, deliver us."³ It seems William Stewart Halstead's wisdom of reconstructing tissue planes is relegated to history.

Even Menasché² in his commentary recognizes that adhesions between the heart and sternum pose a major risk at reentry. He goes on to suggest that, for local problems, you have to find local solutions. I have been closing the pericardium completely for 34 years (more than 12,000 cases) for all operations, including coronary artery bypass grafting. In reoperative surgery, there is absolutely no risk at all of catastrophic hemorrhage if I have done the primary surgery. There is a safety cover over the heart, and amazingly few and flimsy adhesions between heart and pericardium, especially if it was washed thoroughly before closing. In many patients, some pericardial fluid also makes dissection of the heart easy.

The article by Lassaletta and colleagues¹ attempts to offer very imaginative and provocative solutions to postoperative adhesions. The article is not conclusive, because the adhesions were thought to be significantly less, and when the observer was blinded there was no difference between vodka-fed and wine-fed swine. Fortunately, there is no mention of the dose of vodka per kilogram or otherwise. This article might induce surgeons to prescribe a generous dose of vodka to patients who could be candidates for reoperations.

So what is the lesson from this article? Are we to look for exotic solutions anew, or simply follow basic

surgical principles of surgery and restore nature's protection to the heart?

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<http://dx.doi.org/10.1016/j.jtcvs.2012.06.033>

Reply to the Editor:

From the antibacterial properties of penicillin to the sweetness of saccharin, the antiadhesive properties of Teflon (polytetrafluoroethylene), or the now most-prescribed use of Viagra (sildenafil), countless medical and scientific discoveries have been made by the observation of unexpected qualities or side effects in experiments designed for a completely different purpose. In the recent study by Lassaletta and colleagues,¹ the findings of reduced pericardial adhesions and fibrosis with the administration of vodka were both surprising and unexpected in a study designed to evaluate myocardial perfusion and vessel density.² The reductions in pericardial adhesions at reoperative sternotomy and the decreases in intramyocardial fibrosis and transmural collagen deposition all did, however, reach statistical significance. The dose of ethanol administered to both the red wine and vodka treatment groups was 45 mL per day. With animal weights averaging just a bit more than 30 kg at the end of the 11-week experiment, this would translate to roughly 1¼ cups of vodka for an 80-kg person, if the pigs were

drinking these substances directly. In our experiment, however, the ethanol-containing agents were mixed with the chow and allowed to soak before feeding. Because of the volatility of ethanol, the actual amount consumed by the animals was thus likely to be less. For this reason, we measured the blood alcohol levels of the animals in both treatment groups, which averaged roughly 35 mg/dL 1 hour after consumption (equivalent to 0.035 g/100 mL on a standard breath alcohol analyzer, with 0.08 being the legal limit for intoxication in most U.S. states). The blood alcohol concentrations in this study were thus consistent with what most previous studies have considered to fall into the range of "moderate alcohol consumption."

Although our findings of decreased adhesions and fibrosis with vodka administration were significant, a definitive mechanism for the decrease has yet to be established. As addressed in the original article's conclusion,¹ such a mechanism would have to be defined before any progression to a clinical trial. That being said, ethanol is one of the most widely consumed pharmacologic substances in the world, with a well-known safety profile and an abundance of growing evidence demonstrating its cardioprotective properties at moderate doses. Although we aren't suggesting that physicians send their postoperative cardiac patients home with a prescription for vodka, we certainly wouldn't advocate abstinence for current, moderate drinkers.

Closure of the pericardium most certainly can reduce the incidence of opening catastrophe, but it may be associated with short-term hemodynamic compromise. Not all surgeons close the pericardium for this very reason. In this experiment, the pericardium was reapproximated with interrupted, braided nylon sutures, which still allowed drainage of any accumulated pericardial fluid into the left hemithorax.

Finally, it was surprising that the same reduction, or at least some decreased adhesion formation was not seen in the animals that received the same amount of ethanol from red wine, especially because there was some decrease in histologic fibrosis and collagen deposition in that group relative to control animals. For this reason, we are currently looking at the effects of pure ethanol (200-proof U.S. pharmaceutical grade) diluted to the same volume and concentration as in the original study in an experiment specifically designed to evaluate the extent of adhesions at reoperation.

Although the findings in the original experiment were inadvertent, they were recorded, documented, and corroborated with objective and blinded histologic analysis of the myocardium. They thus at the very least warrant sharing with the scientific community. Had someone decided to ignore the odd and controversial side effects of a vasodilator designed to treat hypertension simply because the outcome was not anticipated or because he or she feared public perception of controversial findings, that would have engendered a great disservice—not only to the medical and scientific communities but to the general population and the very patients for which medical research is conducted. As physicians and scientists, we must not be blinded to unanticipated information that may potentially improve patient care.

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<http://dx.doi.org/10.1016/j.jtcvs.2012.06.012>

ATRIOVENTRICULAR VALVE COMPETENCE AND VENTRICULAR GROWTH IN BIVENTRICULAR REPAIR OF UNBALANCED

ATRIOVENTRICULAR CANAL To the Editor:

I read with fascination the excellent contribution by Dr Foker and colleagues¹ to the supplement issue of the April 2012 volume of the *Journal* on the topic of valve repair in unbalanced atrioventricular canal, expanding on a series of infants in whom this same approach was used, which Foker and colleagues first reported in 1999.² The report focuses not only on atrioventricular valve competence but also more importantly on promoting ventricular growth in unbalanced ventricles, with the brave goal of consistently achieving satisfactory biventricular repair. With borderline anatomy, the results of single-ventricle repair are poor,^{3,4} and defining echocardiographic criteria to better select those patients who may benefit from biventricular repair⁵ is important. In patients with overt single-ventricle anatomy, promoting ventricular growth by staged surgery would seem even more attractive, if reproducible, as reported by Foker and colleagues.¹ We have had many of these patients, but honestly I have never had the guts to try this approach, which according to the reported excellent results, would systematically allow for a good biventricular repair. I have some technical and decision-making questions that may help me and many others to enroll more patients in this encouraging pathway.

1. Even after reading and looking at the finely illustrated 1999 article reporting on the initial 9 infants² and now the newer series of 24 patients,¹ I do not understand the way

the authors “partially” close the ventricular septal defect (VSD) in patients with small left ventricles. In the illustration, only the inferior part of the VSD is closed with a “partial patch,” leaving the atrioventricular valves floating on the ventricular side and only attached or reinforced by the atrial septal defect (ASD) patch from above. How do the authors, as they state in the text, achieve stability of the valve repair by partial VSD closure?

2. How big of a residual VSD is left open? I guess it depends on the size of the infant and the initial VSD, because there are no indications regarding patient weight or native VSD size.
3. Are there any contraindications to this approach, that is, in which patients should this not be attempted? Those with a valve *z* score less than -3.0 or a particular ventricular dimension cutoff? Are there particular unsuitable valve morphologies (multiple clefts, chordal anomalies)? Is any pericardial patch augmentation of the leaflet necessary?
4. Making a separate snared ASD fenestration, not in the closed patch, but more laterally in the native septum, is often very near the lateral right atrial wall when the primum ASD is large. Is there a real advantage to doing this and were any disadvantage or injuries encountered? Why not just punch a hole in the ASD patch?

Standardized decision-making and reproducible technical considerations promoting ventricular growth to allow biventricular repair, even at the cost of 2 staged surgeries, would be a huge advance for patients with an unbalanced atrioventricular canal.

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