applicable when considering thoracic endovascular aortic repair (TEVAR) for dissections.² DeBakey type III dissections are divided into 2 groups: type IIIa, in which the dissection is confined to the supradiaphragmatic descending aorta, and type IIIb, in which it extends beyond the diaphragm. Definition is important from the point of achieving false lumen-free treatment or only aneurysm exclusion with TEVAR. According to our modest experience, we³ obtained “cure” with TEVAR in patients with type IIIa dissections by coverage of the proximal tear and complete reattachment of the intima and media levels.²

Endovascular treatment is performed to exclude the aneurysm in type IIIb dissections.^{2,4} However, patency of the false lumen is a major prognostic factor in the mid to long run in patients with type IIIb aortic dissections both after open repair⁵ and after TEVAR.^{2,4} After TEVAR, the proximal false lumen does not always thrombose and blood flow may continue outside the stent graft.² This may increase the size of the aneurysm, lead to a new aneurysm formation at the excluded segment of the aorta, or even lead to collapse of the stent graft system.² The postoperative course marked by such a complication in 1 of our patients (Figure E1), who was further treated with TEVAR.

Even if the proximal false lumen completely thromboses, it is still under the stress of systemic blood pressure. In contrast to the theory that thick thrombus protects from rupture, intra-aortic thrombus diminishes aortic wall nourishment, and ruptures frequently occur in the presence of aortic thrombus.

In brief, TEVAR fails to completely compress the false lumen in chronic type IIIb dissections either because of high pressure inside the false lumen or because it is intended not to, since some visceral arteries may originate from this lumen. The issue is different during surgery or perioperative hybrid therapy. Surgical treatment mediates

replacement of the dilated segment of aorta. Distal anastomosis may provide perfusion of only the true lumen or both lumina.⁵ And TEVAR during surgical treatment differs from conventional deployment, in which stent grafts are delivered and expanded into a pressureless aorta, a technique that has better success rates on follow-up.⁴

Once again, we congratulate the authors for their very valuable contribution. We believe surgical treatment still seems the valid option for the treatment of chronic type B dissections with low complication rates in experienced centers in the current era.⁵

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DECISION MAKING IN ISCHEMIC CARDIOMYOPATHY: IS THE CLEVELAND CLINIC MODEL VALID?

To the Editor:

Clinically significant variables are required before any statistical model can provide useful decision making during surgical management of ischemic cardiomyopathy. Models would otherwise be created with an insufficient database, so that statistically validated results might yield valueless clinical conclusions that might worsen the development of congestive heart failure (CHF) symptoms and fatal ventricular arrhythmias after their application to the enormous population with CHF.

The recent Cleveland Clinic Foundation (CCF) report¹ concludes that coronary artery bypass grafting (CABG) alone or in combination with surgical ventricular restoration (SVR) provides similar long-term results to the recent National Institutes of Health Surgical Treatment of Ischemic Heart Failure (STICH) trial that evaluated quality-of-life outcome, rehospitalization rate, and mortality after surgical management of ischemic cardiomyopathy.² Their findings establish CABG therapy alone as a sufficient treatment, fulfill current angioplasty revascularization goals, diminish cardiologic interest in SVR, and limit expansion of the surgeon's interest in learning a new procedure for CHF management.

The innovative statistical model hypothesis of no outcome difference between CABG versus CABG with SVR becomes invalid if the ischemic cardiomyopathy clinical database does not show increased left ventricular end-systolic volume index (LVESVI) rather than reduced systolic ejection fraction³ as the indicator of heart failure. The original STICH trial entry criteria required a preoperative LVESVI of greater than 60 mL/m², as measured by means of cardiac magnetic resonance imaging or single photon emission computed tomography

(SPECT), and excluded echocardiographic analysis because of volume underestimation from ventricular asynergy.⁴ The extensive CCF report tables do not include this vital component. The STICH trial report excluded volume measurement in 66% of patients having CABG and SVR, and an almost similar exclusion rate occurred in patients having CABG alone. Moreover, the accepted trial design defined a 30% LVESVI reduction as a satisfactory SVR procedure.⁵ An inadequate SVR procedure was done in the STICH trial because volume reduction was only 19%.⁵ The CCF authorship should provide preoperative and postoperative LVESVI measurements so that readers can determine whether the right patients were selected and whether a proper SVR volume reduction was done.

