MEDICAL GRAND ROUNDS

LEFT VENTRICULAR ANEURYSM

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A left ventricular aneurysm (LVA) has been identified in 2 to 30% of patients several months to years after myocardial infarction (6,22,25,28,34,40,53,76,96). Thus, it is one of the two most common mechanical complications of myocardial infarction (the other being papillary muscle dysfunction). Ventricular tachyarrhythmias, congestive heart failure, angina and arterial embolism are complications of LVA which have been treated successfully by surgical correction of the aneurysm. In spite of the prevalence of LVA and the morbidity associated with it, the rate of surgical repair compared to coronary artery bypass surgery is low, less than 3% at referral centers (69). This could be due to differences of definition in prevalence studies compared to surgical reports, a low rate of medically uncontrolled symptoms, or a unclear benefits of surgery. For example, in spite of successful aneurysm reduction surgery, improvement in ventricular function has not been documented consistently. Another frustrating observation in most surgical series has been the high (up to 30%) incidence of serious ventricular arrhythmias and late sudden death. Fortunately, since the mid 1970s there have been substantial advances in the evaluation and treatment of ventricular tachyarrhythmias, and in surgical techniques including intraoperative myocardial protection and coronary bypass surgery. However, The variable definitions of LVA compromise the value of these observations. The objective of this review is to provide a framework for the evaluation of symptomatic patients with ischemic heart disease and LVA, and to suggest an approach to therapy.

1. DEFINITION

Aneurysms of the left ventricle are divided into two groups, true and false. The wall of a true aneurysm is composed of transmural fibrous scar or scar and muscle, and is usually derived from remodeling of the left ventricle after infarction. The wall of a false aneurysm (or pseudoaneurysm) is composed of pericardium and results from perforation of the left ventricle (Figure 1). Fatal cardiac tamponade does not ensue because of adherent overlying pericardium which expands but does not acutely rupture. It is important to appreciate that false aneurysms may occur in patients with a first transmural infarction in a vigorously contracting, nondilated LV, and that prognosis is excellent with surgical intervention. Clinically, the distinctive features of a false aneurysm (compared to a true aneurysm) are: 1) the narrow neck of the false aneurysm detected by contrast ventriculography, and 2) the tendency of the false aneurysm to rupture (137). The only recent report of spontaneous rupture of a true aneurysm was in 1945 (12).

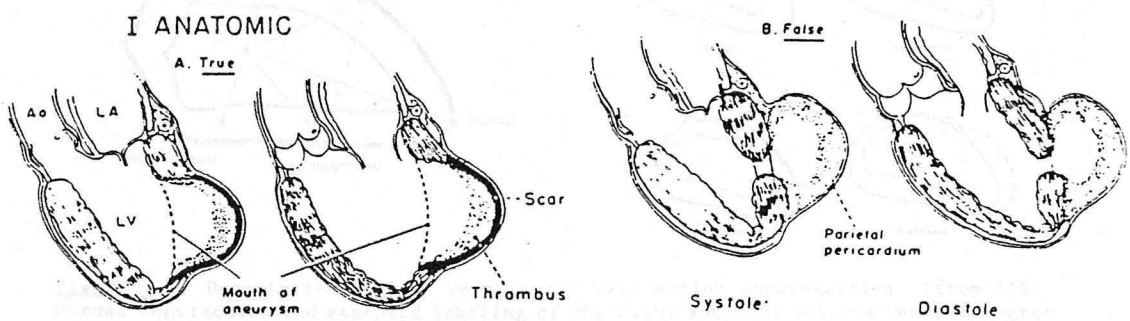

Figure 1. Diagrams of hearts with true and false aneurysms. The true aneurysm has a mouth as wide or wider than the maximal diameter of the aneurysm (22).

Although the differences between true and false aneurysms are clear, the distinction between a true aneurysm of the left ventricle and other regional abnormalities are controversial. Schlicter (124) considered an aneurysm as "...a localized outpouching of the cavity of a cardiac chamber with or without outward bulging of the external surface" which was identified at postmortem. Kirklin and Barratt-Boyes (77) defined a left ventricular aneurysm as "... a well-delineated transmural fibrous scar, virtually devoid of muscle, in which ... the wall is usually thin, and both inner and outer surfaces bulge outward. During systole, the involved wall segments are akinetic (without movement) or dyskinetic (characterized by paradoxical movement)."

