Left Ventricular Reconstruction in Ischemic Cardiomyopathy

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Salim Aziz and Jaishankar Raman

Introduction

Repair of left ventricular aneurysms was reported as early as the late 1950s by Likoff and Bailey [1]. Cooley reported on the success of linear closure of these aneurysms [2].

Josephson and Harken described blind endocardial resection for VT with poor functional results [3]. Gorlin was among the first to show that the ventricular wall after myocardial infarct could be akinetic or dyskinetic [4]. Dor and co-workers described a technique [5] which is illustrated in Fig. 7.1 to close the left ventricle reorganizing a neo left ventricular apex, in an attempt to recreate the normal conical shape in 1985. Jatene also described a plication technique in the same year using an external circular set of sutures [6]. However, it was Batista's bold experiments in dilated cardiomyopathy with heart failure and very public presentations of his "successes" that made people in the heart failure arena consider left ventricular reconstruction seriously [7].

Despite all this intuitive work on preserving left ventricular geometry, there was little interest in adopting these techniques into widespread clinical practice.

The 1980s were marked by two important steps in the knowledge of evolution and prevalence of ischemic failing ventricles:

1. The mechanism of progressive ventricular dilatation following transmural myocardial infarction, also known as remodeling, was well analysed and characterized [8]. The role of neurohormonal activation was established and this helped to provide therapeutic targets [9]. Ischemic remodelling lead to progressive ventricular dilatation which then sets in motion the cascade of heart failure [10].
2. Aggressive treatment of acute myocardial infarction, by revascularization of the occluded artery, has resulted in improved the survival rates. However, even after successful recanalization, the left ventricular wall of survivors, remains affected by scar in 80–100 % of cases; the infarct size varying from 6 to 60 % of the ventricular surface area as indicated by Christian [11].

Various imaging modalities such as cardiac magnetic resonance (CMR) developed as accurate, reliable and reproducible tools [12] to assess

S. Aziz, MD, FACS
Department of Surgery,
George Washington University Hospital,
Washington, DC, USA
e-mail: HLTXAziz@gmail.com

J. Raman, MBBS, MMed, FRACS, PhD (✉)
Cardiac Surgery, Mechanical Circulatory Support and Heart Transplantation, Cardiovascular and Thoracic Surgery, Rush University Medical Center, Chicago, IL, USA
e-mail: jairaman2462@gmail.com

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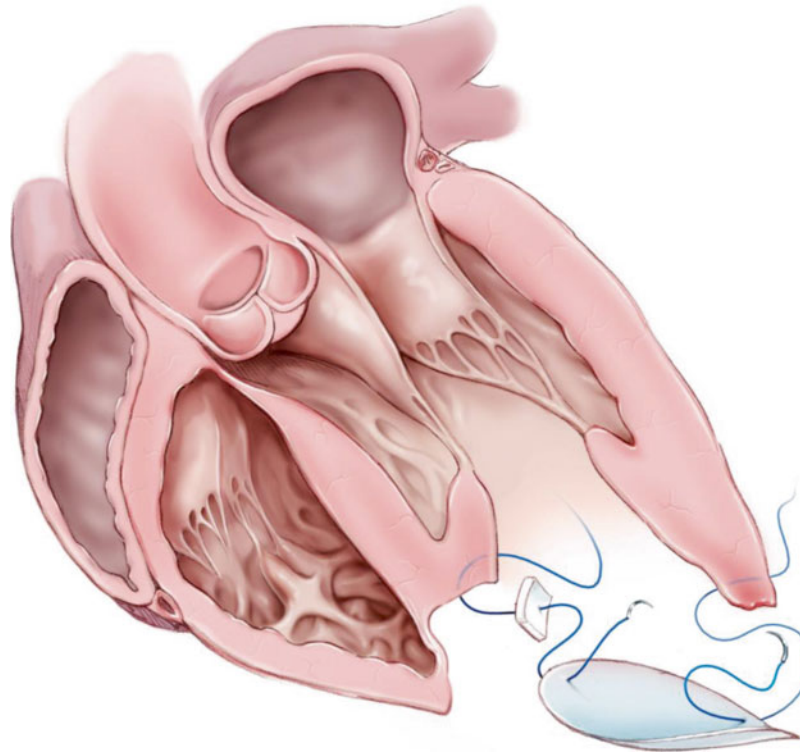

Fig. 7.1 Schematic of the principles of Left ventricular reconstruction (As proposed by Dor in 1985)

both anatomy and performance of the ventricles after infarction. Use of contrast MRI further enhanced the utility of this imaging modality [13].

More recently, three dimensional echocardiography and volume rendered images with multi-detector CT scanning have shown particular promise in this arena.

Fate of the Left Ventricle after Infarction

Gorlin in 1967 observed that:

> when 20 % to 25 % of LV area is asynergic, contraction of the myofibers to maintain stroke volume exceeds pathophysiological limits and cardiac enlargement (by the Frank-Starling mechanism) must ensue to maintain cardiac output.
>
> There are two types of asynergy seen as consequence of ventricular aneurysms: regional akinesis (total lack of wall motion), and regional dyskinesis (paradoxical systolic expansive wall motion.

In spite of revascularization of the culprit artery altering the immediate prognosis, the left ventricular wall may remain diseased [14].

(A) Evolution of the Infarcted Area: The infarcted wall undergoes changes that start with necrosis, progress to fibrosis and, eventually to calcification

1. The typical left ventricular aneurysm is characterized by a transmural infarct (Fig. 7.2).
2. The use of thrombolysis and/or revascularization in the acute phase may prevent transmural necrosis (Fig. 7.2b). Ventricular muscle adjacent to the epicardial artery is salvaged by recanalization, but the subendocardial muscle is necrotic, as described by Bogaert in 1997 [14]. The resultant ventricular wall may have viable myocardium which maybe evident at surgery (or during Thallium test) surrounding an akinetic, necrotic zone. The scarred asynergic ventricular wall may result in dyskinesia or akinesia. In both morphologies, it is important to know the extent of ventricular wall is abnormal, since this determines the indications and prognosis of surgical intervention.
3. Location: The **antero-apical and septal** regions are most commonly involved in

