Favorable effects of left ventricular reconstruction in patients excluded from the Surgical Treatments for Ischemic Heart Failure (STICH) trial

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Objective: We sought to examine the hemodynamic effects at 1 month and 1 year of left ventricular reconstruction by means of endoventricular patch plasty for patients with acute or chronic, very severe post–myocardial infarction heart failure who would have been systematically excluded from the Surgical Treatments for Ischemic Heart Failure (STICH) trial.

Methods: From 2002 to May 2008, 274 patients underwent left ventricular reconstruction for post–myocardial infarction scarring; 117 of these patients would not have been eligible for the STICH trial. The pertinent criteria for exclusion included 12 patients with no coronary vessel suitable for coronary artery bypass grafting; 17 patients within a month of myocardial infarction, including 11 with acute heart failure (8 septal ruptures and 3 cases of ventricular tachycardia); 48 patients receiving intravenous inotropes, intra-aortic balloon pumping, or both; 15 patients with bifocal or posterior scarring; 4 patients scheduled for heart transplantation; and 21 patients meeting 5 other exclusion criteria. These patients (mean age, 64 years; age range, 34–83 years) preoperatively had a mean 49% (range, 35%–75%) scarred left ventricular circumference, as determined by means of magnetic resonance imaging assessment. In the patients with chronic heart failure, the preoperative ejection fraction was 26% ± 4% (range, 9%–34%), the end-diastolic volume index was 130 ± 43 mL/m^2 (range, 62–343 mL/m^2), and the end-systolic volume index was 95 ± 37 mL/m^2 (range, 45–289 mL/m^2). Mitral regurgitation was mild to severe in 56 patients and associated with annular dilatation (≥35 mm) in 51 patients. A strategy of left ventricular reconstruction by means of endoventricular circular suturing and patching excluded the scarred left ventricular wall and was balloon sized to provide a diastolic volume of 50 mL/m^2. Circular patches were used for anteroseptoapical lesions, and triangular patches were used for posterior lesions. The mitral valve was repaired in 51 patients, and coronary revascularization was performed in 105 patients (arterial grafts in 95 and mixed in 12). Seventy-eight patients had endocardectomy, and cryotherapy was used in 39 patients for ventricular tachycardia.

Results: Four in-hospital and 2 delayed deaths occurred during the first year. In 101 patients with chronic heart failure, magnetic resonance imaging revealed that ejection fraction improved from 26% ± 4% preoperatively to 40% ± 8% at 1 month and 44% ± 11% at 1 year postoperatively. At these same time points, the end-diastolic volume index was reduced from 130 ± 43 mL/m^2 to 81 ± 27 and 82 ± 25 mL/m^2, respectively, and the end-systolic volume index was reduced from 96 ± 45 mL/m^2 to 50 ± 21 and 47 ± 20 mL/m^2, respectively.

Conclusions: With minimal associated mortality, left ventricular reconstruction produces durable improvement in left ventricular function in patients with a large scarred ventricular wall. Considering that this patient cohort would have been systematically excluded from the STICH trial, care should be taken not to extrapolate that study's results too widely so as to inappropriately deny selected patients an effective treatment for ischemic cardiomyopathies with an injured ventricle. (J Thorac Cardiovasc Surg 2011;141:905-16)

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The aim of this study is to show by means of hemodynamic data and left ventricular (LV) wall-motion analyses 1 month and 1 year after surgical intervention that in patients with ischemic cardiomyopathies (ICMs) with a widely scarred LV wall and distended viable myocardium caused by eccentric systolic motion, the exclusion of the scar by means of endoventricular patch plasty (EVPP) is followed by a stable regression of LV remodeling and restoration of normal concentric contraction of the LV wall surrounding the scarred area. The pathophysiologies leading to ICM are diverse and complex, including depressed but viable ischemic myocardium, stunned or hibernating muscle, and asynergic fibrous scarring.

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Abbreviations and Acronyms

AHF = advanced heart failure
CABG = coronary artery bypass grafting
caDVI = contractile area diastolic volume index
CMR = cardiac magnetic resonance
EDVI = end-diastolic volume index
EF = ejection fraction
ESVI = end-systolic volume index
EVPP = endoventricular patch plasty
GLE = gadolinium late enhancement
HF = heart failure
ICM = ischemic cardiomyopathy
IFV = ischemic failing ventricle
LAD = left anterior descending coronary artery
LAVI = left atrial volume index
LV = left ventricular
LVR = left ventricular reconstruction
MI = myocardial infarction
MR = mitral regurgitation
MRI = magnetic resonance imaging
NYHA = New York Heart Association
SI = sphericity index
STICH = Surgical Treatments for Ischemic Heart Failure
SVR = surgical ventricular reconstruction

Although coronary artery bypass grafting (CABG) or percutaneous transluminal coronary intervention can treat ischemia, only left ventricular reconstruction (LVR) is specifically designed to address the scarring of the myocardial wall that triggers remote negative LV remodeling.

