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# Architecture of the left ventricle: insights for optimal surgical ventricular restoration

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**Abstract** The normal left ventricular shape has been defined as prolate ellipsoid. This shape is an adaptation to evolution. A knowledge of its unique macro and micro architecture forms the cornerstone in the understanding of its complex function. The left ventricle has a unique architecture with three different myofiber orientations, the longitudinal, circumferential and oblique fibers. The oblique orientation of fibers is essential for effective clockwise and anticlockwise torsional movements during systole and diastole, for optimal ventricular ejection and filling. The orientation and fiber angle decide the shape of the ventricle. An ellipsoid shape is vital for optimal function. Pathological disease states such as ischemic heart disease, valvular heart disease and cardiomyopathies cause a loss of obliquity of the myofibers. The myofibers become more horizontal resulting in ventricular dilatation and increased sphericity. The change from ellipsoid to globular shape with disease heralds the onset of left ventricular dysfunction and initiates the cascade of heart failure. Several strategies have been successful in reverting ventricular dilatation and sphericity to a more ellipsoid geometry. Pharmacological therapies like beta blockade and angiotensin converting enzyme inhibition have proven beneficial in early stages of heart failure with pathological remodeling. However, these agents in isolation are limited in reversing pathological remodeling in advanced heart failure. In some cases of advanced heart failure due to postinfarction left ventricular aneurysms, ventricular volume reduction with restoration surgeries have a role in restoring ventricular geometry with beneficial clinical outcomes. Surgical ventricular restoration has progressively evolved from the 1950s. Initially, aneurysmal resection and linear repair was done. This was gradually replaced by endoventricular patch plasty, which had better results. The resulting left ventricle was smaller in size but still continued to have a spherical configuration. Exclusion of the infarct area with a smaller longitudinal patch results in realignment of the non-diseased ventricular fibers with a resulting ellipsoid shape. This ellipsoid shape ensures clinical benefits. The geometry of the endoventricular patch thus holds the key to optimal ventricular shape in these patients. The technique to optimally restore a diseased ventricle to normal continues to evolve. This requires insights into the normal architecture and function, and the pathophysiologic effects of disease.

**Keywords** Aneurysm · Cardiac anatomy · Heart failure operations · LV reshaping/restoration · Ventricle left

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## Introduction

There are several limitations in defining therapeutic strategies for the syndrome of heart failure. This is a result of

existing lacunae in the understanding of the normal structure and function of the heart in vivo. Despite regional inhomogeneity of the normal left ventricle during systole and diastole, a highly effective global function is maintained. This is due to both structural and functional anisotropy. The effects of structural anisotropy that gets distorted in diseased states, and the impact of surgical ventricular restoration are reviewed here.

## Normal left ventricular structure and shape

The normal left ventricular shape is a prolate ellipsoid with its long axis directed from apex to base [1]. Hutchins and Brawley [2] studied the shape of the LV. By substituting normal values for maximal LV diameter (*d*) and apex to base length (*h*) into the formula for the volume of a prolate ellipsoid, namely, $V = \Pi/6$ d2 h, they obtained 50 ml in systole(observed 62 ml) and 176 ml in diastole(observed 153 ml). The mathematical model of an ellipse has therefore been fairly accurate in defining left ventricular shape.

Its inflow and outflow are at 30° to each other i.e., V shaped. The inlet (mitral valve) and outlet (aortic valve) of the ventricular chamber are in continuity, being separated by a thin membrane—the anterior mitral leaflet. Hence, blood enters and leaves through virtually the same orifice, which makes its flow through the left ventricle bi-directional. The blood flows vertically into the left ventricle through the mitral valve and is propelled out of the left ventricle in the same vertical but opposite direction through the aortic valve, which is continuous with the mitral valve. The left ventricle acts by its unique fiber orientation as a pump to propel blood flow in the reverse direction to its inflow. This bi-directional flow in the left ventricle is required to generate a systolic blood pressure of 120 mm Hg in order to propel blood flow through the entire systemic circulation. The ellipsoid shape is crucial for normal ventricular function, which is an adaptation to evolution and assumption of an erect habitus. The amphibian has a spherical ventricle, man has an ellipsoid systemic ventricle and a giraffe has an extreme ellipsoid (almost cylindrical) ventricle, which is required to generate a systolic blood pressure of 300 mm Hg.

The right ventricle propels blood into the low pressure pulmonary circulation. Hence, it does not require propulsion at high pressures. A peak systolic pressure of 25 mm Hg is necessary for adequate pulmonary blood flow. The inflow and outflow of the right ventricle are at 90° to each other; the tricuspid valve and pulmonary valves in the right ventricle are perpendicular to each other. The blood flows into the right ventricle through the tricuspid valve and flows out through the pulmonary valve. In the right ventricle, the longitudinal fibers are more abundant and their contraction constitutes a milking effect that propels blood flow through the tricuspid valve and out of the pulmonary valve, which are separated by the muscular conus tissue. This flow is linear and is hence termed unidirectional.