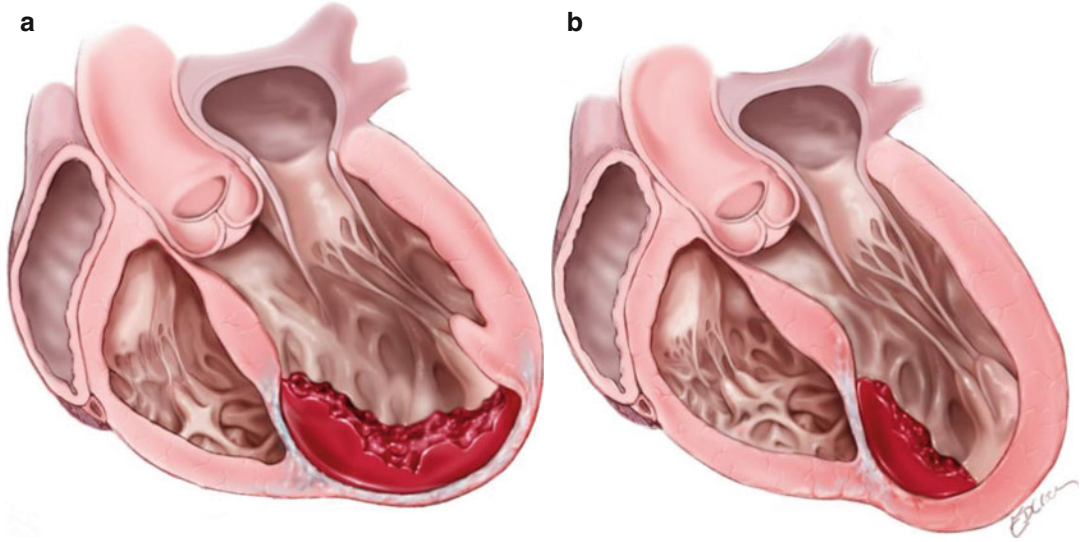

Fig. 7.2 Anteroseptal infarcts with extensive apical and septal involvement (**a**), Anterior infarct with predominantly septal involvement (**b**). Note adjacent mural thrombus formation

anterior infarctions caused by left anterior descending artery occlusion. Involvement of the septum is difficult to assess on ventriculography. The septum is best assessed by biplane angiography, echocardiography or cardiac MRI.

4. The extent of asynery or abnormality governs the prognosis of the left ventricle after infartion. This can be assessed by a variety of imaging techniques. Traditionally angiography using the centerline method in the right oblique projection has been used. However, increasingly, other techniques such as contrast echocardiography, radionuclide ventriculography, multi-slice CT Scan, cardiac magnetic resonance (MRI), provide more information. Analysing the LV wall and the presence of a necrotic scar is very important. The extent of asynergy can be expressed as the ratio between the length of necrotic wall and the total length of the LV circumference. The progression towards severe heart failure is likely when this ratio reaches 50 %.

(B) Fate of the Non-Infarcted Myocardium:

While normal at first, the undamaged myocardium undergoes hypertrophy to compensate for the lack of contractility of the necrotic wall, and finally dilates by a combination of mechanical forces and neurohormonal signaling. The Frank-Starling mechanism explains the early dilatation that temporarily improves the cardiac output and function. LaPlace's Law described increased wall stress and this is the fundamental reason behind the deleterious effects on myocardial contractility due to increased wall tension. Left ventricular remodeling is the term used to describe the progressive dilatation of the heart, and is based on a set of complex inflammatory and neurohormonal processes. Gaudron suggested that the culmination of this process is progressive dilatation of the non-infarcted area with an ensuing spherical shape and akinesia, which occurs, in 20 % of patients treated for myocardial infarction [15].

Harvey White was an early proponent of using left ventricular volume as a sensitive marker of post-infarction ventricular dysfunction [16]. Yamaguchi used left ventricular end-systolic volume as an important predictor for prognosis after surgical repair [17]. Doubling of indices can be considered markers of severe dilatation (normal values are 25–30 ml/m^2

for end-systolic volume index (ESVI) and 50/60 ml/m^2 for end-diastolic volume index (EDVI).

Who Should Be Considered for Surgical Ventricular Restoration (SVR) or Left Ventricular Reconstruction (LVR)?

Currently SVR is largely established for patients with ischemic cardiomyopathy after myocardial infarction, although some have advocated a variant of the technique for patients with idiopathic dilated cardiomyopathy.

Presently, the "ideal" patient should have a dilated ventricle and NYHA class III to IV heart failure symptoms following infarction. As for patients with LV aneurysms, totally asymptomatic patients should not be considered. The SVR/LVR is advocated for patients with a prior left anterior descending artery territory infarct (anterior wall, septum region). Patients with both LAD and circumflex artery occlusions may not be suitable candidates. The infarcted segment can either be akinetic or dyskinetic.

The timing of surgery is also important. Conventional wisdom states that 6 weeks following infarction should elapse before SVR is considered. There is, however, one small series of seven patients that underwent surgery soon after a large anterior infarction with encouraging results [18]. Anecdoctal cases have been performed in the early post-infaction period (2–14 days), particularly in the setting of low cardiac output as salvage therapy.

Preparation for Surgery

1. **Pre-operative preparation**: Patients that have been inpatients in cardiology for recurrent episodes of CHF and titration of medical therapy, often need tuning up. A variety of tests may be of benefit:
   - Measurement of pulmonary arterial pressure (PAP) by right heart catheterisation and the response to vasodilator therapy, and/or oxygen administration helps in stratifying patients.
   - Detailed echocardiographic assessment including three-dimensional echocardiography is of great benefit. Mitral regurgitation has to be assessed by pre-operative echocardiography or trans-esophageal echocardiography if necessary.
   - The extent of the scar and its location are important in planning the operation. Establishing viability of the remote non-infarcted segments is crucial because often there are regional wall motion abnormalities in these areas. Gadolinium enhanced magnetic resonance imaging is a good test of viability. Segments that are hypokinetic predictably improve if there is no hyperenhancement. If MRA is contraindicated due to implantation of a defibrillator or pacemaker, multi slice CT scan or 3-D echocardiography may be used. However, these tests may only assess contractility of the remote segments.
   - A coronary angiogram is mandatory to delineate the coronary anatomy.
2. **Preoperative medical treatment is continued** except for cessation of anti-platelet and anticoagulant therapy.
   - The intra-aortic balloon pump (IABP) should be used whenever there is hemodynamic compromise, such as evolving infarction without remission, CHF not improved by medical therapy, patients with a mechanical complication of myocardial infarction, or incipient renal failure. Some have found it useful in all SVR procedures as adjuvant therapy.
3. **Specific operative procedures**: A femoral arterial line is inserted for monitoring purposes and to allow quick access for insertion of a balloon pump if required. The patient is prepared for saphenous vein harvest if required. Cannulation for cardiopulmonary bypass is routine, utilizing an aortic cannula and a dual stage right atrial cannula. Monitoring includes arterial line, central venous pressure and Swan Ganz catheter.

Trans-esophageal echocardiography is used routinely.

- For the management of cardiopulmonary bypass, we minimize crystalloid use. Retrograde autologous priming of the circuit helps remove the crystalloid that is added to prime the circuit. Hemofiltration during bypass in helpful in reducing myocardial edema.

Left ventricular reconstruction (LVR): (Fig. 7.3)

Sequence of surgical steps may depend on the acuity of the patient, the extent of ischemia, presence or absence of left ventricular thrombus, etc.

1. The repair may be conducted utilizing the open-beating or cardioplegic arrest methods of myocardial protection (Fig. 7.4).
2. Grafting all coronary arteries with meticulous myocardial protective strategies is important. Retrograde cardioplegia preserves septal contractility as is crucial in preventing low output states postoperatively. Particular care is taken to revascularize the LAD, or diagonal if possible, thereby increasing septal blood flow.

