

# Left Ventricular Volume Reduction by Radiofrequency Heating of Chronic Myocardial Infarction in Patients With Congestive Heart Failure

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**Background**—Myocardial infarct expansion and left ventricular (LV) remodeling are integral components in the evolution of chronic heart failure and predict morbidity and mortality. Radiofrequency (RF) heating and patch placement of chronic LV aneurysms caused a sustained reduction in LV infarct area and volume in an ovine infarct model. This study evaluated the effect of RF heating and epicardial patch as an adjunct to coronary artery bypass graft on LV volumes in patients with prior myocardial infarction, evidence of akinetic/dyskinetic scar, and LV ejection fraction  $\leq 40\%$ .

**Methods and Results**—Ten patients (3 female; mean age,  $64 \pm 11$  years) scheduled for coronary artery bypass graft were enrolled (Canadian Cardiovascular Society angina class  $2.1 \pm 1.1$ ; New York Heart Association class  $3.1 \pm 0.5$ ). Intraoperative digital photography demonstrated an acute 39% reduction in infarct area ( $n=5$ ;  $P=0.01$ ), and transesophageal ECGs demonstrated a 16% acute reduction in LV end-diastolic volumes ( $n=9$ ;  $P=0.002$ ) after RF treatment. There were no intraoperative or procedure-related postoperative complications, and during an average follow-up of  $>180$  days, there have been no safety issues. All patients had complete relief of their angina and improvement in exercise tolerance. Serial transthoracic ECGs over the 6 months of follow-up after RF treatment demonstrated persistent reductions in LV end-diastolic volume (29%;  $P<0.0001$ ) and LV end-systolic volume (37%;  $P<0.0001$ ) with improved ejection fraction ( $P<0.02$ ).

**Conclusions**—RF heating and patch placement in these 10 patients resulted in acute reduction in infarct area and ventricular volumes that were maintained 180 days after procedure. This technique may reduce the incidence of congestive heart failure and mortality in these patients and warrants investigation in larger clinical trials. (*Circulation*. 2002;105:1317-1322.)

**Key Words:** heart failure ■ myocardial infarction ■ remodeling ■ surgery ■ cardiac volume

Congestive heart failure afflicts almost 5 million people in the United States, and despite recent advances in pharmacological approaches to its treatment, the 1-year mortality rate remains high.<sup>1</sup> Ventricular remodeling plays a central role in the development of heart failure, and multiple experimental and clinical studies have shown that ventricular volumes are the most powerful predictors of mortality.<sup>2-7</sup> In addition, the pharmacological agents that have been successful in reducing mortality in this condition have also had significant effects on ventricular remodeling.<sup>8-10</sup> These observations have led to the hypothesis that interventions that beneficially influence ventricular remodeling may result in improved survival in patients with left ventricular (LV) dysfunction.

Multiple surgical techniques have evolved based on this hypothesis. These techniques often produce acute volume

reductions and, in some cases, improved ejection fractions. Unfortunately, many of these techniques, such as LV aneurysm repair, have at least modest operative mortality and limited potential to be performed in a minimally invasive manner and ultimately result in subsequent LV redilation.<sup>11,12</sup> However, the compelling promise of the hypothesis that ventricular volume reduction should be beneficial and the limitations of present surgical approaches have encouraged the search for surgical ventricular volume reduction techniques that have a lower operative mortality and improved long-term results.

In a recent study, we (M.B.R., A.W.W., and J.R.T.) demonstrated in an ovine model of myocardial infarction and heart failure that radiofrequency (RF) heating of myocardial infarct scar resulted in acute scar shrinkage and volume reduction that was sustained over a 10-week period.<sup>13</sup> RF

