

# Organ-conserving cardiac surgery

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## ABSTRACT

The goal of organ-conserving cardiac surgery is the preservation of heart function in the setting of heart failure. One way to improve heart function is left ventricular surgical reconstruction. This article briefly reviews the pathophysiology of left ventricular cardiomyopathy and the evolution of surgical left ventricular reconstruction techniques. These techniques are now part of a well-accepted concept with satisfactory perioperative mortality and morbidity. Further studies are warranted before definitive recommendations regarding surgical ventricular reconstruction can be given.

**Keywords:** *left ventricular surgical reconstruction, surgical techniques, heart failure.*

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## INTRODUCTION

There are a variety of options for patients with severe cardiomyopathy being evaluated for surgical therapy. These include cardiac resynchronization therapy, surgical coronary revascularization, valve repair or replacement, implantation of ventricular assist devices and heart transplantation. One of the most challenging tasks for cardiac surgeons, however, is organ-conserving cardiac surgery, which should be considered before the ultimate treatment with assist device implantation or heart transplantation comes to mind. The term “conserve” is defined by the Oxford Dictionaries, <http://oxforddictionaries.com> (accessed 18 January 2013) as protection from harm or destruction. Organ-conserving surgery is usually applied to the field of oncology

and means surgery designed to preserve, or to remove, diseased or injured organs or tissues with minimal risk. In cardiac surgery, the term “organ conserving strategy” may refer to the preservation of heart function in the setting of heart failure. In patients with complications after myocardial infarction (MI), such as severe remodeling, left ventricular dilatation, functional mitral insufficiency and impaired left ventricular (LV) function, one technique used in the organ-conserving strategy is surgical ventricular reconstruction (SVR).

This article briefly reviews the pathophysiology of LV-remodeling and the evolution of surgical techniques used for SVR.

### ***Pathophysiology of LV remodeling***

In ischemic cardiomyopathy, LV remodeling begins within the first few hours after a myocardial infarction (1). Remodeling is, in fact, a complex phenomenon, which involves myocyte stretch and is associated with increased expression of angiotensin II, norepinephrine and endothelin

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(2). These neurohormonal changes lead to myocyte hypertrophy, collagen synthesis, fibrosis and remodeling of the extracellular matrix.

The changes at the molecular and cellular levels lead to structural myocardial alterations, including chamber dilatation, altered configuration and increased wall stress. This domino effect starts with occlusion of one of the main coronary arteries. To compensate for the acutely reduced ventricular contractility, the volume increases, which is interpreted as an effort to maintain normal cardiac output.

If the left anterior descending artery is involved, the apex loses its spiral orientation and becomes rounded (3). Thinning and dilation of the infarcted region are usually accompanied by eccentric hypertrophy of the non-infarcted remote regions, resulting in increased wall mass and, effectively, in shape distortion of the entire heart with volume-overload hypertrophy of the non-infarcted myocardium (3). In patients with continuing heart dilatation, the LV end-systolic volume index (LVESVI, mL/m<sup>2</sup>) increases and LV ejection fraction (EF) drops as a result of the loss of myocyte contractile function (4).

From the physical point of view, the best way to interpret the changes in the LV remodeling process is to apply Laplace's law: LV wall tension is the product of the pressure within the ventricle, multiplied by the volume within the LV chamber and divided by the wall thickness.

The initial increase in wall thickness in non-infarcted regions seems to be a compensatory mechanism to reduce wall tension. However, ongoing dilatation of the LV chamber and the increasing radius lead to an increase in wall tension, which is unavoidable in maintaining systolic function. These pathophysiological pathways are part of a vicious cycle, in which cardiac output is reduced as oxygen requirements are

increased (5). Surgical resection of dysfunctional myocardium reduces the size of the LV cavity and, according to Laplace's law, may reduce wall stress. When performing SVR, it is essential to consider the physics of remodeling based on LV volume/mass ratio, in order to improve cardiac work efficiency.

### ***Surgical procedures***

The surgical techniques of ventricular reconstruction were initially used for repairing ventricular aneurysms. Ventricular aneurysmectomy was popularized in the 1950s by Cooley (6) and these techniques were further developed and improved over the years by Jatene, Dor and Buckberg.