The law of Laplace [3, 4] can be used to explain the great variation in thickness of the ventricular wall. In the portions that are very curved (small radius of curvature) as at the apex, the walls though thin can still produce sufficient tension during contraction, to develop enough pressure in the contents. When the wall is relatively less curved as at the base (large radius of curvature), the tension developed must be much greater to produce the same pressure. Hence, the wall should be and is correspondingly thicker. This transition in thickness is gradual from base to apex.

A knowledge of the orientation of its muscle fibers is vital in understanding its complex function. Torrent Guasp et al. [5, 6] proposed a model where the continuum of myocardial architecture was depicted as a muscle band that was organized spatially into two distinct helicoids, extending from beneath the pulmonary valve across the septum, to beneath the aortic valve. Some authors [7–9] do not agree with this concept of a single helical muscle band. Several studies (autopsy and tagged MRI) [10–14] have proved that the LV comprises two helical fiber geometries that are continuous, i.e. a right-handed helix in the subendocardium that gradually changes to a left-handed helix in the subepicardium. Mathematical models have proved that the counterdirectional helix is energetically efficient and equalizes redistribution of stress and strain during the cardiac cycle [11, 15] Streeter et al. [16] studied the double helix. The helix angle changes continuously from subendocardium to the subepicardium, ranging from +60° to −60° [17, 18]. This change in helix angle is due to the 3-dimensional sheet architecture of the myofibers. The base and upper septum have more circumferential fibers and from mid-wall to apex, the fibers run obliquely.

This double helix is embryologic in origin [19]. The first region of differentiation of myoblasts into striated fibers is in the epicardium. They have an obliquely horizontal orientation. The LV empties laterally into the right ventricle through the interventricular canal. The fibers are parallel to the path of ejection. Weiss [20] demonstrated that cell lines are laid parallel to the direction of mechanical tension applied. The inner fibers are laid when the LV has developed considerably i.e., as it becomes larger, the interventricular canal becomes smaller and the aorta becomes the main pathway of ejection. The endocardial helix is therefore in a different direction from the epicardial helix.

## Normal left ventricular deformation during the cardiac cycle

Nuclear magnetic resonance tagging with 3D MRI enables non-invasive tracking throughout the LV myocardium during the cardiac cycle [21].

### Radial displacement

This is directed inward throughout the LV. The magnitude is greatest in the apical inferior and lateral walls and least in the septum and apicoanterior wall, reflecting contraction and bulk rotation about a septolateral axis with anterior motion at the apex. Shortening strains are maximal at the apex, moving axially from apex to base, causing descent of the mitral annulus.

### Longitudinal deformation

Shortening along the long axis occurs by descent of the base toward the apex. The displacement magnitudes are greatest at the base, decreasing linearly toward the apex.

### Circumferential deformation

When viewed from the base, it is clockwise initially, and anticlockwise upto end-systole. More apically, the initial rotation is more prominent [22–25]. The magnitude of circumferential deformation is maximal at the base of the ventricle.

### Torsion

Torsion is the rotation of a level about the long axis with respect to the base. It is greater in the endocardium than the epicardium [26]. It is a function of the oblique ventricular fibers. In systole, the apex has a brief initial clockwise twist (torsion) followed by a predominant anticlockwise twist. The base twists in a clockwise direction (reverse of apical twist) causing a wringing effect enhancing ventricular ejection. In diastole, LV torsion occurs in the reverse direction-both the apex and the base twist in reverse of the systolic twist. This untwisting is maximal during early relaxation and augments LV filling by a suction effect [27].The orientation of the oblique myocardial fibers is decisive of ventricular shape and function.

The fiber angle is crucial for fiber obliquity. The normal oblique fiber angle is 60°. When heart failure ensues, the fiber orientation changes from oblique to transverse as detailed in the following paragraphs. Sallin [28] demonstrated that a myofiber contraction of 15% in a ventricle with a normal short/long axis ratio (sphericity index) of 0.5 (ellipsoid shape), generated an ejection fraction of 62%. At the same 15% fiber contraction, the ejection fraction fell below 40% if the sphericity index approached 1 (spherical shape) and went up to ≥80% if the sphericity index approached 0 (extreme ellipsoid; Fig. 1).

Despite different torsions from base to apex and between epicardium and endocardium, these areas are subject to a constant mean shear. Shear at a point is dependent on the torsion angle, the distance between the point and the center of the ventricular cavity®, and the distance between the point and the base (*h*; Fig. 2). The torsion increases as *h* increases and as *r* decreases. The non-linear increase in torsion, with greater increments at the apex than at the base, may be due to tapering at the apex. This maintains constancy of mean shear [26]. This is the principle by which stress is equalized along the spatially non-homogenous LV wall during normal ejection. The torsion of the epicardial fibers exceed that of the endocardial fibers [21, 26], as they are at a greater radius from the LV central long-axis and so have longer lever arms to produce greater momentum.