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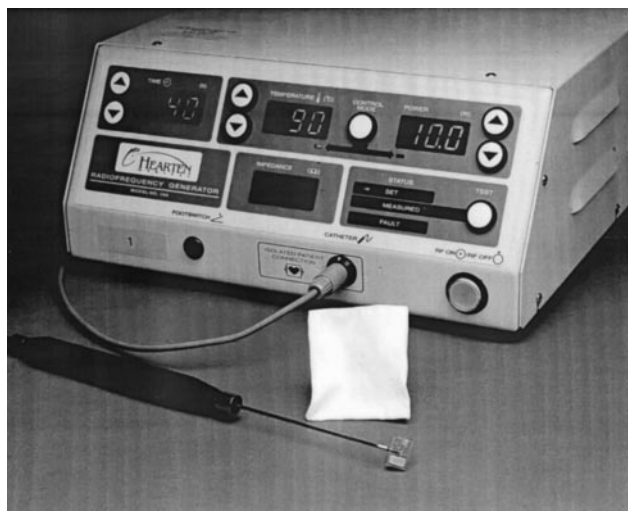
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**Figure 1.** Photograph of the Myotech cardiac restoration system with the RF generator, MyoMend RF applicator wand, and MyoGuard polyester patch material.

remodeling techniques are based on the principle that collagen denatures and contracts when heated above 65°C.<sup>14,15</sup> However, RF heating also makes collagen more likely to creep under load,<sup>16</sup> a finding that was also confirmed in our previous ovine study.<sup>13</sup> Consequently, application of a restraining patch was incorporated into the technique to prevent dilation of the treated scar area in the immediate posttreatment period.

The purpose of this pilot study was 3-fold: (1) to demonstrate the feasibility of applying this technique to patients, (2) to assess in a preliminary fashion safety concerns, and (3) to evaluate the effects of this technique on ventricular remodeling. The hypothesis of this study is that RF treatment of myocardial infarct scar with the MyoTech cardiac restoration system (Figure 1; Hearten Medical, Inc) as an adjunct to coronary artery bypass grafting in patients with prior myocardial infarction, evidence of an akinetic/dyskinetic scar, and a LV ejection fraction of  $\leq 40\%$  will produce significant and sustained reductions in left ventricular volume.

## Methods

### Patient Selection

Patients were enrolled in the study if they required nonemergent coronary artery bypass graft (CABG) surgery and had a prior history of myocardial infarction ( $\geq 10$  weeks earlier), evidence of chronic dilated LV infarct scar as defined by a LV ejection fraction  $\leq 40\%$ , and an akinetic/dyskinetic scar on ECG. Patients were excluded if there was evidence of calcification in the scar or thrombus as identified by echocardiography or contrast ventriculography. All patients gave informed written consent to participate in the study. The protocol was approved by the ethical and scientific committee of the Hospital de Especialidades del Centro Médico Nacional de Occidente, del Instituto Mexicano del Seguro Social and conformed to the International Scientific and Ethic Regulations.

### Preoperative Evaluations

Patients underwent cardiac catheterization with right anterior oblique (RAO) contrast ventriculograms at the referring hospitals as well as a history including New York Heart Association (NYHA) classification, physical examination, chest x-ray, ECG, exercise treadmill

(in patients who were able to walk) or dipyridamole stress testing with thallium perfusion imaging, and two-dimensional and Doppler transthoracic ECG. Patients still eligible by the inclusion and exclusion criteria were selected to undergo the intraoperative evaluation.

### Hospital Course

A brief repeat history and physical examination were performed, and blood chemistries, complete blood count, and ECG were obtained. After general anesthesia and sternotomy but before bypass and RF heating, a transesophageal ECG (TEE) was obtained in each patient to measure LV volumes. After isolation and inspection of the scar area, the RF device (MyoMend Wand; Hearten Medical, Inc) was then applied to the myocardial scar on the beating heart without ventriculectomy at normal body temperatures. The desired power, temperature, and application time were selected on the RF generator, and the hand-held wand electrodes were held against the infarcted scar tissue. A thermocouple monitored the temperature of the tissue during treatment to automatically maintain the desired conditions, and the power automatically shut off when the elapsed time had passed. Treatment was repeated in an overlapping, outward spiral pattern to cover the entire infarct zone. After treatment, the MyoGuard Patch (Hearthen Medical, Inc) was sutured within the perimeter of the scar to provide reinforcement during the patient's initial recovery. Intraoperative digital photography of the myocardial infarction scar before and after treatment to measure acute shrinkage was performed in the last 5 patients. In these patients, the patch was initially sized to the pretreatment scar area and then digitally photographed on a flat surface with a ruler. After treatment, the patch was trimmed to the posttreatment scar area and another digital photograph was obtained on the flat surface with the ruler. Planimetry of these areas yielded estimates of pretreatment and posttreatment scar areas. After the treatment was complete, cardiopulmonary bypass was initiated and standard CABG was performed. Posttreatment/post-CABG TEE off cardiopulmonary bypass was performed to measure the acute changes in LV volumes.