What is the best resultant shape/volume of the operated left ventricle? What sort of patch should be used? These are interesting questions that are yet to be resolved conclusively. Although some authors have suggested that the LV can be "tailored" without a patch, this can be difficult, especially for the novice surgeon. This technique promoted by McCarthy is described in the next chapter. The size of the patch and the stiffness of the patch also may have a bearing on the outcome long-term, and these matters are addressed in the next chapter.

Management of Associated Mitral Regurgitation

Mitral regurgitation is found often in these patients, either due to the nature of the remodeling or due to involvement of the infero-basal wall of the left ventricle. Our personal preference to correct any degree of mitral regurgitation that is greater than moderate or 2+. Typically, this is done by utilizing a flexible posterior band or a remodeling annuloplasty ring. At times a simple Alferi stitch maybe resorted to as well. The mitral valve can be accessed and repaired through the ventriculotomy or via a separate incision in the left atrium or a trans-septal approach. The limitation of the ventricular approach is that access to the valve may be suitable only in very large scars that are more apico-septal in orientation (Fig. 7.5).

1. **The LVR technique**: Dor reported the technique of endo-ventricular patch patch plasty in 1989 [19]. In this classic technique, the ventricular wall is opened at the center of the scarred area, which often appears as a dimpled area once a left ventricular vent is placed after aortic cross clamping (Fig. 7.3b). Any clots present are removed. It is important to note that some fragmented and friable small clots may not be seen on trans-esophageal or epicardial echocardiography, especially if the thrombus is non-homogenous or small. The endocardial scar is dissected and resected if the scar is calcified (Fig. 7.3e) or if there is evidence of ventricular tachycardia (VT). Ablative treatment at the edge of scar usually at the borderzone, with radio-frequency energy or cyoablation is a short adjunctive procedure shown to limit postoperative dysrrhythmias.

   The reconstruction of the left ventricular cavity is started using a continuous suture 2-0 monofilament purse-string suture (Fig. 7.3d) with bites going into the muscle at the borderzone (the junction between the scar and normal myocardium). Typically, this suture is run as a continuous purse-string suture is tightened over a rubber balloon inflated within the cavity at the theoretical diastolic left ventricular volume (50–60 ml per sq.m. of BSA). This technique was introduced to avoid making the residual cavity too small [20]. This is a good guide to surgeons with limited experience. The endoventricular circular suture is also known as the "Fontan stitch" (Fig. 7.3d)

and helps optimize orientation of the patch while selecting its shape and size.

The septum and apex are more involved than the lateral wall in the antero-septo-apical type of scarring. In these cases, the suture, thus placed deeply in the septum, totally excludes the apex and the posterior wall below the base of the posterior papillary muscle, and only a small portion of lateral wall above the base of the antero-lateral papillary muscle. Therefore, the orientation of this new neck (and of the patch) roughly follows the axis of the septum.

Dor described the use of a Dacron patch that is sutured to the Fontan Stitch. The excluded, redundant bits of scar are then sutured over the patch to aide in hemostasis. The traditional Dacron patch is quite stiff, and various modifications of the technique include use of Gortex, bovine pericardium or no patch at all.

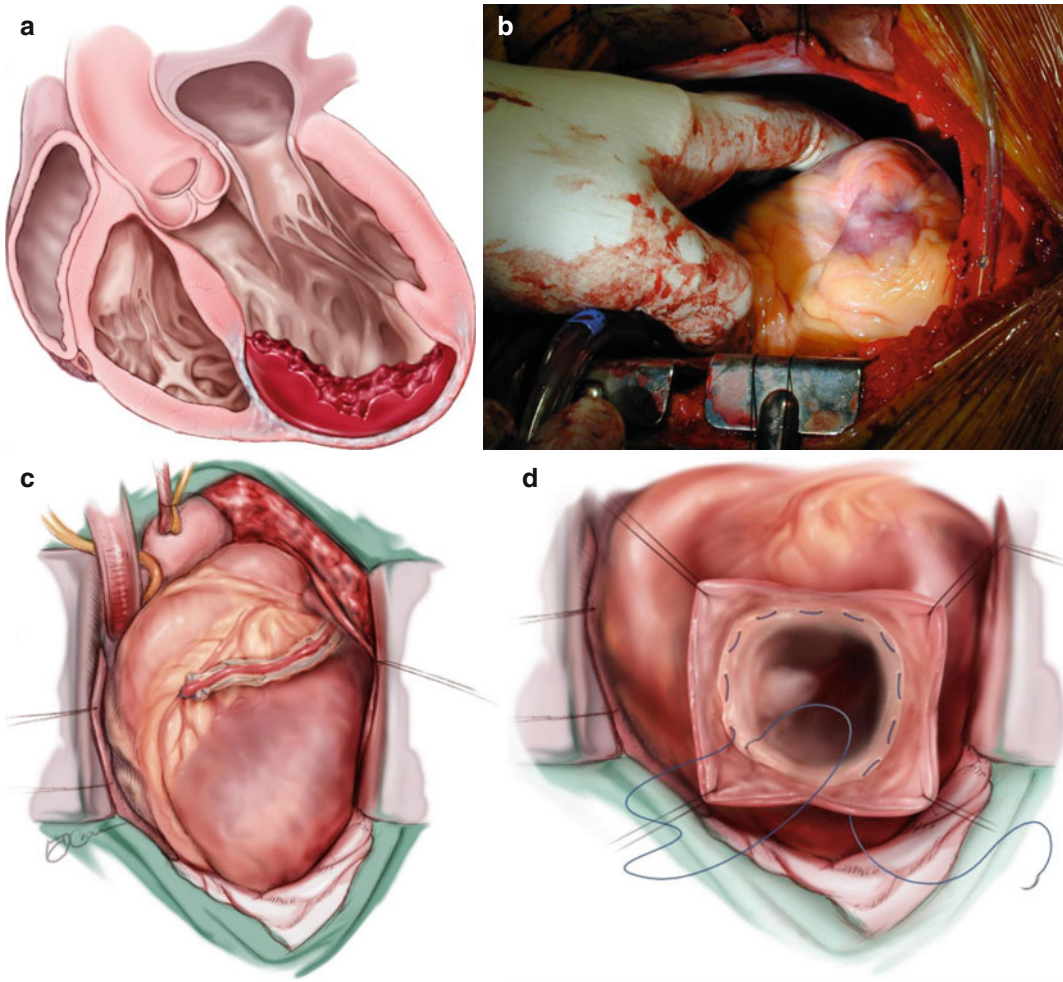

Fig. 7.3 Steps of surgery for Left Ventricular Reconstruction (LVR): Left Ventricular Reconstruction: (**a**) The antero septo apical aneurysm with mural thrombi: the dilatation also affects the non-scarred myocardium on septum (S) and lateral wall (L). (**b**) Intra-operative photograph showing dimpling of Left ventricular scar with use of vent. (**c**) Coronary revascularization accomplished first on arrested heart. (**d**) The continuous purse-string suture at the limit between fibrous and normal myocardium (Fontan "Trick"). (**e**) possible endocardectomy if needed. (**f**) The suture is tied on a rubber balloon inflated to 50 mm per square meter of the body surface area (normal diastolic volume). The shortening of the SL length illustrates the reorganization of the curvature. (**g**) The Dacron patch anchored on the suture. The right ventricle apex projects beyond the new LV apex