First demonstrated in 1967 by Klein and associates[1] and confirmed by McKay and coworkers,[2] irreversible heart failure (HF) occurs when post–myocardial infarction (MI) scar involves 20% of the LV area or 40% of its perimeter. Since 1967, knowledge of post-MI HF has been expanded by numerous studies showing that even after recanalization of the culprit artery at the acute phase of MI, the LV wall almost always becomes permanently scarred. In 80% to 100% of such cases,[3] the scarred wall shows extension from 6% to 80%. Twenty-seven percent of patients with restoration of Thrombolysis in Myocardial Infarction grade 3 flow after MI[4] have LV dilatation with HF, a finding that correlates with the conclusion of Gaudron and coworkers[5] that at 18 months after MI, 20% of patients are in congestive HF.

Since 2002, our institutional assessment of the scarred left ventricle after MI (with or without successful coronary recanalization) has been conducted in 840 patients by means of magnetic resonance imaging (MRI). The extent of gadolinium late enhancement (GLE) in 4 planes (2- and 4-chamber views, LV outflow tract, and short axis at the level of the papillary muscle base) allowed us to build a GLE map with the percentage of scarred LV perimeter (Figure 1) that correlates to the classical centerline method.[6] Ninety percent of patients have 10% or greater scarred LV perimeter (Figure 2), and 22% of these patients are in advanced HF (AHF), according to the criteria of the European Society of Cardiology,[7] or in acute HF. AHF exceeds 53% for those patients with a scarred LV perimeter of greater than 50%. The degree of AHF and the percentage of scarred circumference are more increased in those patients (n = 229) who had recanalization of an occluded coronary artery during the acute phase of MI (Figure E1, *A* and *B*). Recanalization, per se, does not carry deleterious effects (unless performed too late in a totally necrotic territory initiating the "myocardial reperfusion induced injury"[8]) but emergency recanalization was applied to the sickest patients.

These highly scarred left ventricles in patients with chronic HF and New York Heart Association (NYHA) class III or IV, in those with AHF, or in those with acute HF are often beyond any response to medical treatment, to resynchronization, or to revascularization therapies.[9] They represent ischemic failing ventricles (IFVs), the most severe category of ICM.

MATERIALS AND METHODS

From 2002 to May 2008, 274 patients after MI underwent LVR with EVPP at our institution.[10] One hundred seventeen patients had LV scarring of greater than 35%, (chronic NYHA class III/IV or acute HF) and met the exclusion criteria for the Surgical Treatments for Ischemic Heart Failure (STICH) trial, as listed in the "STICH investigator's handbook." The pertinent exclusion criteria are listed in Table 1.

All 117 patients had left and right heart catheterization, coronary angiography, and programmed ventricular stimulation to detect a potential ventricular arrhythmia induced by the scar. Asynergic scarring (dyskinetic or akinetic), systolic function (ejection fraction [EF], end-diastolic volume index [EDVI], and end-systolic volume index [ESVI]) and diastolic function (left atrial volume index [LAVI])[11] were assessed by means of MRI to be more reliable.[12] A GLE map was established for all patients with a 1.5-T imager (Siemens Sonata). For patients with intra-aortic balloon pumping or previous pacemaker or defibrillator implantation contraindicating MRI, the hemodynamic parameters were assessed by means of echocardiographic analysis, angiographic analysis, and computed tomographic scanning.

Table 2 shows the clinical characteristics and hemodynamic data for the 106 patients with chronic AHF and 11 patients with acute HF. Mitral insufficiency (56 patients) was quantified by using proximal isovelocity surface area, and the distribution of classification[13] is also presented in Table 2.

The preoperative assessment confirmed the fact that the trigger of HF was a large scarred akinetic wall (mean, 49.5% of LV perimeter; range, 35%–75%) and also that the mechanism that induces ventricular dilatation is (functionally but not histologically speaking) a deleterious systolic eccentric (centrifugal) motion (Figure E2 and Videos 1-3) of the noninfarcted myocardium surrounding the scar. This eccentric motion is best seen on MRI, immediately after MI, when the wall is still necrotic (no reflow or microvascular obstruction). The ongoing dilatation process is correlated to the amount of myocardial loss; it might take only a few weeks if this loss exceeds 50% of the left ventricle (evolving aneurysms) or up to months and years. This ineffective contraction is explained by "the extent of shortening of the remaining myocardium exceeding its physiological limits."[1]