Postoperatively, patients were monitored for 24 hours with a Holter. Patients were followed daily during their hospital courses, and adverse events were recorded. On postoperative days 1, 3, and 5, physical examinations were performed, and blood chemistries, complete blood counts, and electrocardiograms were obtained.

### Follow-Up

Patients were followed by one of the operating surgeons (O.A.V.) as well as their referring cardiologists for at least 6 months after the procedure. Serial transthoracic ECGs were performed on days 10, 30, 90, and 180 after the procedure. At 6 months, repeat RAO contrast ventriculograms as well as history, including New York Heart Association (NYHA) classification, and physical examination, chest x-ray, ECGs, 24-hour Holter examinations, and exercise treadmill stress testing were also performed.

### Statistical Analyses

The single-plane RAO ventriculograms were obtained at different referring sites without uniform calibration, so changes in absolute volumes could not be determined. However, these studies were analyzed by 1 investigator (E.G.), and ejection fractions were calculated by planimetry using the area-length method. All of the serial ECGs were analyzed by echocardiography technicians at the local sites, whereas the baseline and 6-month transthoracic ECGs were analyzed at an investigator's (J.R.T.) remote core echocardiographic laboratory by a technician in a random, blinded fashion averaging 3 representative beats. All analyses were based on standard biplane apical views and volumes calculated by modified Simpson's rule. Results are expressed as mean  $\pm$  SD unless otherwise specified. Comparisons of baseline to postprocedure values are performed with a paired *t* test, whereas results from the serial ECGs were analyzed with a repeated-measures ANOVA. Statistical significance was considered attained when  $P < 0.05$ .

**TABLE 1. Patient Medications**

	On Admission	On Discharge	6 Month Follow-Up
ACE inhibitor (ARB)	8	8	4 (1)
β-Blockers	1	1	1
Long-acting nitrates	10	7	3
Digoxin	9	9	0
Loop diuretics	3	3	1
Amiodarone	1	1	0
Aspirin	10	10	10

ARB indicates angiotensin receptor blocker.

**Results**

**Demographics**

Ten patients (age 63±11 years; 3 women) were enrolled in the study, all of whom successfully completed the procedure and the 6-month follow-up period. The patients on average had a moderate degree of angina (Canadian Cardiovascular Society [CCS] class 2.1±1.1), were 2.8±2.9 years from their most recent myocardial infarction, and had moderate-to-severe heart failure symptoms (NYHA class 3.1±0.5). Half of the patients had hypertension, and diabetes mellitus (4 patients), hypercholesterolemia (3 patients), stroke (2 patients), and severe chronic obstructive pulmonary disease (2 patients) were also present. The patients' medications on admission are listed in Table 1.

**Preoperative Evaluations**

The 10 patients had a significant limitation in daily exercise tolerance (1.7±1.0 blocks without stopping). Eight of the patients were able to perform exercise treadmill tests on a modified Bruce protocol for 6.4±3.2 minutes (Table 2). The 2 other patients were unable to exercise because of musculoskeletal disorders. Single-plane RAO contrast ventriculography demonstrated decreased ejection fractions (31±6%).