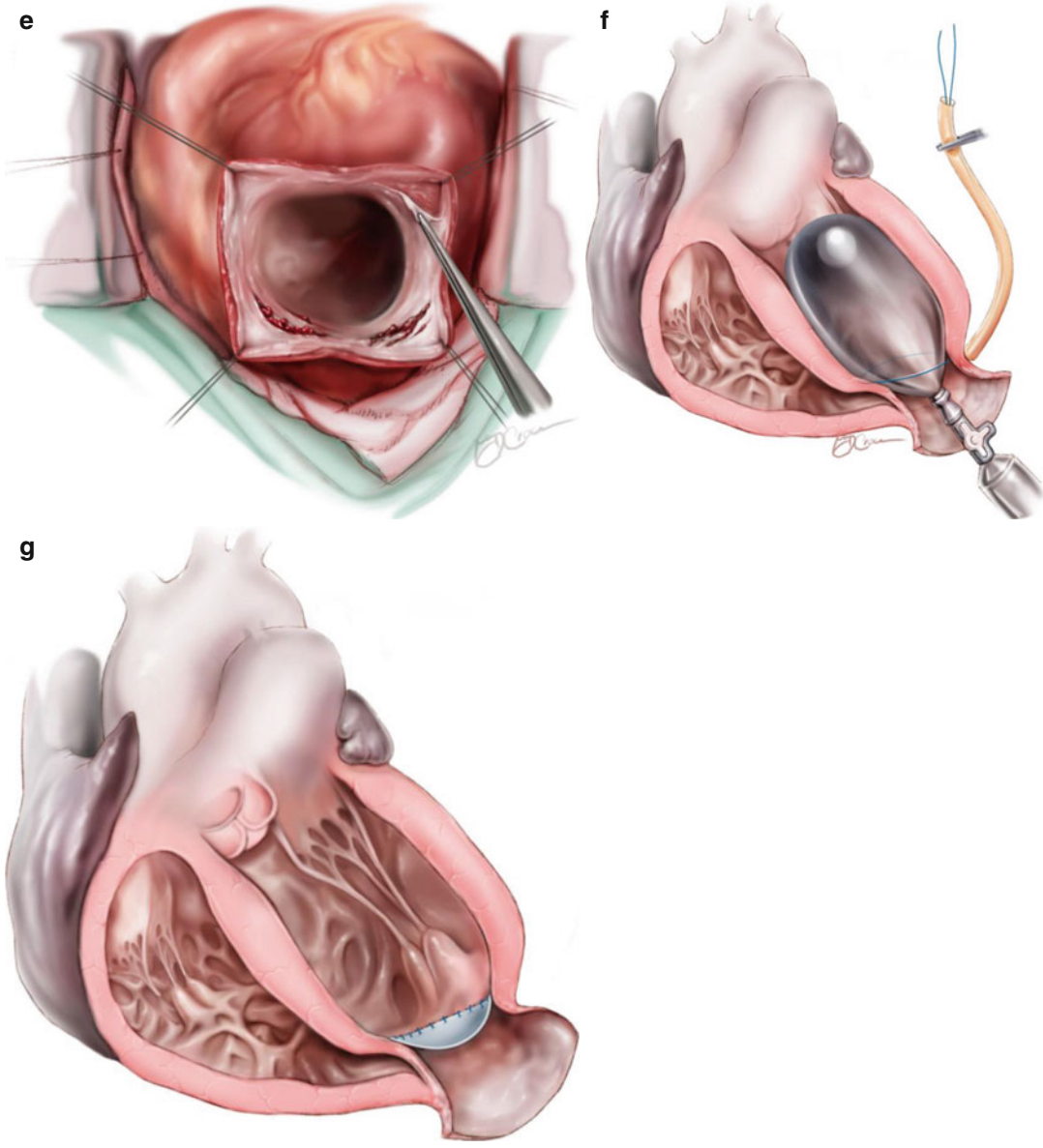

Fig. 7.3 (continued)

2. **Wean off CPB**: Typically, weaning from bypass is slow and gentle to allow complete recovery of both ventricles. Frequent use of antegrade and retrograde blood cardioplegia every 8–10 min helps preserve biventricular function and allows quicker recovery of the heart. Our choice of inotrope typically involves milrinone and dobutamine. Often, norepinephrine and/or vasopressin are required as an adjunct to counter the hypotension that is seen due to the effect of milrinone. Liberal use of the balloon pump has been found to be very useful. Prophylactic atrial and ventricular pacing wires are used to ensure a regular rhythm. If there is a history of atrial fibrillation or ventricular arrhythmia, prophylactic use of amiodarone helps reduce the risk of lethal arrhythmias.

