EDITORIAL COMMENT

Surgical Ventricular Reconstruction for Heart Failure
Is There Life After STICH?*

Robert O. Bonow, MD
Chicago, Illinois

The major advances in medical therapy that have occurred over the past few decades have not diminished the impact of heart failure as one of the leading causes of death and disability in the developed countries of the world. Mortality rates remain high in patients with left ventricular (LV) systolic dysfunction, even among those receiving evidence-based medical therapies. Adverse LV remodeling is continuous and unrelenting in all too many patients with ischemic or dilated cardiomyopathy.

The progressive nature of LV chamber dilation, with its concomitant neurohormonal activation, myocardial fibrosis, and hypertrophy, have generated considerable interest in surgical techniques to alter LV volume and geometry toward more normal size, shape, and physiology. During the previous decade, surgical ventricular reconstruction (SVR) procedures were developed for this purpose and in small, unblinded series resulted in improvement in both LV function and quality of life (1–4). However, it was uncertain whether SVR combined with coronary artery bypass surgery (CABG) would result in improved outcomes of patients with ischemic cardiomyopathy compared with CABG alone, especially when combined with evidence-based medical management. This equipoise led to the multicenter, prospective, randomized STICH (Surgical Treatment for Ischemic Heart Failure) trial, sponsored by the National Heart, Lung, and Blood Institute, which randomized 1,000 patients to CABG or to CABG plus SVR (5). Patients in both groups also received aggressive, evidence-based medical therapies. The STICH trial reported no benefit of SVR plus CABG over CABG alone in terms of mortality, hospitalization, or quality of life over a median follow-up period of 4 years. Despite these definitive findings, the results of the STICH trial have engendered considerable discussion and controversy among proponents of SVR (6–9).

Alternative surgical techniques to reshape the left ventricle have also been promulgated. Unlike the standard SVR procedure, in which an endocardial patch is placed before reshaping the ventricle, in 2004 Matsui et al. (10) reported early experience with a modified method of SVR, termed overlapping left ventriculoplasty (OVLP), in which the myocardial layers were overlapped without the placement of an endocardial patch. Short-term experience with this procedure in small numbers of patients, with or without associated mitral valve repair for functional mitral regurgitation (MR), was favorable in terms of operative mortality, symptom improvement, and hemodynamics (10,11). Operative mortality with this procedure varied between 8.0% and 12.5%, compared with 5.0% in patients treated with CABG plus SVR in the STICH trial (5,10,11).

In this issue of iJACC, Sugiki et al. (12) provide further unique information regarding the functional results of the OLVP method of SVR. Using a combination of echocardiography and positron emission tomography (PET), the investigators studied LV function, volume, and mass along with assessment of oxidative metabolism using carbon-11 acetate. They report that the reduction in LV volume and mass after the surgical remodeling procedure is associated with both a reduction in global oxidative metabolism and an increase in cardiac efficiency or “work metabolic

*Editorials published in JACC: Cardiovascular Imaging reflect the views of the authors and do not necessarily represent the views of JACC: Cardiovascular Imaging or the American College of Cardiology.

From the Center for Cardiovascular Innovation, Department of Medicine, Northwestern University Feinberg School of Medicine, Chicago, Illinois. Dr. Bonow has reported that he has no relationships to disclose.
index” (WMI). These results represent an impressive body of work from an experienced team of surgeons, cardiologists, and imaging experts. These apparently favorable results underscore the unique potential of PET in assessing the effects of medical or surgical interventions in heart failure. Whether these results also provide new impetus for further exploration of the role of surgical remodeling techniques in the treatment of heart failure, however, is less certain and will require additional evidence.

Equally important would be findings in patients treated with aggressive medical therapy. In the current study by Sugiki et al. (12), 7 of 12 patients (58%) received standard medical therapy for heart failure in terms of beta-blockers and angiotensin receptor blockers (ARBs), but 42% did not: 2 patients did not receive beta-blockers, 2 patients did not receive ARBs, and 1 patient did not receive either a beta-blocker or an ARB. Spironolactone was also not consistently used. This is an important issue because medical therapy for heart failure will not only improve symptoms and LV systolic function, but will also improve oxidative metabolism and cardiac efficiency, as pointed out by the authors (13–15).