The normal right ventricular free wall also exhibits torsion, but of a lesser magnitude [29]. In patients who underwent Mustard or Senning repair for transposition of great arteries, the systemic right ventricle exhibited greater circumferential than longitudinal strain, in contrast to the normal right ventricle that has greater longitudinal than

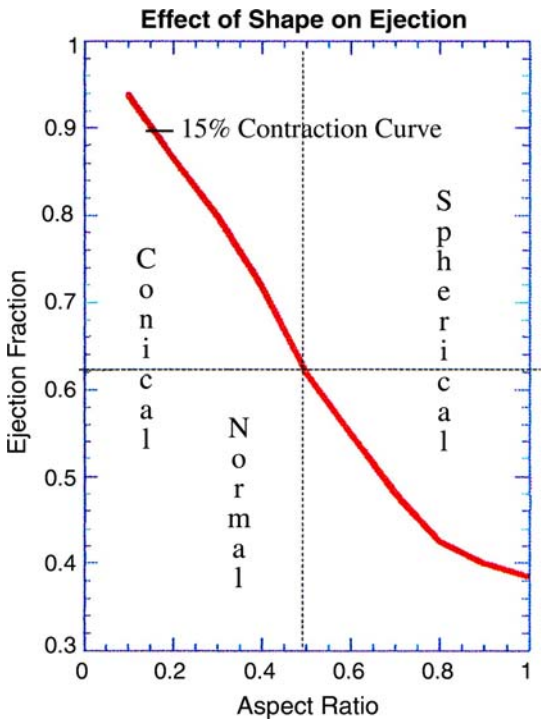


**Fig. 1** Relationship of fiber contraction to LVEF in circular and ellipsoid ventricles

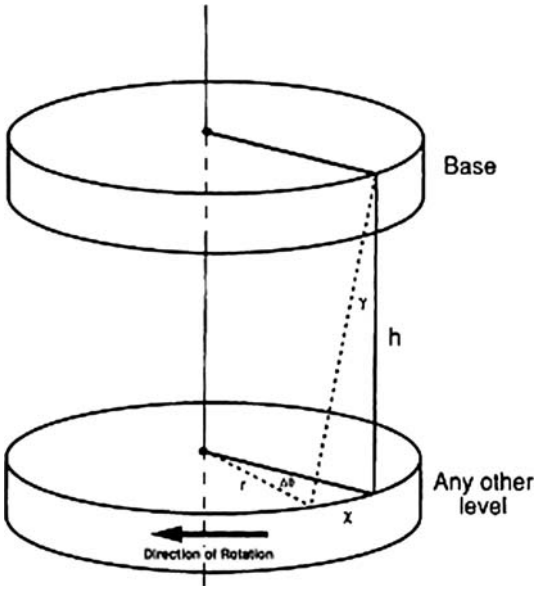


**Fig. 2** Endocardial torsion angle ($\Delta\theta$) is greater than the epicardium with equal shear ($\gamma$)

circumferential strain. This could be an adaptation to a higher resistance circulation. However, the systemic right ventricle did not exhibit any torsional movement. Torsion is a prerequisite for energy efficient LV ejection and relaxation. This is explained by the double helical myofiber structure and the acute angle between the LV inflow and outflow. This is impaired in myocardial infarction, cardiomyopathy and aortic stenosis, and is more marked for diastolic untwisting, forming the basis of diastolic dysfunction in these disease states [30–32].

## Altered LV geometry and function in heart failure

### Altered geometry

In chronic heart failure, there is dilatation of the cardiac chambers, which leads to distortion of the LV ellipsoid geometry [33]. At the base, dilatation leads to straightening of the angle between the LV inflow and outflow. The oblique fibers from mid-wall to apex become more horizontal [32–34]. A theoretic analysis can demonstrate that an oblique fiber angle on a surface with a certain radius of curvature (Fig. 2) can attain a narrower angle, as the surface projects onto one with a larger radius of curvature as in a dilated spherical ventricle. For example, for a normal 60° fiber angle, increase in short axis dimension of the ellipsoid by 3 cm can decrease the fiber angle by 10° that is from 60° to 50°. Minor fiber angle changes of 5°–10° can substantially affect ventricular torsion and performance [28].

### Altered function

As the normal LV inflow and outflow are in continuity, there is need for torsion in systole to propel blood into the aorta and a similar torsion in the opposite direction for negative suction during LV filling. In the spherical dilated heart, due to horizontal orientation-'creep' of the fibers, both systole and diastole are affected. In diastole, the circumferential strain is reduced, leading to reduced amplitude of untwisting of the base. The negative suction vortex is not optimal with additional impaired lengthening of the LV leading to abnormal early filling.

During systole, the apical systolic torsion is reversed. The apex continues to rotate clockwise with reduction of maximum positive torsion. The maximum positive torsion is delayed until after end-ejection. This signifies an increasing positive torsion during isovolumetric relaxation and early diastole affecting both effective ventricular ejection and filling. As radial, circumferential and longitudinal strains are also reduced, there is a non-uniformity of mean shear along the LV wall increasing wall stress. There is decreased longitudinal shortening of the lateral wall with systolic ascent instead of the normal descent of the mitral annulus [35]. In addition, there is abnormal stretching of the apex. The apex no longer retains its maximal curvature and hence is no longer an optimum fulcrum for ventricular contraction.