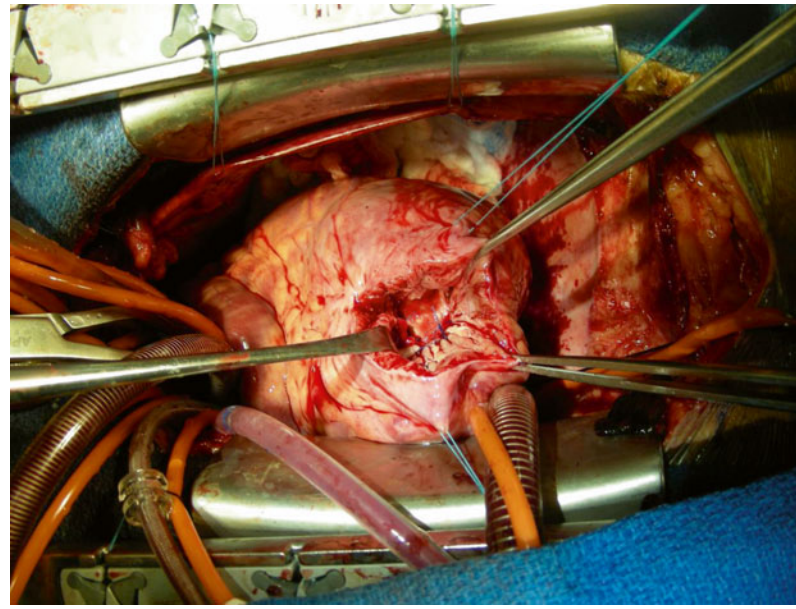

Fig. 7.4 Completed patch implant

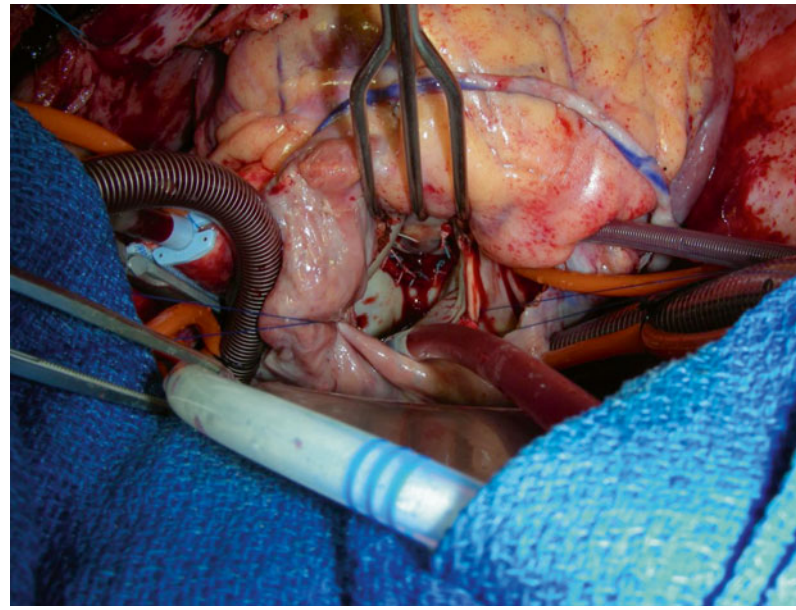

Fig. 7.5 Completed mitral valve repair. Note vein graft to inferior to a branch of the RCA and trans-septal approach to mitral valve

3. Specific modifications: (Fig. 7.6)
   (a) Changes based on alterations of the ventricular wall
      • Autologous tissue has been used for the patch: either a semicircle of the fibrous endocardial scar mobilized with a septal hinge if this scar is strong, or autologous pericardium. Figure 7.7a, b show a septal hinge being used to patch the defect.
      • When the tissues are soft and necrotic, during the repair of an acute mechanical complication of myocardial infarction (exclusion of septal rupture or treatment of free wall fissures), the patch has to be inserted into healthy tissue by deep stitches

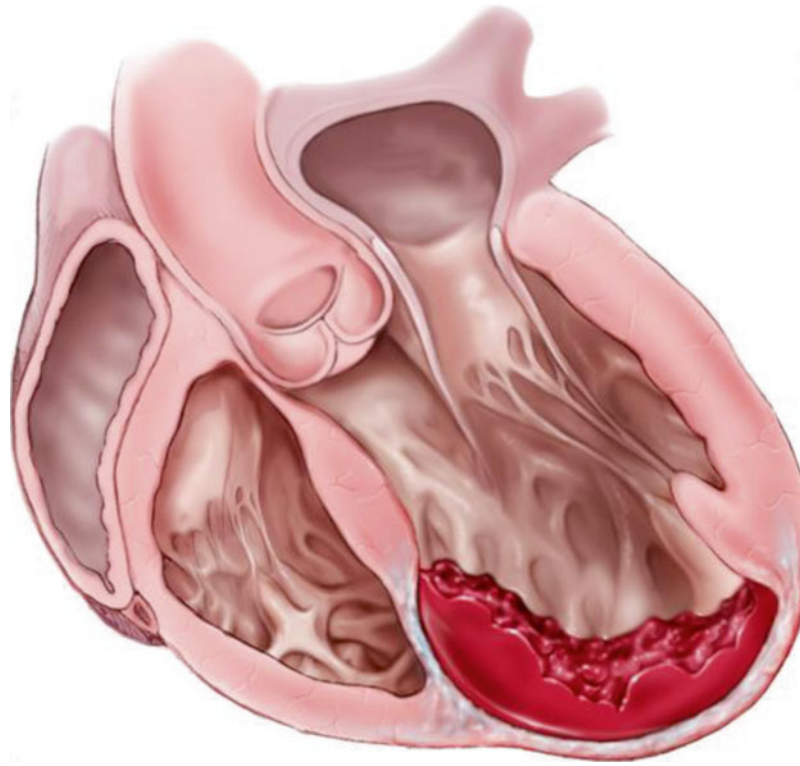

Fig. 7.6 This shows the left ventricle in longitudinal section with mural thrombus along the septum and antero-apical wall

reinforced with Teflon pledgets. The patch is anchored above the septal rupture which is excluded from the LV cavity (Fig. 7.7d).

Figure 7.7c, d show a septal infarct that has been excluded with a pledgetted plicating suture and subsequent implantation of a patch to exclude the septal infarct.

(b) Amount of Scar Exclusion: In cases of a large amount of asynergy (above 50 % of LV cavity) surgery is accomplished with some modifications. These patients typically are in Class III or IV heart failure and on inotropes. Mean pulmonary artery pressure is often above 25 mmHg, ejection fraction (EF) below 30 %, EDVI above 150 ml/m^2, and ESVI above 60 ml/m^2. Ventricular tachycardia may be present in nearly 50 % of cases and this should preferably be addressed by ablative strategies and/or endocardectomy. Mitral insufficiency has to be repaired in the majority of cases. Mitral valve repair in this instance is performed as a remodelling annuloplasty, with Bolling and his followers advocating rigid, shaped rings. David and his group have shown good results with flexible posterior bands. If the presentation is acute or if there is difficulty in repairing the valve, a valve replacement with chordal preservation should be considered. Theoretically, the exclusion of all scarred areas may lead to a very small LV cavity with a high risk of immediate or delayed diastolic dysfunction. The Fontan stitch is placed slightly beyond the edge of healthy muscle at the transitional area. The use of a mandril or balloon inside the LV, inflated at the theoretical diastolic volume of the patient is a useful guide to optimize tension on the suture. The patch can be slightly more redundant (3–4 cm in diameter) than in the usual technique.

If the septum cannot be excluded easily, the redundant septum can be plicated or imbricated separately or at the edge of the patch as described by Jatene.

(c) **Inferior and posterior scars** (Fig. 7.8b): An oblong or triangular patch, with its base aligned along the posterior or postero-lateral mitral