In practice, an anatomical emphasis is less useful than a dynamic nomenclature. Cardiologists generally have defined aneurysms based on contrast left ventriculograms, as shown in Figure 2. Gorlin et al. (53) considered an aneurysm as "either akinesis or paradoxical pulsation of a portion of the left ventricular wall." Cheng (25) stated that "the diagnosis of aneurysm [is] obvious by visual inspection of the cine ventriculogram ... with areas of either totally noncontracting (akinetic) or less frequently paradoxically systolic expansile (dyskinetic) wall." Johnson et al. (67) state that "this diagnosis does not require diastolic deformity of the ventricle,... nor does it require that dyskinesis of the scar occur during systole."

Each of these definitions have in common the feature of a discrete region of abnormal myocardium which is distinguished clearly from surrounding, functionally normal myocardium. The distinguishing feature is either the absence of contraction during systole or a circumscribed region of fibrous tissue. According to definitions based on the ventriculogram, the diastolic outline of the endocardial surface may be normal, and an akinetic segment qualifies as an aneurysm. This definition, which will be used in this review, has the weakness of not including an estimate of the size of the aneurysm or the functional state of the uninvolved tissue. However, it is general enough to encompass all of the surgical reports summarized in Section 7.

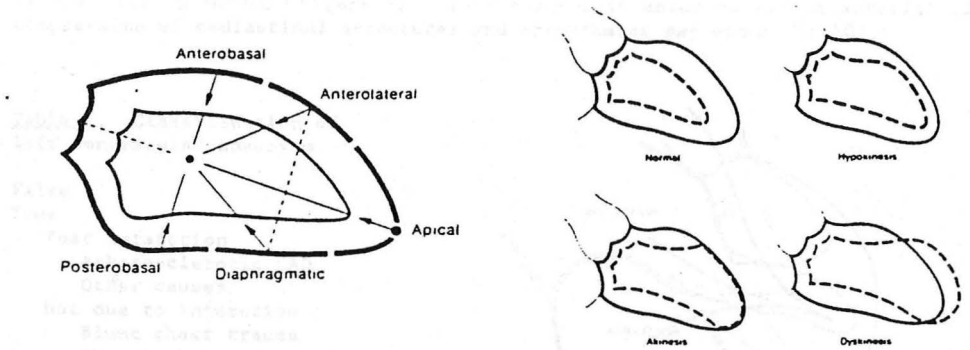

Figure 2. Description of left ventricular wall motion abnormalities (from 115). Normal contraction and standard labeling of the right anterior oblique ventriculogram is shown at the left. Patterns of LV wall motion are shown at the right (solid line, end diastole, broken line, end systole).

2. ETIOLOGY

The great majority of left ventricular aneurysms are due to myocardial infarctions secondary to coronary artery disease. Nonatherosclerotic causes of myocardial infarction also could result in aneurysm formation. Seven conditions have been reported as nonvascular causes of true left ventricular aneurysms (Table 1).

Blunt chest trauma may cause such a severe contusion that myocardial necrosis occurs and a localized ansurysm may form. It may be asymptomatic, and detected within 6 weeks after the injury, or it may be detected several years later (87). Because they are thin-walled, they probably are likely to rupture, and should be resected. In one case report the patient was asymptomatic, the electrocardiogram was normal, and the aneurysm was identified on an incidental chest x-ray.

Chagas' heart disease in patients dying with congestive heart failure is a dilated cardiomyopathy. However, patients who die suddenly have relatively normal size hearts with aneurysmal dilation of the apex. Sarcoid heart disease is usually a focal or multifocal replacement of the myocardium by granulomatous tissue or fibrosis. LVA may occur, but the most common complications are ventricular ectopy, atrioventricular block or fascicular block, and heart failure (24). Tuberculosis was identified as the cause of left ventricular aneurysm in 3 young patients without tuberculous pericarditis (120). Syphilis has also been stated (67) to cause LVA.

