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LETTER TO THE EDITOR

Postinfarction Heart Failure: A New Calling for Cardiac Surgeons

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I noted with interest the New Technology Center of *The Heart Surgery Forum* (http://newtech.hsforum.com/) and the currently featured TR^{3}I SVR (Chase Medical, Richardson, Texas, USA) system of surgical ventricular restoration (SVR) using an endoventricular shaper device. This technology accompanies a renewed interest in heart failure as the number of cases increases and the limitations of medical therapy are recognized. I have begun to use this procedure in patients for whom there are otherwise few realistic treatment options. Embracing this surgical option involved a reorientation of thinking to accept that physiological abnormalities of both shape and size are amenable to surgical correction. The safety and efficacy of surgical reconstruction of the ventricle has been demonstrated by several groups, including the RESTORE Group (reconstructive endoventricular surgery, returning torsion original radius elliptical shape to the left ventricle [LV]), and cardiac surgeons may offer the next major advancement in the clinical course of therapy for ischemic heart failure [Athanasuleas 2001].

Ventricular reconstruction techniques have evolved to include multifaceted approaches to restore LV size, shape, and function with the goal of improving the structural basis of the pathophysiology of the failing heart [Dor 2002, Kaza 2002]. These approaches include coronary revascularization to relieve ischemia, ventricular volume decrease, restoration of the ventricle to a more normal geometry, and volume overload decrease through mitral valve repair. Although there have been no randomized trials completed to date, experience with SVR in post–myocardial infarction patients has shown encouraging 1-year and 5-year survival rates of 98% and 82%, respectively, following an initial overall operative mortality of 7% to 8% [Athanasuleas 2001, Calafiore 2003, Di Donato 2001, Dor 2002]. Coronary artery bypass grafting is routinely and successfully performed to revascularize the ischemic heart, but it often leaves patients facing a lengthy battle with progressive ventricular dysfunction and advancing heart failure.

The importance of restoring the geometry of the dilated ventricle to normality of both size and shape has been emphasized by Buckberg [2001], who suggests that more attention should be given to size as measured by left ventricular end-systolic volume index rather than ejection fraction. The LV has a distinct shape that gives the heart its enormous pumping efficiency. Remodeling increases hemodynamic wall stress, decreases mechanical performance, and increases regurgitant flow through the mitral valve. Physiological, structural, mechanical, genetic, and biochemical processes sustain and exacerbate the remodeling process [Francis 2001]. Cardiac remodeling is now recognized as a major determinant of the clinical course of heart failure [Cohn 2000].

The recognition that both akinetic and dyskinetic tissue should be excluded represents an advancement of SVR beyond traditional aneurysmectomy [Dor 2001]. Today, fewer patients present with distinct LV aneurysms, because early intervention saves the epicardium. The saved epicardium may appear to be healthy tissue but may disguise the diseased tissue underneath that contributes to the progressive worsening of heart failure. Reperfusion produces endocardial necrosis while sparing epicardial tissue, resulting in asynergy.

The TR^{3}I SVR endoventricular shaper device adds precision to the SVR technique and supports the surgeon's ability to consistently and optimally resize, reorient, and reshape the LV. During SVR, a significant challenge is reconstruction of the ventricle after identification of the akinetic and dyskinetic tissue to be excluded. The shaping device is used to more precisely determine LV size and orientation as well as to identify the location of the new apex. A ventricle that is too large, too small, or box shaped results in suboptimal or deleterious outcome [Dor 2001].

Clearly, some questions remain regarding ventricular reconstruction. An important factor relative to acceptance of SVR is our ability to more accurately identify patients likely to benefit from the procedure. Data regarding preoperative risk factors have been published [Menicanti 2002]. These include a worsening functional class, ejection fraction less than 20 percent, age greater than 70 years, urgent intervention,

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mitral procedure, systolic pulmonary hypertension greater than 60 mm Hg, the number and sites of previous myocardial infarctions, and right ventricular dysfunction. Subsequent quantitative indications and relative contraindications have also been identified. However, surgeons may need to take a more aggressive approach to encourage evaluation of ventricular function and directly screen patients who may benefit from SVR.

Other ventricular remodeling techniques have been or are currently being evaluated. These include cardiomyoplasty, LV resection (Batista) and variations, splint and compression devices, and left ventricular assist devices [Vitali 2003]. In patients with conduction delay, cardiac resynchronization with biventricular pacing has resulted in substantial clinical improvement and reversal of remodeling [Yu 2002]. Continued rigorous evaluation of SVR and these procedures will be necessary to define specific patient selection and determine which technique(s) may benefit the largest number of patients with heart failure.

Cardiac magnetic resonance imaging is probably the best way to obtain quantitative measurements and to provide the most discrete visual images to assess wall motion and performance volumes of the LV before and after the procedure, and thus it should be used more [Kim 2000]. The quantification of multiple aspects of cardiac geometry, architecture, and function will likely provide invaluable information to guide patient selection and the reconstruction procedure.

While we wait for further pharmacologic and technologic advances, patients are dying. The necessity for alternative treatments is indisputable. Heart failure affects nearly 5 million people in the United States, and the incidence is increasing annually. By 2010, the number of patients suffering from heart failure is projected to increase by 40% to nearly 7 million. This growth is driven by two trends: the aging of our society and advances in the treatment of other cardiovascular disorders, preventing premature death. Unfortunately, current medical therapeutic options do not correct the underlying structure of the failing heart and, as a result, fail to change the slope of survival curves for heart failure. Overall 1-year and 5-year mortality rates (1990-1999) for heart failure are 25% and 50%, respectively [Levy 2002]. Transplantation is the gold standard for end-stage heart failure. In the year 2000, only 2202 heart transplantations were performed in the United States, demonstrating that it is not a viable option for the vast majority of heart failure patients. Survival rates for transplantation patients are 81% to 85% in the first year and 70% to 71% at 5 years. It is time to rethink the pathophysiology of heart failure, to imagine that postinfarction remodeling is an anatomical maladaptation, and to consider that surgical restoration offers a lasting benefit that is not available through any other form of treatment for heart failure.

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