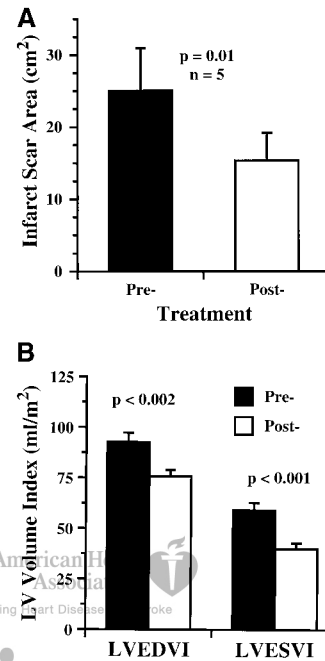
**Intraoperative**

Patients underwent 18±7 applications of the MyoMend wand followed by suturing of the patch in place, for a total treatment time of 40±17 minutes for the RF procedure, patch placement, TEE assessment, and scar sizing. All 10 patients completed the surgical procedure with an average total cardiopulmonary bypass time of 90±33 minutes and cross-clamp time of 51±23 minutes with 2.8±1.2 grafts. There were no intraoperative complications. Intraoperative digital photography was performed in the last 5 patients, demonstrat-

**TABLE 2. Symptom Assessments and Exercise Tolerance**

	Preprocedure	6 Month Follow-Up	P
Angina (CCS Class)	2.1±1.1	0±0	<0.0001
Congestive heart failure symptoms (NYHA Class)	3.1±0.5	1±0	<0.0001
Walking distance, blocks	1.7±1.0	14.4±8.4	0.0015
Exercise treadmill test tolerance, min	6.4±3.2	11.1±3.0	0.03

Values are expressed as mean±SD. Preprocedure and 6-month follow-up values are compared with a paired t test.

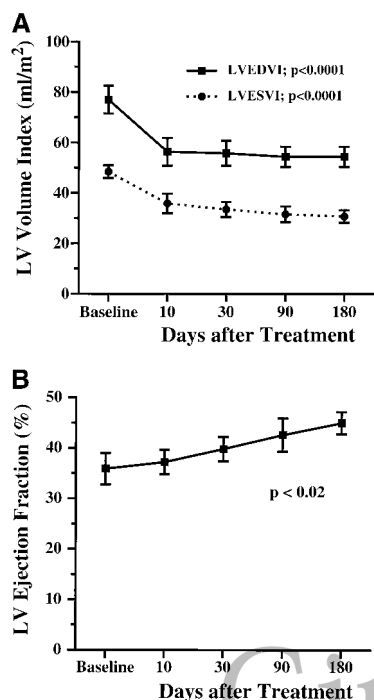


**Figure 2.** Acute effect of RF heating on left ventricle. A, Infarct scar area as measured by digital photography before and after RF heating. B, LV volumes before and after RF heating and CABG as measured by intraoperative transesophageal ECGs. Results expressed as mean±SEM.

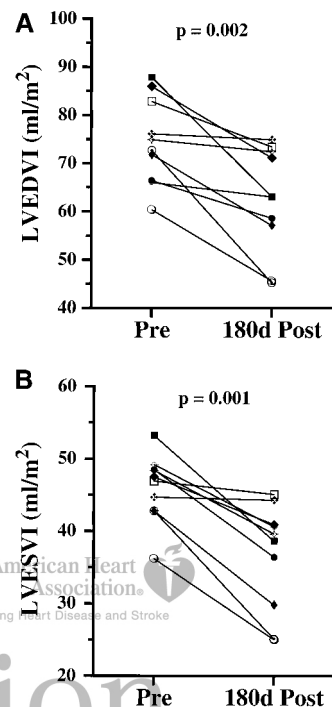
ing an immediate 39% reduction in scar area (Figure 2A). TEE volumes measured before and after the operative procedure showed commensurate acute reductions in ventricular volumes (Figure 2B; 16% reduction in LV end-diastolic volume [LVEDVI], 32% reduction in LV end-systolic volume [LVESVI]). There was minimal ( $r=0.12$  to  $0.41$ ;  $P=NS$ ) correlation between various measures of the extent of the acute reduction in ventricular volumes and the extent of scar shrinkage. Using a simple spherical model, a reduction of surface area of 9.7 cm² from a sphere with a volume of 156 mL (the mean preoperative TEE LVEDV) would result in a commensurate 17-mL volume reduction (less than the observed 30-mL reduction in LV volume).