Congenital aneurysms of the left ventricle cover a broad spectrum of morphologies and sites of origin in the ventricle. All are rare. The most common is an aneurysm of the membranous ventricular septum which can be detected by two-dimensional echocardiography and is clinically insignificant. A muscular diverticulum originating at the left ventricular apex has been reported. Congenital LV aneurysm in the adult may present as ventricular dysrhythmia. Calcification may occur (55). A congenital aneurysm may have a narrow neck (5). Finally, submitral aneurysms usually occur among Africans living in equatorial regions. This aneurysm arises at the base of the heart, typically below the mitral valve, and has a narrow neck which must be distinguished from a false aneurysm (Figure 3). This subannular aneurysm may be anterior (37). Compression of mediastinal structures and arrhythmias may occur (26,50).

Table 1. Classification of left ventricular aneurysm

- False
- True
  - Post infarction
    - Atherosclerotic CAD
    - Other causes
  - Not due to infarction
    - Blunt chest trauma
    - Chagas' heart disease
    - Sarcoidosis
    - Tuberculosis
    - Syphilis
    - Congenital
    - Submitral

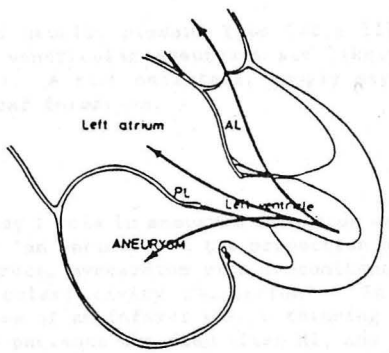

Figure 3. Submitral left ventricular aneurysm (4).

3. ANATOMY, PATHOLOGY AND PATHOGENESIS

Pathology

The aneurysm is usually located on the anterior or apical surface of the heart. Left ventricular volume and weight are increased (34). Hypertrophy of the uninvolved myocardium occurs because of overall dilation of the ventricle and increased wall tension due to LaPlace's law. Grossly, the aneurysm is a thin, homogeneous and well-demarcated fibrous scar (110). The endocardial surface is smooth and frequently covered by adherent thrombus. In some cases there is a well defined, shiny white endocardial peel which extends a few millimeters beyond the border of the aneurysm to cover normal myocardium. The overlying pericardium is often adherent, and the pericardium or thrombus may calcify.

Microscopically, the aneurysm is dense fibrous and elastic tissue. Frequently there are scattered residual myocytes or islands of cells within the fibrous tissue, often exhibint vacuolar degeneration.

Recently, Hochman et al. (61) suggested that the pathological features of LVA correlate with some clincal findings. This study, which included post mortem specimens as well as specimens obtained at the time of surgery, implies that detection of certain endocardial abnormalities could identify a group of patients at high risk for ventricular arryhthmias. It also suggests that the development of thrombus is not solely due to wall motion abnormality.

Pathological type	I	II
elastic tissue	extensive	minimal
thrombus	no	yes
risk of VT	high	low

Table 2. Endocardial abnormalities in left ventricular aneurysm and correlation with ventricular tachyarrhythmia (61).

Coronary Arteries

Multivessel coronary artery disease is usually present (see Table 11). Cheng suggested that patients who develop left ventricular aneurysms are likely to have particularly poor collateral arteries (25). A rich collateral supply may preserve islands of viable myocytes which resist scar formation.

Infarct Expansion

Infarct expansion a process which may play a role in aneurysm formation was defined originally by Hutchins and Bulkley (64) as "an increase in the proportion of surface area of the left ventricle occupied by necrotic myocardium with concomitant thinning of the infarcted wall, [and left ventricular] cavity dilatation". In essence, expansion is an increase in the surface area of an infarct due to thinning. Infarct expansion occured in 59% of 76 consecutive patients who died after MI, and expansion was most common with large transmural, first infarctions (64). Early dilatation of the heart which is due to infarct expansion is not associated with stretching of the uninvolved myocardium. Expansion is often asymptomatic, but may be associated with

chest pain or hypotension, and should be differentiated from extension (141). The physical properties of the infarcted tissue, modification by the healing process, and mechanical loading of the heart are thought to influence infarct expansion.