In postinfarction LV aneurysms, there is an additional load of akinetic or dyskinetic aneurysmal segments. Klein [36] studied the hemodynamics of LV aneurysms. When the aneurysmal area was 20–25% of the surface area of the LV, the extent of shortening of the remaining normal LV myocardium should exceed physiologic limits i.e., 30% of maximal initial muscle length. The increased radius of curvature should therefore increase the systolic tension to 2–2.5 times normal. But, only a portion of contractile myocardium is available to respond to increased stretch (Starling's law). Hence, the effects of systolic tension (afterload) to inhibit ejection may exceed the effects of preload to increase it [37, 38] leading to ventricular dilatation involving the contractile myocardium.

## Surgical ventricular restoration

The various techniques of surgical ventricular restoration have undergone an evolutionary change from the times of Likhoff and Bailey [39]. At first, linear plication and repair of anteroapical dyskinetic aneurysms was done under cardiopulmonary bypass. This technique involved linear plication of the aneurysmal portion at the border zone, excision of the dyskinetic segment and repair. Various modifications were introduced by Cooley et al. [40], Linda

Mickelesborough [41] and Jatene [42]. Savage et al. [43] in their eloquent study, described the changes in ventricular mechanics, hemodynamics and oxygen consumption with linear repair. Plication decreased the circumferential perimeter of the ventricle, decreasing both end-diastolic and end-systolic volumes, not increasing the stroke volume. It did not increase systolic wall thickness, resulting in a smaller but more spherical ventricle. Some patients had distortion of ventricular geometry with disruption of papillary muscles. This indicates that impaired systolic torsion and diastolic untwist of the LV persisted, causing both systolic and diastolic dysfunction. This was despite decrease in wall stress according to Laplace's law. This technique also limits infarct exclusion in the septum. Kramer et al. [44] studied LV mechanics by MRI tagging in anteroapical dyskinetic aneurysms, following linear repair which included parts of the septum. There was improvement in longitudinal strain in the middle, basal and inferior parts of the LV. There was better diastolic torsion or untwisting at the base, with no effect on systolic torsion [45].

Jatene recommended insertion of patches in large LV cavities. Dor [46] revolutionized the technique of surgical ventricular restoration (SVR) with introduction of endoventricular circular patch plasty. Here, the LV was opened parallel to the left anterior descending artery (LAD), the LV cavity was assessed with a collapsible measuring device of 55 ml/m2 body surface area, which was inflated in the LV cavity. A circular Fontan suture was taken at the border zone of the infarcted and viable myocardium. This suture served to decrease the perimeter of the border zone. A circular Dacron patch was sutured over the circular suture line, creating a neo-ventricular apex. The residual LV cavity of 55 ml/m2 BSA was maintained. The infarcted myocardium was sutured over the patch in a linear repair to ensure hemostasis. Although there is widespread use of LV cavity measuring devices by several surgeons (Menicanti [47]), an optimal LV cavity size has not been quantified. The RESTORE group has adopted a similar endoventricular circular patch plasty for anterior aneurysms-the SAVER technique [48]. This ensures a more physiologic ellipsoid shape to the LV than linear repair, as evidenced by quantitative indices like the sphericity index [49]. This technique has been applied to both dyskinetic and akinetic scars by Dor. Results have proven reverse remodeling in the remote myocardium i.e., reorientation of myofibers to a more oblique direction seen at long term following SVR. Revascularisation of the LAD territory is possible which was ineffective with linear repair. The prerequisite for effective SVR seems to be an end-systolic volume $\geq$60 ml/m2 BSA with viable remote myocardium. The clinical outcomes were improved functional class and long term survival [50, 51]. Tulner et al. [52] studied the LV function immediately following endoventricular circular patch plasty by pressure–volume loops. The use of pressure–volume loops accurately reflects intrinsic LV function and is relatively load independent [53, 54]. The slope of the end-systolic pressure–volume relation, the end-systolic elastance, a load-independent parameter of systolic function that increased significantly. The end-systolic pressure–volume relation was significantly shifted to the left, also indicative of improved systolic function. There was a leftward shift in the end-diastolic pressure–volume relation also. The diastolic wall stiffness increased despite a decrease in end diastolic volume, similar to the mechanics of diastolic heart failure. However, there were significant decreases in wall stress, mechanical dyssynchrony with improved myocardial performance. As this was done acutely after SVR, the effects of myocardial edema and stunning after surgery complicate these results. Moreover, from the presentations by Dor, it is known that excessive reduction of ventricular chamber size is associated with a low output state and restricted LV filling(diastolic dysfunction) [55]. At this juncture, it is unknown what factors determine the optimal residual LV volume for individual patients. At present, linear repair is recommended for small aneurysms i.e., regions of asynergy without ventricular dilatation to avoid diastolic dysfunction [56]. Endoventricular circular patch plasty is recommended if the area of akinesis or dyskinesis is atleast 35% of the ventricular perimeter [56]. It is not known what patch size is most appropriate for reconstruction of the LV. If the patch size is large to maintain the original end diastolic contour of the contractile portion of the LV, the patch area will comprise a large akinetic area. If the patch size is maximally reduced, the repair represents essentially linear repair that may deform the ventricular contour, as in linear repair. Kawata et al. [57] used a mathematical model proposed by Watson et al. [58], and derived an optimal patch area to be less than 50% of the surface area of the aneurysmal region. Although there was early improvement in systolic and diastolic functions, the patch size was still very large in some patients. The LV shape changes induced by endoventricular circular patch plasty were studied by Dor et al. [59], by angiographic projections. The improved LV function was mainly due to increased systolic shortening of the inferior wall. There were marked reductions in the end-diastolic volumes, shift of the angiographic apex counterclockwise i.e., toward the aortic corner and disappearance of the rim with negative curvature corresponding to the infero-apical border of the aneurysm, which assumed a normal outward convexity. Thus, the postoperative ventricle is smaller, but still retains a spherical shape. The clinical improvements are attributable to decrease in wall stress, but the hemodynamics studied has been suboptimal, which may have a bearing on the long term outcomes. As