**Hospital Course**

All of the patients did well postoperatively, and postoperative complications were limited and believed to be unrelated to the RF treatment. Complications included the following: 1 patient with transient supraventricular tachycardia; 1 transient disorientation on postoperative day 3 to 5; 2 possible small perioperative myocardial infarctions diagnosed solely by troponin-I levels (2 patients with troponin-I levels of 4.90 ng/mL [normal range, 0 to 1.5 ng/mL] on postoperative days 3 and 5, respectively); and 1 nosocomial pneumonia. Postoperative pulmonary artery thermodilution catheter demonstrated a cardiac output of 3.7±0.4 L/min per m², and there was no evidence of perioperative low-output state requiring intra-aortic balloon pump nor LV assist device implantation. Nine patients were discharged from the hospital without prolongation of hospital stay, whereas 1 patient with prior pulmonary disease was hospitalized for >1 month because of respiratory problems unrelated to the procedure.



**Figure 3.** Chronic effects of RF heating on LV volumes. Serial echocardiographic volume (A) and ejection fraction (B) measurements over 6 months of follow-up. Results are expressed as mean  $\pm$  SEM. Repeated measures ANOVA performed to analyze differences through time.



**Figure 4.** LVEDVI and LVESVI volumes indexed for body surface area before and 6 months after RF heating as measured by independent, blinded two-dimensional ECGs. Individual cases presented with paired *t* test comparison.

### Six-Month Follow-Up

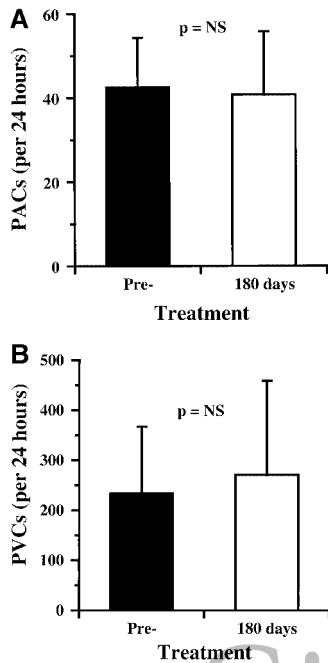
During an average follow-up of  $>180$  days, there were no safety-related issues (no hemorrhage, rupture, tamponade, stroke, renal failure, or patch-related infection) and no rehospitalizations for cardiac cause. All patients had complete relief of their angina (CCS class 0), and all patients were NYHA class I. In addition, the 8 patients able to exercise had a marked improvement in their exercise duration (73% increase to  $11.1 \pm 3.0$  minutes;  $P=0.03$ ) using the modified Bruce treadmill protocol (Table 2). Repeat single-plane RAO contrast ventriculograms demonstrated significant increases in ejection fraction at 6 months (baseline,  $31 \pm 6\%$ ; 6 months,  $39 \pm 7\%$ ;  $P=0.01$ ). Analysis of the serial ECGs obtained at baseline through 180 days after the procedure revealed the time course of these marked, sustained decreases in both LV end-diastolic (29% reduction at 6 months; repeated measures ANOVA,  $P<0.0001$ ) and end-systolic (37% reduction at 6 months; repeated measures ANOVA,  $P<0.0001$ ) volumes (Figure 3A) with no evidence of progressive ventricular dilatation, as well as time-dependent increases in LVEF (Figure 3B; 25% increase at 6 months; repeated measures ANOVA,  $P<0.02$ ). There was a positive trend toward an additional decrease in LVESVI from day 10 to day 180 after the procedure ( $P=0.06$ ; repeated measures ANOVA). Blinded analysis of the baseline and 6-month ECGs confirmed these significant reductions, demonstrating a 16% reduction in LVEDVI (Figure 4A; mean difference,  $12.1$  mL/m<sup>2</sup>; baseline,  $74.5 \pm 9.0$  versus 180 days after procedure,  $62.4 \pm 10.9$  mL/m<sup>2</sup>;  $P=0.002$ ) and a 21% reduction in LVESVI (Figure 4B; mean difference,  $9.5$  mL/m<sup>2</sup>; baseline,

$46.0 \pm 4.6$  versus 180 days after procedure,  $36.5 \pm 7.4$  mL/m<sup>2</sup>;  $P<0.001$ ) and increased ejection fraction (mean difference,  $3.9$ ; baseline,  $37.8 \pm 6.6$  versus 180 days after procedure,  $41.7 \pm 4.0$ ;  $P=0.06$ ). Stroke volume was maintained at all time points. Analysis of the Holters from baseline and at 6 months showed no significant change in either atrial or ventricular ectopy (Figure 5).