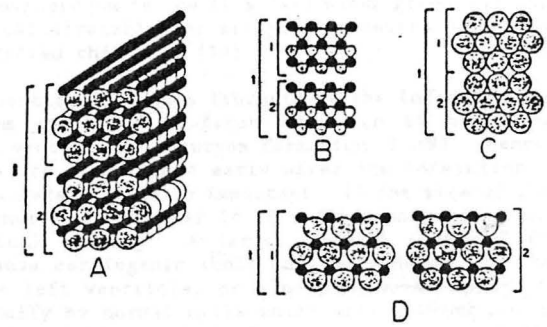

Figure 4. Cellular basis of infarct expansion (140). A normal architecture showing dark tubes (vascular spaces) and striped tubes (myocytes).

The physical properties of infarct depend on multiple interrelated factors. A transmural infarction is less likely to be supported by islands of normal myocytes or a subepicardial rim of normal tissue which will tend to support the myocardium. A first infarction in a particular region will, by definition, not have intermingled fibrous tissue from a previous infarction. Although a large infarction does not necessarily expand in animal studies, clinical studies indicate that large anterior infarcts in humans are prone to expand. The severity of coronary disease, collateral reserve and myocardial hypertrophy may also influence expansion.

The normal healing process includes an early (first week) phase of acute inflammation, which is followed by chronic inflammation, and, during the third and fourth week, collagen deposition. Since expansion occurs in the early phase of healing, it is not surprising that anti-inflammatory drugs may play a role in modifying the healing process and contribute to expansion (20).

Stretch and distension of the infarcted segment are also probably influenced by mechanical stress. In principle, heart rate, preload and contractility (estimated by dP/dt) may influence expansion. Little is known about the relative importance of each factor. Nevertheless, left ventricular rupture is more likely in the hypertensive individual, and therapy which reduces afterload (angiotensin converting enzyme inhibitors) may reduce LV dilatation after MI. Since expansion begins early after infarction, early intervention may limit progression to LVA.

The acute remodeling of the left ventricle which expands has been examined by Weisman et al. (140). Thinning of the infarct zone could be due to 1) myocyte necrosis; 2) reduction in intercellular space; 3) stretch of myocytes; or 4) slippage of myocytes without loss of myocyte architecture. These are shown schematically in Figure 4. In a study of rats and a small number of humans, cell slippage was the dominant cause of thinning which was attributed to slippage of adjacent myocyte bundles.

Pathogenesis of True Left Ventricular Aneurysm

One view of the pathogenesis of LVA is that it represents a delayed complication of myocardial infarction. According to this formulation, the collagen scar which replaces necrotic myocardium in the first 4-8 weeks gradually stretches. The concept of early pathological stretching or slippage of sheets of myofibrils, i.e., infarct expansion, has modified this view (59).

The essential question is: does fibrosis of the infarcted tissue occur before or after the aneurysm develops? Infarct expansion is now thought to provide the substrate for left ventricular aneurysm formation (6,59). Hence, the conditions for aneurysm formation are established early after the infarction. There are probably five variables which are clinically important: 1) the size of the infarct, 2) whether or not it is transmural, 3) whether it is a first infarction in that region, and 4) associated mechanical loading. A large infarct (>40% of the mass of the left ventricle) will cause cardiogenic shock and early death. A small infarction (<15% of the mass of the left ventricle) or a nontransmural infarction are likely to be supported mechanically by normal cells which will prevent aneurysm formation. The concommitant use of steroids, or, experimentally, other antiinflammatory agents, may increase the risk of aneurysm (20,28).