the ventricular contour continued to be distorted in the apico-anterior portion by the endoventricular circular patch plasty technique, Cooley [60] advocated the use of tear drop shaped patches instead of circular patches. The tear drop shaped patch was sutured into the LV cavity, by wedging the apex of the tear drop cephalad in the ventriculotomy incision. This was found to result in a better ellipsoid geometry with lesser apicoanterior distortion. The technique of surgical ventricular restoration is continuing to evolve.

The restoration of near normal ventricular geometry is the primary goal. Optimal function follows optimal form. The geometry of the endoventricular patch determines ventricular anatomy postsurgery. A rectangular linear patch has shown better hemodynamics than a circular patch as determined by mathematical formulae, which need clinical validation.

## Mathematical model for linear endoventricular patch plasty

There is a mathematical basis to our technique of linear endoventricular patch plasty, explained by the model of ventricular aneurysm developed by Vayo [36] and Klein [61]. The ventricle was arbitrarily divided into an akinetic region with the remaining portion having normal contraction and relaxation. To simplify analysis, the ventricular chamber was assigned the shape of a sphere, although the aneurysm has a more complex geometry. Figure 3 illustrates a LV with a circular patch and a rectangular patch of akinetic myocardium, respectively. During systole, the ventricular size decreases except the portion of akinetic myocardium, which remains fixed at end-diastolic length from the center of the chamber. This akinetic area can be considered as the akinetic endoventricular patch. When the per-cent inactive muscle was compared with per-cent active muscle for aneurysms of different configurations i.e., circular versus rectangular (Fig. 4), there was more per-cent active muscle with a rectangular akinetic area than with a circular akinetic area. Klein [61] studied the hemodynamics of LV aneurysms. If the akinetic area was 20–25% of the ventricular area, there was optimal physiologic stroke volume without need for further ventricular dilatation. In endoventricular circular patch plasty, a circular patch was used, with resulting early decrease in stroke volume and impaired systolic torsion [43, 52]. Some workers report a long term positive remodeling (re dilatation) of the ventricle following circular endoventricular patch plasty [50]. We used a 3 cm× 10 cm rectangular patch in all our patients, which was <25% of the ventricular area following infarct exclusion.

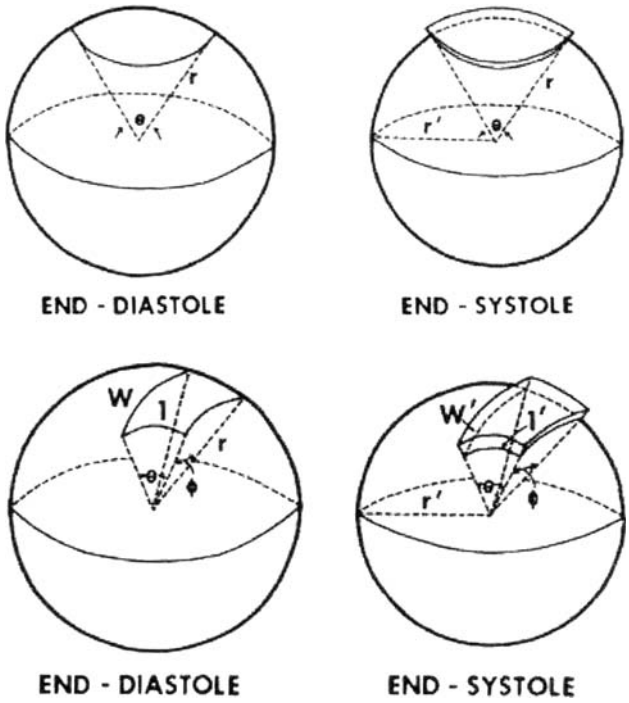


**Fig. 3** Chambers with circular and rectangular akinetic areas. With permission from Klein et al. [61]

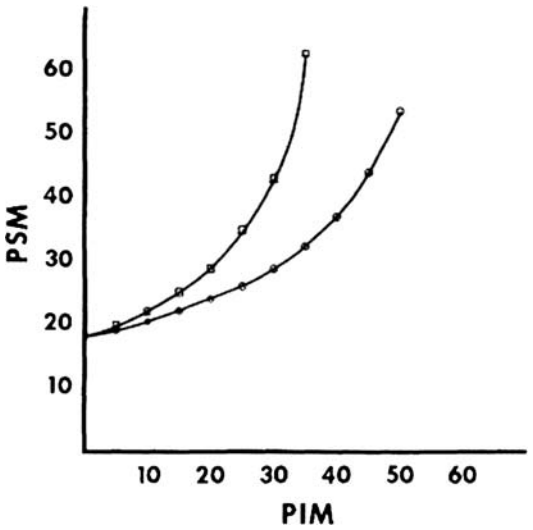


**Fig. 4** Relationship of per-cent inactive muscle *PIM* to per-cent shortening of active muscle(PSM). *Circles* = circular aneurysm, *Squares* rectangular aneurysm. With permission from Klein et al. [61]