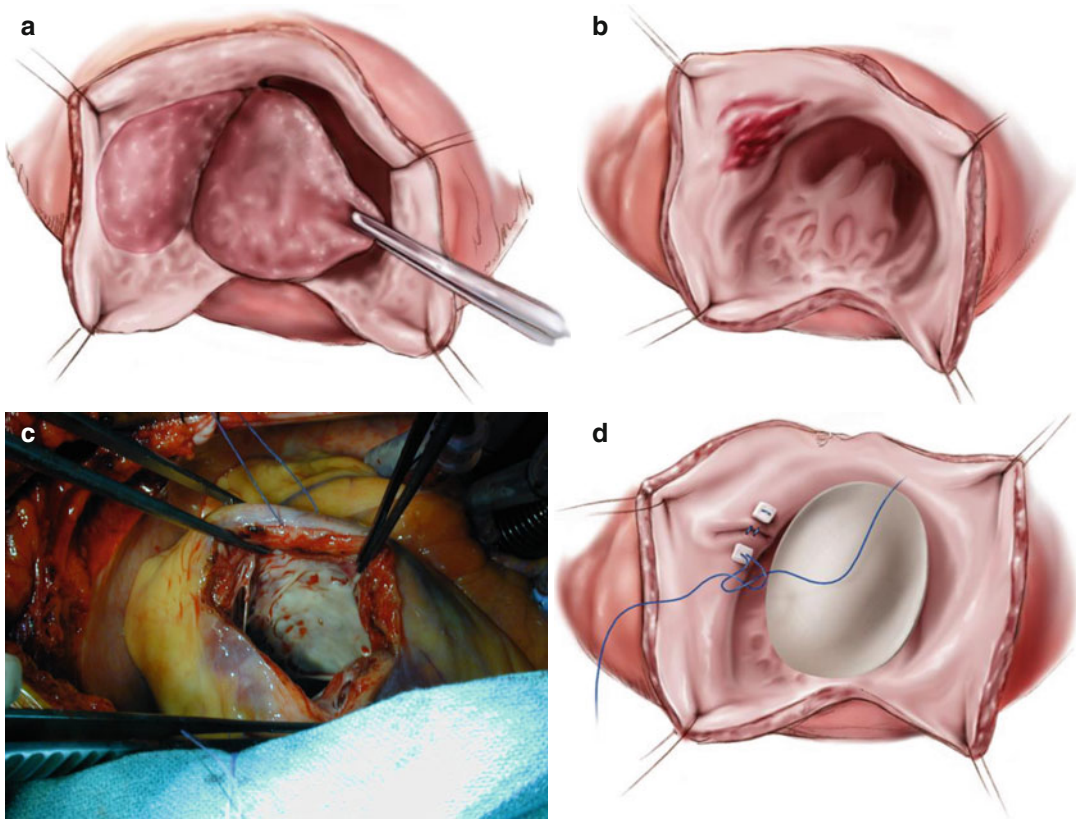

Fig. 7.7 Particular cases of Endoventricular patch reconstruction: (**a**) utilization of the septal scar as an autologous patch. A semicircular portion of the scar mobilized from the septum with a septal hinge is sutured on contractile muscle inside the left ventricle. (**b**) Septal Involvement after opening up the left ventricular scar. (**c**) Operative photograph of septal infarct and scar. (**d**) Septal Involvement. The patch is anchored above the septal repair, which is excluded from the left ventricular cavity

annulus and its apex close to the base of the posterior or antero-lateral papillary muscle is the typical orientation. This allows the reconstruction to follow a geometric pattern restoring shape to normal. If the posterior papillary muscle is totally involved in the resected scar, the mitral valve can be replaced by a prosthesis, implanted through the ventriculotomy.

Figure 7.8 a–c show repair of a postero-inferior scar with replacement of the mitral valve and implant of a triangular patch.

Early Results

Early results with simple linear closure were variable and often suboptimal. Left ventricular reconstruction is a complex procedure performed on patients with significantly impaired ventricular function and is frequently associated with an operative mortality risk of about 7 %. The risk profile is dependant on the extent of scarring, the degree of heart failure, the amount of remaining normal ventricle, the presence of arrhythmias and the amount of mitral régurgitation. Hospital mortality can be stratified into three categories:

- Very severely depressed EF <30 %: mortality of 12–15 %.
- EF of 30–40 %: mortality of 7 %
- EF >40 %: mortality of 1.3 %

LVR Outcomes (Restore Group)

Dor's contributions inspired a multinational study of ventricular restoration. A collaborative

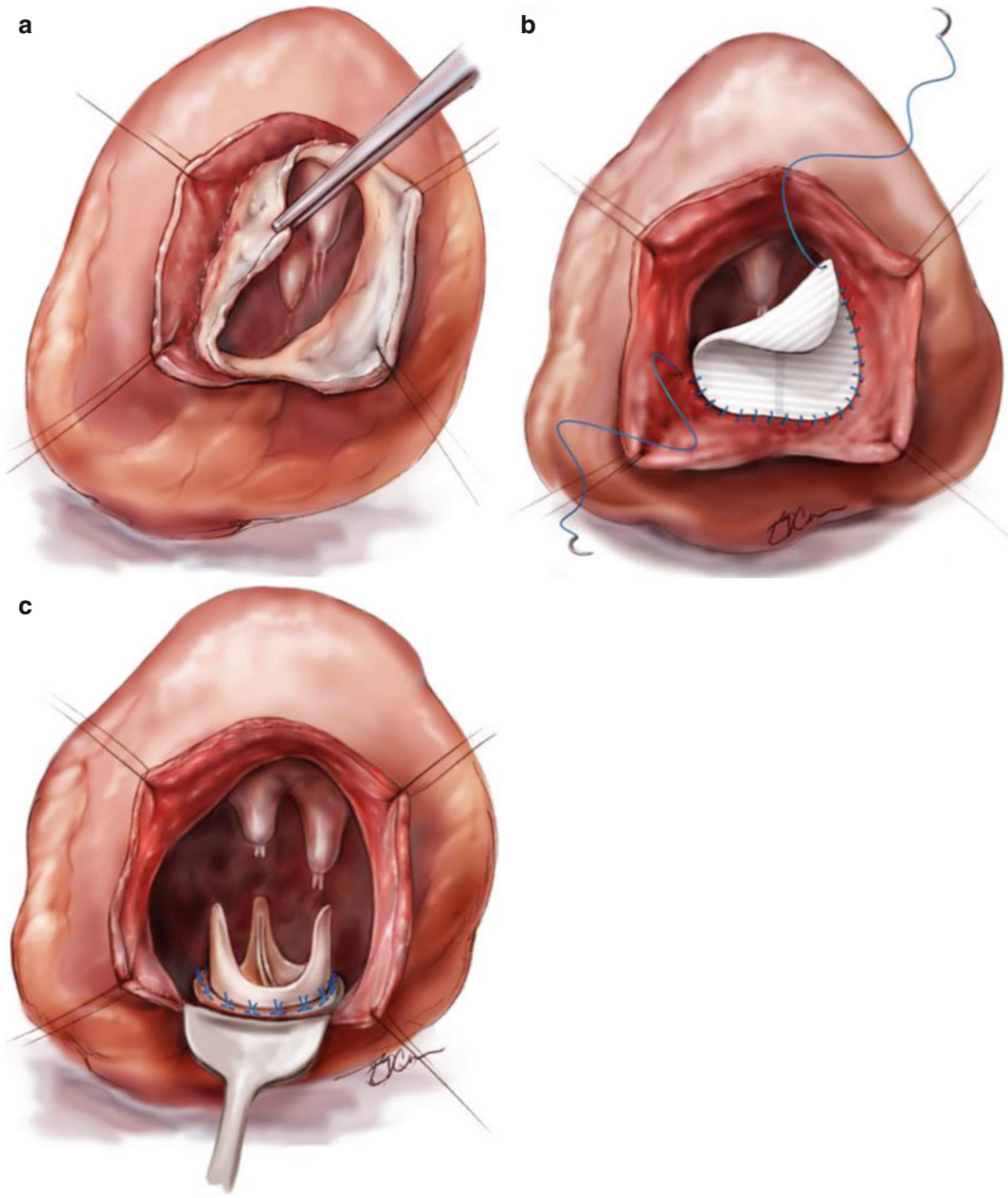

Fig. 7.8 (**a**) LVR for posterior aneurysm: positioned for posterior incision and endocardectomy. (**b**) Triangular patch closure of the posterior left ventricular scar. (**c**) Mitral valve replacement through a posterior scar

group of cardiologists and cardiac surgeons (the RESTORE Group) from four continents (US, Europe, Asia, South America) applied SVR in 1,198 patients between 1998 and 2003 [21]. Patients were included in the registry if SVR was performed with the following criteria: prior anterior myocardial infarction, significant ventricular dilation (LVESVI ≥60 ml/m^2), and a regional asynergic (non-contractile) area of ≥35 %. SVR was most often done as a concomitant procedure: 90 % received coronary bypass grafting and mitral valve repair was required in about 20 %.

