THE LEFT VENTRICLE: TO RECONSTRUCT OR NOT—LESSONS FROM THE STICH TRIAL

To the Editor:

Recently at the 89th Annual Meeting of The American Association for Thoracic Surgery in Boston, May 9–13, 2009, there was a very clarifying debate on the STICH trial¹ (Surgical Treatment for Ischemic Heart Failure) between Dr Robert H. Jones (pro) and Dr Gerald D. Buckberg (con), moderated by Dr Andrew S. Wechsler, which sparked the following thoughts.

Clearly, surgeons involved in the STICH trial had a difficult job: they were instructed to include patients in the trial if they honestly believed they could undergo coronary artery bypass grafting (CABG) either alone or in combination with surgical ventricular reconstruction (SVR) with safety. It would seem likely that, ethically, patients with either global hypokinesis (no clear-cut aneurysm) or those with large aneurysms would be hard to include in this trial. Also, results of the two patient groups (CABG alone vs CABG+SVR) are uncannily similar: identical improvements in congestive heart failure and angina class for both groups (1.7 and 1.0, respectively), increase in distance on the 6-minute walk test (48 vs 52 m), death or hospital readmission for cardiac cause (59% vs 58%), death from any cause (28% vs 28%), hospitalization for cardiac cause (42% vs 41%), and operative mortality as-

treated (5% vs 6%) or as intention-to-treat (5% vs 5%). These results are practically identical as if coming from a single patient population divided in half....because *they are*. They were selected by ethical surgeons aware of the extensive current literature by many expert surgeon-scientists working on hibernating myocardium (no aneurysm, poor ventricles) and overgrown ventricles resulting from infarction (aneurysm causing poor function). Perhaps during the conception of STICH such knowledge was not available, but perhaps the performance of SVR (or not) has reached the level of the “randomized controlled study of the parachute.” *The real message of the STICH trial is this:* if one is pondering treatment of patients with poor ventricular function and mild aneurysmal dilatation, do not perform the SVR surgery, because if you do not send the patient to heaven, you will only prolong his or her and your own suffering (increased crossclamp, operative, and intensive care unit times and total length of stay) with no clinical benefit in ensuing years. The STICH investigators and surgeons are to be congratulated for their ethical dealing with this controversial topic. However, the message that *CABG surgery alone is enough for patients with coronary artery disease and left ventricular aneurysms* should not be interpreted from the STICH trial, as many patients with disabling left ventricular aneurysms would be at risk for the wrong treatment.

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Reference

1. Jones RH, Velazquez EJ, Michler RE, Sopko G, Oh JK, O'Connor CM, et al. Coronary bypass surgery with or without surgical ventricular reconstruction. *N Engl J Med*. 2009;360:1-13.

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CLARIFICATION OF STATEMENTS MADE REGARDING INVESTIGATION INTO AMPLATZER DEVICE COMPLICATION INCIDENCE AND COMPARISON WITH THE SOCIETY OF THORACIC SURGERY DATABASE

To the Editor:

As the corresponding first author and presenter of the article regarding the Amplatzer device (AGA Medical Corporation, Plymouth, Minn) complications and comparison with the Society of Thoracic Surgery database,¹ I must clarify some statements made regarding the work of previous authors on the complications of the Amplatzer device.² In formulating a calculation that was used to estimate complication frequency, we based our mathematics on estimates that were previously used by Amin and colleagues.² Our point in noting that there was financial disclosure in the previous work was not to suggest that the data were not accurate. In fact, we were counting on the accuracy of these data because we also used the data for our calculation. Rather than questioning the work of these authors, we were in fact relying on the integrity of their estimates to make our own calculations. The authors most closely associated with this device serve as the best possible source of such information, and highlighting this relationship was only intended to add validity to our own calculation.

There was absolutely no intended implication of foul play or acting in conflict of interest in the case of these authors and indeed no such accusation was directly made. That some complications go unreported to the MAUDE database is most probably true but this has nothing to do with the work of Amin and colleagues,² and these must be kept separate in the readers' minds to understand the spirit of this work. The previous publications on the complications with Amplatzer devices were as accurate as any, and our addition to the literature presents the most up to date. That the frequency

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of said complications is increasing may be true, but this again had nothing to do with the integrity of previous estimates made in a good faith effort to study results. I hope this clarifies any question in the readers' minds as to the intention of my statements regarding this issue.