The fifth condition may be the patency of the infarct-related artery. The metabolic consequences of coronary occlusion are of course time dependent, and myocyte salvage after, at most, 1 hour of total ischemia, is nil. Clinically, it is difficult to show benefit of reperfusion therapy if reperfusion is initiated 4 to 6 hours after the onset of a documented infarction. However, it has long been appreciated that reperfusion of irreversibly injured myocardium changes the pattern of necrosis from coagulation to contraction band necrosis. Coagulative necrosis results from sustained severe ischemia. The ischemic cells are arrested in the relaxed state and appear stretched or attenuated. Contraction band necrosis develops after severe ischemia followed by reflow. The cells are no longer viable, but the myofibrils are hyper-contracted, and sites of calcium deposition in the mitochondria are characteristic. The cells are arrested in the contracted state.

These observation suggest that late reperfusion may alter the mechanical state of the nonviable myocardium. Little is known about the effects of late reperfusion on the risk of aneurysm formation, but Hochman and Choo have found in a rat model that late reperfusion does not alter infarct size or extent of transmural injury, but infarct expansion is inhibited (60). If these observations can be explored in humans, indications for reperfusion therapy other than direct benefit to regional wall motion may be identified (15).

4. PATHOPHYSIOLOGY AND CLINICAL FEATURES

Classically, symptoms include angina, congestive heart failure, ventricular arrhythmias and thromboembolic disease. Rarely, infection of the aneurysm has been reported (127,130). These four syndromes may also occur in patients with multiple infarct cardiomyopathy or myocardial infarction with minimal residual wall motion abnormalities. The presenting symptoms of LVA are not diagnostic, although nearly all patients have a history of myocardial infarction.

Nonspecific Evidence of Left Ventricular Scar Formation

The physical exam may be abnormal, but there are no specific features which distinguish LVA reliably from multiple infarct cardiomyopathy. In both conditions the first heart sound may be soft, and third and fourth heart sounds are common. Systolic and diastolic murmurs have been reported, but it its not clear if mitral regurgitation was excluded. Examination of the precordium is not necessarily abnormal; holosystolic apical expansion or dyskinetic regions may be detected.

The standard chest x-ray may shows evidence of the left lateral border and calcification in the left ventricle which may be in scar tissue or thrombus within the aneurysm.

The ECG almost uniformly shows a previous myocardial infarction, typically anterior Q waves. Chronic ST elevation has been empirically associated with LVA, and is defined as > 1 mm (0.1 mV) elevation with convexity upward more than one month after the infarction. In a two-dimensional echo study, this finding in the precordial leads correlated best with dyskinesis of the anterior wall (7). In a radionuclide ventriculography study of patients with anterior MI, the presence of ST elevation did not reliably identify segmental wall motion abnormality. The develop-ment of ST elevation during exercise is also associated with LVA, but of course is nonspecific (63).

Table 3. Relation between ST elevation and LV function in patients with anterior Q waves more than 8 weeks after infarction (data from ref. 86).

	n	mean ST	EF	akinesis or dyskinesis
ST > 0.1 mV	12	0.23 mV	41 ± 8%	67%
ST < 0.1 mV	12	0.03 mV	43 ± 14%	50%

Effect of Aneurysms on Left Ventricular Function

The consequences of LVA for myocardial perfusion and function are complex and depend on its size. Klein and Gorlin suggested that the physiological consequences of LVA are not detectable until 20% of LV mass is involved (78). LVA may cause angina and heart failure through several related mechanisms: 1) increased myocardial oxygen consumption, 2) hypertrophy of the uninvolved myocardium, 3) mitral regurgitation, 4) filling of the noncompliant pericardium by a large aneurysm, 5) reduction of forward stroke volume (impaired systolic function), 6) impaired diastolic function, and 7) geometry of the residual myocardium.

LVA increases end diastolic volume. To maintain a normal forward output, stroke volume must be maintained (with resulting increased wall stress) or heart rate must increase. In either case, myocardial oxygen consumption must increase. If the residual (nonaneurysmal) segments are perfused by diseased coronary arteries, angina and ischemic dysfunction may result. Hypertrophy of the uninvolved myocardium is presumably due to chronic increased wall stress because of distortion of left ventricular geometry and the LaPlace relationship.

Mitral regurgitation is commonly associated with inferior or inferolateral wall motion abnormalities or aneurysms.