In the RESTORE registry, 86 % of patients had NYHA class III/IV symptoms of CHF preoperatively. Such patients have a high mortality with medical therapy, or with surgical revascularization without left ventricular restoration, where late deaths are attributable to CHF. Echocardiography,

ventriculography or magnetic resonance angiography (MRA) was used to confirm the asynergic area and calculate the ejection fraction (EF). LVESVI was determined by ventriculography or MRA.

Hospital mortality after SVR was 5.3 %, and increased as preoperative ventricular volume rose: 2.3 % if LVESVI was <60 ml/m^2, 5.7 % if LVESVI was 60–90 ml/m^2, 8.1 % if LVESVI was 90–120 ml/m^2 and 8.4 % if LVESVI was >120 ml/m^2. These findings are comparable to recently reported mortality rates (5.5–11 %) in ischemic patients with left ventricular dysfunction (EF <35 %) undergoing coronary artery bypass grafting alone. The addition of mitral valve repair influenced early outcome. Hospital mortality was 8.7 % with mitral repair vs. 4.0 % without repair ($p<0.001$). Perioperative mechanical support with intra-aortic balloon pumping was uncommon (<9 %). Global systolic function improved postoperatively as EF increased from 29.6 ± 11.0 % preoperatively to 39.5 ± 12.3 % postoperatively ($p<0.001$). LVESVI decreased from 80.4 ± 51.4 ml/m^2 preoperatively to 56.6 ± 34.3 ml/m^2 postoperatively ($p<0.001$).

The overall 5-year probability of survival after SVR was 68.6 ± 2.8 % and confirms Dor's extensive experience and is unprecedented in the treatment of advanced ischemic cardiomyopathy. The multivariate analysis of SVR demonstrated that major risk factors were age, preoperative EF, LVESVI, and NYHA functional class.

The RESTORE data further emphasized the importance of measuring LVEVI as a surrogate marker of left ventricular function. EF and LVESVI are not directly related, as there is a wide variation in volume for a given EF. Patients with preoperative LVESVI ≤80 ml/m^2 had long-term survival of 79.4 ± 3.3 % as compared to 67.2 ± 3.2 % for those with larger hearts. Preoperative NYHA functional class was also predictive of outcome with decreased 5-year survival in patients with preoperative class IV symptoms (49.7 ± 5.8 % vs. 69.9 ± 4.7 % in class III). These predictors of long-term outcome after SVR are similar to those previously reported. A small number of patients (9 %) in functional Class I underwent SVR as an adjunct to CABG because ventricular dilation (LVESVI >60 ml/m2) has been shown to be a precursor of late development of CHF and early death.

1. **Cardiac morphology and performance**:
   - The most striking findings on imaging are the relative normalcy of ventricular shape and function postoperatively. If a localized scar is effectively excluded with restoration of normal shape, the heart failure symptoms are dramatically ameliorated. **Improvement in Systolic function**: Often, the mean increase in ejection fraction early after reconstruction is between 10 to 20 %. Improvement is similar for dyskinetic as well as akinetic lesions [22].
   - **Improvement in Diastolic function**: The peak filling pressures and filling pressures in the left atrium as surrogate markers of left ventricular diastolic function are relatively normal a few months after operation.
   - **Efficiency**: The elimination of the dead space of the scar helps improve the efficiency of ventricular contraction by:
   – Elimination of the asynergic scar.
   – Restoration of the curvature of the ventricular wall: Analysis of pressure-volume curves has shown reduction in wall stress [23].
   – Mechanical synchrony is restored.

Intermediate and Long Term Results

1. Despite acceptable early morbidity and mortality, the intermediate and long-term results are not uniform. They are based on a variety of comorbidities and factors. Late mitral regurgitation has been noted to recur in some series [24]. We could speculate that this might be related to the stiffness and size of the patch. This may also occur in very large ventricles where there has been a delay of more than 40 months between infarction and surgical repair. Progression in remodeling may depend on mechanical causes or neuro-hormonal activation [25]. Continued medical therapy with diuretics, vasodilators, beta-blockers and ACE inhibitors are vital in ensuring good long-term outcomes.

2. **Improved left ventricular function and dimensions**.

Our experience along with those of others, have shown that there is reverse remodelling of the reconstructed left ventricle over a period of weeks and months. There is a steady improvement in functional status and exercise tolerance in the majority of patients. However, there is a small group of patients where the results may be mediocre, with functional status remaining static or with some deterioration. These are usually patients with diastolic dysfunction or a significant amount of myocardial fibrosis. Occasionally, the underlying cause may be the use of too large a patch or residual mitral regurgitation.

McCarthy reported on the Cleveland Clinic's experience with a variation of the technique whereby the left ventricle was reconstructed without a patch, where they analysed the first data for the first postoperative year [26]. These patients had a mean EF 23.9 % and mean EDVI 140 ml/m^2, that changed to 36 % and 90 ml/m^2 respectively. The levels of norepinephrine, plasma rennin activity, angiotensin and brain natriuretic peptide decrease significantly, confirming the regression of neurohormonal activation.

3. **Long-term results**

Global life expectancy at 5 years, in a series of 207 surviving patients analysed from 1991 to 1998, was 82 %. In another consecutive series of 245 patients from 1998 to 2003 (analysed to assess the impact of diastolic balloon sizing), the life expectancy at 5 years, hospital death included, is 85 % for the global series and 70 % for patients with very poor ventricular function (ESVI >120 ml/m^2). At 10 years, in the last category of very largely dilated failing ventricle, the percentage of survivors is 50 %, while it is 80 % for patients with ESVI <90 ml/m^2.

The Surgical Treatment for Ischemic Heart Failure (STICH) trial was designed to define the role of cardiac surgery in the treatment of patients with heart failure and coronary artery disease. One of the two major hypotheses of this trial (Hypothesis 2) was that surgical ventricular reconstruction, when added to CABG, would decrease the rate of death or hospitalization for a cardiac event, as compared with CABG alone.