## Surgical technique of linear endoventricular patch plasty

We modified the technique of endoventricular patch plasty advocated by Cooley [60], by making the patch linear to address the dyskinetic/akinetic area, and adding a Teflon buttressed linear repair to the ventriculotomy site [62]. The LV cavity was opened by a linear incision parallel to the

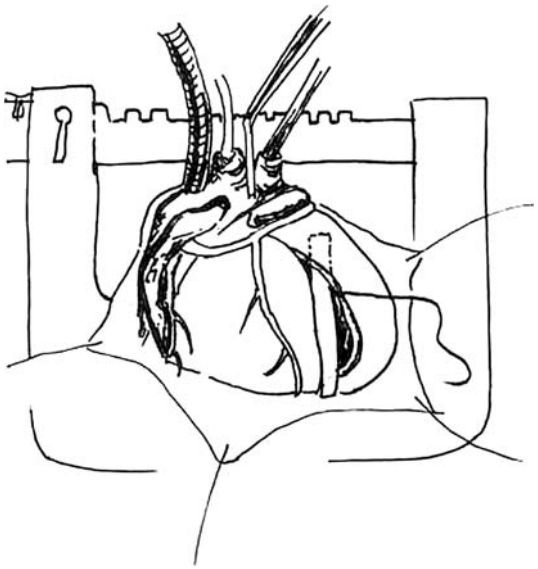

**Fig. 5** Linear endoventricular patch plasty

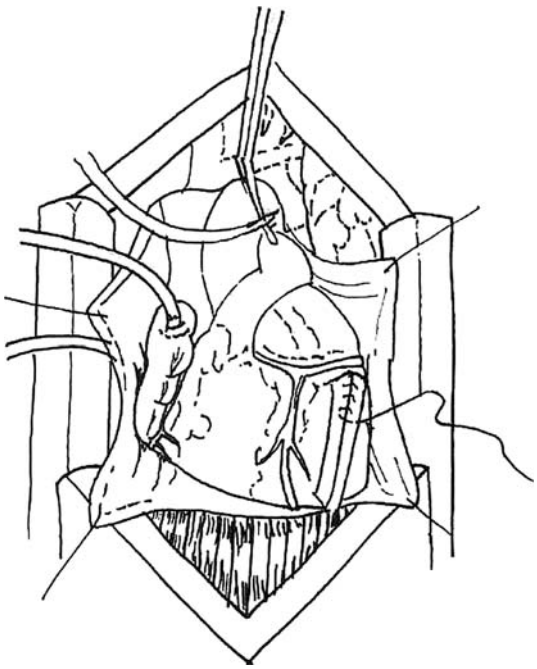

**Fig. 6** Linear repair after endoventricular linear patch plasty

LAD. The residual LV cavity was assessed visually. We did not use any residual LV cavity measuring devices, as we believe that the accuracy of measurement by these devices is fallacious in the cardiopleged heart. A 3 cm× 10 cm linear hemashield patch was used for LV reconstruction. Demarcation of the infarct zone was not done. A linear patch was sutured into the LV cavity (Fig. 5) with an oblique lie from base to apex. With the rectangular patch sutured to the border zone, the uninfarcted myofibers get reoriented to an oblique lie from their horizontal position. The aneurysmal apex was excluded by creating a neo-apex with a smaller radius of curvature. The ventriculotomy site was closed with a Teflon buttressed linear repair (Fig. 6).

## Validation of Vayo's mathematical model—left ventricular shape analysis following aneurysm repair by rectangular endoventricular patch

The ellipsoid ventricular shape is essential for optimal ventricular function, as described. As evidenced by the mathematical model of Vayo et al. [36], a rectangular endoventricular patch of about 3 cm× 10 cm would result in optimal exclusion of the infracted segment, limit the area of akinesis to <25% of the ventricular area and increase the ratio of uninfarcted (contractile) myocardial segments to akinetic myocardial segment (patch).