If a true aneurysm develops rapidly, it is conceivable that a component of pericardial constriction may play a role in symptoms suggestive of heart failure.

Reduction of forward stroke volume is commonly attributed to LVA. Tyson et al. examined distensible (homologous aortic arch) and nondistensible (woven Teflon) sacs communicating with the left ventricle of dogs (133). Only the distensible sacs were associated with depression of LV function. Pairolero et al. could not show significant improvement in LV function after large (3 x 5 cm) akinetic segments were excised in their dog model (108). These studies suggest that the benefits of aneurysmectomy (defined by improvement in left ventricular systolic function) may be limited to patients with dyskinetic aneurysms.

The influence of LVA on left ventricular diastolic function, independent of the above factors, is unknown.

The size of a myocardial infarction and the resulting aneurysm may be modeled mathematically. Radhakrishnan et al. developed a theoretical analysis of the mechanics of LVA (117). The left ventricle was represented as three ellipsoidal layers of myocardium. Variable in this analysis include the shape of the uninvolved ventricle (ratio of major to minor semiaxis lengths), and the size of the infarct (angle subtended by the infarction and the transmural extent). Wall tension in the aneurysm (tendency to expand) was sensitive to the aspect ratio of the ventricle: the more spherical the ventricle, the lower the systolic stress in the aneurysm.

Aneurysms and the Substrate for Ventricular Tachycardia

Ventricular tachycardia (VT) in patients with healed MI is thought to occur because of microscopic reentry which originates near the border between infarcted (fibrotic) and normal myocardium. If this formulation is correct, then the substrate for reentry is present in all patients with LVA. In fact, ventricular arrhythmias are a major problem for these patients, but the factors that determine which patients with this electrically inhomogeneous tissue will develop ventricular arrhythmias are unknown. It is important to point out that clinical observations - the risk of VT after simple aneurysmectomy - as well as recent electrophysiology studies support the notion that the functional as well as anatomic substrate is present in many patients with LVA but without symptomatic VT. This interpretation of the electrophysiology studies assumes that inducible sustained monomorphic VT implies that the substrate for clinically important VT is present.

To assess the risk of VT in this setting, patients with coronary disease and LVA (defined as dyskinesis on contrast ventriculography) but without symptoms of VT underwent electrophysiology study (143). Sustained monomorphic VT was observed in 50%; other details are summarized in Table 4. Interestingly, a sophisticated computer-based analysis of the left ventriculogram (the centerline method) was used to assess the discreteness of the aneurysm which was defined as the shortest distance between dyskinetic and adjacent normal myocardium. VT was induced in the patients with the most discrete LVA.

Table 4. Clinical variables of patients with LVA and coronary disease, without symptoms of VT (143). SMVT, sustainted monomorphic VT.

	SMVT inducible	SMVT not inducible
n	11	11
age (years)	62	58
EF	34%	33%
Number of diseased coronaries	2.4	2.7
LVEDP (mmHg)	19	20
Size of dyskinetic region (%)	22	23

Another study of patients with LVA and coronary artery disease but without clinical VT examined the incidence of inducible tachycardia during aneurysmectomy. Again, a significant fraction of patients (40%) had inducible VT. Furthermore, clinically important sustained VT soon after aneurysmectomy (31) or sudden death (see section 7) are not infrequent. Since VT often originates in regions which are not resected in simple aneurysmectomy, the origin may have been present prior to surgery. Taken together, these studies suggest that the substrate for sustained monomorphic VT is present in a large fraction of patients with left ventricular dyskinesis, and that late arrhythmias after aneurysmectomy may represent a late manifestation of a progressive disease. Aneurysmectomy itself, of course, may contribute to injury of the subendocardium.

5. EVALUATION

Left ventricular aneurysm should be suspected in a patient with previous myocardial infarction and congestive heart failure, angina which is poorly controlled, ventricular ectopy or, rarely, arterial thromboembolism. Once the diagnosis is established, therapeutic planning requires an assessment of 1) coronary anatomy, 2) the site and size of the aneurysm, 3) function of the residual left ventricle, and 4) the risk of serious ventricular arrhythmias.