This was conducted as a multicenter, nonblinded, randomized trial at 127 clinical sites in 26 countries. The trial was sponsored by the National Heart, Lung, and Blood Institute (NHLBI) of the National Institutes of Health.

Between September 2002 and January 2006, a total of 1,000 patients with an ejection fraction of 35 % or less, coronary artery disease that was amenable to CABG, and dominant anterior left ventricular dysfunction that was amenable to surgical ventricular reconstruction were randomly assigned to undergo either CABG alone (499 patients) or CABG with surgical ventricular reconstruction (501 patients). The primary outcome was a composite of death from any cause and hospitalization for cardiac causes. The median follow-up was 48 months.

Surgical ventricular reconstruction reduced the end-systolic volume index by 19 %, as compared with a reduction of 6 % with CABG alone. Cardiac symptoms and exercise tolerance improved from baseline to a similar degree in the two study groups. However, no significant difference was observed in the primary outcome, which occurred in 292 patients (59 %) who were assigned to undergo CABG alone and in 289 patients (58 %) who were assigned to undergo CABG with surgical ventricular reconstruction (hazard ratio for the combined approach, 0.99; 95 % confidence interval, 0.84–1.17; P = 0.90).

Adding surgical ventricular reconstruction to CABG reduced the left ventricular volume, as compared with CABG alone. However, this anatomical change was not associated with a greater improvement in symptoms or exercise tolerance or with a reduction in the rate of death or hospitalization for cardiac causes. (ClinicalTrials.gov number, NCT00023595.) [27]

The results of the STICH trial while not reflective of the experience or practice of many skilled heart failure surgeons, was a body blow to the

whole field of left ventricular reconstruction. There were many flaws in this study, which are addressed below.

Firstly, that there was great reliance on imaging using SPECT nuclear scans and dobutamine stress echocardiography. These techniques have their limitations, as alluded to in the publications in the New England Journal.

Second, there was a range of techniques used and residual volumes accepted as part of the study.

Third, the surgical strategy was different in various centers, with little standardization.

Fourth, there was a selection bias in that many centers that were experienced in left ventricular reconstruction tended to randomize patients who were in the grey zone. These patients who often ended up having that procedure, may not have been representative of the patients with actual akinetic left ventricles that would have benefited from LVR,.

Discussion

(A) **Other surgical techniques**: Beck first described a technique for repair of ventricular aneurysm before the age of the heart-lung machine [28]. This early technique utilized a clamp for resection and repair of aneurysms. Thereafter, the technique evolved to linear closure with use of cardiopulmonary bypass. Eventually, the techniques evolved into a variety of geometric repairs to recreate the conical shape of the left ventricle.

It is difficult to compare linear suture and circular repair, as these techniques can be utilized for the following good indications:

- A distal anterior and apical bulging true dyskinetic aneurysm can be repaired by resection of fibrous exteriorised scar followed by direct suture of the "neck" of the aneurysm.
- When the septum is widely involved, geometric reconstruction is the best solution in a great majority of cases. Linear repair produced poor physiological results [29]. Jakob et al. [30], Grossi [31], and Lundblad [32] are among authors that have reported positive experiences with endoventricular plasty and geometric repair over the past 15 years. Interestingly, Shapira [33], Kesler [34], and Tavakoli [35], showed no difference in the results between linear suture and patch repair. The reasons for equivocal results between two distinctly different techniques might be due to
- The retrospective nature of the comparisons,
- Small number patients in each series,
- The procedure being used only on dyskinetic bulging aneurysms.
- The series often spanning a long time period
- Left ventricular ejection fraction is used to report the data, without detailed information on the technique of assessment (angiography, radioisotope, or echocardiography) or ventricular volume measurements. The imaging techniques are often not consistent and, therefore, not comparable.
- Some suggestions have been made to improve poor results of linear suture. Stoney described a sandwich technique [36], for repairs of septal scars. Cooley described plication of the free ventricular wall in his original publication and this technique is still a very useful but fails to address the issue of the akinetic or dyskinetic septum. Mickelborough uses reinforcement of the septum with a patch [37] and also advocates a tailored "convergent suture" from outside to inside to reduce the length of the vertical suture.
- Athanasuleas and Buckberg promoted beating heart repair during the ventricular repair phase of the operation in an attempt to better preserve ventricular function and to also assess areas of potentially viable myocardium by palpation [38]. There are many advantages and disadvantages of beating heart repair. The theoretically benefits of a perfused myocardium over cardioplegic arrest, may not be clinically obvious in the majority of patients [39].

Some of the contentious discussion points are:

1. The exact location and the extent of the asynergic area cannot be reliably and reproducibly identified by palpation alone. The extent of scar should be carefully analysed preoperatively on imaging studies with multiple projections and a plan formed as to the actual areas that need to be excluded.
2. Diastolic balloon sizing of the residual left ventricle is more cumbersome when performed on a beating heart. This may be an important step for inexperienced or occasional surgeons, to ensure good early and intermediate results.

Adjunctive Therapies in Ischemic Cardiomyopathy

- Medical treatment with the appropriate medications form the cornerstone of chronic management [40]. Many of the drugs have a positive impact on controlling symptoms and reducing the metabolic demand on the heart, thereby allowing greater efficiency of cardiac function. Newer approaches such cardiac resynchronization or bi-ventricular pacing may be effective in about one third of ischemic patients, with a potential improvement of 1.5–5 % in EF without any effect on 1 year mortality [41].
- Improving blood flow to the ischemic areas with percutaneous intervention or surgical revascularisation, without addressing the enlarged heart, does not significantly improve dilated ischemic myocardium [42].
- There is much promise in the arena of cellular therapy, during acute, healing and the chronic phases after myocardial infarction. The hope is that this will reduce the extension of the scar, and prevent remodelling. None of the trials of regenerative therapy have been significant clinically [43].
- There is a revolution in the realm of mechanical assistance with a variety of small pumps that show great promise. For the moment, these devices are building on the promise shown by the Heartmate in the REMATCH trial [44].
- Ventricular containment or passive constraint maybe of interest in the treatment of dilated cardiomyopathies [45]. Containment of a reconstructed scar with the addition of cellular regenerative therapy maybe a future therapy to watch. This may even be applied early after a myocardial infarction to reduce remodelling and improve borderzone function [46].

Key Points to Remember

Left ventricular volume, not ejection fraction determines prognosis in dilated ventricles after myocardial infarction.

Left ventricular reconstruction began with repair of left ventricular aneurysms in the 1950s by the linear closure technique.

The contributions of Dor and Jatene suggested that the best approach would be to attempt to recreate the conical shape of the left ventricle.

Jatene's proposed imbrication of the septum to eliminate dyskinesia of this segment.

Geometric principles essentially utilize small patches of various kinds or purse string sutures to get reshape the ventricle into a conical form.

Reconstruction of scarred ventricles should be advocated soon after the ventricle dilates without waiting for symptoms of heart failure and decompensation.

Reconstructive therapies owe their success to a combination of modification of the anatomical deformation of the dilated ventricle, physiologic improvement of geometric efficiency and appropriate medical therapies.

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