Surgical Ventricular Reconstruction for Heart Failure

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Congestive heart failure is one of the leading causes of death and complications in the developed world, and coronary artery disease is the major cause of heart failure. Efforts to improve ventricular function, symptoms, and clinical outcomes in patients with heart failure have included neurohormonal inhibition with angiotensin-converting–enzyme inhibitors, angiotensin-receptor blockers, beta-blockers, and aldosterone antagonists, as well as cardiac resynchronization therapy. All these therapies have been shown in randomized clinical trials to be beneficial. However, none of them specifically address the coronary disease responsible for ischemic cardiomyopathy and myocardial infarction.

Coronary-artery bypass grafting (CABG), which was first used to ameliorate symptoms, is now used to improve survival in selected patients with ischemic heart disease, although the improved outcome of percutaneous coronary intervention (PCI) has resulted in a reduction in the number of CABG procedures that are performed. CABG remains the standard of care for patients with disease involving at least three vessels or the left main coronary artery. Revascularization with CABG can improve the perfusion of viable myocardium but does not restore function in areas of infarction.

The large, aneurysmal myocardial segments that were seen in the past in patients with myocardial infarction are seldom seen in the current era of coronary reperfusion. However, regional zones of myocardial dysfunction are still frequently observed. These areas of regional dysfunction may result in adverse cardiac remodeling and the progression of heart failure. A surgical procedure to reconstruct dysfunctional myocardial segments and favorably remodel the ventricle, known as surgical ventricular reconstruction, has been developed and is performed in selected patients, usually in conjunction with CABG. Previously reported clinical studies of surgical ventricular reconstruction were not randomized and were conducted either in a single center or in multiple centers as observational studies.

In this issue of the Journal, Jones et al. report the results of the Hypothesis 2 substudy of the Surgical Treatment for Ischemic Heart Failure (STICH) trial (ClinicalTrials.gov number, NCT00023595). This substudy compared CABG alone with the combined procedure of CABG with surgical ventricular reconstruction. Patients were required to have coronary disease amenable to CABG, a left ventricular ejection fraction of 35% or less, and a dominant anterior region of myocardial akinesia or dyskinesia that was amenable to treatment with surgical ventricular reconstruction. All patients received standard medical and device therapy for heart failure. The results of the Hypothesis 1 substudy of the STICH trial, which compared medical therapy plus CABG with medical therapy alone, are not reported.

In the Hypothesis 2 substudy, 1000 patients were recruited from 96 medical centers in 23 countries. The patients in the two study groups were closely matched in terms of demographic characteristics, coexisting illnesses, the proportion who were receiving specific heart-failure medications, the Canadian Cardiovascular Society (CCS) angina class, the New York Heart Association (NYHA) heart-failure class, coronary anatomy, and the extent of anterior myocardial akinesia or dyskinesia. Although Jones et al. indicate that the use of evidence-based medical therapy (including heart-failure medications, pacemakers, and cardioverter–defibrillators) was monitored throughout...
Figure 1. Surgical Ventricular Reconstruction.

An incision is made through the area of scarring in the left ventricle (Panel A), which is opened to identify the boundary between damaged and healthy myocardium (Panel B). A purse-string suture is placed to encircle the scar, and the healthy portions of the ventricular wall are brought together (Panel C). The suture is tightened or a patch is placed (Panel D), and the scarred sections left outside the chamber are closed (Panel E).
the trial, they have not provided information about rates of use of these therapies over time.

Both CABG alone and the combined procedure were equally successful in improving the post-operative CCS angina class and NYHA heart-failure class. The two groups had similar improvements in the 6-minute walk test and similar reductions in symptoms. There was a greater reduction in the end-systolic volume index with the combined procedure (16 ml per square meter of body-surface area), as compared with CABG alone (5 ml per square meter). Unfortunately, these data were obtained from only 373 patients at baseline and at 4 months.

The primary outcome of the trial was a composite of death from any cause or hospitalization for cardiac causes. There was no difference in the occurrence of the primary outcome between the CABG group (59%) and the combined-procedure group (58%). The 30-day surgical rates of death for CABG alone (5%) and for the combined procedure (6%) were similar and low overall, and no difference in the rate of death from any cause was observed in a median follow-up period of 48 months. Subgroup analyses showed no individual variables interacting significantly with study-group assignment.

This large clinical trial had many strengths, including an up-to-date approach to CABG, certification of surgeons and interventionalists in both PCI and CABG, and an attempt to standardize heart-failure therapies according to guidelines throughout the entire study population. The study also had some limitations, including sparse data regarding medical or device therapy used in each study group. The diversity of patients who were enrolled might have limited the ability of the investigators to identify particular groups or types of patients who might benefit from the combined procedure. Finally, more complete and longitudinal information on the end-systolic volume index might have provided an indication of whether ongoing ventricular remodeling occurred, which might have negated or mitigated the difference seen in the early postoperative period.

On the basis of this trial, the routine use of surgical ventricular reconstruction in addition to CABG cannot be justified. Potential explanations for the lack of added efficacy of the combined procedure include the fact that current heart-failure therapies are very effective at limiting adverse remodeling. The addition of CABG may have enhanced this process, leaving little room for additional benefit from surgical ventricular reconstruction. We will have to wait for the results of the Hypothesis 1 substudy of the trial to know for sure.

It is also apparent that surgical remodeling of the ventricle is different from remodeling induced by heart-failure therapies or cardiac resynchronization therapy and that the abrupt geometric reduction in ventricular dimensions with this procedure does not mimic the benefits derived from neurohormonal inhibition and perhaps revascularization. The extent of volume reduction from surgical ventricular reconstruction also may not be consequential. There may be specific subgroups of patients who might benefit from the combined procedure, but such an effect is not apparent so far in the results of the STICH trial and may be difficult to detect, given the diversity of the study population.

Dr. Eisen reports receiving consulting and lecture fees from Medtronic and grant support from Medtronic and St. Jude. No other potential conflict of interest relevant to this article was reported.

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This article (10.1056/NEJMe0901815) was published at NEJM.org on March 29, 2009.


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