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References

1. DiBardino DJ, McElhinney DB, Kaza AK, Mayer JA. Analysis of the U.S. Food and Drug Administration MAUDE Database for adverse events involving transcatheter septal occluder devices and comparison to Society of Thoracic Surgeons Congenital Cardiac Surgery database. *J Thorac Cardiovasc Surg.* 2009;137:1334-41.
2. Amin Z, Hijazi ZM, Bass JL, Cheatham JP, Hellenbrand WE, Kleinman CS. Erosion of Amplatzer septal occluder device after closure of secundum atrial septal defects: review of registry of complications and recommendations to minimize future risk. *Catheter Cardiovasc Interv.* 2004;63:496-502.

doi:10.1016/j.jtcvs.2009.06.012

MICHAEL E. DEBAKEY

The obituary of Dr DeBakey was very interesting.¹ One of Dr DeBakey's pioneering efforts does need special emphasis. By using autologous saphenous vein(s), on November 23, 1964,² DeBakey performed the first human coronary artery bypass graft surgery to the left anterior descending coronary artery in an asymptomatic patient after myocardial infarction.

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References

1. Frazier OH, Michael E. DeBakey, 1908–2008. *J Thorac Cardiovasc Surg.* 2008;136:809-11.
2. Garrett EH, Dennis EW, DeBakey ME. Aortocoronary bypass with saphenous vein grafts: seven-year follow-up. *JAMA.* 1973;223:792-4.

doi:10.1016/j.jtcvs.2009.05.022

PULMONARY ARTERIAL HYPERTENSION AND CONGENITAL HEART DISEASE: TARGETED THERAPIES AND OPERABILITY

To the Editor:

We read with great interest the case report of Hoetzenecker and colleagues¹ of a patient with severe pulmonary arterial hypertension associated with an atrial septal defect and the beneficial effect of bosentan, which allowed for closure of the defect. This is a very interesting topic that raises a lot of controversies in the field of pulmonary arterial hypertension associated with congenital heart disease.²

I would like to comment on the hemodynamic data presented in Table 1. Total pulmonary resistance at baseline is calculated at 460 dynes $\cdot$ s⁻¹ $\cdot$ cm⁻⁵, but pulmonary vascular resistance, taking the mean left atrial pressure in the calculation, which is commonly done in congenital heart disease, would give a value of 311 dynes $\cdot$ s⁻¹ $\cdot$ cm⁻⁵ or 3.9 Wood units, which is very close to a value of 3 Wood units considered as totally safe for surgical repair. If we calculate the ratio of pulmonary to systemic blood flow (Qp/Qs), we have a value of 2.7, which is also considered a value that allows for repair.

Moreover, when we look at the data during the nitric oxide testing, these values reach a pulmonary vascular resistance of 1.78 Wood units or 142 dynes $\cdot$ s⁻¹ $\cdot$ cm⁻⁵, the Qp/Qs is 3.25, and the pulmonary arterial saturation reaches 84.5%. All these values are consistent with a reactive pulmonary vascular bed and are values that will clearly allow closure of the shunt for a congenital cardiologist.^{3,4} It would be of interest also to report the ratio of pulmonary over systemic vascular resistance, as a ratio < 0.33 indicates good prognosis after closure of the shunt.⁴

Maybe the authors used the definition of reactivity used for other forms of pulmonary arterial hypertension, al-

lowing for the use of calcium channel blockers, but this may not be applied for the evaluation of operability in patients with congenital heart disease. Why did the authors think that this particular patient required bosentan treatment based on these measurements? If we look at the data after treatment and just before repair, the hemodynamic does not look much better than with nitric oxide, as the pulmonary vascular resistance is 2.6 Wood units or 205 dynes $\cdot$ s⁻¹ $\cdot$ cm⁻⁵ and Qp/Qs is 2.24. Why was the patient considered operable with these values but not before? If this is based only on mPAP, I would suggest the authors should discuss the fact that mLAP also had a significant decrease.

Finally, if we analyze the measurement 8 months after repair, the pulmonary vascular resistance is 4.2 and the mean pulmonary arterial pressure is 35 mm Hg—still not normal. We clearly hope that these values will remain at these levels and not rise again in the coming month, as the outcome, at least in pediatric patients with recurrent pulmonary hypertension after repair, is dismal, as shown recently by Haworth and colleagues.⁵

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References

1. Hoetzenecker K, Ankersmit HJ, Bonderman D, et al. Atrial septal defect repair after a 10-month treatment with bosentan in a patient with severe pulmonary arterial hypertension: a case report. *J Thorac Cardiovasc Surg.* 2009;137:760-1.
2. Dimopoulos K, Peset A, Gatzoulis MA. Evaluating operability in adults with congenital heart disease and the role of pretreatment with targeted pulmonary arterial hypertension therapy. *Int J Cardiol.* 2008;129:163-71.
3. Berner M, Beghetti M, Spahr-Schopfer I, Oberhansli I, Friedli B. Inhaled nitric oxide to test the vasodilator capacity of the pulmonary vascular bed in children with long-standing pulmonary