Klinik für Herz und Gefäßchirurgie

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Surgical treatment of left ventricular aneurysms: Results of a long term study over 25 Years.

Chirurgische Therapie des linksventrikulären Aneurysmas: Ergebnisse einer Langzeitstudie über 25 Jahre.

Wael Bedda

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Surgical Treatment of left ventricular aneurysms:
Results of a long term study over 25 Years

1. Introduction:

1.1 Background:

Recently, endoventricular pericardial patch plasty has been proposed as a more physiologic repair of post infarction left ventricular aneurysm than is linear repair. My aim is to present our institutes long-term results in surgical treatment of left ventricular aneurysm comparing two techniques.

The first successful repair of left ventricular aneurysm was performed by Likoff and Bailey [46] in 1955. Linear repair of left ventricular aneurysm using cardiopulmonary bypass was reported by Cooley and associates [12] in 1958. The operative technique remained unchanged until the mid-1980s, when it became apparent that the clinical results were suboptimal. Early mortality was relatively high, in the range of 10% to 20%, and late results were also unsatisfactory, with many patients having persistent symptoms of congestive heart failure [38]. Attention was then focused on finding new methods of reconstruction to restore left ventricular geometry. These concepts were introduced by Jatene [38] and later modified by Dor and colleagues [22]. In 1989, more than 3 decades after his original report, Cooley abandoned linear repair for a new technique, which he termed Intracavitary repair or Endoaneurysmorrhaphy [14].

History Of left ventricular resection for LV. Aneurysms		
1944	*Beck*	*Fascia lata reinforcement*
1955	*Likoff-BaiIey*	*1st closed resection*
1958	*Cooley*	*1st open resection*
1973	*Stoney*	*" In coat " plicature*
1977	*Dagget*	*Posterior patch*
1979	*Levitsky*	*Anterior patch*
1980	*Hutchkins*	*Influence of cardiac geometry*
1984	*Jatene*	*Circular reduction*
1985	*Dor*	*Endoventricular patch plasty*
1989	*Cooley*	*Endoaneurysmorrhaphy*

Table 1: Trend towards LV Reconstruction [1]

1.2 Definition:

A *post infarction aneurysm of the left ventricle is a well delineated transmural fibrous scar, virtually devoid of muscle. During systole, the involved wall segments are akinetic or dyskinetic. Scars and infarcts are not considered aneurysms, but this is controversial. Johnson and colleagues define aneurysm as a large single area of infarction (scar) that causes the left ventricular ejection fraction to be profoundly depressed (35% or lower) [43]*

However, imprecise definition of a problem may be the largest obstacle to its resolution. Similarly, the solution of a problem is determined by a precise view of its cause.

Left ventricular wall ischemic asynergy can be dyskinetic or akinetic but there is actually a continuum between pure dyskinetic and pure akinetic due to the thickening of the endocardium, the calcification or localisation of the scar on the septum and also due to the variations in the LV wall involvement (transmural or only subendocardial scar). There is also a continuum in time as a dyskinetic area progressively provokes a global akinesia of the ventricle. [30]

The centreline ventriculogarphic definition makes akinesia and dyskinesia the same consequence of LV scar and leaves the appearance of bulging or akinesia as only radiologic or surgical definition. More important, this changes completely the conceptual understanding of akinesia versus dyskinesia and defines aneurysm as a noncontractile segment. [30]

The value of this more global definition is that the operation must exclude the noncontractile septum.

This more physiologic concept exceeds the limitations of current ventriculographic and surgical description, which do not quantify the muscle involved. The centreline method describes motion of the septum and all LV segments. This shows that akinesia and dyskinesia are part of the same process. Clearly an anterior aneurysm is a noncontractile or asynergic muscle that includes the septum as well as the anterior wall and apex.

We must therefore reclassify our concept of aneurysm to indicate scar or noncontractile segment. This asystolic region makes the remaining ventricle enlarge and compensate for the inability of the scarred segment to help generate cardiac output. When this occurs, we will then recognize that akinesia versus dyskinesia is a verbal and not operative description; both led to remodelling and subsequent cardiac failure. [30]

1.2 Epidemiology:

Incidence and Natural history:

Aneurysmal dilatation of the left ventricle occurs in between 10 and 35% of the patients experiencing a transmural myocardial infarction. The reported incidence varies depending upon the definition of aneurysm utilized by the author. Different types of ventricular contractility alterations may result from myocardial infarction [1].

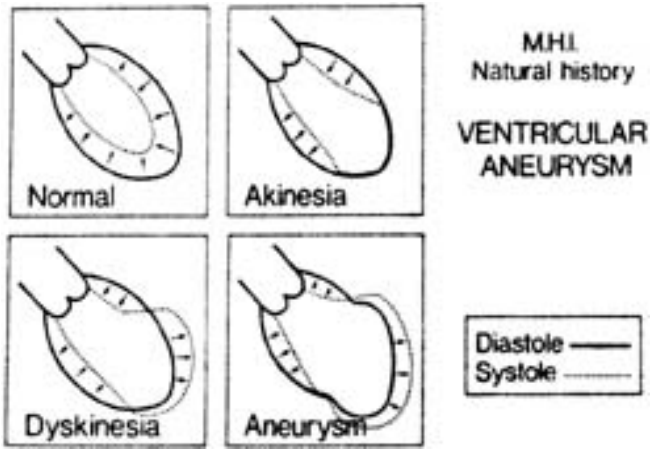


Figure 1: Natural history of Ventricular aneurysm [13]

The most frequent are akinesia (noncontractile area) and dyskinesia, defined as an area that does not contract but rather expands during systole. Although ischemic, traumatic or congenital aneurysms of the right ventricle do occur, these are most uncommon.

Although earlier autopsy series reported relatively poor survival in patients with medically managed left ventricular aneurysms (12 percent at 5 years), most recent studies report 5-year survival from 47 to 70 percent. Causes of death include arrhythmia in 44 percent, heart failure in 33 percent, recurrent myocardial infarction in 11 percent, and noncardiac causes in 22 percent [23].

Variable mortality rates are reported in clinical and necropsy series. Schlichter and colleagues reviewed 102 necropsy cases and observed that in 73%, the aneurysm had been present less than 3 years and in 88%, less than 5 years [57]. Proudfit, in 1978, studied a group of 74 patients with angiographically proven ventricular aneurysms and found a mortality rate 53% at 5 years and 88% at 10 years [56].

Bruschke and colleagues demonstrated different mortality rates in patients with left ventricular aneurysms and concomitant one, two, or three-vessel coronary artery disease [6]. Both the survival rate and the quality of life can be significantly affected by the complications of LV aneurysm: Cardiac insufficiency, arrhythmias, arterial embolization, and the occurrence of angina.

The excellent prognosis of asymptomatic patients with ventricular aneurysms who were treated medically was demonstrated in a series of 40 patients followed for a mean of 5 years. Of 18 initially asymptomatic patients, 6 developed class II symptoms while 12 remained asymptomatic. Ten-year survival was 90 percent for these patients but was only 46 percent at 10 years in patients who presented with symptoms [33].

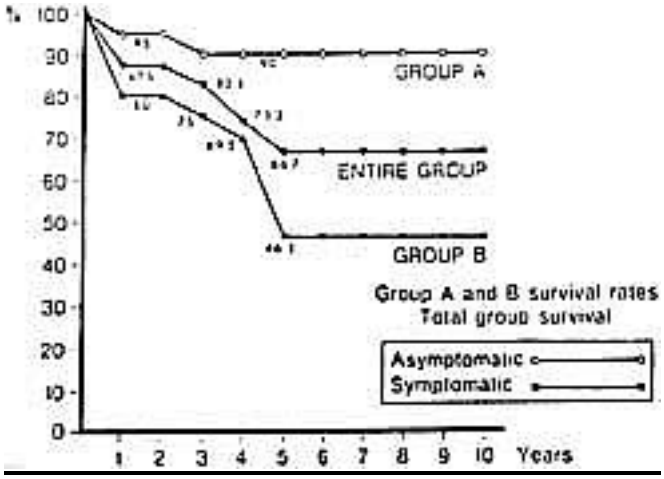


Figure2: **Survival in medically treated patients with left ventricular aneurysm based on presence (group B) or absence (group A) of symptoms [13].**

Meizlish and colleagues demonstrated that early formation of a functional aneurysm occurs frequently after anterior myocardial infarction and carries a high risk of death within one year that is independent of ejection fraction. In addition, the absence of a functional aneurysm identifies a large group with low one-year mortality despite a markedly impaired ejection fraction.

Patients undergoing cardiac catheterization in the CASS study, 7.6 percent had angiographic evidence of left ventricular aneurysms [50].

Factors that influence survival with medically managed left ventricular aneurysm include age, heart failure score, extent of coronary disease, duration of angina, prior infarction, mitral regurgitation, ventricular arrhythmias, aneurysm size, function of residual ventricle, and left ventricular end-diastolic pressure [33], [50]. Early development of aneurysm within 48 hours after infarction also diminishes survival [50].

The absolute incidence of left ventricular aneurysms may be declining due to the increased use of thrombolytics and revascularization after myocardial infarction [15], [10].

In general, the risk of thromboembolism is low for patients with aneurysms (0.35 percent per patient-year), and long-term anticoagulation is not usually recommended. However, in the 50 percent of patients with mural thrombus visible by echocardiography after myocardial infarction, 19 percent develop thromboembolism over a mean follow-up of 24 months. In these patients, anticoagulation and close echocardiographic follow-up may be indicated. Atrial fibrillation and large aneurysmal size are additional risk factors for thromboembolism [45].

Aetiology and risk factors:

Left ventricular aneurysm (LVA) is an important adverse development after acute myocardial infarction, being associated with an increased risk of thrombus formation [48] and sudden cardiac death (within an increased cardiac mortality [35]), independent of the left ventricular function.

There may also be an increased incidence of chronic heart failure [48]. It is also known that ejection fraction, the presence of sustained ventricular tachycardia and the presence of right coronary artery disease are independently predictive of death due to left ventricular aneurysm [48].

The literature contains conflicting reports of those factors that might most obviously affect left ventricular aneurysm formation, namely the patency of infarct-related artery [47, 37,66] and the extent of coronary collateral circulation [48, 3] . The role of an open infarct-related artery is probably favourable, as suggested from a number of papers.

In particular, in a substantial study by White et al. [65], where more than 300 patients were investigated one month after first acute myocardial infarction (AMI).

Among these patients, after multivariate analysis, ventricular function and patency of the infarct-related artery were independently predictive of survival during a 3 year follow-up period. Of interest, the beneficial effect of restoration of patency of the infarct-related artery was most when the ejection fraction was below 50% and when the infarct-related artery supplied more than 25% of the myocardium. In other words, patients with large anteroseptal infarcts are those most likely to benefit [65].

Intuitively, the influence of a well-developed coronary collateral circulation should be benign, reducing infarct size by preservation of the ischemic border zone between infarcted and non-infarcted myocardium and thereby also lowering the incidence of left ventricular aneurysm. However, the objective data are less clear cut.

Thus, Banerjee et al. [3], 8 years ago, reported that the collateral circulation had no demonstrable effect on the incidence of LVA, although the average infarct zone was said to be smaller [3]. Similar findings with respect to the collateral circulation were reported by Mariotti and colleagues [48].

Tikiz et al. describe a retrospective study of over 800 patients with left ventricular aneurysm after acute myocardial infarction [61]. The coronary anatomy of these patients and ventricular function were defined by angiography and ventriculography and the data compared to results from 435 other patients with anterior acute myocardial infarction, but in whom a left ventricular aneurysm did not develop.

The significant discriminators between the groups were: absence of previous angina, single vessel coronary disease, total occlusion of the infract-related artery and female gender in the LVA group compared to the control group.

In Tikiz et al.'s study [61], the status of the coronary collateral circulation and conventional risk factors for coronary artery disease were not significant discriminators.

It is well known that Left ventricular aneurysm is rare other than in the context of coronary artery disease and acute myocardial infarction [62].

Dilatation of the left ventricle after acute myocardial infarction is a two phase process, an early stage of infarction expansion, followed by a later phase of changes in the left ventricular shape, size and properties which constitute remodelling of the left ventricular myocardium remote from the site of infarction. In a sense, LVA formation can be considered to be an extreme manifestation of unfavourable LV remodelling. One might imagine that the main factors promoting LVA would be the same as those promoting left ventricular remodelling after infarction.

Over 95 percent of true left ventricular aneurysms reported in the English literature result from coronary artery disease and myocardial infarction. True left ventricular aneurysms also may result from trauma, Chagas' disease, or sarcoidosis. A very small number of congenital left ventricular aneurysms also have been reported and have been termed diverticula of the left ventricle [59].