Noninvasive Evaluation of Left Ventricular Geometry

Two dimensional echocardiography and color-flow Doppler provide an excellent assessment of left ventricular wall motion and many of the conditions which may be associated or confused with LVA (23,29,135,136). Specifically, it is important to assess four features. First, the site and size of an aneurysm should be determined and a pseudoaneurysm excluded (but see 32 and 83). Second, the function of residual myocardium must be determined (135,136). Third, the persistance of systolic wall thickening in the abnormal segments suggests residual functioning myocardium (hibernating myocardium, 14). Finally, the severity of mitral regurgitation or other valvular abnormalities should be determined.

Gated radionuclide ventriculography will detect diffuse hypokinesis, although the diagnosis of dilated cardiomyopathy is quite secure by two-dimensional and M mode echocardiography. Left ventricular ejection fraction may be determined and occasionally thrombus may be detected. Fourier analysis of the ventriculogram has been suggested (85,106,145). LVA may also be detected by thallium scintrigraphy, CT or MRI (2,100,107,116,146).

Cardiac Catheterization

A complete right and left heart catheterization is necessary. A right heart catheterization will exclude unsuspected intracardiac shunts or abnormalities of pulmonary vascular resistance. In combination with left ventriculography, the severity of mitral regurgitation (commonly associated with inferior aneurysms) can be determined quantitatively. Systemic vascular resistance and cardiac output measurements will allow medical therapy (e.g., vasodilators) tailored to the hemodynamic state.

In addition to the standard right anterior oblique projection, the left ventriculogram should include, if practical, a left anterior oblique view in order to assess motion of the interventricular septum. This may be important in planning the surgical approach to these patients since Mullen et al. (102) have reported a high mortality for patients with septal involvement combined with the usual anteroapical aneurysm.

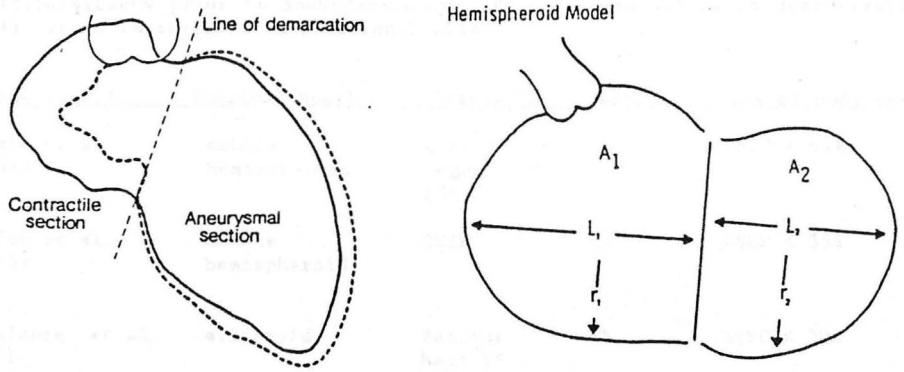

Figure 5. Left ventriculogram and the corresponding double hemispheroid model (from 139).

Various methods (Figure 5, Figure 6 and Table 5) have been suggested for quantitative evaluation of residual LV function. Feild and Dowling suggested a the simplest method (42). They assumed that the ventricle could be represented by a sphere and that the contrast left ventriculogram can be used to measure the circumference of the sphere. The fraction of the perimeter of the ventricle in diastole which does not contract was defined as the abnormal segment (AS), and reported as a fraction of the ventricular perimeter. Thus, in a normal ventricle or a ventricle with diffuse hypokinesis, AS = 0. For a large LVA, AS = 0.25. They derived a simple relationship which predicts the consequences of an abnormal segment

on ejection fraction, assuming that the remainder of the ventricle is normal: $PEF=NEF(1-AS)^3$, where PEF is the predicted ejection fraction and NEF is the normal ejection fraction (about 0.65). Lee et al., used this approach to predict mortality of aneurysmectomy, and found that if the difference between PEF and actual ejection fraction (the excess ejection fraction, or XEF) was less than 10%, mortality was increased.