### Discussion

This study of ten patients undergoing concurrent coronary artery bypass graft surgery demonstrated that treatment of a chronic myocardial scar with RF heating resulted in acute reduction in infarct scar area with resultant decreases in ventricular volumes. This acute reduction in ventricular volumes was sustained for at least 6 months and was associated with marked improvement in all measures of symptoms and exercise tolerance. There were no safety-related concerns that were discovered during  $>6$  months of follow-up.

The primary finding of this study was that RF heating treatment of myocardial infarct scar resulted in significant reductions in ventricular volumes that were sustained over at least 6 months of follow-up. The 39% acute reduction in infarct scar area was nearly identical to the 34% reduction in the sheep infarct model using the same RF system,<sup>13</sup> as was the 16% to 37% decrease in LV volumes at 6 months after the RF treatment in patients compared with a 20% to 34% reduction in sheep at 10 weeks after the procedure.<sup>13</sup> In both studies, not only was there preservation of the initial relative volume reduction, but there was no evidence of progressive dilation as is typically seen in the setting of large infarctions,



**Figure 5.** Atrial (A) and ventricular (B) ectopy at baseline and 6 months after RF heating of myocardial scar. Results expressed as mean  $\pm$  SEM.

suggesting that the procedure may have interrupted the pathophysiological process of deleterious ventricular remodeling. In fact, there was evidence of progressive decreases in LVESVI from day 10 to day 180 after the procedure, with a stable LVEDVI, suggesting a possible improvement in LV function.

These results were clearly statistically significant, but are they clinically relevant? In the study by White et al<sup>5</sup> using contrast ventriculography, a 35-mL increase in LVESV resulted in an 8-fold increase in mortality at 5 years. In the present study, RF heating resulted in up to a 30-mL reduction in LVESV ( $-18 \text{ mL/m}^2$  LVESVI), as measured by biplane echocardiography. Some additional context for this question might be found in the pharmacological heart failure trials. In the first ACE inhibitor trials to assess the effects of captopril on ventricular enlargement, captopril treatment for 6 months demonstrated a 9.4-mL/m<sup>2</sup> reduction in LVESVI,<sup>17</sup> whereas another study demonstrated an 11-mL (4.3%) decrease from placebo with 1 year of captopril treatment.<sup>18</sup> In a substudy of the SOLVD treatment trial,<sup>9</sup> enalapril treatment for 1 year resulted in a 26-mL/m<sup>2</sup> (17%) decrease in LVEDVI compared with placebo-treated patients. Recent studies with  $\beta$ -blocker therapy in patients with chronic heart failure have shown similar results. In a study by Metra et al,<sup>19</sup> the administration of metoprolol or carvedilol for 13 to 15 months resulted in 9% (15 mL/m<sup>2</sup>) and 12% (20 mL/m<sup>2</sup>) reductions in LVEDVI. ACE inhibitors and  $\beta$ -blockers obviously have other effects than volume reduction, but the 16% to 37% sustained volume reduction using the RF heating technique in this study was similar to that produced by these agents and supports the hypothesis that these volume reductions may be clinically significant.

The principle by which RF heating causes reductions in ventricular volumes is based on the property of collagen to denature and contract at temperatures  $>65^\circ\text{C}$ . The extent of contraction is modified by the amount of cross-linking, age of scar, pH, temperature, load during heating, and water content.<sup>14,15,20</sup> Unloaded bovine chordae tendineae contract  $\approx 65\%$  when chordae are heated to  $85^\circ\text{C}$ ; similar findings were observed in tendons from the ovine glenohumeral capsule.<sup>20</sup> In this study, epicardial temperature at the treatment site was maintained at  $95^\circ\text{C}$ , identical to the temperature in the sheep studies.<sup>13</sup> Histological studies in the RF-treated sheep demonstrated evidence of the heating extending 50% of the thickness of the infarct scar. Because all patients are alive and well, there are no histological specimens available for the present study, but the findings in the sheep suggest that more aggressive heating might be able to increase infarct shrinkage even more. Of course there is the legitimate concern that full-thickness heating could result in rupture, but because the procedure is performed in the beating heart with the large heat sink of circulating blood, the thermocouple is able to regulate the temperature such that full-thickness heating can be avoided.