False aneurysms of the left ventricle result most commonly from contained rupture of the ventricle 5 to 10 days after myocardial infarction and often occur after circumflex coronary arterial occlusion. False aneurysm of the left ventricle also may result from submitral rupture of the ventricular wall, a dramatic event that generally occurs after mitral valve replacement with resection of the mitral valve apparatus [59].

Pathological anatomy and Pathophysiology:

Post infarction aneurysm of the left ventricle is a consequence of the transmural fibrous scar that forms following ischemic myocardial damage. There are few or no residual muscle cells within the aneurysm wall. This may lead to left ventricular remodelling with global and regional LV dysfunction, ventricular arrythmias, or thromboembolic complications [1].

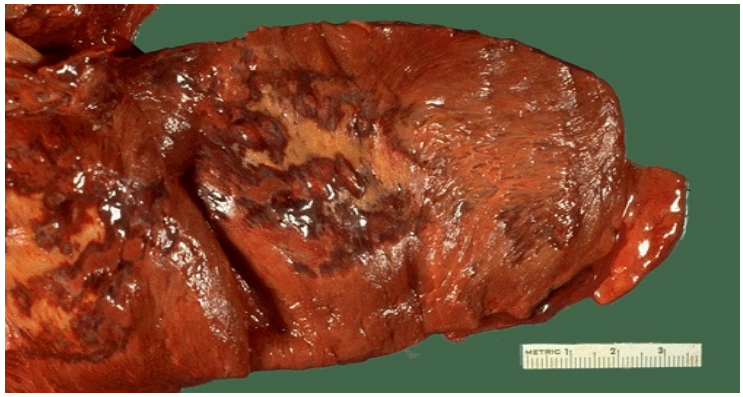


Figure 3: This is the left ventricular wall which has been sectioned lengthwise to reveal a large recent myocardial infarction. The centre of the infarct contains necrotic muscle that appears yellow-tan. Surrounding this is a zone of red hyperemia. Remaining viable myocardium is reddish brown
(Source: Internet Pathology Laboratory for Medical Education Florida State University College of Medicine)

For acutely ischemic myocardium, the pathophysiology of the global LV dysfunction has been linked to regional dysfunction in the border zone (BZ) region of normally perfused but poorly functioning myocardium. Some investigators have estimated increased wall stress in the border zone and attributed the associated global LV dysfunction to increased wall stress [1].

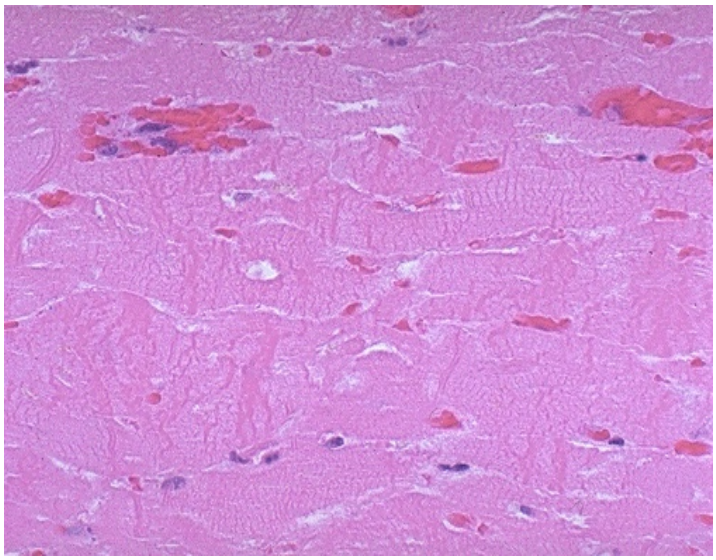
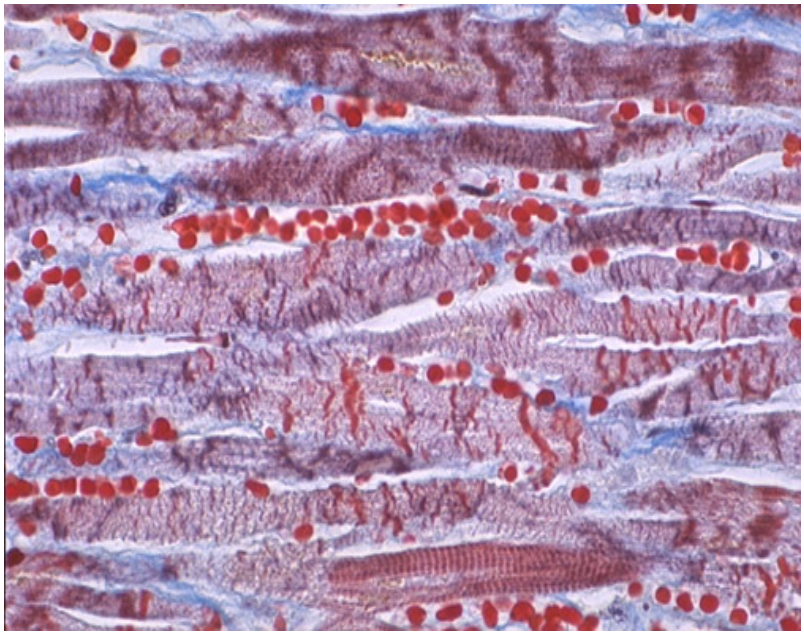


Figure 4: The earliest change histologically seen with acute myocardial infarction in the first day is contraction band necrosis. The myocardial fibers are beginning to lose cross striations and the nuclei are not clearly visible in most of the cells seen here. Note the many irregular darker pink wavy contraction bands extending across the fibers.
(Source: Internet Pathology Laboratory for Medical Education Florida State University College of Medicine)



Failure to normalize regionally increased wall stress results in progressive dilatation, recruitment of BZ myocardium into the scar, and deterioration in contractile function. Consequently, myocardial stress, pathologic cardiac remodelling, and LV decompensation are closely related [59].

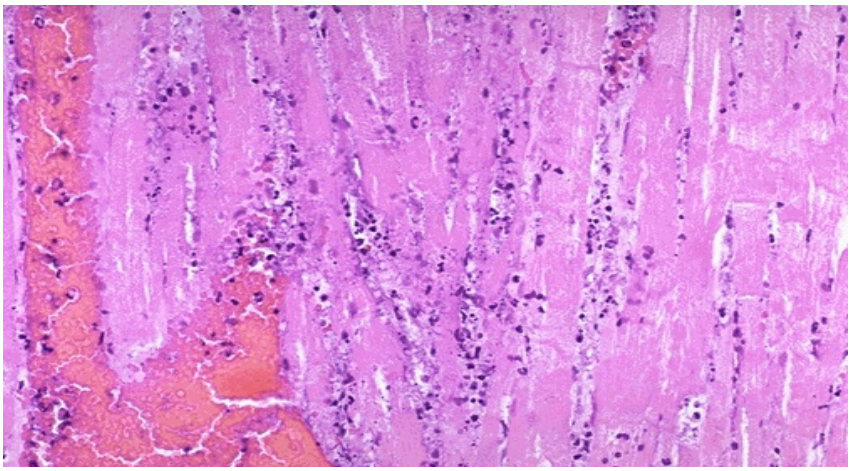


Figure 5: This myocardial infarction is about 3 to 4 days old. There is an extensive acute inflammatory cell infiltrate and the myocardial fibers are so necrotic that the outlines of them are only barely visible.
(Source: Internet Pathology Laboratory for Medical Education Florida State University College of Medicine)

Cardiac rupture usually occurs only during the acute phase of myocardial infarction. Ventricular rupture is, therefore, not a routine indication for surgery on a ventricular aneurysm, although it certainly can lead to pseudoaneurysm that requires treatment. Rupture of a mature ventricular aneurysm is a distinctly infrequent event [59].

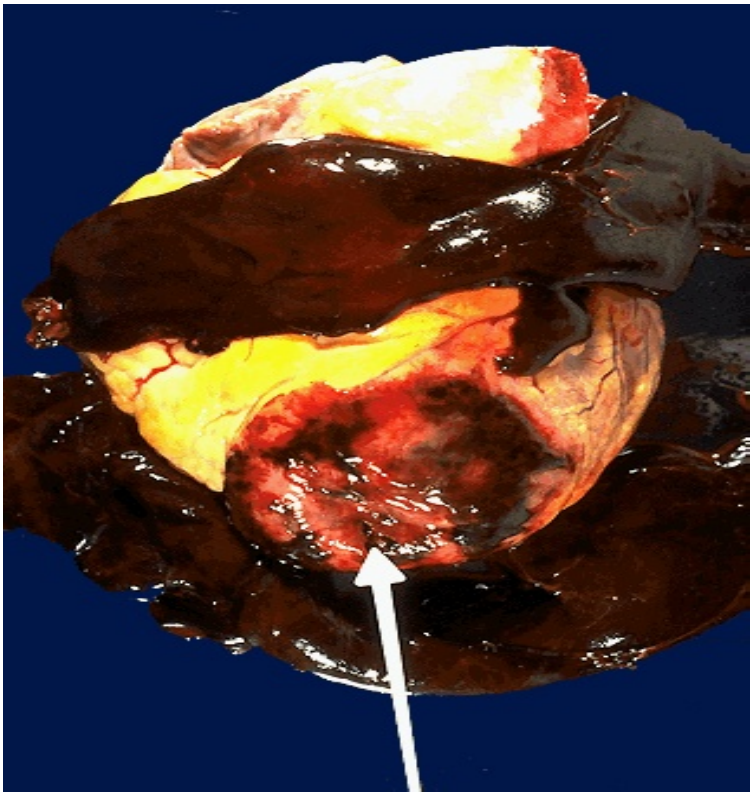


Figure 6: One complication of a transmural myocardial infarction is rupture of the myocardium.
(Source: Internet Pathology Laboratory for Medical Education Florida State University College of Medicine)

This is most likely to occur in the first week between 3 to 5 days following the initial event, when the myocardium is the softest. The white arrow marks the point of rupture in this anterior-inferior myocardial infarction of the left ventricular free wall and septum. Note the dark red blood clot forming the hemopericardium. The hemopericardium can lead to tamponade.

An accurate determination of regional ventricular wall stress has considerable potential to characterize and quantify the post infarction remodelling process and help to elucidate the mechanism underlying the global LV dysfunction associated with LVA.

Gorlin pointed out that when 20% of the ventricle surface is diseased, it leads to a progressive dilatation and impairment of the global ventricle. LV wall asynergy is large when more than 50% of the left ventricle circumference analyzed on an angiogram by centreline method is diseased [32].

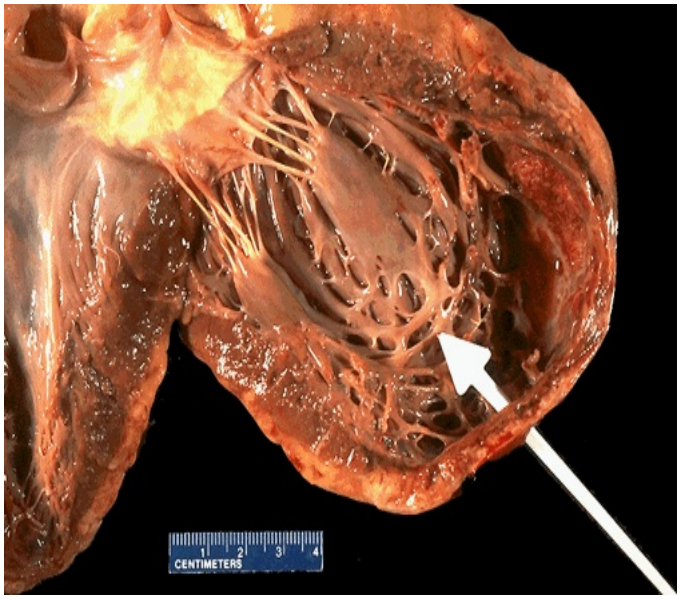


Figure 7: In cross section, the point of rupture of the myocardium is shown with the arrow. In this case, there was a previous myocardial infarction 3 weeks before, and another myocardial infarction occurred, rupturing through the already thin ventricular wall 3 days later.
(Source: Internet Pathology Laboratory for Medical Education Florida State University College of Medicine)

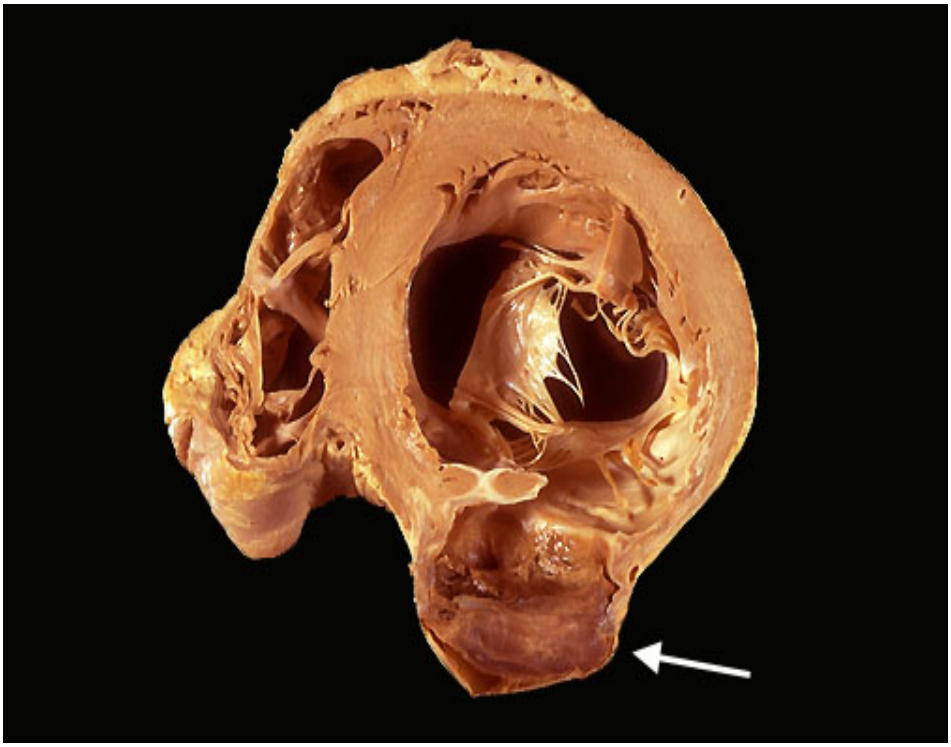


Figure 8: A cross section through the heart reveals a ventricular aneurysm with a very thin wall at the arrow. Note how the aneurysm bulges out. The stasis in this aneurysm allows mural thrombus, which is present here, to form within the aneurysm.
(Source: Internet Pathology Laboratory for Medical Education Florida State University College of Medicine)

Mills and his associates pointed out that the presence of a left ventricular aneurysm involving more than 25 % of the LV surface, burdens nonaneurysmal myocardial fibers, and ultimately decreases the ejection fraction of the noninfarcted area of the heart. Mills has emphasized that some authors have advised repair of left ventricular aneurysms only if medical measures fail. A more current understanding of the pathophysiology has led clinicians to advise repair before such irreversible changes secondary to progressive impairment of nonaneurysmal muscle by increased wall tension, if an acceptable mortality rate can be achieved by the surgical team [55].

The development of a true left ventricular aneurysm two principal involves phases:

Early Expansion Phase:

The early expansion phase begins with the onset of myocardial infarction. Ventriculography can demonstrate left ventricular aneurysm formation within 48 hours of infarction in 50 percent of patients who develop ventricular aneurysms. The remaining patients have evidence of aneurysm formation by 2 weeks after infarction [50].

True aneurysm of the left ventricle generally follows transmural myocardial infarction due to acute occlusion of the left anterior descending or dominant right coronary artery. Lack of angiographic collaterals is strongly associated with aneurysm formation in patients with acute myocardial infarction and left anterior descending artery occlusion, and absence of pre-formed collateral circulation is probably a prerequisite for formation of left ventricular aneurysm [29] .

At least 88 percent of ventricular aneurysms result from anterior infarction, while the remainder follow inferior infarction. Posterior infarctions that produce a distinct left ventricular aneurysm are relatively unusual [55].

In experimental transmural infarction without collateral circulation, myocyte death begins 20 minutes after coronary occlusion. Infarctions that result in aneurysm formation are almost always transmural and may show gross thinning of the infarct zone within hours of infarction. Within a few days, the endocardial surface of the developing aneurysm becomes smooth with loss of trabeculae and deposition of fibrin and thrombus on the endocardial surface in at least 50 percent of patients. While most myocytes within the infarct are necrotic, viable myocytes often remain within the infarct zone. In a minority of patients, extravascular haemorrhage occurs in the infarcted tissue and may further depress systolic and diastolic function of involved myocardium. Inflammatory cells migrate into the infarct zone by 2 to 3 days after infarction and contribute to lysis of necrotic myocytes by 5 to 10 days after infarction.

Electron microscopy demonstrates disruption of the native collagen network several days after infarction. Collagen disruption and myocyte necrosis produce a nadir of myocardial tensile strength between 5 and 10 days after infarction, when rupture of the myocardial wall is most common. Left ventricular rupture is relatively rare after the ventricular aneurysmal wall becomes replaced with fibrous tissue.

Loss of systolic contraction in the large infarcted zone and preserved contraction of surrounding myocardium cause systolic bulging and thinning of the infarct. By Laplace's law (T = Pr /2 h), at a constant ventricular pressure P , increased radius of curvature r and decreased wall thickness in the infarcted zone both contribute to increased muscle fibre tension T and further stretch the infarcted ventricular wall. relative to normal myocardium.

Ischemically injured or infarcted myocardium displays greater plasticity or creep, defined as deformation or stretch over time under a constant load [31].

Thus increased systolic and diastolic wall stress in the infarcted zone tends to produce progressive stretch of the infarcted myocardium (termed infarct expansion) [27] until healing reduces the plasticity of infarcted myocardium.

Transmural infarction without significant "hibernating" myocardium within the infarct region is necessary for subsequent development of a true left ventricular aneurysm. Angiographic ventricular aneurysms with evidence of hibernating myocardium (lack of Q waves or presence of uptake on technetium scan) may resolve over several weeks and thus do not represent true left ventricular aneurysms by strict criteria [36].

Due to increased diastolic stretch or preload and elevated catecholamines, remaining noninfarcted myocardium may demonstrate increased fiber shortening and, ultimately, myocardial hypertrophy in the presence of a left ventricular aneurysm. This increased shortening and increased wall stress increase oxygen demand for noninfarcted myocardium and for the left ventricle as a whole.

In addition to increased regional wall stresses, left ventricular aneurysm can increase ventricular oxygen demand and decrease net forward cardiac output by producing a ventricular volume load because a portion of the stroke volume goes into the aneurysm instead of out the aortic valve. Net mechanical efficiency of the left ventricle (external stroke work minus myocardial oxygen consumption) is decreased by reducing external stroke work (volume times pressure) and increasing myocardial oxygen consumption.

Left ventricular aneurysms can produce both systolic and diastolic ventricular dysfunction. Diastolic dysfunction results from increased stiffness of the distended and fibrotic aneurysmal wall, which impairs diastolic filling and increases left ventricular end-diastolic pressure.

Late Remodelling Phase:

The remodelling phase of ventricular aneurysm formation begins 2 to 4 weeks after infarction when highly vascularized granulation tissue appears. This granulation tissue is subsequently replaced by fibrous tissue 6 to 8 weeks after infarction. As myocytes are lost, ventricular wall thickness decreases as the myocardium becomes largely replaced by fibrous tissue. In larger infarcts, the thin scar is often lined with mural thrombus [49].

After acute myocardial infarction, animal studies show that ventricular load reduction with 8 weeks of nitrate therapy may reduce expected infarct thinning, decrease infarct stretch, and lessen hypertrophy of noninfarcted myocardium. Interestingly, nitrate therapy for only 2 weeks after infarction does not prevent aneurysm formation [39].

This observation emphasizes the importance of late remodelling from 2 to 8 weeks after infarction. Angiotensin converting-enzyme (ACE) inhibitors also reduce infarct expansion and subsequent development of ventricular aneurysm. Because animal studies show that ACE inhibitors non-specifically suppress ventricular hypertrophy, it is not clear whether suppression of the compensatory hypertrophy of surrounding myocardium is ultimately beneficial or harmful.

Lack of coronary reperfusion is probably prerequisite for development of left ventricular aneurysm. In humans, reperfusion of the infarct vessel spontaneously, [36] by thrombolysis, [41] or by angioplasty [9] has been associated with a lower incidence of aneurysm formation. It is speculated that coronary reperfusion as late as 2 weeks after infarction prevents aneurysm formation by improving blood flow and fibroblast migration into the infarcted myocardium. The role of delayed infarct healing in aneurysm development is supported by observations that steroids after myocardial infarction may increase the likelihood of aneurysm formation [7]. Arrhythmias such as ventricular tachycardia may occur at any time during the development of ventricular aneurysm, and all these patients have the substrate for re-entrant conduction pathways within the heterogeneous ventricular myocardium. These pathways tend to involve border zones surrounding the ventricular aneurysm.

Reperfusion produces epicardial and, occasionally, mid-myocardial sparing while leaving endocardial necrosis. Persistence of viable ventricular muscle frequently trades left ventricular dyssynergy for asynergy, with some preservation of wall thickness.

When one third or more of the ventricular perimeter is involved, left ventricular volume markedly increases, the apical and basal portions become rounded, and pump function is globally depressed.

This condition resembles dilated nonischemic cardiomyopathy more than classic dyskinetic aneurysm.

Extension of modified aneurysm surgery to large dilated ventricles has been made possible by the Dor procedure [24, 25], Di Donato has demonstrated that outcome in a large series of surgically treated patients is more strongly linked to the extent of asynergy than to the type of asynergy (akinetic vs. dyskinetic). This leads to the following principles

<u>Relieve ischemia</u>

Coronary revascularization should be as complete as possible [62]. Grafting the left anterior descending coronary artery (LAD) is important since the high portion of the septum, which is almost always functioning, needs to be perfused. In their early experience 70 patients underwent pre-coronary and post-coronary angiography. All patients had a critically stenosed LAD, and in 35 of them the LAD, distal to the lesion, was not visible at angiography. All had left internal thoracic artery grafts and 27 of 35 patients had a patent graft with a 3-month patency of 77% [53].

Diminish ventricular volume

Ventricular volume should be reduced in its septal and anterior components without deforming the chamber [62]. If the residual volume is too small, the results will be catastrophic, resulting in the physiology of a restrictive cardiomyopathy. This risk is particularly great if the preoperative chamber is only moderately dilated. If the residual chamber is too large, the benefit will be limited.

To diminish this risk, Dor introduced the use of an intraventricular balloon filled to a known volume of 60 mL/m^2, to guide the restoration and to leave an adequate residual chamber. The volume 60 mL/m^2 was chosen after study of postoperative angiograms. This value may be too small if the preoperative volume is very large; thus, when they approach preoperative volumes greater than 150 mL/m^2, they add 15% to the volume of the balloon (approximately 70 mL/m^2) [52].

Reduction of ventricular volume has two important effects. First, based on the Laplace equation, which relates wall stress inversely to wall thickness and directly to chamber radius, volume reduction diminishes wall stress and thereby reduces myocardial oxygen consumption. Minimizing the mass of abnormal myocardium improves wall compliance, reduces filling pressure, and further enhances diastolic coronary flow. Second, reduction of wall stress, as a critical determinant of afterload, enhances contractile performance of the ventricle by increasing the extent and velocity of systolic fiber shortening [19].

Restore shape

The Dor's procedure was initially perceived as functional amputation of the ventricle with exclusion of the entire akinetic or dyskinetic scar. This led to increased sphericity of the ventricle in some patients, but in general the volume reduction still improved function. However, a suboptimal short axis/long axis ratio may influence the development of late moderate mitral regurgitation [16].

The objective in optimizing the shape of the ventricle should be the proportional reduction of both the short and the long axes. There are limits to the extent to which the short axis can be reduced. Circumferential shortening is maintained by the upper part of the septum and by the inferior or lateral wall, which are often sound. Their motion is crucial for a good outcome. It is therefore necessary to find equilibrium between the exclusion of akinetic or dyskinetic regions and the reduction of the longitudinal axis that is determined by the position of the new apex. To overcome this problem, great attention should be paid to positioning the patch with an oblique orientation, toward the aortic outflow tract. In this way you can avoid creating a boxlike shape of the ventricle that may occur when the orientation of the patch is almost parallel to the mitral valve [52].

In a normal heart, myocardial fibers have a spiral direction from the base to the apex with two opposite layers and well-defined intersecting angles.

This double spiral allows 30% fiber shortening to yield a 60% ejection fraction [52].

When the heart dilates and this spiral architecture is lost (especially at the apex), ventricular function is impaired and ejection fraction and stroke volume decrease. This starts a vicious circle, with dilatation of the ventricle and activation of peripheral and central neurohumoral mechanisms that characterize left ventricular remodelling and lead to clinical heart failure [52].

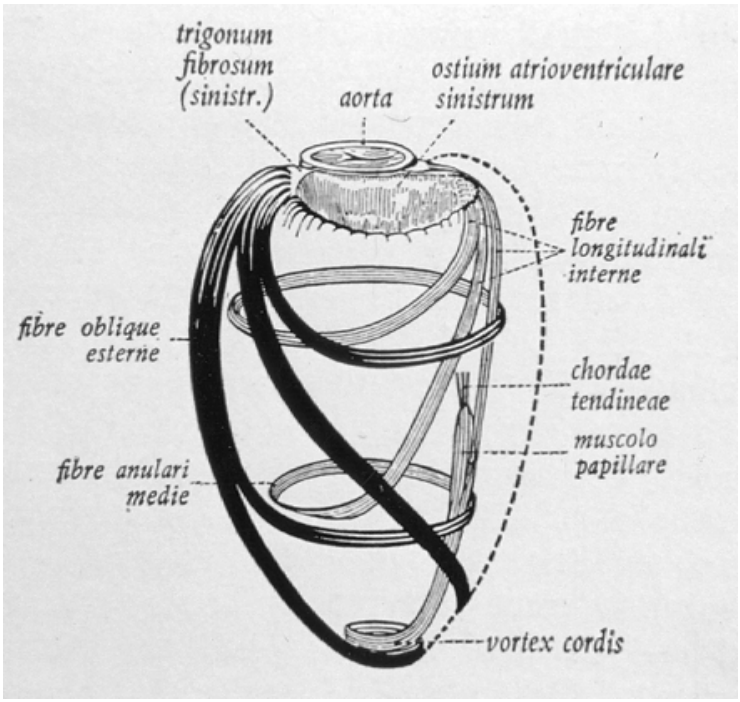


Figure 9: Drawing showing that the myocardial fibers of the normal heart have a spiral direction from the base to the apex with two opposite layers and well-defined intersecting angles *(From Benninghoff-Goertler, Atlas of anatomy, Vol II, 1996, Piccin Editor [52]).*

If you compare two different architectural arches, one with an acute and the other with a spherical form (ie, the Gothic and the Roman arch) and you apply an identical force at the vertex, this force is split into two components: one longitudinal and one lateral force. For a given longitudinal component the lateral force will be higher in the spherical arch; thus, if we consider the apex of the heart as analogous to the vertex of the arch, a rounded, spherical apex will have a higher lateral force (stress) than an elliptical ventricle [52].

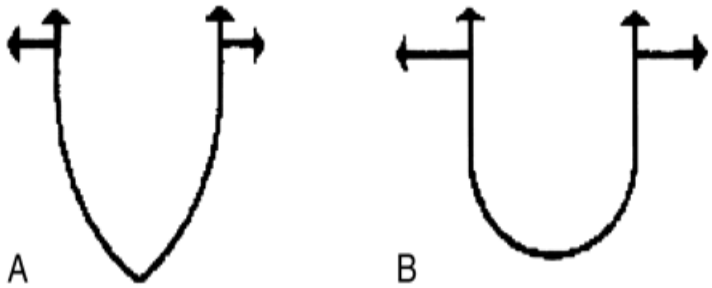


Figure 10: The Gothic (A) and Roman (B) arches. Arrows indicate longitudinal and lateral forces. See text for explanation [52].

The shape of the ventricle is also important for normal functioning of the mitral valve. Ischemic functional mitral regurgitation is more frequent in dilated, spherical than elliptical ventricles. In a spherical heart, papillary muscles are displaced together with the lateral wall, losing their normal orientation toward the apex and increasing the distance between them.

In this condition the posterior leaflet of the valve is retracted, the posterior annulus is dilated, the cooptation point is lost, and the valve becomes incompetent [52].

Repair mitral regurgitation

if mitral regurgitation is present, repair is performed from the ventricle if mitral regurgitation is greater than 2+ or if the anulus is larger than 35 mm. The advantages of repairing the valve from the ventricle are that the trigones are better visualized and the procedure is faster [51].

Following infarction, the normally smooth endocardial surface is transformed into a damaged, inflamed surface that promotes platelet adherence and aggregation. Contractility changes in the myocardium as well as geometric changes in the left ventricular configuration may result in relative stasis of the blood. These two factors often lead to development of thrombus adherent to the rough endocardial surface. Although, in the majority of cases this progresses harmlessly toward thrombus organisation, portions of the thrombus can break off and embolize into the systemic, mesenteric, or cerebral circulations, often resulting in dire clinical consequences. The incidence of this occurrence is exceedingly low [2]

Clinical features and Diagnosis:

The most frequent clinical manifestation of LV aneurysms is congestive heart failure. Angina pectoris is also a frequent symptom, occurring in 44% to 98% of patients with LV aneurysm due in most cases to concomitant obstructive lesions in noninfarct-related arteries [8]. Significant ventricular arrhythmias appear less frequently, more often when the septum is involved, occurring in about 20% of large aneurysms and 3% of smaller aneurysms. Small and moderate sized aneurysms often have no specific associated symptoms.

On physical examination, signs of congestive heart failure may be present. In cases of anterior wall aneurysms, diffuse apical systolic thrust and double impulse are frequently present. On auscultation, heart murmurs are seldom heard except when associated mitral regurgitation is present.

Persistent ST segment elevation and T-wave changes are electrographic signs suggestive of ventricular aneurysm.

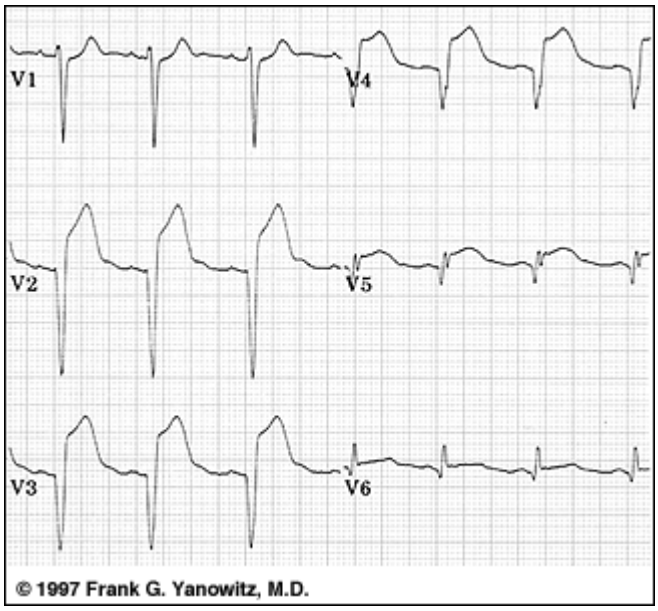


Figure 11: Persistent ST elevation after acute MI suggests ventricular aneurysm

In patients with left ventricular aneurysm, persistent or 'Freezing' ST-segment elevation appears clearly independent of ischemia; however, it did not make sense that ST elevation, which is experimentally a current of injury, would come from scar tissue in the absence of injured cell. If we reconsider the mechanism of ST segment elevation, while a strict relation was demonstrated between ST-segment elevation and an increase in extra cellular potassium due to the opening of K ATP channels, the link between a stress induced ST-segment shift and ischemia is lower, because non-ischemic mechanisms, i.e. mechanical trauma, can also open K ATP channels.

It was hypothesized that an abnormal stretch on the tissue adjacent to the aneurysm may also alter cellular activity to generate injury currents and the triggering of so-called stretch-activated channels, caused by the outward bulging of the aneurysm, could induce a diminution in magnitude of the action potential plateau and consequently, ST-segment elevation.

Fluoroscopy and chest roentgenograms may demonstrate bulging of the heart contour.

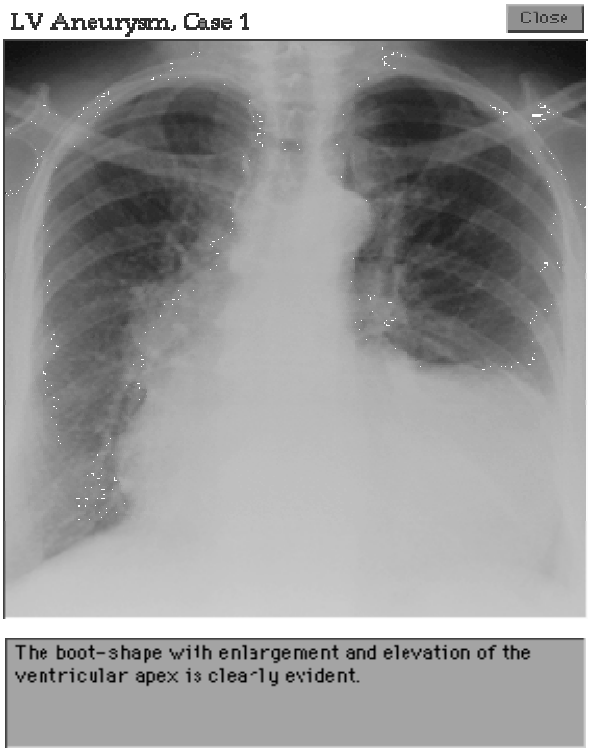


Figure 12: The boot shape with enlargement and elevation of the ventricular apex is clearly evident.
(Source: Yale University School of Medicine, Cardiothoracic Imaging)

Echocardiography provides a non-invasive and precise evaluation of the ventricular shape and, to some extent, function. Two dimensional echocardiography may allow identification of the aneurysm sac and can often detect the presence of mural thrombosis on the endocardial surface.

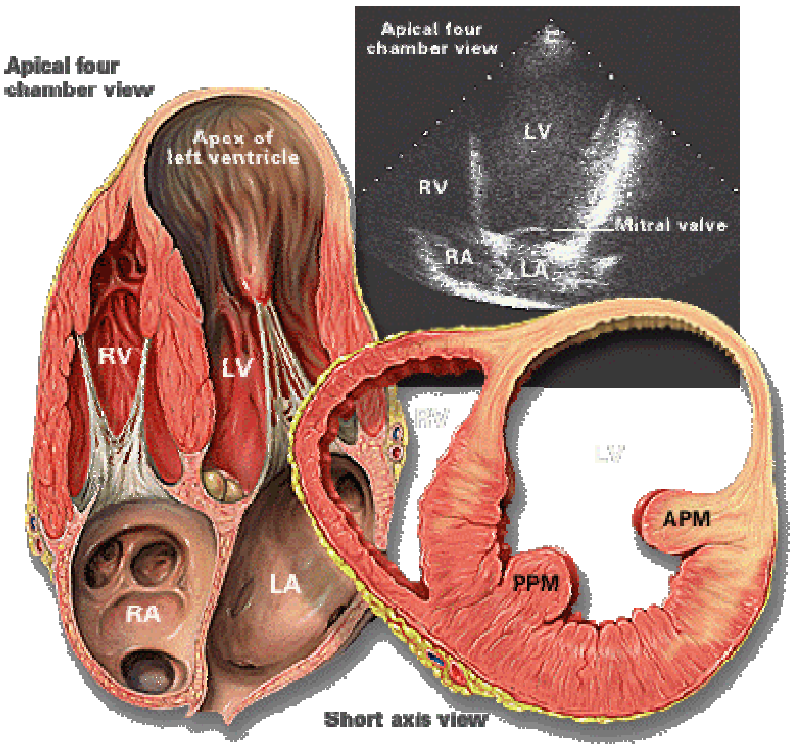


Figure 13: Two dimensional echocardiography may allow identification of an aneurysm.
(Source: Yale University School of Medicine, Cardiothoracic Imaging)

Aneurysms usually result from large areas of fibrotic myocardium scarred by ischemic infarction.

A common area for aneurysm includes the antero-apical segments which create a distortion of the ventricular shape both in systole and diastole and prevent effective ventricular pumping since their wall motion is often paradoxic. Postero-basal aneurysms also occur and are well defined by the long axis view.

The availability of intraoperative transoesophageal Doppler echocardiography may allow even more precise anatomic and functional evaluations to be made. Also the presence of mural thrombus and degree of mitral insufficiency can be accurately determined.

Global left ventricular function can be noninvasively evaluated using the gated blood pool scintigraphy. This scan is highly accurate for the identification of aneurysms in the anterior and apical area but somewhat less sensitive in the posterobasal area. Because it is safe, accurate, and non-invasive technique for identifying ventricular aneurysms and evaluating function, it is useful as a screening test. However, because it does not delineate the coronary artery anatomy, it does not replace contrast Ventriculography.

Because of the sharp contrast between flowing blood and static tissues in an MRI scan, this modality is particularly well-suited to delineate the anatomy of an aneurysmal sac. However, it is an expensive and time consuming test to perform, which gives little or no information as to cardiac function or coronary anatomy and, therefore, has no real role in the diagnosis of left ventricular aneurysms [2].

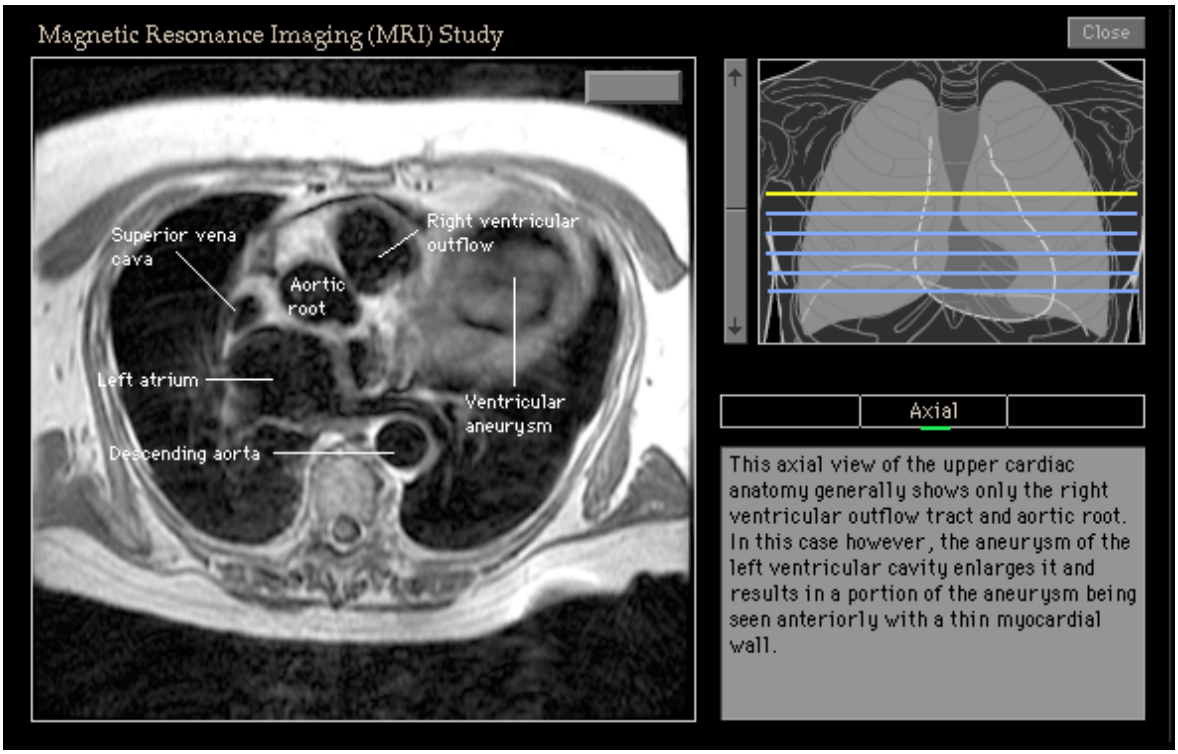


Figure 14: Magnetic Resonance Imaging (MRI) Study.
(Source: Yale University School of Medicine, Cardiothoracic Imaging)

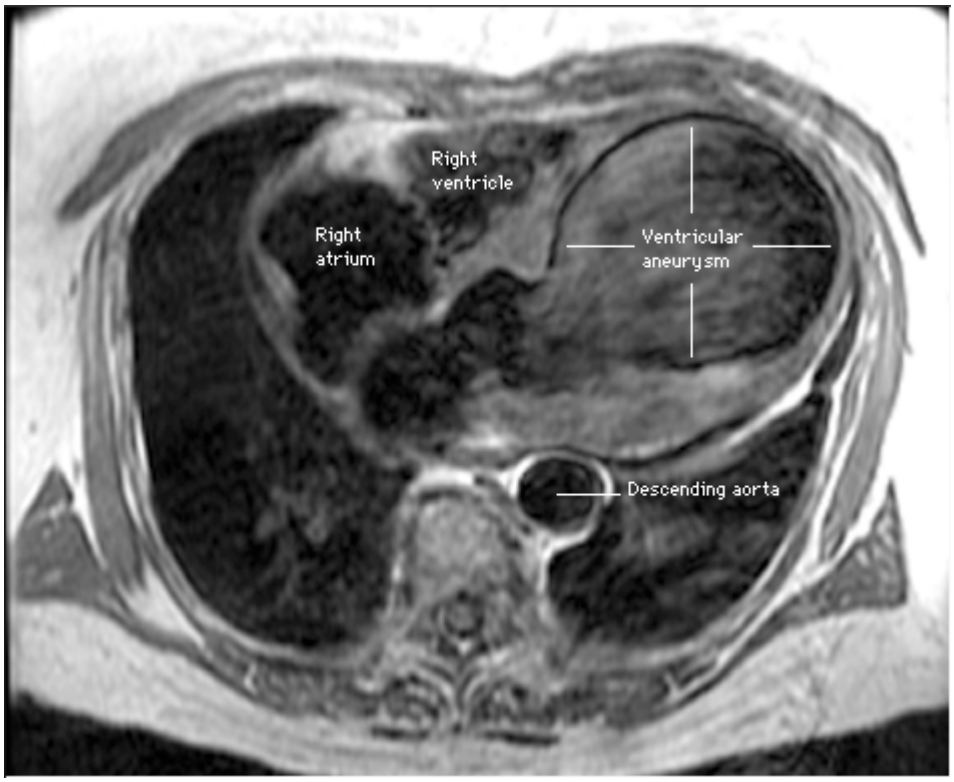


Figure 15: This radiograph was obtained from a 70 y.o. previously well black female who presented in congestive heart failure and was diagnosed as having a massive aneurysm of the left ventricle.
(Source: Yale University School of Medicine, Cardiothoracic Imaging)

Computed tomography scans and magnetic resonance imaging of the chest are useful only in delineating anatomy.

Contrast Ventriculography remains the single most useful test in the workup of left ventricular aneurysm. It defines the cardiac function and coronary anatomy. These are critical determinants to make, since it is on the basis of these findings the surgeon must decide whether or not to operate and what procedure should be undertaken. If one looks only at the standard ventriculogram, it is possible to misinterpret how much muscle has been destroyed and replaced by fibrosis, especially if the aneurysm is a large one [2].

It is important that Ventriculography be undertaken from several views lest superimposition of the fibrosis and normal muscle lead to an inaccurate assessment. If misinterpretation occurs, patients eligible for aneurysmectomy may be inappropriately rejected or referred for heart transplantation. The motion of the coronary arteries supplying myocardium outside the infarcted area is particularly important to determine. The shape and movement of these arteries during cardiac cycle are different from those in the hypokinetic or the non-involved area. It is possible to make a reasonable distinction between distension and muscular destruction by assessing the motion of these arteries. When one looks at late results following surgery, the amount of preserved muscle and the technique of reconstruction are the main two contributing factors to beneficial outcome [2].

Naturally it is extremely important that the anatomy of coronary arteries and the sites of obstruction be identified so that concomitant revascularisation can be undertaken at the time of aneurysmectomy.

Indications and Contraindications for surgery:

The majority of patients with symptomatic or asymptomatic moderate to large size aneurysms should be treated surgically, particularly if they have associated coronary artery disease. If these patients are managed medically, it is possible that over time the contractile ejection fraction decreases, thus increasing the subsequent operative risk. As pointed out by Dor and his associates, patients can be treated surgically even if the ejection fraction is between 25 and 30%, provided that the mean pulmonary pressure is less than 40 mmHg and the cardiac index greater than or equal to 2.1L/min per square meter [24]. However, in selected patients transplantation should be performed as a treatment of choice when contractile ejection fraction less than 25% and when there is right ventricular dysfunction, permanent mitral insufficiency, and poor target coronary arteries for bypass [4].

Di Donato and associates [52] mentioned that according to their experience they consider the following to be the indications for surgery. In patients of any age:

- Anteroseptal infarction and dilated left ventricle (end-diastolic volume index > 100 mL/m^2), and
- Depressed ejection fraction (even below 20%), and
- Left ventricular regional asynergy, either dyskinesia or akinesia, greater than 35% of the ventricular perimeter, and
- Symptoms of angina, heart failure, arrhythmias, or a combination of the three, or
- Inducible ischemia on provocative tests in asymptomatic patients.

If patients are asymptomatic and results of provocative tests are negative, they suggest monitoring them by an echocardiographic study every 6 months. If the ventricle tends to dilate and ejection fraction tends to decline, these patients should be offered the procedure. Intervention may limit the progressive deterioration in clinical status.

Di Donato, also proposed the following criterias as relative contraindications for surgical intervention:

- *Systolic pulmonary artery pressure more than 60 mm Hg (when not associated with severe mitral regurgitation).*
- *Severe right ventricular dysfunction as assessed by TAPSE (tricuspid annulus plane systolic excursion). This is a simple index of function, and when it is less than 10 mm the intervention is contraindicated because failure involves largely the septum and both ventricles. In this condition left ventricular repair is not effective and mortality is high.*
- *Regional asynergy without dilation of the ventricle (risk of too small a residual chamber) [52].*

Historical development and current standard of surgery:

Left ventricular aneurysm resection is one of the oldest and simplest techniques that can be seen in all cardiac surgery departments and is illustrated in plenty of books.

It consists of removing the maximum of fibrous scarred tissue and of closing the left ventricle hole by a long linear suture with or without felt reinforcernent.

History Of left ventricular resection for LV. Aneurysms		
1944	*Beck*	*Fascia lata reinforcement*
1955	*Likoff-Bailey*	*1st closed resection*
1958	*Cooley*	*1st open resection*
1973	*Stoney*	*„ In coat „ plicature*
1977	*Dagget*	*Posterior patch*
1979	*Levitsky*	*Anterior patch*
1980	*Hutchkins*	*Influence of cardiac geometry*
1984	*Jatene*	*Circular reduction*
1985	*Dor*	*Endoventricular patch plasty*
1989	*Cooley*	*endoaneurysmorrhaphy*

Table 1: Trend towards LV Reconstruction *[22]*

As far as the technique itself is concerned, the general review of Mills [55] enables to make a clearer distinction between the various operations. There are roughly three types of surgeries:

a) The LINEAR SUTURE, quite widely used during 40 years, has its advantages: easy and quick techniques. But has also its disadvantages: only exteriorized scar is resected and LV cavity is distorted by the linear suture and by the non exclusion of the septal scar. This technique has still its supporters such as Elefteriades [28] and Kesler [42].

But the series of these authors, even comparative, are poorly significant; the series are short, based on inhomogeneous cases and with quite limited parameters of post-operative control [63]. The results published since 1958 after the first description of linear resection by Cooley [12] were so much questionable as far as ventricular morphology was concerned, hemodynamic improvement and benefit in terms of survival, that they lead the cardiologists to limit the indications of ventricular aneurysms to only very severe cases.

b) The CIRCULAR EXTERNAL SUTURE of the ventriculotomy, at the limit of the incision was described by Jatene in 1984 [38]. It consists in sewing a suture at the edge of the resection in order to restore the curvature of the wall, as it was before the infarct, the opening itself being closed by U stitches rested on Teflon and joint together in a linear way. A patch was only used in 10 to 15% of cases when the opening was more than 3 cm in diameter in order to ensure the tightness of this suture.

Advantages: it restores a more normal left ventricular shape. Disadvantages: it does not allow revascularization of the diseased Left Anterior Descending artery (LAD), the intraventricular sulcus being involved by the stitches. This technique also does not allow repairing or excluding the septal endoventricular part of the fibrous scar, which is therefore left or plicatured or lined with a patch and remains therefore in the ventricular neo cavity. More recently, Mickleborough [54] published an important series with good results, a similar method, but only in restructuring in a partially circular way the vertical opening by sutures passed more widely in the muscle than in the Teflon strip which is smaller, this leading to an oval reduction of the opening. These Jatene type techniques bring a definite improvement in the real forms of dyskinetic aneurysms, mainly with apical localization, but have limited advantages in more diffuse forms of ischemic scars and mainly septal.

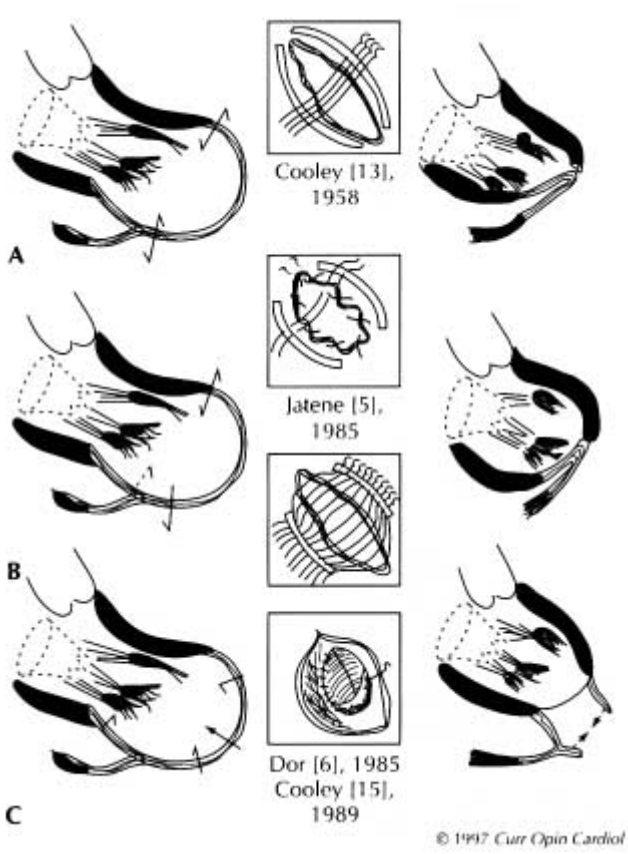


Figure 16: Types of surgery for repair of the left ventricular wall. A, Linear suture. B, Circular external suture. C, Endoventricular patch plasty *[31].*

c) the ENDOVENTRICULAR PATCH PLASTY, as it has been described since 1985 by Dor [21], has been named "endoaneurysmorrhaphy" by Cooley [11] and consists in suturing inside the ventricle a patch within the limits of the fibrous scar, this having the advantage of both reorganizing in a circular way the sound muscle, as in the Jatene technique, but excluding the septal areas.

This technique which is simple and logical, has been progressively adopted by most of the surgical teams and named, depending on the publications, "endoventricular circular patch plasty", "endoventricular remodelling" (Grossi), endoaneurysmorrhaphy", (Shapira), "Intracavitary repair" (Cooley), "endoventriculoplasty" (Salati) and was very often just named "Jatene" by many groups, even if the endoventricular patch repair has not been described by this author [63].

Apart from being simple, this method has the advantage of being easily reproduced, restoring the ventricular morphology, improving the ejection fraction, improving the late results and allowing a complete coronary revascularization, including the LAD territory.

During the procedure, if ventricular arrhythmia, spontaneous or inducible, is present, a subtotal endocardectomy, with or without mapping, is easy to accomplish, with, in the postoperative period, absence of inducible ventricular tachycardia in nearly 90% of cases, as it was shown by Mickleborough, Grossi, Jakob and Dor. The theoretical disadvantage is the use of a synthetic tissue inside the left ventricular cavity but this tissue can be covered by pericardium or replaced by autologous tissue, such as pericardium or a piece of endocardial scar, and the experience shows that this tissue gets very quickly covered by a neoendothelium.

Material and Methods:

Patient characteristics and preoperative data:

From May 1974 through December 2000, 305 consecutive patients underwent surgical repair of post infarction dyskinetic left ventricular aneurysm at the German Heart Centre in Munich. In all patients dyskinetic LVA was confirmed at operation.

In 200 (66 %) patients linear resection with standard closure, as described by Cooley and coworkers, was performed [group L] [54]. 105 (34 %) patients underwent aneurysmectomy with endoventricular patch repair according to the technique described by Dor and associates [9] [group D]. Linear closure was performed since 1974; from 1985 the Dor-technique was applied as an alternative procedure.

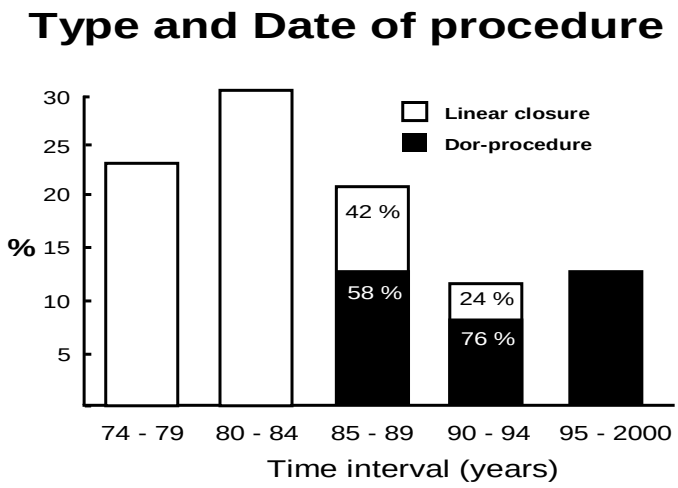


Figure 17: Type & Date of Procedure

All patients had preoperative coronary angiography and left ventriculography. Clinical symptoms, hemodynamic data, functional outcome and survival rate were analyzed from medical records, patient's follow-up visits, and communications from the referring physicians. In 74 (24 %) patients, cardiac performance was assessed postoperatively by ventriculography. Congestive heart failure (CHF) was defined according to New York Heart Association (NYHA) functional class. The average age of the patients was 57 ± 9.6 years (range 26.3 to 79.7 years). 126 patients (41 %) were older than 60 years. Mean age in group L was 54.5 ± 8.6 years compared to 62 ± 9.6 years in group D (p = 0.001). 253 (83 %) were men and 52 (17 %) women [5:1]. The percentage of female patients was significantly higher in group D (24%) compared to group L (13 %) [p = 0.023]. 81.5 % of the patients were in New York Heart Association functional class III or IV. Diabetes mellitus was present in 21 % of the patients, lipid abnormalities in 57 %,
hypertension in 45 % and a history of smoking in 43 %. Preoperative clinical and hemodynamic data of the study groups are summarized in Table 2.

Table 2. Preoperative clinical and hemodynamic data in 305 patients with LV-aneurysm

	All patients (n = 305)	Group L (n = 200)	Group D (n = 105)	p-value
Age(mean ± SD)[years]	57 ± 9.7	55 ± 8.6	62 ± 9	0.001
Sex (w / m) (%)	17 / 83	13 / 87	24 / 76	0.023
Delay from MI [years]	3.1 ± 4.8	2.3 ± 3.1	3.9 ± 5.9	
NYHA functional class [%]				NS
Class III	54	58	49	
Class IV	28	28	27	
Site of infarction [%] (anterior / posterior)	88 / 8	91 / 6	82 / 12	NS
Coronary artery disease [%]				NS
Left main stenosis	6	5	8.0	
Triple vessel	31	31	33	
Double vessel	39	41	35	
Single vessel	24	23	25	
EF (mean ± SD) [%]	34 ± 12	35 ± 14	33 ± 11	NS
LVEDP (mean ± SD) [mmHg]	20 ± 10	20 ± 11	20 ± 9	NS

MI = myocardial infarction; NYHA = New York Heart Association; EF = ejection fraction; LVEDP = Left ventricular enddiastolic pressure

Single indications for operation were angina in 33 %, congestive heart failure in 32.5 %, and severe rhythm disturbances in 8 %. A combination of angina, congestive heart failure and VT constituted the indication in the remaining patients. 38 patients (12 %) had sustained ventricular tachycardia or fibrillation preoperatively. 13 patients (4.3 %) had undergone cardio-pulmonary resuscitation.

The average interval between preoperative infarction and operation was 3.1 ± 4.8 years.

The site of the aneurysm was anteroapical in 88 % of the patients and posterior in 7.8 %.

6 % of the patients presented with left main disease, 24 % had single-vessel disease; 39 % had two-vessel, and 31 % three-vessel disease.

Operative Technique

Aneurysmectomy, as classically described, is a resection of the fibrotic area leaving a 1 to 2 cm border of fibrous tissue. The closure of the ventricular chamber is then accomplished utilizing interrupted mattress sutures tied over Teflon felt strips.

All operations were performed on cardiopulmonary bypass and moderate systemic hypothermia (26 to 32 °C). Cold crystalloid cardioplegia (Bretschneider solution) was used in 106 patients (35 %) [5]. The remaining patients were operated with the heart arrested by hypothermic fibrillation without aortic occlusion. In cases with concomitant bypass grafting, the distal anastomoses were performed first. Then the left ventricular aneurysm was opened and resection of the endocardial scar was performed.

In cases of linear closure, after removing all mural thrombus, the aneurysmal wall is trimmed, leaving a 3-cm rim of scar to allow reconstruction of the normal left ventricular contour. Care is taken not to resects too much aneurysmal wall and overly reduce ventricular cavity size. Anterior aneurysm defects are closed vertically between two external 1.5-cm strips of Teflon felt, two layers of 0 monofilament horizontal mattress sutures, and finally, two layers of running 2-0 monofilament vertical sutures with large-diameter needles.

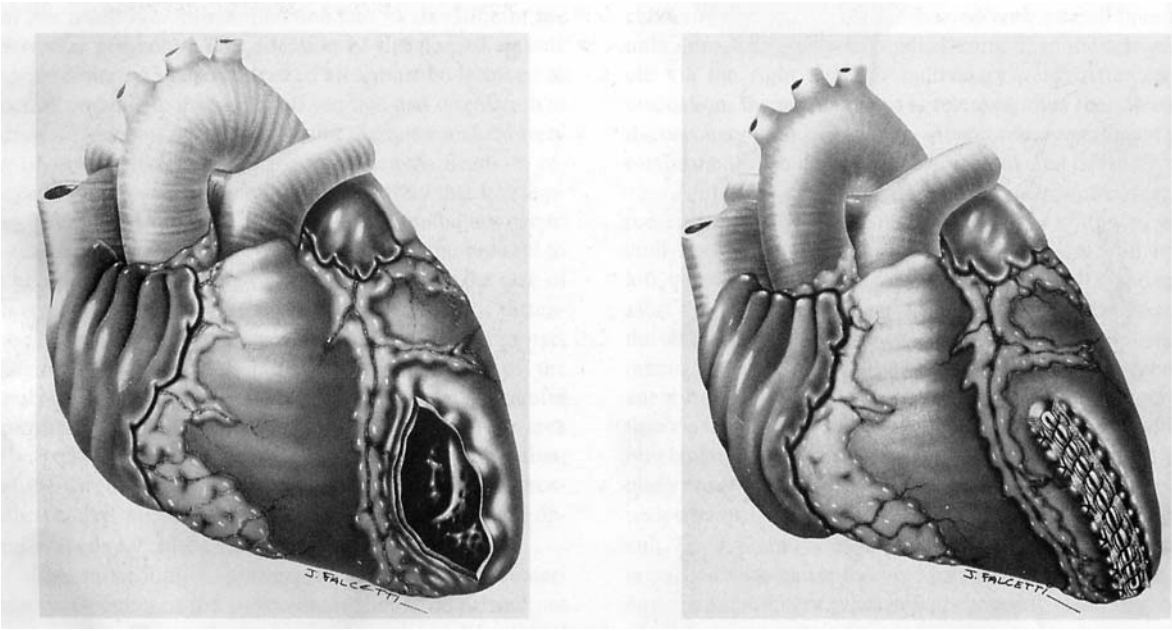


Figure 18: Linear repair of LV aneurysm with longitudinal distortion of normal muscle orientation *[2].*

In the cases where the Dor-technique was applied, after debridement of thrombus, a Teflon felt patch is cut to size sufficient to restore normal ventricular size and geometry when secured to the aneurysmal rim. The patch (sometimes covered with autologous pericardium) is sutured to normal muscle at the aneurysmal circumference using a running 3-0 polypropylene suture that is secured with single sutures at three or four places around the patch circumference.

Interrupted 3-0 sutures are placed as needed to ensure good fit. Care is taken not to distort the papillary muscles. The aneurysmal rim is trimmed to allow primary closure of the native aneurysmal wall over the patch using two layers of running 2-0 monofilament suture without pledgets.

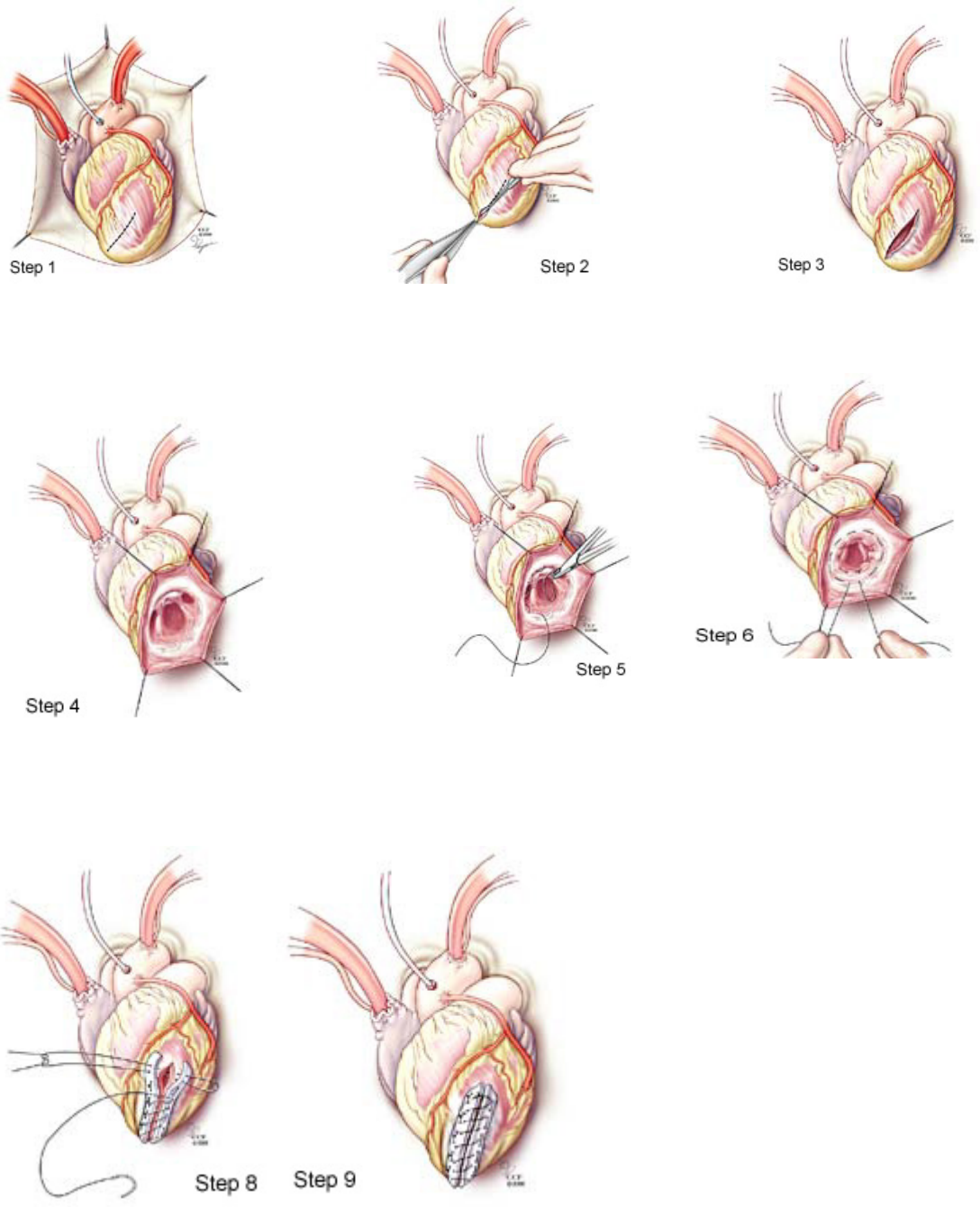


Figure 19: Endoventricular Patch Repair

As compared with linear and circular patch techniques, the endoventricular patch technique has technical advantages. An endoventricular patch preserves the left anterior descending artery for possible grafting and leaves no external prosthetic material to produce heavy pericardial adhesions. The technique facilitates patching the interventricular septum, and is suitable for acute infarctions when tissues are friable.

Associated procedures included intraoperative electrophysiological studies and cryotherapy in six patients who had documented preoperative ventricular tachycardia or fibrillation, ventricular septal defect closure in 11 patients, mitral valve replacement in 6, mitral valve repair in two and aortic valve replacement in three patients. In 63 patients a mural thrombus attached to the endocardial surface was removed with the resection of the aneurysm.

207 patients (68 %) underwent associated coronary grafting with a mean number of 1.5 ± 1.4 bypass grafts. The average number of grafts did not differ between both patient-groups. Associated CABG was performed in 65 % of the patients in group L and 73 % of group D, respectively (NS). The internal mammary artery was used for grafting in 21 % of the group D patients compared to 7 % in group L (p = 0.001). Repair of LVA alone was performed in 98 patients (32 %). The mean aortic cross-clamp time was 64.7 ± 40 minutes and the cardiopulmonary bypass time was 110 ± 48.3 minutes (group L (79 ± 36 min) and group D (124 ± 48 min) (p = 0.001). 15 % of the patients were operated on an emergency basis. Intraaortic balloon pump was inserted postoperatively in 15 patients because of low cardiac output.

Statistical Analysis

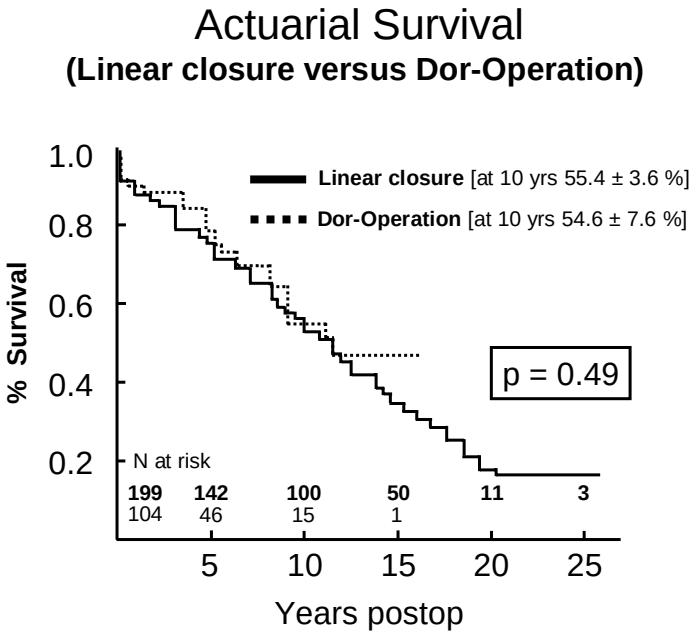
Kaplan-Meier analysis was used to study patient and event-free survival status. Differences between patient groups were analyzed using a log-rank test. The Chi-square test (for categorical data) and Student's t-test (for measured data) were used to determine statistical significance. Differences resulting in a P value of less than 0.05 were considered significant. All data were analyzed with SPSS software, release 11 (SPSS, Chicago, IL).

Results

Mean age at operation was significantly higher in group D (62 ± 9.6 years) compared to group L (54.5 ± 8.6 years) ($p = 0.001$). There was also a higher percentage of female patients in group D (24 %) compared to group L (13 %) ($p = 0.023$). No significant differences between both patient groups were found regarding the site of infarction, preoperative NYHA functional class, LV ejection fraction or the risk factors for coronary artery occlusive disease.

The overall 30-day mortality was 6.2 %. In group L 13 (6.5 %) patients died early compared to 6 (5.7 %) in group D. 156 patients (51 %) died late after in average 7.5 ± 5.8 years. Overall actuarial survival after 10 years was 55.6 ± 3.2 %. Patients in group L showed a 10-year actuarial survival-rate of 55.4 % compared to 54.6 % in group D. The difference did not reach significance ($p = 0.49$) (see Fig.20).

Fig.20



Operative and late mortality were mainly due to cardiac failure. Perioperative complications are reported in Table. 3

Table 3. Prevalence of postoperative complications

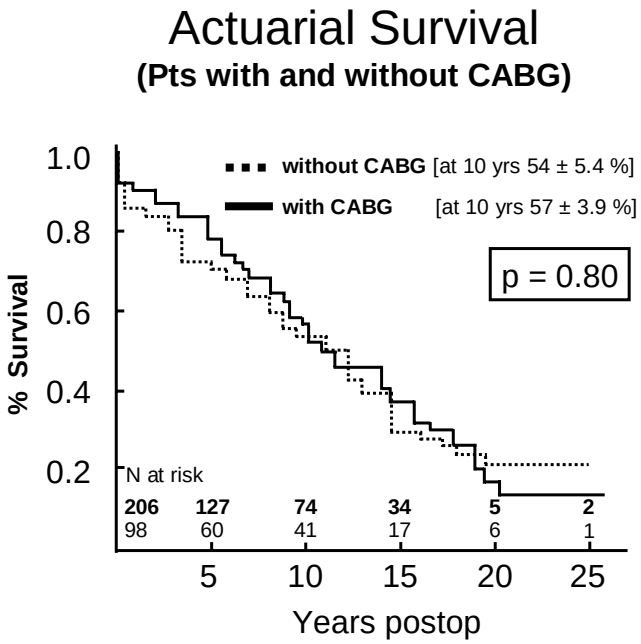
	All patients (n = 305)		Group L (n = 200)		Group D (n = 105)	
Complication	No.	%	No.	%	No.	%
Reexploration of the mediastinum for bleeding	14	4.6	8	4.0	6	5.7
Intraaortic balloon pump	17	5.6	12	6.0	5	4.8
Pacemaker Implantation	7	2.3	4	2.0	3	2.9
AICD Implantation	9	3.0	4	2.0	5	4.8

AICD = Automatic implantable cardioverter defibrillator

There were no significant differences in postoperative complications between both groups. Concomitant mitral valve replacement was the only significant risk factor for early mortality, we could identify. In our study, factors as age > 60 years, EF > 25 %, NYHA functional class IV, site of infarction had no significant influence on early mortality.

There was also no difference in long-term survival between patients with and without concomitant CABG (Fig. 21)

Fig.21



Follow up

Follow up extends up to 26 years (mean 10 ± 6.9 years, with a cumulative total of 2520 patient-years. Patients spent an average of 4.4 ± 2.5 days on the ICU. Mean length of hospital stay was 16.3 ± 11 days. Postoperatively 15 patients required IABP support. As of July 2002, 135 patients are alive. Symptomatically, patients in group L and D showed a significant improvement. Overall 78 % of the survivors are in NYHA functional class I-II. Questionnaire analysis revealed that more than 2/3 of the patients felt a significant improvement of their clinical condition. There was no statistically significant difference in postoperative NYHA functional class between both groups.

The following questionnaire was sent to the patients:

Sehr geehrte Frau, sehr geehrter Herr,

Bei Ihnen wurde in unserer Klinik eine Herzoperation durchgeführt. Zur Kontrolle unserer Behandlungsverfahren bitten wir Sie, den Fragebogen auf der Rückseite auszufüllen und in beiliegendem Freiumschlag möglichst bald an uns zurückzusenden.

Ihre Mitarbeit hilft uns bei unserem Bemühen die Therapieverfahren und Techniken ständig zu verbessern. Durch ihre Aussage leisten Sie einen wesentlichen Beitrag und helfen uns bei der Behandlung anderer Patienten.

Es wäre für uns auch von größter Bedeutung, wenn Sie sich zu einer Kontrolluntersuchung in unserer Klinik bereiterklären würden. Für Rückfragen und zur Terminabsprache stehen wir Ihnen gerne unter der Rufnummer **089/12183106** zur Verfügung.

Vielen Dank im Voraus

Fragebogen Zutreffendes bitte ankreuzen ☐

1) Verspüren Sie zurzeit ☐ Atemnot, ☐ Brustschmerzen, Druck- oder Engegefühl in der Brust? ☐ ja ☐ nein

falls ja: ☐ in Ruhe
☐ bei Belastung → ☐ Gehen in der Ebene

→ ☐ nach einem Stockwerk Treppensteigen
→ ☐ nach drei Stockwerken Treppensteigen

2) Wurde Ihre Befinden durch die Operation gebessert ?
☐ deutlich gebessert, ☐ gering verbessert, ☐ keine Änderung ☐ Verschlechterung des Befindens

3) Nehmen Sie zur Zeit Medikamente ein ? ☐ ja ☐ nein

Bitte geben Sie den Namen des Präparates an

.....
....

.....
....

4) Wann wurde die Herzfunktion zuletzt untersucht ? ..... / ..... / ..... (Datum)

bei welchem Arzt / in welcher Klinik ?
.....

mit welchem Ergebnis ?
.....

5) Wurde nach der Operation nochmals eine Herzkatheter-Untersuchung durchgeführt?

☐ ja ☐ nein

Wenn ja, wann ? ..... wo ?
.....

6) Sind Sie zwischenzeitlich nochmals am Herzen operiert worden ?
☐ ja ☐ nein

Wenn ja, wann ? ..... wo ?
.....

Grund ?
.....

7) Haben Sie seit der Operation andere Erkrankungen durchgemacht?
☐ ja ☐ nein

Wenn ja, welche ?
.....

.....
....

8) Bitte tragen Sie hier noch Ihre derzeitige Adresse und die Ihres Hausarztes ein

Name, Vorname: ..... Name, Vorname: .....
.....

Strasse: ..... Strasse:
.....

PLZ / Ort: ..... PLZ / Ort:
.....

Tel: ..... Tel :
.....

Raum für Mitteilungen
.....

.....
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Datum: **Unterschrift:**

Reoperations

Within the observation period a total of 13 patients (4.3 %) required a reoperation 3 months to 11.6 years (mean 5.5 ± 4.6 years) after the initial operation. 7 (3.5 %) patients in group L and 6 (5.7 % in group D. Reoperations included CABG n = 3, transplantation n = 3, AVR n = 1, mitral valve repair or replacement n = 5, other n = 1). Overall freedom from reoperation after 10 years was 95 ± 1.4 %. In group L freedom from reoperation at 10 years was 96.5 % compared to 95.1 % in group D. (see Fig. 22). The difference was not significant (p = 0.055).

Fig.22

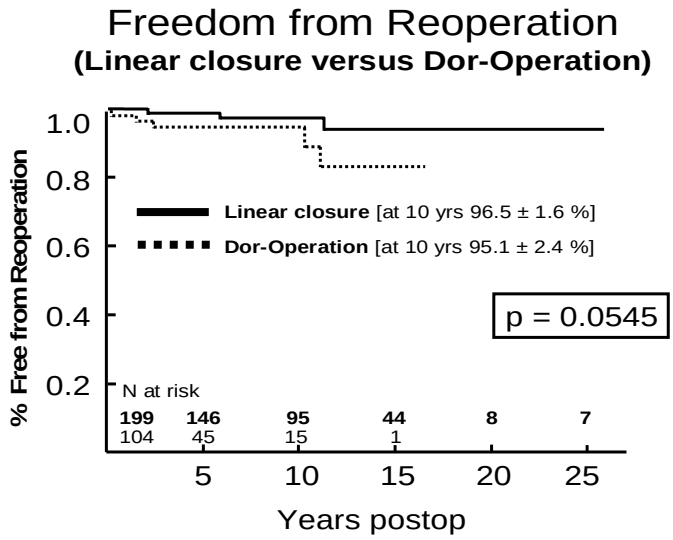

Table 4: Reoperations

	All patients (n = 305)		Linear closure (n = 200)		Dor-technique (n = 105)	
Reoperation	No.	%	No.	%	No.	%
CABG	3	1.0	3	1.5		
Transplantation	3	1.0	2	1.0	1	1.0
AVR	1	0.3			1	1.0
MV-repair	1	0.3	1	0.5		
MVR	4	1.3	1	0.5	3	3.0
Other					1	1.0
Total	13	4.3	7	3.5	6	5.7

Discussion

10 % to 35 % of patients, who have had transmural myocardial infarction, develop a ventricular aneurysm [55]. Studies on the natural history of LV aneurysms report a 5-year survival of 12 % to 47 % [57, 56]. Since the late 1950s, when LVA resection under cardiopulmonary bypass was first performed, surgical results improved considerably. Improvements in operative technique, anesthesiologic management and postoperative intensive care had a continuous positive impact on surgical results. Jatene and associates reported a reduction in operative mortality from 12.6 % to 3.5 % in a series of 508 patients who underwent circular reduction and closure with or without a prosthetic patch [38]**.** Newer surgical techniques of LVA repair with the aim of restoring left ventricular geometry proved to be valuable even in patients with severely impaired left ventricular function [38; 44, 18]. However there is an ongoing debate regarding the influence of surgical technique on long-term survival.

This report is a review of our experience with different methods of LVA-repair. Beginning in the year 1974, linear closure of LVA was the technique of choice. Since 1985 the technique described by Vincent Dor was applied as an alternative Method [23]. At our institution overall operative mortality was 6.2 % and 10-year survival 55.6 ± 3.2 %. Our results compare favorably with those of other authors, who report an operative mortality between 3,5 and 8 % and a 10-year survival between 44 - 63 % [38, 60, 64, 44] In our series we could not identify a significant difference in operative mortality and long-term survival between both surgical techniques. This confirms results of other autors [60, 64]. Both patient-groups in our study were comparable regarding the preoperative NYHA functional class, ejection fraction, site of infarction and the extent of coronary artery disease. However, those patients, who underwent the Dor procedure, were usually older and the percentage of female patients was higher**.** Komeda and associates also found no influence of the technique of LVA-repair on operative mortality. However analyzing a subgroup of their patients with poor LV-function (LV-EF < 20 %). Operative mortality after conventional closure versus inverted T-closure or endocardial patch repair differed significantly. (12.6 % versus 6.5 %) [44] In a series of 118 patients with LVA analyzed by Sinatra and coworkers none of the patients who had endoventricular patch plasty died in hospital. Hospital mortality in those patients, who underwent linear closure, was 10.3 % [58].

Various authors underlined the importance of concomitant complete revascularization in patients with LVA [44, 4]. Right ventricular dysfunction is common after repair of LVA and Komeda and colleagues therefore strongly recommend revascularization of the right coronary artery [44]. 68 % of our patients underwent concomitant bypass grafting. However there was no statistical difference in survival between patients with and without CABG. Vural and coauthors had similar results. [64]

Different risk factors for operative and late mortality have been investigated [62, 63]. Komeda and associates, analyzing 336 patients operated with different surgical techniques, identified poor left ventricular function (EF < 20 %), age greater than 60 years, previous myocardial revascularization, lack of angina pectoris and NYHA functional class IV as independent predictors of operative mortality. The presence of left main coronary artery stenosis was a predictor of late mortality after repair of LVA [44]. Non-anterior location of myocardial infarction and a history of thromboembolic event were independent risk factors for overall mortality in a recent study [60]. The presence of a posterior aneurysm is associated with a higher mortality and those patients come to the operation in a more compromised state. In our series concomitant MVR was the only significant factor influencing early mortality.

Various studies showed a significant clinical and hemodynamic improvement after LVA resection with different surgical techniques. [28, 40, 34]. Dor and coworkers demonstrated a significant increase in ejection fraction and a reduction in ventricular volumes as well as an improvement in NYHA functional class. Patients, who benefit most from the operation are those with more severe preoperative ventricular dysfunction (EF < 30 %). Patients who did not have a satisfactory improvement of EF were more likely to have multivessel disease and, in particular, critical involvement of the right coronary artery [26]. Vural and associates found a better functional improvement in patients with circular patch closure [64].

We could also demonstrate a subjective and objective improvement in functional status. However there was no significant difference between both patient-groups.

Study limitations

As many other studies our study also has the known limitations of any retrospective nonrandomized analysis. We compared two different surgical techniques for LVA repair. Linear closure was performed since 1974; from 1985 the Dor-technique was applied as an alternative procedure. Patients in the linear repair group had a longer follow up and the sample sizes of both patient-groups L and D were different. This might have influenced the late clinical results. On the other hand, there are only a few studies with a 25-year follow up available. A prospective randomized trial would obviously provide more definitive conclusions regarding the superiority of a particular technique. With the decreasing number of LVA this might be possible only in a multicenter study.

Conclusion

LVA resection is a definite procedure with a low reoperation rate and can be performed with low operative mortality even in severely compromised patients. The clinical status of the patients improved significantly. In regard to the long-term survival and the reoperation rate we could not demonstrate a difference related to the surgical technique. With both surgical techniques comparable results regarding operative mortality, improvement in clinical status and survival can be achieved.

Summary:

OBJECTIVE: Various surgical techniques have been introduced for patients with postinfarction LV aneurysm (LVA). Compared to conventional linear closure, endoventricular patch repair according to Dor is advocated to maintain LV geometry, allowing a more physiologic repair of LV aneurysm. However the impact of these different techniques on long-term results is discussed controversially. In this study we retrospectively analyzed patients with postinfarction LVA operated with different surgical techniques during the last 25 years.

METHODS: The study included 305 pts. operated between 1974 and 2000. Mean age was 57 ± 9.6 years. 17 % were female and 83 % male. 200 pts (66 %) underwent linear resection of LVA (group L) and 105 (34 %) had aneurysmectomy with endoventricular patch according to the technique described by Dor and associates (group D). Repair of the LVA alone was performed in 98 patients (32 %). 207 patients (68 %) had associated coronary grafting with a mean number of 1.5 ± 1.4 grafts. 81.5 % of pts. were in NYHA class III or IV, and median preoperative ejection fraction was 34 ± 12 %. The vast majority of aneurysms were in the anterior region. Six patients underwent epicardial electrophysiological mapping and cryotherapy intraoperatively. The follow up period extends up to 25 years (mean 10 ± 6.9 years) with a cumulative total of 2520 patient-years.

RESULTS: Overall 30-day mortality was 6.2 % (19/305). Early mortality did not differ in group L (6.5 %) and D (5.7 %). Actuarial survival after 10 and 20 years was 55,6 ± 3,2 % and 16,7 ± 3,5 % respectively. There was also no significant difference in survival between patients of group L and D (55.4 % vs. 54.6 % at 10 years, p = 0.49). 13 patients (4.3 %) required a reoperation after in average 5.5 years.

Freedom from reoperation after 10 years was 95.8 ± 1.4 % and did not differ between both patient groups (group L 96.5 % vs. group D 95.1 % at 10 years, p = 0.06). As of December 2002, 130 patients (43 %) are alive. 83 % of the survivors are in New York Heart Association (NYHA) functional class I-II

CONCLUSIONS LVA resection is a definite procedure with a low reoperation rate and can be performed with low operative mortality even in severely compromised patients. The clinical status of the patients improved significantly. In regard to the long-term survival and the reoperation rate we could not demonstrate a difference related to the surgical technique. With both surgical techniques comparable results regarding operative mortality, improvement in clinical status and survival can be achieved.

Zusammenfassung:

ZIEL:

Für Patienten mit Postinfarkt-LV-Aneurysma (LVA) gibt es verschiedene Operationstechniken. Im Vergleich zum konventionellen linearen Verschluss besteht der Vorteil der endoventrikulären Korrektur mit Patch nach Dor im Erhalt der LV-Geometrie, d. h. die Dor-Technik erlaubt eine bessere physiologische Korrektur von LV-Aneurysmen. Die Auswirkung dieser beiden Operationstechniken auf das Langzeitergebnis ist jedoch umstritten. In unserer Studie untersuchten wir rückwirkend Patienten mit Postinfarkt-LV-Aneurysma, die in den letzten 25 Jahren mit den beiden Operationstechiken operiert wurden.

METHODEN:

In unserer Studie begutachteten wir 305 Patienten, die zwischen 1974 und 2000 operiert wurden. Das Durchschnittsalter lag bei 57 ± 9,6 Jahren. 17 % der Patienten waren weiblich und 83 % männlich. 200 Patienten (66 %) unterzogen sich einer linearen LVA-Resektion (Gruppe L) und 105 (34 %) einer Aneurysmektomie mit einem endoventrikulären Patch, d. h. der von Dor et.al. beschriebenen Technik (Gruppe D). Bei 98 Patienten (32 %) wurde nur das LVA operiert, während bei 207 Patienten (68 %) gleichzeitig Bypässe angelegt wurden, und zwar im Durchschnitt 1,5 ± 1,4 Bypässe. 81,5 % der Patienten entsprachen der NYHA-Klassifikation III oder IV und die durchschnittliche präoperative Auswurffraktion (EF) betrug 34 ± 12 %. Die überwiegende Mehrheit der Aneurysmen befand sich in anteriorer Position.

Sechs Patienten unterzogen sich intraoperativ einem epikardialen elektrophysiologischen Mapping und einer Kryotherapie. Der Follow-up-Zeitraum erstreckt sich auf bis zu 25 Jahre (im Durchschnitt 10 ± 6,9 Jahre), woraus sich eine Gesamtsumme von 2520 Patienten-Jahren ergibt.

ERGEBNISSE:

Die Gesamtmortalität innerhalb von 30 Tagen betrug 6,2 % (19/305). Die Frühmortalität unterschied sich in Gruppe L (6,5 %) und D (5,7 %) nicht. Die Überlebensrate nach 10 und 20 Jahren betrug 55,6 % ± 3,2 % bzw. 16,7 % ± 3,5%. Auch hier bestand kein wesentlicher Unterschied in der Überlebensrate der Patienten in Gruppe L und D (55,4 % bzw. 54,6 % nach 10 Jahren, p = 0,49). 13 Patienten (4,3 %) mussten nach durchschnittlich 5,5 Jahren reoperiert werden. Nach 10 Jahren benötigten 95,8 % ± 1,4 % der Patienten keine Reoperation, wobei hier kein Unterschied zwischen den beiden Patientengruppen bestand (Gruppe L 96,5 % bzw. Gruppe D 95,1 % nach 10 Jahren, p = 0,06). Bis Dezember 2002 hatten 130 Patienten (43 %) überlebt. 83 % der Überlebenden entsprachen der NYHA-Klassifikation I - II.

FOLGERUNGEN:

LVA-Resektion ist eine Technik mit geringer Reoperationsrate, die auch bei Patienten mit schlechtem Allgemeinzustand mit geringer Mortalitätsrate durchgeführt werden kann. Der klinische Zustand der Patienten verbesserte sich deutlich. Im Bezug auf die Langzeitüberlebensrate und die Reoperationsrate konnten wir keinen Unterschied bei den verschiedenen Operationstechniken feststellen. Mit beiden Operationstechniken lassen sich bezüglich der Mortalität, des klinischen Zustandes und der Überlebensrate vergleichbare Ergebnisse erzielen.

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Figures & Tables Index

Abbreviations:

ACE = Angiotensin Converting Enzyme

AICD = Automatic implantable cardioverter defibrillator

AMI = Acute Myocardial Infarction

APM = Anterior Papillary Muscle

BZ = Border Zone

CABG = Coronary Artery Bypass Grafting

CASS = Coronary Artery Surgery Study

CHF = Congestive Heart Failure

EF = ejection fraction;

IABP = Intra Aortic Balloon Pump

LA = Left Atrium

LAD = Left Anterior Coronary Artery

LV = Left Ventricle

LVA= Left Ventricular Aneurysm

LVEDP = Left ventricular enddiastolic pressure

MI = myocardial infarction

MRI = Magnetic Resonance Imaging

NYHA = New York Heart Association

PPM = Posterior Papillary Muscle

RA = Right Ventricle

RV = Right Ventricle

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