Several caveats are warranted in interpreting the oxidative metabolism results reported by Sugiki et al. (12). As noted by the authors, their sample size was small (N = 12), especially considering the subgrouping of patients according to ischemic and nonischemic etiologies, and larger numbers of patients will be needed to draw meaningful conclusions and support these preliminary positive findings. In addition, the follow-up period of 1 month is too brief to determine the longer term effects on cardiac function, oxidative metabolism, and work efficiency. There are other issues worthy of discussion.

It is noteworthy that the carbon-11 acetate data revealed no change in oxidative metabolism per gram of tissue (Kmono) from before to after surgery. As the global oxidative metabolism and the WMI are defined as: global oxidative metabolism = (Kmono)(LV mass) and WMI = [(forward stroke volume)(systolic blood pressure)(heart rate)]/Kmono, it is apparent, in the absence of any change in Kmono, that the decrease in global oxidative metabolism is driven entirely by the reduction in LV mass, and the increase in WMI is driven entirely by the increases in forward stroke volume and heart rate. Thus, the PET data actually yield little insight beyond the insights provided by the echocardiographic data alone. These latter data are also worthy of further scrutiny.

It is uncertain whether LV mass can change as abruptly within 1 month of surgery as reported by Sugiki et al. (12), given that the operation does not actually remove any LV myocardium but simply folds it upon itself. More likely, the changes in calculated LV mass are more apparent than real and reflect the impact on the determination of LV mass of the significant and immediate reduction in LV volume stemming from the volume-reducing surgery.

Similarly, the WMI equation may not provide an accurate evaluation of overall LV work when there is significant MR because only the forward stroke volume measured in the LV outflow tract is accounted for. Thus, although it is good to know that forward stroke volume increased post-operatively (a true benefit of the operation), it is not certain whether total LV stroke volume actually increased. Total LV stroke volume may actually decrease after elimination of MR. The total stroke volume (forward plus regurgitant) might have been used in the calculation of the pre-operative WMI had the authors computed regurgitant volume according to current societal recommendations (16–18) rather than a semiquantitative assessment of MR color flow using the older recommendations of the 1997 American College of Cardiology/American Heart Association guidelines on echocardiography (19).

Interpretation of the post-operative increase in heart rate is equally difficult because no patient received post-operative beta-blockers according to the surgical protocol, including the 9 patients receiving these medications pre-operatively. Thus, the increase in heart rate could reflect, at least in some patients, the effects of beta-blocker withdrawal. These issues regarding the post-operative changes in LV stroke volume and heart rate make it uncertain whether the increase in calculated WMI represents a true benefit of surgery.

As the authors note, further experience in larger numbers of patients with longer follow-up periods is necessary to determine the relevant role of surgical OLVP and mitral valve repair in patients with ischemic and nonischemic LV systolic dysfunction. The significant reduction in LV end-diastolic and end-systolic volumes at 1 month after OLVP is an expected result of an operation designed to achieve this endpoint. More important would be the effect on late LV remodeling and whether the changes and LV volumes and function are sustained with longer follow-up periods. It is noteworthy that no long-term data have yet been reported regarding LV function and clinical outcomes (such as mortality) in patients undergoing the OLVP procedure,
including the patients reported initially in 2004 and 2005 (10,11), for whom such data should now be available. Thus, in the absence of such data and comparison with a medically treated control group, the use of OLVP or other forms of SVR remains an unproven strategy in the management of patients with heart failure. As far as patients with ischemic LV dysfunction are concerned, the STICH trial (5) provides strong and thus far definitive evidence that such approaches are not superior to CABG alone in patients who are also receiving aggressive medical therapy.

**Reprint requests and correspondence:** Dr. Robert O. Bonow, Center for Cardiovascular Innovation, Northwestern University Feinberg School of Medicine, 645 North Michigan Avenue, Suite 1006, Chicago, Illinois 60611. E-mail: r-bonow@northwestern.edu.

---

**Key Words:** heart failure • positron emission tomography • surgical ventricular reconstruction.

---

**REFERENCES**