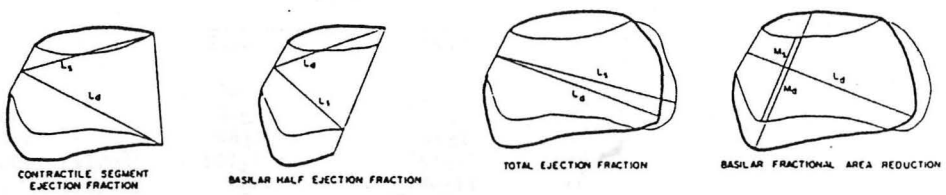

Figure 6. Methods for assessing function of uninvolved myocardium based on RAO ventriculogram (71).

Table 5. Studies of abnormal regional myocardial function by contrast left ventriculography prior to aneurysmectomy. Poor outcome refers to death (early or late), or no improvement in functional class.

Author (ref.)	Physical Model	Variable	#Pts	Index of poor outcome
Watson et al. (139)	double hemispheroid	contractile segment EF (CSEF)	9	CSEF < 44%
Kiefer et al. (75)	double hemispheroid	CSEF	42	CSEF < 35%
Kapelanski et al. (71)	ellipsoid	Basilar half EF (BHEF)	45	BHEF < 30%
Ryan et al. (121)	ellipsoid	BHEF	44	BHEF < 37%
Lee et al. (84)	sphere EF	excess	64	XEF < 10%

In spite of the complexity of these methods, measurements of column and pressure may be adequate (Table 6). Kiefer et al., did show excellent predicted value of CSEF coupled with LVEDP (Figure 7).

Table 6. Preoperative cardiac catheterization results in 42 patients with left ventricular aneurysm. Poor outcome is defined as perioperative death or late death or no improvement in functional class (75). CSEF, contractile segment ejection fraction; EDVI, end diastolic volume index (normal <90 ml/m^2); ESVI, end systolic volume index (normal <30 ml/m^2); EDP, end diastolic pressure.

	Outcome		
	Good	Poor	p
n	33	9	
EF	37±2	22±2	<0.01
CSEF	48±2	38±1	<0.01
EDVI (ml/m^2)	109±7	165±12	<0.01
ESVI (ml/m^2)	72±7	134±11	<0.01
EDP (mm Hg)	15±1	26±2	<0.01
bypass grafts	2.8	2.2	ns

High Resolution ECG Recordings of Late Cardiac Potentials

Ventricular tachycardia in patients with healed MI are thought to occur on a microscopic re-entrant basis which originates in the border between fibrotic and normal myocardium. This region of inhomogeneously excitable myocardium (which would not be resected in a standard aneurysmectomy) may not cause symptomatic arrhythmias until after surgery. A noninvasive test which reliably detects this substrate would, of course, be helpful. The absence of sustained VT as a single Holter monitor does not exclude this substrate.

Microelectrode studies in intact animals have shown that days after a myocardial infarction, irregular high-frequency potentials may be detected in the infarcted tissue which may extend beyond the QRS complex. This feature is a late potential which is present in every sinus beat.

The high resolution ECG (HRECG) is obtained by computer averaging multiple beats recorded by a low-noise electrocardiograph. Typically, three orthogonal bipolar leads are recorded and the analog signals are converted to digital representation with a wide dynamic range. These signals are transmitted to a computer where premature or late beats are rejected and the onset of the QRS in the accepted beats is aligned. The sum of 100-300 QRS complexes represents the signal averaged ECG (10,17-19,21,33).

Repeated signal accumulation reduces random signals (i.e., noise, either electrical or from skeletal muscle) and allows the detection of reproducible low voltage components of the QSR complex and ST segment. Electrical or software filters which remove low frequencey components of the QRS and ST segment allow the detection, in some patients, of high frequency potentials late in the QRS complex. This component of the QRS is the late potential.

The importance of late potential in patients with aneurysms is unknown. However, animal and human studies indicate that late potentials are associated with a high risk of ventricular tachycardia. