Review of surgical ventricular restoration: A procedure to treat cardiac failure

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Abstract Severe heart failure refractory to medical treatment necessitates the use of other modalities of treatment. Surgical ventricular restoration (SVR) surgery can provide relief until donor hearts are available or when transplantation is contraindicated. In this review we look at the physiological basis for improvement in cardiac function and cardiac and haemodynamic changes that occur after SVR. We also compile the reported factors affecting surgical outcome, criteria for patient selection and predictors of postoperative mortality. NYHA class, presence of diastolic failure, severe impairment of contractile function and presence of mitral regurgitation were the most important determinants. Various different procedures for achieving SVR are briefly discussed. Importance of mitral valve repair/replacement, coronary artery bypass grafting and intraoperative echocardiography in improving postoperative result is highlighted. Role of perioperative medication (anti-arrhythmics, ACE inhibitors and β -blockers) is briefly studied at the end of the review.

Keywords Ventriculectomy · Cardiac failure · Surgical ventricular restoration

Background

Partial left ventriculectomy (PLV) was first advocated and performed by a Brazilian surgeon, Randa J. V. Batista, M.D. [1]. PLV involves excision of a portion of the left ventricular (LV) wall, sparing the papillary muscles, also referred to as the Batista procedure. Originally, only patients with Chagas disease, who were not candidates for cardiac transplantation because of the infectious nature of the disease, were enrolled for PLV. Multiple surgical techniques to reduce LV volume have been proposed. The term surgical ventricular restoration (SVR) would be more appropriate as the thrust of the procedures is on re-establishing more normal ventricular geometry, i.e. an ellipsoidal rather than a spherical shape [2].

End stage LV failure implies severe heart failure with low ejection fraction that is refractory to medical treatment with increased pulmonary artery pressure and mitral regurgitation (MR) [3]. SVR may prove useful where cardiac transplant is not available, is contraindicated or as a bridge to transplant when donor hearts are not readily available [1].

Rationale

Batista's explanation for post-SVR functional improvement was based on Laplace's law [1], which states

$$\text{LV Wall Tension} = \frac{\text{LV intraventricular pressure} \times \text{radius of LV cavity}}{2 \times \text{LV wall thickness}}$$

Wall tension and LV wall stress are decreased by reduction of the ventricular radius, when other variables in the above equation remain unchanged.

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Decreased LV volume also leads to improved left ventricular ejection fraction (LVEF). However, LVEF is a poor index of functional ventricular improvement after surgery, because LVEF is primarily increased due to reduction in left ventricular end diastolic volume (LVEDV). Absolute indices like cardiac index, left ventricular stroke volume (LVSV) or exercise capacity may be more suitable variables for assessment of postsurgical functional improvement [1].

Left ventricular energy efficiency and mechanical function is improved by making the ventricular shape more ellipsoidal. Myofibril shortening of 15% generates a global EF of 30% in a spherical ventricle but 60% in an ellipsoidal ventricle [2]. SVR improves LV energy efficiency by improving the ratio of stroke work to total pressure-volume work [4]. This benefit occurred even when stroke volume was reduced or unchanged after SVR.

Changes in haemodynamic and cardiac variables after SVR

Three months after PLV, Schafers et al. demonstrated significant reduction in left ventricular end diastolic dimension (LVEDD) and left ventricular end systolic dimension (LVESD), which remained unchanged for up to 1 year [5]. Similar changes were noted in LVEDV, LVESV, LVEF and LV myocardial volume as measured by gated perfusion single photon emission computerized tomography (SPECT). SPECT further demonstrated that the volume of the surgical scar did not increase from 3 to 12 months postoperatively. Kawaguchi et al. found that on post-PLV follow up, LVESV and LVESD were decreased more than the LVEDV and LVEDD [6]. This could explain the observed improvement in LVSV, contractile excursion, LVEF and LV fractional shortening.

Setser et al. employed MRI at 3 months post-PLV and found LV mass, LVEDV, HR and LVEDV/mass significantly reduced [7]. At 12 months LV mass was still reduced but HR, LVEDV, LVEDV/mass had returned to baseline values. Global and regional wall stress was measured in nine quadrants of the LV (base, mid and apical regions of the anterior, inferior and septal walls). They found significantly lower stress in all parts of the LV, except at the base. Percentage of LV circumferential shortening was significantly increased in all regions at 3 months but returned to baseline at the base after 12 months.

In a group of 95 patients, Suma et al. found that LVEF and cardiac index increased significantly over a 6 month postoperative period and LVESV index, LVEDV index, diastolic dimension, pulmonary artery systolic pressure and brain natriuretic peptide levels decreased significantly [8]. The Osaka Cardiovascular Surgery Research (OSCAR) group observed significant decreases in LVEDD and LVESD with increased LVEF postoperatively [9].

These LV functional improvements persisted for 3 years. Bove et al. reported decreased LVEDV and LVESV indices along with improved LVEF after SVR [10]. LVEDV and LVESV increased slightly during the first postoperative year and then remained stable for the next year. Fractional shortening and regional wall motion improved significantly postoperatively and showed further improvement over 2 years.

SVR has also been shown to improve right ventricular (RV) function. Tulner et al. demonstrated decreases in RV annular diameter, RV short and long axes, severity of tricuspid regurgitation, and pulmonary artery pressure after SVR [11].

Patient selection and prediction of morbidity and mortality

Kawaguchi et al. noted that contractile function was improved after SVR, but the effect of SVR on diastolic performance was highly variable, and low preoperative diastolic compliance hampered functional improvement and long-term survival [4]. Regression analysis by the RESTORE (Reconstructive Endoventricular Surgery returning Torsion Original Radius Elliptical shape to the left ventricle) group identified preoperative LVEF <30%, LVESV index >80 ml/m², advanced NYHA functional class and age >75 years as risk factors for postoperative mortality [2]. Suma et al. noted that higher NYHA class and higher brain natriuretic peptide (BNP) levels preoperatively were also significant in predicting a poorer prognosis [8].

Analysis of data in the International Registry of Partial Left Ventriculectomy led Gradinac et al. to conclude that preoperative duration of heart failure symptoms (>48 months compared to <48 months) was a significant predictor of survival [12]. Further analysis showed that poorer outcomes were associated with worse preoperative patient condition, as indicated by NYHA Class IV, LV fractional shortening <5% and severe MR (grade >2) [13, 14]. Recently, Bove et al. identified EuroSCORE (a cardiac surgical risk prediction model), preoperative LVEDV index, LVESV index, NYHA functional class and associated non-cardiac morbidity as significant risk factors by univariate analysis, but only EuroSCORE remained a significant risk when multivariate analysis, which is a more appropriate statistical test for any outcome that can be influenced by multiple factors, was used [10].

Higher preoperative wall stress at mid level in the lateral wall predicted a significantly higher likelihood of an adverse cardiac event within 6 weeks after surgery [7]. A high positive value for percentage shortening in the inferior wall at the base of the heart, and a negative value for percentage shortening in the basal septum, i.e. systolic stretching, predicted a longer postoperative period free of adverse events.

McCarthy et al. had suggested that patients showing improvement in contractility on administration of sympathomimetics might be suited for SVR [1]. One, 3 and 5 years survival rates were significantly higher in patients who were not inotrope dependent compared to those who were inotrope dependent before surgery [8]. Suma et al. recommended that patients should not undergo SVR if they had an entirely akinetic LV that showed no increase in wall thickness during systole as seen on echo/MRI or had pulmonary artery hypertension in the absence of significant MR.

Vural et al. identified NYHA class IV, congestive hepatomegaly, LVEDP >25 mmHg, left atrial diameter >55 mm, and pulmonary artery systolic pressure >40 mmHg as risk factors for higher mortality after SVR [15].

In summary, the following indices indicate an adverse prognosis post-SVR.

- Higher NYHA class, poorer EUROScore.
- Diastolic failure (congestive hepatomegaly, LVEDP >25 mmHg, LA diameter >55 mm, pulmonary artery systolic pressure >40 mmHg).
- Severely impaired contractile function as identified by LV fractional shortening <5%, wall stress in mid lateral area more than 250×10^3 dynes/cm², positive value of percentage of shortening at base of septum, inotrope dependence, LVESV index >80 ml/m².
- MR greater than grade 2.

Thus, criteria have been identified to distinguish patients who could potentially benefit from SVR without need for subsequent cardiac transplantation.

International Registry Reports identified multiple factors that did not influence the outcome of SVR [13, 14]. Gender, body surface area and age were not significant in determining survival. Underlying cardiac pathology categorised as idiopathic dilated cardiomyopathy, ischaemic cardiomyopathy, valvular disease or unknown aetiology did not affect postoperative survival. Preoperative LV dimensions, echo derived LV mass and presence or absence of cardiac transplant indications did not affect morbidity or mortality. Thus age, sex, aetiology and cardiac transplant indications would not be useful to identify the subset of patients who would benefit from this procedure.

Procedure selection

All SVR procedures reduce radius to wall ratio, decreasing wall stress. Smaller ventricles requiring less myocardial resection had better postoperative survival. However, if papillary muscles and mitral apparatus are excised a poorer survival was observed [13, 14]. New procedures to reduce LV wall stress are illustrated in Figure 1 and described below [4]:

- clasp - where a clasp is placed on opposite sides of LV to compress the LV,

Fig. 1 Various procedures for surgical ventricular restoration. (Figure reproduced with permission from Wiley-Blackwell publications. Illustration from article titled “Ventricular volume reduction procedures.” by Kawaguchi AT, Bergsland J, Linde LM. in *J Card Surg.* 2003 Jul-Aug;18 Suppl 2:S29-32 published by Wiley-Blackwell, John Wiley & Sons, Inc., UK.)

- plicating - where a small portion of the LV wall is excluded from the cavity by suturing the margins of the area to be excluded with one another,
- piercing - where sutures are run from one surface of the LV to the opposite surface in order to reduce the LV diameter,
- banding - where an elastic compression bandage is wrapped around the LV,
- overlap - where instead of excising the LV tissue, a flap is created and resutured at a position distal to the original line of incision.

These less invasive procedures have a similar limitation as PLV, viz. decreased diastolic compliance. The advantages of these new modifications are reversibility and adjustability [4].

Another surgical strategy proposed by Dor et al. (Dor procedure) to treat ischaemic cardiomyopathy involves selective excision of fibrotic myocardium followed by circular endoventricular patch plasty with purse string sutures [3, 16]. This differs from the classical Batista procedure, which considers the pathology to be evenly distributed about the dilated chamber. It has been shown that dilated cardiomyopathy is, in fact, a non-homogeneous process with fibrotic patches varying widely in different areas of the ventricle. This would explain adverse results seen in previous studies if normal LV wall was excised while retaining a diseased LV wall and septum. Dor also proposed that a balloon should be inflated inside the LV to the theoretical target LVEDV index (50–60 ml/m²) before suturing the heart wall to prevent excess reduction in the chamber size [17]. This is called “diastolic balloon sizing”. Ferrazzi et al. have

proposed a modification of Dor procedure called “horseshoe repair” [18]. In this procedure, two parallel purse string sutures placed in a horseshoe pattern reduce ventricular volume without patch plasty, reducing the amount of foreign material introduced into the heart.

Suma et al. introduced the concept of intraoperative echocardiographic assessment of regional LV wall function, while LV volume was decreased with partial cardiopulmonary bypass [3]. Changes in the LV wall thickness and motion in response to decreased volume load were utilised to delineate the most akinetic region for excision. Whenever the septum or anterior LV wall was found to be the most akinetic, a procedure called septal anterior ventricular excision (SAVE) was undertaken. In SAVE, instead of the lateral LV wall being excised, the anterior wall is sutured to the posterior part of the septum, thus overlapping a portion of the LV anterior wall and the anterior septum.

Hospital mortality decreased in elective cases from 22% to 5% by using SAVE where it was appropriate [3]. LVEF, LVEDD, LVEDV and LVESV all were significantly improved at the end of 6 months postoperatively. At 3 years postoperatively, cardiac index and LVEF were increased, LVEDP and brain natriuretic peptide were decreased and synchronicity of ventricular conduction and NYHA classification were improved. Once the appropriate procedure was chosen, univariate analysis showed that type of procedure (SAVE vs PLV) was not a significant predictor of postoperative mortality [8].

The “overlap” technique for SVR, in which radius is reduced without resection of myocardium, was found to be successful in non-ischaemic cardiomyopathy [4]. Ueno compared the Dor, SAVE and overlap procedures during postoperative follow up [19]. They found that LVEDV and LVESV increased postoperatively in patients who underwent Dor procedure; diastolic deceleration time, an index of myocardial compliance, did not increase after SAVE and LV sphericity index increased with the Dor and SAVE procedures. The overlap procedure did not suffer from any of these drawbacks and was rated the preferred procedure.

According to the Torrent-Guasp, the ventricular myocardium exists as basal and apical helices [20]. The apical helix spans the LV lateral wall, papillary muscles and ventricular septum. Nishina et al. argued that conventional PLV may result in splitting this helix into two separate bands, with a loss of functional efficiency [21]. To avoid this, Nishina developed an apex-sparing SVR procedure that maintains the continuity of the apical helical band. They reported a significant improvement in LVEDD, LVEF and NYHA class that persisted 2 years after surgery. They reported an 84% 5-year survival rate with this procedure, but this study had only 13 patients and did not compare apex sparing surgery to conventional PLV.

These modifications may have better outcomes than the original PLV and should be further evaluated in clinical

trials. Although it has not been studied, another potentially helpful modification would be to make the LV incision in a watershed zone between coronary arteries. This should minimise the zone of necrosis adjacent to the incision.

In any SVR procedure, care must be taken to maintain normal mitral valve function. Chordae tendinae laxity due to change in orientation of the papillary muscles should be avoided, since this could lead to leaflet prolapse and regurgitation. Also LV incisions should be made, such that tissue damage near the incision does not involve the papillary muscles and result in postoperative MR [1]. Apex sparing SVR procedure reverses the MR seen in ischaemic cardiomyopathy that occurs due to increased separation of papillary muscle tips [21].

Bove's and Izzat's groups found out that postoperative MR led to poor diastolic function and late onset postoperative cardiac failure [10, 22]. They recommended that mitral valve repair should be undertaken at the time of the SVR surgery if necessary. Correction of preoperative MR alone without SVR failed to improve survival [8, 13, 14]. Patients who underwent mitral valve annuloplasty had greater improvement in parameters of LV function (LVEF, LVEDD, LVESD) than patients who did not undergo annuloplasty with SVR [23].

McCarthy et al. performed mitral valve repair in all patients undergoing SVR, and they used LV assist devices or cardiac transplantation to rescue patients that showed signs of heart failure after PLV [1]. A 1 year survival rate of 87%, which is similar to that of cardiac transplantation, was obtained in patients that underwent mitral valve repair with SVR. This surgical trial was abandoned after <60 operations, because this group thought their survival rate of 53% at 3 years was too low. However, this low survival rate could have been due to the early learning curve experienced by most clinics [1]. As the International Registry reported, significantly poorer outcomes occurred in the initial 5 cases at each hospital as compared to the 30th or more cases at the same hospital. This later improvement demonstrated the existence of a learning curve for the hospital surgical staff [13, 14].

Suma et al. found mitral valve repair during SVR to be a better option than mitral valve replacement, since survival was significantly higher at 1, 3 and 5 years [8]. This conclusion is confounded by the fact that valvular replacements were performed during the early part of the study (learning curve effect?) and in patients with higher NYHA class. Suma et al. strongly recommend performing mitral valve reconstruction at the time of SVR based on their excellent results in terms of survival and functional parameters [24]. Bove et al. found that neither type nor size of mitral annuloplasty ring device were significant factors for determining recurrence or severity of MR [10].

In a group of patients studied by Suma et al. all patients had mitral valve repair or replacement and intra-aortic balloon counterpulsation or LV assist devices

were used when deemed necessary [8]. Biventricular pacing was performed in patients with NYHA class III or IV with QRS duration >130 ms and LVEF <35%. After recovery, β -blockers, ACE inhibitors, and other medications for heart failure were reinstituted. Amiodarone was prescribed for all patients perioperatively, and, when ventricular fibrillation or tachycardia occurred postoperatively, a class III antiarrhythmic was used. This management protocol resulted in improved perioperative survival. Carvedilol administration at high doses led to significantly improved diastolic function and lesser myocardial fibrosis in rat models of ischaemic heart failure (coronary artery ligation induced aneurysm formation) post-SVR [25]. Hospital mortality was 6.6% ($n = 76$) in elective SVR and 31.6% ($n = 19$) in emergency SVR [8]. Extensive LV resection was another significant risk factor for higher hospital mortality.

Role of coronary artery bypass grafting (CABG)

Bax et al. found that revascularisation alone failed to improve LVEF in patients who had higher LVESV (mean of 140 ml compared to mean of 109 ml) preoperatively and furthermore, improvement in LVEF was not correlated with myocardial viability [26]. Higher occurrence of adverse events was observed in patients with lower myocardial viability and higher preoperative LVESV.

Maxey et al. found significant difference in postoperative improvement in LVEF in patients undergoing SVR with CABG as compared to those undergoing CABG alone [27]. Postoperative heart failure and survival was significantly improved in the group that underwent SVR in addition to CABG.

Prucz et al. compared two groups of ischaemic cardiomyopathy patients: one that underwent CABG alone and another that had SVR with CABG [28]. Postoperative MR grade decreased and LVEF increased in both groups, but the change was significantly greater in the group that had SVR with CABG. Survival after 3 years was 85% for those who underwent SVR with CABG as compared to 72% for CABG alone.

In Boves study, patients undergoing CABG alone and CABG with SVR had similar maximal oxygen consumption values and comparable exercise performance and NYHA class after their surgeries, even though preoperatively patients in the SVR group had poorer NYHA class than CABG group [10]. Similarly although the SVR group had poorer systolic and diastolic indices preoperatively when compared to CABG group, all the systolic functional parameters (fractional shortening, regional wall motion, LVEF), as well as, LV relaxation capacity were similar for both groups 2 years after surgery. It is likely that improvement in diastolic mechanics may have a greater role in the benefit due to SVR than improvement in systolic contractility.

A large multicentric trial “Surgical Treatment for Ischaemic Heart Disease” (STICH) recently compared the efficacy of SVR with CABG to CABG alone [29]. Reduction in LVESV index was significantly greater in patients who underwent SVR, but the differences in case of NYHA class, 6 minute walk test, operative mortality and postoperative survival were not significantly different. Although the results indicate that SVR is not essential in case of ischaemic cardiomyopathy we must consider some confounding factors. The authors acknowledged the fact that it is likely that experienced surgeons enrolled patients in whom SVR was unnecessary in this trial but also operated on patients who would benefit from SVR without enrolling them in the trial. Another factor is a decrease in diastolic compliance postsurgery. Considering that the procedure most commonly used was Dor’s endoventricular patch plasty, which has been shown to be inferior to the overlap technique, beneficial effect of SVR might have been observed if more modern methods had been used [19, 30]. Also the surgeons certified to perform SVR surgeries had to have previously performed more than five surgeries with decrease in postoperative LV volume. It is likely that some of the surgeons were in the earlier part of the learning curve and beneficial effects might have been observed if the patients were subdivided into groups according to surgical experience of more than 30 cases [13, 14, 30].