The RF heating system was designed to be widely applicable to a large group of patients. There are  $>7$  million patients with myocardial infarction in the United States alone and  $>1$  million new myocardial infarctions per year. Present treatment strategies have reduced the death rate by 26%, resulting in an increase in the number of myocardial infarction survivors.<sup>21</sup> Although the ratio of nontransmural to transmural myocardial infarctions is increasing<sup>22</sup> and most infarcts seen in the operating room no longer are confluent scar, available evidence suggests that RF heating of salt-and-pepper infarcts that are mostly collagen will still benefit despite the myocyte loss. In a study by Nagueh et al,<sup>23</sup> hibernating myocardium with  $>25\%$  fibrosis did not recover with revascularization. Furthermore, the extent of fibrosis correlated well with areas that failed to thicken ( $r = -0.83$ ,  $P < 0.01$ ) during noninvasive dobutamine echocardiography. Thus, there seems to be a noninvasive way to assess areas that would not improve from revascularization and might benefit from this procedure. In addition, RF heating of ankle ligaments has been performed through an arthroscope,<sup>20</sup> and the present RF heating system was designed to be adaptable to minimally invasive cardiac surgical techniques. Because the procedure can be performed off bypass, there is the possibility that future applications could be performed as minimally invasive, stand-alone procedures.

This study has some important limitations. First, it is a small study that cannot fully evaluate the safety and efficacy of this technique. RF heating has been used safely in other applications, and in the present study of 10 patients, RF heating was associated with no evidence of any adverse effects during the 1 year of follow-up. However, this study is too small to be confident regarding safety issues. Although the study was designed to enroll consecutive patients meeting the enrollment criteria, the group of patients in this study was selected and may not be representative of the population as a whole. Second, echocardiographic follow-up was limited to 6 months. As noted above, most previous studies of ventricular

remodeling have demonstrated additional dilation during a 6-month follow-up period, so we suggest that there should have been some evidence of dilation noted in the present study during this follow-up period. Third, the absence of a control group is an important limitation. Although most studies suggest that patients with large myocardial infarctions continue to have significant dilation over 6 months, the CABG procedure itself may have resulted in the reduced volumes observed in this study. Unfortunately, the serial changes in ventricular volumes after CABG are relatively unknown, especially in this patient population. However, we suggest that this mechanism is unlikely given the acute volume reductions observed by TEE and the recent studies that show no change in resting ejection fractions in patients similar to those enrolled in this study.<sup>24</sup> Finally, this study did not investigate the underlying mechanism for the improvement in ventricular volumes. As noted in the results, the extent of volume reduction was disproportionate to that expected given the reduction in surface area from scar shrinkage. This disparity could be attributable to many factors, including technical limitations in the scar area measurements, which may have underestimated the scar area reduction, or improvements in ventricular geometry or loading characteristics, resulting in beneficial remodeling. The present study is solely hypothesis generating, but it seems to have developed the intriguing hypothesis that RF heating in these patients significantly reduced LV volumes and was safe.

This study demonstrates that RF treatment of myocardial infarct scar with the MyoTech system as an adjunct to CABG resulted in significant and sustained reductions in LV volume without significant treatment-related adverse events in patients with prior myocardial infarction, evidence of an akinetic/dyskinetic scar, and LV ejection fraction of  $\leq 40\%$ . The ability of this treatment to be applied to millions of patients and the potential to be accomplished with minimally invasive techniques suggests that RF heating of infarct scar may be an important technique to reduce ventricular volumes and possibly reduce the incidence of congestive heart failure and mortality in this high-risk group of patients. Larger controlled clinical trials are being planned to measure the effect on ventricular remodeling and patient outcomes.

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