To test this, the LV shape was assessed by calculating the wall motion by the centerline method on the LV angiograms. The LV shape was determined by the centerline method on contrast ventriculography [8]. Here, motion was measured along 100 chords drawn perpendicular to a centerline constructed midway between the end-diastolic and end-systolic contours of the left ventriculogram on the 30° RAO projection. The measured motion of the 100 chords was normalized for heart size by dividing by the length of the end-diastolic perimeter. As normal motion varied from chord to chord, the normalized motion at each chord was converted into units of normal standard deviations from the normal mean motion at each chord. Wall motion in each of five regions of the LV contour was determined by averaging the motion abnormality, expressed in SD, of chords 1–16, 17–32, 33–48, 49–64 and 65–80, corresponding to the anterobasal, anterolateral, apical, inferior and posterobasal regions, respectively. The hypokinetic chords were designated as negative and hyperkinetic chords positive. There was an improvement in the size (EDV and ESV) and in LV function (LVEF). The improved LV performance was also evidenced in a significant reduction of hypokinetic segments. As the patch was akinetic, the decrease in akinetic segments was not significant (Table 1). The EDV and ESV were measured from contrast ventriculography in the RAO projection. These volumes were also calculated based on the formula of volume of an ellipse. The calculated ESV was 41 ml (observed: 48 ml) and the EDV was 64 ml (observed: 68 ml), conforming to an ellipsoid geometry. To further validate these findings, ventricular shape analysis was performed, which was modeled on the technique used by Kass et al. [63]. The digitized LV contour was traced in systole and diastole. The center moment of the digitized contour was calculated, and using this as origin, radii

**Table 1** Clinical characteristics and contrast ventriculographic data of our patients

| | | | | |
|---|---|---|---|---|
| *Clinical characteristics* | | | | |
| Total patients | | 102 | | |
| Controls | | 0 | | |
| Age | | | | |
| Mean age | | 43.2 ± 8.3 years | | |
| Sex | | | | |
| Males | | 82 (80.3%) | | |
| Females | | 20 (19.6%) | | |
| Symptoms | | | | |
| Cardiac failure | | 99 (98%) | | |
| Anterior myocardial infarction | | 89 (95%) | | |
| Inferior myocardial infarction | | 7 (7%) | | |
| Diabetes mellitus | | 65 (69.1%) | | |
| Concomitant CABG | | 67 (65.7%) | | |
| Preoperative NYHA class | | 4:61 (60%); 3:41 (40%) | | |
| Postoperative NYHA class | | 1:62 (92%); 2:5 (8%) | | |
| Postoperative cardiac morbidity | | | | |
| IABP | | 5 (4.9%) | | |
| LVAD | | 0 | | |
| Rehospitalization for cardiac failure | | 4 (4.5%) | | |
| Postoperative cardiac mortality | | | | |
| In-hospital | | 8 (7.8%) | | |
| Mid-term | | 5 (3%) | | |
| | Preoperative | Postoperative (30 days) | Mid-term follow up (4 years) | *P*-value |
| *LV geometry (echocardiography)* | | | | |
| LVEF | 31.5 ± 6.5% | 34.2 ± 5.9% | 38.4 ± 4.5% | <0.001 |
| LVIDd | 60.2 ± 7.5 mm | 55 ± 7 mm | 51.2 ± 6.6 mm | <0.001 |
| LVIDs | 48.1 ± 7.9 mm | 43.4 ± 7.7 mm | 38.4 ± 6.9 mm | <0.001 |
| EDV | 140.3 ± 38.3 ml | 100.8 ± 3.5 ml | 68.4 ± 12.4 ml | <0.001 |
| ESV | 95.1 ± 26.1 ml | 66 ± 21.7 ml | 54.2 ± 16.4 ml | <0.001 |
| Sp.ind. | 0.9 ± 0.02 | 0.79 ± 0.04 ($p$ = NS) | 0.62 ± 0.02 | <0.01 |
| *LV geometry (contrast ventriculography)* | | | | |
| Hypo. Ch | | 61.1 ± 12.8 | 36.5 ± 14.8 | <0.01 |
| Akin/dys. Ch | | 32.5 ± 11 | 25 ± 8.1 | NS |

were drawn to each of the digitized points on the perimeter of the contour. The length of each radius® and the angle ($\theta$) relative to a given orientation was measured and an $r(\theta)$ function was generated. The first digitized point (aortic valve-anterobasal wall intersection) was chosen as the 0° position and was continued counter-clockwise from that location to generate a polar representation (Fig. 7). When the preoperative and postoperative polar representations were generated, there was a significant decrease in the anterolateral, anterobasal and inferior areas. This proves the mathematical model of a rectangular area of akinesis <25% of the ventricular area has better hemodynamics as it has a greater ratio of uninfarcted (contractile) myocardial segments to akinetic segments. In addition, there is realignment of the anterior, antero basal and inferior non-infracted myocardial segments to a more physiologic ellipsoid geometry.

