

Surgical Remodeling of the Left Ventricle in Heart Failure

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Introduction

Despite significant advances in medical management, heart failure (HF) remains a significant cause of morbidity and mortality. The incidence of HF is reaching epidemic proportions, with more than 500,000 new cases diagnosed yearly in North America.¹⁾ The Framingham study showed that both increased age and hypertension are significant risk factors for developing HF.²⁾ Costs associated with treating HF exceed \$35 billion/year. Five-year mortality in advanced stages of HF is less than 50%.^{3,4)}

Coronary artery disease is the principle cause of HF with left ventricular (LV) dysfunction.⁵⁾ Yoshida and Gould⁶⁾ demonstrated a significantly worse 3-year survival in patients with LV dysfunction associated with infarction. To compensate for reduced ventricular function, the LV dilates to increase stroke volume, a process known as post infarction remodeling. There are two distinct phases: early remodeling (within 72 hours) which is characterized by infarct expansion with wall thinning and ventricular dilatation, with subsequent increases in wall stress; and late remodeling (beyond 72 hours) in which cardiomyocytes hypertrophy in response to the increased wall stress.⁷⁾ These phases are governed by a complex series of processes involving mechanical, genetic, and neurohormonal factors. Unfortunately, remodeling leads to alterations in the LV architecture, such that it takes on a spherical rather than elliptical form, further increasing wall stress, resulting in a

vicious cycle ultimately leading to HF. Thus, procedures to reduce the diameter of the LV and return it to a more physiologic structure appear to be a treatment option. Cooley et al.⁸⁾ reported the first successful excision of a post-infarction LV aneurysm with the use of the newly developed cardiopulmonary bypass circuit.

The recent publication of the results of Hypothesis 2 of the Surgical Treatment for Ischemic Heart Failure (STICH) trial has focused attention on the efficacy of surgical remodeling of the LV.⁹⁾ This is the first large-scale randomized trial of the efficacy of surgical remodeling in patients with post-infarction ventricular dilatation. This review will examine the different surgical techniques for LV remodeling, as well as newer devices currently undergoing investigation for clinical application.

Partial Left Ventriculectomy (Batista procedure)

In the 1990's Batista et al. developed a procedure to reduce ventricular volume in patients with end-stage dilated cardiomyopathy with ejection fractions < 20%.¹⁰⁾ In this operation, a wedge of LV muscle between the papillary muscles is resected from apex to mitral annulus. If papillary muscles are resected (extended procedure), the mitral valve is replaced. A report of the outcome of this procedure from two centres (Campina Grande, Brazil and Buffalo, New York) in 120 patients in New York Heart Association (NYHA) class IV HF showed a 30-day mortality of 22% and 2-year survival rate of 55%.¹¹⁾ Dramatic improvements in NYHA functional class were seen in 90%. Measurements in these patients undergoing this procedure showed that wall stress was reduced by up to 64% depending on the extent of chamber diameter reduction.¹²⁾ This was associated with significant improvements in both stroke volume and cardiac index, and significant reductions in pulmonary artery pressures. Schreuder et al.¹³⁾ also found significant increases in LV ejection fraction and decreased LV wall stress 5 days after partial left ventriculectomy. Caution must be used in interpreting any index of cardiac

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Table 1. Current indications and contraindications for SVR^{18,19)}

Indications	Contraindications	
	Relative	Absolute
Symptoms of HF	Severe pulmonary hypertension not associated with MR	Severe RV dysfunction
Prior anterior MI		Severe regional asynergy without ventricular dilatation
LV dysfunction with dilatation LVEDVI > 100 ml/m ² Regional LV akinesia/dyskinesia Angina/HF/arrhythmias Inducible ischemia		Restrictive diastolic pattern associated with high functional class and MR

SVR, surgical ventricular restoration; HF, heart failure; MR, mitral regurgitation; RV, right ventricular; MI, myocardial infarction; LV, left ventricular; LVEDVI, LV end-diastolic volume index.

function that relies on the use of ejection fraction. A reduction in LV volume results in a decrease in end-diastolic volume, thereby increasing ejection fraction without necessarily increasing myocardial contractility. While others have also reported improved postoperative cardiac function and reduced ventricular dimensions following partial left ventriculectomy, concerns have been raised about the unpredictable outcome of this operation and the lack of clear indications for its use. The Cleveland Clinic reported on 62 patients with dilated cardiomyopathy (59 eligible for transplantation, all NYHA class III or IV) who underwent this procedure with concomitant mitral valve surgery.¹⁴⁾ There were 2 in-hospital deaths. Survival at 1- and 3-years post-surgery was 80% and 60%, respectively. This was associated with an 18% rate of LV assist device (LVAD) implantation (11 patients). However, freedom from a return to class IV failure was 57% at 1 year and 42% by 3 years; overall event-free survival (encompassing all cause mortality, LVAD implantation, return to class IV HF, automatic implantable cardioverter defibrillator [AICD] implantation) was 49% by 1 year and only 26% by 3 years. Overall, this group concluded that the Batista procedure is not a suitable alternative to cardiac transplantation for end-stage HF. Currently, partial left ventriculectomy has largely been abandoned. The most recent American College of Cardiology/American Heart Association (ACC/AHA) guidelines do not recommend the use of this procedure in adult patients with non-ischemic cardiomyopathy and refractory HF (class III, level of evidence C).¹⁵⁾

Left Ventricular Aneurysmectomy (Dor Procedure)

LV aneurysms develop in areas of transmural infarction,

where necrotic tissue is gradually replaced by scar tissue, producing an area of akinesis/dyskinesis. Although the first successful aneurysmectomy was performed in 1955,¹⁶⁾ Cooley et al. developed the first procedure for surgically treating LV aneurysms that utilized cardiopulmonary bypass, in which the dyskinetic region is resected and the ventricle is closed linearly.⁸⁾ This technique is not capable of dealing with aneurysms involving the interventricular septum and does not restore the normal elliptical shape of the LV cavity. Therefore, in the 1980's Dor developed a new procedure in which the aneurysm is resected and a circular dacron or pericardial patch is used to reconstruct the ventricle.¹⁷⁾ This technique is referred to known as the Dor procedure, endoventricular circular patch plasty (EVCPP), or surgical ventricular restoration (SVR). Table 1 shows the indications and contraindications for SVR based on recent work.^{18,19)} Mickleborough et al.²⁰⁾ described a technique using the linear technique combined with septoplasty for cases of LV aneurysm involving the interventricular septum and reported good long-term outcomes.

The Dor procedure has the advantage of excluding akinetic septal regions of the LV, and restores LV chamber size and shape to a more physiologic condition. Ventricular volume is usually established at 60 ml/m² by inserting a balloon of known volume into the ventricle during reconstruction.²¹⁾ Normal LV end-systolic volume index (LVESVI) is 24 ± 10 ml/m².²²⁾ When combined with coronary revascularization, this procedure results in significantly improved LV function, reduced ventricular volumes, and improved NYHA class at 1-year post-surgery.²³⁾

A number of studies have compared the linear versus endoventricular patch repair for LV aneurysms. A retrospective examination of 95 consecutive patients with

post-infarction LV aneurysms operated at a single institution from 1989 to 1998 found no difference between groups in perioperative mortality (patch 12% vs linear 7%).²⁴⁾ Choice of technique was at the discretion of the surgeon. Long-term survival was not different up to 10 years post-surgery. However, other, larger retrospective studies have shown significant differences in early mortality and 5-year survival.^{25,26)} Differences in these results may be related to the smaller size of the negative study, or the differences in the era in which these operations occurred, particularly due to improved methods of myocardial protection. A recent meta-analysis showed a higher relative risk of early and late mortality in patients undergoing LV reconstruction for post-ischemic aneurysm via the linear procedure.²⁷⁾

The Reconstructive Endoventricular Surgery returning Torsion Original Radius Elliptical shape to the LV (RESTORE) group published 5-year results of a large, multinational study on the efficacy of SVR on early and late survival in 1,198 post-anterior infarction patients with congestive HF (CHF).²⁸⁾ They found an overall 30-day mortality of 5.3% and a 5-year survival of 68.6%, and significantly improved LV function and reduced dimension. The use of perioperative intra-aortic balloon pumps was 8.2%, LVAD 0.7%, and extracorporeal membrane oxygenation (ECMO) 0.3%. Risk factors for death were an ejection fraction $\leq 30\%$, preoperative NYHA class III/IV, preoperative LVESVI ≥ 80 ml/m², and age ≥ 75 years. Overall, these studies of surgical reconstruction suggest it is an effective therapy for dilated cardiomyopathy with excellent 5-year outcomes and low perioperative mortality. This formed the basis of the design of the STICH trial.

STICH is the first multicentre, randomized controlled trial to address whether the addition of SVR to coronary artery bypass grafting (CABG) in patients with LV anterior dysfunction will improve survival compared with CABG alone.²⁹⁾ Inclusion criteria were age > 18 years, LV ejection fraction $\leq 35\%$, and coronary artery disease suitable for revascularization. SVR was performed with or without a patch. The results of this part of the study (Hypothesis 2) were surprising. While CABG + SVR (n = 501) reduced LVESVI by 19% vs 6% by CABG alone (n = 499), over a median follow-up of 48 months there was no significant difference in the primary outcome of a composite of death from any cause and hospitalization for cardiac causes (58% CABG + SVR vs 59% CABG only). It was hypothesized that SVR may impede LV filling, thereby counteracting any beneficial effect on systolic function. It is important to note that total pump time was 25% longer



Fig. 1. Acorn CorCap Cardiac Support Device.
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($p < 0.001$) and aortic cross clamp time was 29% longer ($p < 0.001$) in the CABG + SVR group. Cardiac care unit/intensive care unit (CCU/ICU) stay was also significantly longer in patients receiving SVR. Whether these increased times affected outcome is unknown.

Cardiac Support Devices

In recent years a number of cardiac support devices have been developed to provide circumferential support to decrease LV wall stress in the hope of reducing or preventing dilatation and HF. Among these is the CorCap Cardiac Support Device (Acorn Cardiovascular, Inc., St. Paul, MN) (Fig. 1). This device consists of a polyester mesh bag that is surgically implanted such that it surrounds the heart, passively providing mechanical diastolic support. This is similar to cardiomyoplasty using the latissimus dorsi muscle to wrap the heart. Due to its passive nature, the CorCap does not support systolic function. The device covers the heart from apex to atrioventricular groove and is adjusted at implantation by the surgeon to fit snugly at end-diastole, thereby resisting dilatation.³⁰⁾ Investigations using a sheep model of HF secondary to acute anterior infarction showed that this cardiac support device significantly decreased end-diastolic volume by 39% and increased ejection fraction by 90%.³¹⁾ Clinical safety and feasibility studies of the CorCap device in 48 patients with dilated cardiomyopathy (LV end-diastolic diameter: 71.3 ± 7.1



Fig. 2. Paracor HeartNet on heart model.
(Photo courtesy of Paracor Medical, Inc.)

mm) and dysfunction (ejection fraction: $22.3 \pm 8.9\%$) showed that average implantation time was 27 minutes, and that survival was 73% at 1 year and 68% at 2 years.³²⁾ Mann et al.³³⁾ reported the first multicentre, prospective randomized controlled trial of the Acorn CorCap. Three hundred patients with NYHA class III and IV HF were enrolled to test the safety and efficacy of the device. Patients requiring mitral valve surgery were allocated to undergo either mitral valve surgery or mitral valve surgery + the Acorn device, while those not requiring mitral surgery were allocated to either optimal medical management or medical management + the Acorn device. Overall, 148 patients received the Acorn CorCap. The primary endpoint was a composite of death, the need for major cardiac procedures (biventricular pacing, CABG surgery, repeat mitral valve surgery, tricuspid valve surgery) for worsening HF, and a change in NYHA class. The odds ratio for the primary endpoint favoured treatment with the CorCap (1.73, CI 1.07 to 2.79, $p = 0.024$). Although there was no difference in survival between control and treatment groups, those receiving treatment had significantly fewer cardiac procedures and improved NYHA class compared with patients in the control group. LV end-diastolic and end-systolic volumes were also significantly less in the treated group up to 12 months after surgery. Adverse event rates were similar between groups. These results are encouraging, suggesting that the Acorn CorCap cardiac support device is safe to use in cases of dilated cardiomyopathy in symptomatic patients receiving optimal medical therapy and has a favourable affect on LV remodeling.

A different ventricular support system is the Paracor

HeartNet (Paracor Medical, Inc., Sunnyvale, CA) (Fig. 2). Constructed of a fine nitinol mesh which is highly elastic, the HeartNet is designed to cover both the left and right ventricles, providing a small but continuous epicardial force. Nitinol is radio-opaque, echogenic, and MRI compatible, and has proven biocompatibility in humans.^{34,35)} Feasibility studies in an ovine model of dilated cardiomyopathy showed no evidence of device migration, injury to coronary vessels, cardiac perforation, or myocardial damage after 6 weeks.³⁶⁾ Unlike the Acorn CorCap, which requires sternotomy and cardiopulmonary bypass, the HeartNet is designed to be implanted through a left mini-thoracotomy. Sizing can be determined pre-implantation via echocardiography. After a small pericardial incision, the apex of the heart is fixed by a suction device and the HeartNet is stretched over the epicardial surface of the ventricles using a deployment system. Correct placement can be verified via direct visualization, fluoroscopy, or echo. Safety and efficacy was investigated in 51 patients with symptomatic HF (NYHA class II/III, ejection fraction $\leq 35\%$) and dilated cardiomyopathy.³⁷⁾ Implantation was successful in 98%. Adverse events included 2 deaths within 30 days from pulmonary complications, 2 cases of epicardial laceration at implantation requiring repair, phrenic nerve injury (4%) and arrhythmias (27%). Four patients experienced worsening HF symptoms, with 3 ultimately requiring placement of a ventricular assist device. At 6 months, significant improvements were seen in 6-minute walk test and LV echocardiographic indices. Based on these encouraging results, a randomized controlled trial to investigate the HeartNet in patients with

symptomatic HF due to ischemic or nonischemic cardiomyopathy is underway. The Prospective Evaluation of Elastic Restraint to Lessen the Effects of Heart Failure (PEERLESS-HF) trial is currently recruiting patients. Completion of the study is expected in 2010.

An alternative to these cardiac support devices is the Myosplint (Myocor, Inc., Maple Grove, MN) (Fig. 3). This consists of a tensioning rod with epicardial pads at either end and is intended to be placed as a transcavitary restraining system. Typically, three are placed in the ventricle to draw the anterior and posterior walls together, creating a bilobular LV cross-section. This effectively reduces the radius of the chamber, reducing wall stress. Proof of concept studies at the Cleveland Clinic using a dog model of pacing-induced HF showed that the Myosplint improved LV ejection fraction, reduced LV end-systolic volume, and reduced LV wall stress, and that these changes were sustained for 1 month.³⁸⁾ Safety and feasibility in humans was investigated in 21 symptomatic patients with idiopathic dilated cardiomyopathy (NYHA class III/IV), with 9 receiving the device only and 12 receiving the device + mitral valve repair.³⁹⁾ Patients were followed at 3 and 6 months. Implantation required from 40 to 50 minutes; 6 patients required intraoperative repositioning of the device to optimize LV bisection. Three deaths occurred during follow-up; 2 from sepsis and 1 due to sudden ventricular tachyarrhythmia. Patients receiving the Myosplint had significantly reduced LV end-systolic and end-diastolic volumes at 3 and 6 months. NYHA functional class was also significantly improved. In 2008, Myocor suspended clinical trials of the device and Myocor's intellectual property assets were acquired by Edwards Lifesciences.

Conclusion

The incidence of HF will likely continue to rise as the population ages. With increased survival post-myocardial infarction, it is also likely that increasing numbers of patients will experience the deleterious effects of ventricular remodeling, namely, ventricular dilatation. Although both the Batista and Dor procedures have been shown to have clinical efficacy, intensive investigation has failed to demonstrate convincing evidence that either of these operations improves long-term survival. New devices are currently being investigated that may prove to be beneficial. Passive ventricular support devices appear to be effective in preventing progression of dilatation, but clinical trials are ongoing. Considerable work is still needed to determine the optimal surgical management of this high-risk patient population.

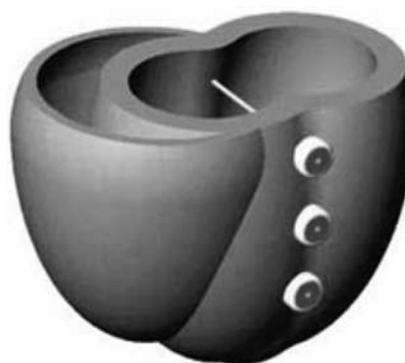


Fig. 3. Myocor Myosplint.
(Used with permission of Edwards Lifesciences)

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