The STICH trial required that an area of myocardial necrosis of greater than 35% be present to qualify for randomization to CABG or CABG plus SVR. Temporary akinesia, dyskinesia, or both can result from stunning or hibernation of nonscarred muscle. The STICH trial did not report myocardial necrosis. The only entry criterion for SVR was dysfunction caused by akinesia or dyskinesia without necrosis documentation and included 13% of patients without previous myocardial infarction. Furthermore, the CCF reports that only 153 patients had necrosis determined by means of gadolinium cardiac magnetic resonance imaging because the 111 dobutamine echocardiographs cannot be used quantify the area of necrosis. How can the authors be sure the 6-cm left ventricular end-diastolic diameter was due to scar rather than stunning?

The role of experience is another barometer of the STICH trial's validity that was recently questioned.⁵ This inquiry is related to the learning curve because the STICH trial recruited 96 centers to perform an innovative procedure in 490 patients. The CCF is a single institution and thereby par-

tially addresses this question. There is historic precedent for the role of experience at CCF because Sheldon and colleagues' 1976 report⁶ established the vital importance of CABG therapy by demonstrating excellent graft patency and 1% mortality in 1000 consecutive patients. Most importantly, this classic study contradicted the National Institutes of Health Veterans Hospital CABG report showing that inexperienced centers recorded a higher graft occlusion rate and mortality.⁷ Unfortunately, the current CCF report does not follow the strategy of Sheldon and colleagues⁶ because long-term mortality is reported without evaluating the quality of the SVR procedure in sufficiently reducing left ventricular volume. Moreover, LVESVI measurements in the CCF database would allow readers to compare their findings with the approximately 40% LVESVI reduction (30%–58% range) achieved by 1500 patients reported from 12 international centers,⁵ the database for which demonstrated improved late outcomes in patients having CABG plus SVR compared with late mortality outcomes from patients having CABG alone.^{8,9}

The effect of a satisfactory volume reduction rate after SVR was confirmed by the 2001 Di Donato and associates' report¹⁰ of the Dor experience, whereby higher late mortality occurred when postoperative LVESVI was greater than 60 mL/m². Di Donato and coworkers' recent 2010 registry report¹¹ from Milan demonstrates significantly higher late SVR mortality rates if postoperative volume reduction of 40% attained an LVESVI of greater than 60 mL/m² versus less than 60 mL/m². Consequently, the question is not "Have you done SVR?" but rather "Did you do an adequate SVR procedure?"

This CCF analysis indirectly addresses the role of ventricular volume by showing that CABG and mitral valve procedures (MVP) have inferior late mortality results when compared with CABG, CABG and SVR, and

heart transplantation. Markedly increased LVESVI from mitral insufficiency is the most likely cause, even though LVESVI was not described. The surgical approach of addressing the "vessel valve and ventricle"^{8,12} applies to patients undergoing simultaneous CABG, MVP, and SVR, but the article does not indicate analysis of these patients. In contrast, the Reconstructive Endoventricular Surgery, Returning Torsion, Original Radius, and Elliptical Shape to the LV (RESTORE) analysis shows that their 5-year mortality was similar to that of patients undergoing CABG with SVR and SVR alone.⁸ The surgical theme of reducing ventricular volume by means of SVR thereby applies to patients with MVP in ischemic cardiomyopathy, yet this decision option that also solves mitral insufficiency volume expansion is not considered.

The CCF statistical analysis contributions are superb gifts to our specialty because they have improved our decision-making process. However, the hallmark of a validated prediction model in decision making for ischemic cardiomyopathy must become linked to the established clinical variables of importance, namely LVESVI, necrosis, and adequate volume reduction rate. These important variables were not included in the CCF study. Their model might potentially mislead the surgical and cardiology community.

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