Mitral valve repair was not performed on all patients and could have led to poorer results after SVR. Similar patient groups were used for undergoing CABG and SVR with CABG, but it is possible that if the groups were subdivided into higher LVESV and lower LVESV then the difference might have become significant. More arterial conduits were used for patients undergoing CABG alone, and this might have denied the full benefit of revascularisation to patients undergoing SVR. Thus, although the STICH trial failed to show benefit in patients who underwent SVR in addition to CABG for ischaemic heart failure, it would be hasty to dismiss the role of SVR in ischaemic cardiomyopathy.

Relapse and failure

The most common cause of death was postoperative recurrence of cardiac failure. Most deaths occurred perioperatively and were significantly associated with higher NYHA class, larger ventricles with reduced LV contraction and with more severe preoperative MR [10, 13, 14]. Spironolactone prevented adverse postoperative remodeling after SVR in rats [10].

Postoperative recurrence of heart failure could be attributed to: a) recurrence of volume overload due to MR, b) excision of too much LV tissue, leaving a small ventricle with restricted filling, c) excision of too little LV tissue, leaving a large ventricle with elevated wall stress and d) retention of LV tissue with poor contractile function [3].

With more complete knowledge of factors that could be responsible for heart failure after surgery and with optimal patient selection criteria, the incidence of postoperative heart failure should decrease.

The second most common cause of death after SVR was cardiac arrhythmias. Most of these deaths occurred during the first postoperative year and were usually accompanied by the same factors associated with cardiac failure [13, 14]. The increased incidence of postoperative arrhythmias could be due to residual areas of fibrosis giving rise to re-entrant pathways. Early treatment with anti-arrhythmics or implantation of automatic defibrillators might decrease mortality due arrhythmias [1].

Conclusion

Taking into account the painstakingly obtained data in the above-mentioned studies, the following approach to SVR surgery seems reasonable. After choosing the appropriate patient group for surgery, the most akinetic area for excision should be identified using echocardiography and MRI. Surgery should be planned early, before the patient becomes inotrope dependent or decompensates, so an emergency procedure is not necessary and myocytes do not become fibrotic. β -blockers (Carvedilol), ACE inhibitors, and other medication should be continued till surgery and reinstituted after the procedure. Spironolactone should be the diuretic of choice postoperatively. Amiodarone should be used perioperatively and anti-arrhythmic therapy should be continued postoperatively if indicated.

During the operative procedure, all patients should have mitral valve repair or replacement. At this time, there is insufficient data to know whether valve repair or replacement would offer a better prognosis. For ischaemic cardiomyopathy CABG is a necessary component. Whether CABG alone is sufficient is still a matter of debate.

LV wall kinetics should be monitored intraoperatively with echocardiography. Partial cardiopulmonary bypass during surgery is helpful in assessing the potential change of wall kinetics that would be produced by SVR. With this information, the surgeon can identify the optimal LV region for excision. Haemodynamic support during the early postoperative period with intra-aortic balloon counterpulsation or LV assist devices should be provided when necessary. Biventricular pacing might prove beneficial in patients of NYHA class III or IV with QRS duration >130 ms and LVEF <35%. In the event of postoperative ventricular fibrillation or tachycardia, a class III anti-arrhythmic should be administered.

The efficacy of newer SVR procedures has to be established by means of clinical trials. Other new modalities of treatment, like the use of stem cells and novel drugs to treat heart failure, should be constantly assessed as potential alternative treatment options. Research to compare the

benefits of one modality over another must be conducted to define which therapeutic option would be of maximum benefit to each specific case.

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