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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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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Reply to the Editor:

We thank Dr Dodge-Khatami for his interest and insightful questions. Because the methods of inducing catch-up growth of the hypoplastic ventricles and valves in patients with unbalanced atrioventricular canal (UAVC) defects are not yet standard procedures, the technical details are of considerable importance.

There are also several important general points about these operations that should ease the concern about embarking on this approach.

The general principle is to increase flow across the hypoplastic atrioventricular (AV) valve, which generates the ventricular growth signal and induces catch-up growth of the hypoplastic ventricles and valves.¹ Even the smallest of these structures have enormous growth potential, and with the growth conditions met, the hypoplastic valves and ventricles will catch up surprisingly quickly.

While catch-up growth is occurring, septal defects are left behind to allow intracardiac shunting that will maintain adequate systemic and pulmonary blood flows. After the first stage, the patients predictably will be

stable because of the intracardiac shunting. There is no need to “bite the bullet” in an all-or-nothing operation and hope that a complete repair will be tolerated.

A restrictive atrial septal defect (ASD) is used to both allow shunting and increase flow across the hypoplastic AV valve to generate the growth signal. In right ventricle (RV)-dominant UAVC defects, a ventricular septal defect (VSD) is also left behind so that the RV can augment left ventricular (LV) flow and maintain an adequate cardiac output (CO).¹

The AV valve repair itself is helpful whether the end result is a 2-ventricle or single-ventricle repair. A UAVC lesion is a known risk factor for a Fontan procedure, at least in part because of AV valve problems, and valve repair should reduce this risk.

Finally, these growth-inducing operations do not irreversibly commit patients to a 2-ventricle repair, and a single-ventricle track remains available if necessary.

The following is in regard to your specific questions:

1. The partial VSD repair: A VSD is left behind only in RV-dominant UAVC defects where the CO must be augmented by the RV.¹ A complete VSD patch closure is done first in the usual fashion for a 2-patch AV canal defect repair, which stabilizes the valves. It also allows the upper edge of the patch to be placed away from the hypoplastic component when dividing the common AV valve leaflet, so it will enlarge the area of the hypoplastic valve.²
2. The residual VSD is created by cutting a hole in the center of the VSD patch. The hole must be big enough to allow augmentation of LV output, so it depends on the size of the patient. The size is not exact, but it should be at least 5 mm in diameter. The hole should not be so big, however, that it discourages transmitral flow. These patients will have pulmo-

nary artery bands, so the pulmonary vasculature will be protected.

When growth is adequate, the hole in the patch can be closed surgically. Because the septal rim of the VSD is patched, there will not be a risk of heart block during closure of the hole.

In an LV-dominant UAVC lesion, augmentation through a residual VSD is usually not necessary. Only approximately half a CO will provide sufficient pulmonary blood flow, and if more is needed, a shunt can be placed to allow time for the RV to grow. These patients do require a restrictive ASD to encourage tricuspid flow and contribute to the CO by right-to-left shunting.

3. Limits to the degree of hypoplasia in the growth of UAVC defects: No patients have been turned down, even with a ventricular z score of less than -7 or a rudimentary mitral valve. The hypoplastic valves and ventricles will reliably grow. We all grow 20- to 30-fold after birth, so a lot of potential growth is available.

Our article illustrates a tiny mitral valve, which would have been highly obstructive after a complete repair. To provide adequate mitral flow, the large tricuspid valve was split in 2, which left the patient with 2 mitral valves. As shown, the tiny mitral valve grew well and reached normal size.²

Most of the hypoplastic AV valves in our series were only small, but a few were also deformed. Among the deformities repaired were a 2-layer mitral valve and several with significant regurgitation. Most are repairable, but if not, a replaced mitral valve in a 2-ventricle repair should still be better than a single ventricle repair, especially one with bad AV valves.

4. Snared ASD: The reason for keeping the ASD snare mechanism separate from the primum ASD patch is that the snare is adjusted off