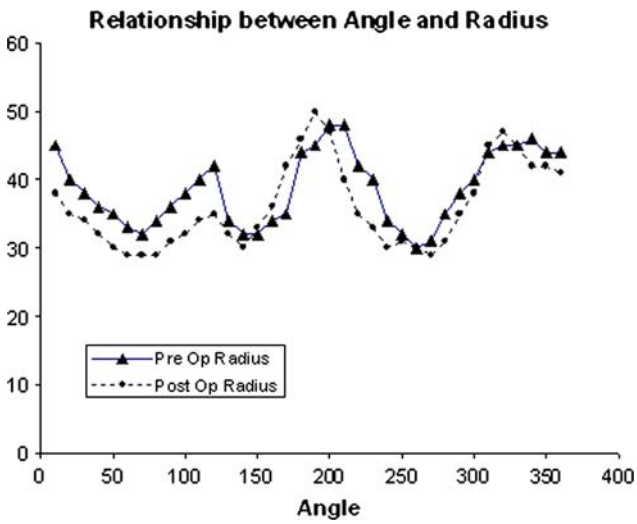


**Fig. 7** LV shape analysis

## Impact of left ventricular shape on clinical outcomes in heart failure

During transition from normal ventricular function to ventricular dysfunction, the normal ellipsoid shape is distorted to a more spherical geometry. The onset of clinical symptoms of heart failure occurs as the sphericity of the ventricle increases [64]. The symptoms of exercise intolerance are more prevalent in patients with ventricular dysfunction whose sphericity increases during exercise testing [65]. These findings indicate proven clinical benefits of ventricular ellipsoid geometry. Surgical restoration of ventricular geometry has proven long term clinical benefits.

Clinical trials randomizing patients to either therapy alone in order to compare the efficacy of individual therapies would lead to confounding results, which might obscure optimal therapy for patient subgroups. The reasons for this are multifactorial. The patients with heart failure constitute a diverse population. In advanced heart failure, surgical therapy needs to be individualized to specific patient groups. The recently concluded Hypothesis 2 of the STICH trial investigators only highlights this. The STICH trial [66] had three arms comprising medical therapy, coronary revascularization (CABG) and CABG with surgical ventricular restoration. They concluded that there was no difference in clinical outcomes with CABG alone as compared to CABG and surgical ventricular restoration. The population of patients assigned to either arm was diverse. This might have obscured the benefits of combined CABG and surgical ventricular restoration, which has shown benefit in specific patient subgroups. Cardiac remodeling has also not been assessed in totality. Longitudinal data on ESVI is not available. The ESVI was assessed only in the early postoperative period (upto 4 months), which does not estimate cardiac remodeling due to neurohormonal inhibition and revascularization. Ventricular restoration was accomplished by the circular endoventricular patch plasty, which is known to result in a spherical ventricle.

Some of the patients with postinfarction LV aneurysms demonstrate normal or recanalised coronary arteries, where coronary revascularisation has no role.

The two surgeries are diverse and serve to address different patient substrates. The trial has attempted to randomize patients who qualify as two different substrates for different surgical therapies.

Revascularisation by percutaneous intervention or surgery is an adjunct to standard heart failure therapy in a specific population with demonstrable reversible ischemia and is based on the anatomy and functional pathology of the coronary tree.

Surgical ventricular restoration was initially advocated for patients with postinfarction LV aneurysms, with discrete akinetic/dyskinetic areas and remaining areas of contractile myocardium. Later, it was done in patients with dilated hypokinetic ventricles where the benefits were seen due to decrease in wall stress by decreasing the ventricular radius (Laplace's law).

The technique of surgical ventricular restoration has been under focus recently. Several authors [62, 67] have shown better results in terms of ventricular geometry and clinical outcome with narrow endoventricular patches as compared to circular endoventricular patches. The near normal geometry of the left ventricle can be achieved with rectangular endoventricular patch plasty as evidenced by our results.

We studied 102 consecutive patients over 3–4 years. Ours was an observational study where all patients with postinfarction left ventricular aneurysms qualifying the inclusion criteria were studied. We did not have a control arm in our study for reasons explained above. All our patients had suffered a previous transmural myocardial infarction and had significant left ventricular dilatation with significant LV dilatation with LV end-systolic volume index $\geq$60 ml $m^{-2}$, and large akinetic or dyskinetic segments. Most patients were in advanced heart failure. Some also presented with angina. Our patient clinical characteristics and contrast ventriculographic data are listed in Table 1. Those of our patients [67(65.7%)] with significant coronary stenosis and presence of viable myocardium assessed by nuclear scanning underwent SVR and concomitant CABG. Following surgery, there were 8 (7.8%) in—hospital deaths. There was a decrease in the ESVI from $95.1 \pm 26.1$ to $66 \pm 21.7$ ml $m^{-2}$ ($p < 0.001$). During follow up, there were 5(3%) mid-term deaths. There was a further decrease in ESVI from $66 \pm 21.7$ to $54.2 \pm 16.4$ ml $m^{-2}$($p < 0.001$). There was continued beneficial remodeling with near physiologic ellipsoid ventricular shape as evidenced by the shape analysis on contrast ventriculography. There were significant clinical improvements in symptoms at 4 years following surgery as evidenced by the NYHA class. There were 62 (92%) patients in NYHA class 1 and 5 (8%) patients in class II. Cardiac failure requiring hospitalization developed in 4 (4.5%) patients. The interval from SVR to development of cardiac failure was $9.2 \pm 1.2$ months.

## Comment

Our technique of surgical ventricular restoration is original and ensures a near physiologic ellipsoid LV shape, which persists 4 years after surgery. The techniques of surgical ventricular restoration continue to evolve toward

restoration of a near normal physiologic ventricular shape [68]. Several technical considerations need to be individualized for specific patient groups.

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