*Batista procedure.* The Batista procedure was developed in 1994 for use in patients with non-ischemic cardiomyopathy. This technique is a partial left ventriculectomy, which involves resecting a portion of the LV lateral wall between both papillary muscles from the apex to the mitral annulus with or without repair or replacement of the mitral valve.

The removed myocardium is usually not a scar, but poorly functioning tissue. The result of the Batista procedure is reduced wall stress by reducing LV volume, which was assumed to improve the LV function (7). However, the postoperative outcome was not satisfactory. Several high-volume centers presented their results with the Batista approach, showing an increase in LV systolic function and reduced volume of the LV chamber.

Disappointingly, event-free survival rates remained low with increased long-term mortality that even reached 60% at 1 year (8-10). Currently, the Batista procedure is no longer recommended for patients with advanced heart failure (5).

*Surgical ventricular reconstruction.* In 1985, Dor developed an approach to surgical ventricular reconstruction in the set-

ting of LV aneurysm after a myocardial infarction. This technique is defined by exclusion of the scarred left ventricle, prevention of progressive dilation and the creation of an appropriate diastolic chamber size (11). The scarred LV wall is excluded by an endoventricular circumferential suture (Fontan technique), which “should be placed at the border between the scarred and normal myocardium” (11). The result of the Dor technique is a more physiological LV chamber geometry. Dor underlined that this technique must be accompanied by eliminating mitral insufficiency and achieving complete coronary revascularization, including anastomosing a graft to the left anterior descending artery. To maintain a normal diastolic volume, he promoted preoperative ventricular sizing, suggesting a reduction of the volume to 60 ml/m<sup>2</sup> and 70 ml/m<sup>2</sup> in patients with preoperative LVEDVI <150 ml/m<sup>2</sup> and LVEDVI >150 ml/m<sup>2</sup>, respectively. To achieve the proper volume, he suggested tying the circumferential suture on a rubber balloon inflated to the target volume. Late mortality reported by Dor et al. was under 5% and the patients presented significant improvement of ejection fraction.

These techniques were further modified by Buckberg and the RESTORE group showing excellent results with these SVR techniques, provided that they were applied carefully and the reduction in LV dimensions was more than 30% (12). Detailed descriptions of the surgical techniques used for SVR have been published (12, 13). Based on the favorable results of these and other published reports, a multicenter, international, prospective, randomized study was initiated (Surgical Treatment for Ischemic Heart Failure, STICH) (14) to test the hypothesis that the addition of SVR to coronary revascularization is beneficial for the long-term survival in patients with ischemic cardiomyopathy

and severely enlarged LV. Unfortunately, many methodological flaws of the STICH trial (15) (study design, change of the inclusion criteria after start of the trial, etc.) prevented the drawing of any meaningful conclusions. In the meantime, several studies have been reported showing that good results can be obtained by using the proper inclusion criteria and adequate intraoperative techniques (11, 16). Left ventricular reconstruction techniques can also be used with achieving of a conical shape of the LV apex with septal reshaping (17).

#### ***Practical considerations***

The 3 main contraindications for LV reconstruction surgery are cardiogenic shock, emergency procedure and end organ failure (renal, hepatic, pulmonary) (18).

Based on our experience, SVR should be performed no earlier than 3 months after an acute myocardial infarction. We usually perform all of these procedures on cardiopulmonary bypass with the beating heart. In patients with ventricular thrombi, we use the cross-clamp under blood cardioplegic arrest (ante- and retrograde).

As SVR is a relative rare procedure in cardiac surgery, we highly recommend the use of a checklist (webappendix 1) for better planning of pre- and postoperative diagnostic examinations.

#### **CONCLUSION**

The techniques used for SVR now represent a well-accepted concept as part of a series of organ-conserving cardiac surgery strategies. SVR can be performed with acceptable perioperative mortality and morbidity. Short- and midterm results are promising. Since the results of the STICH trial are not reliable for drawing practical conclusions, a new trial should be initiated before making definitive recommendations.

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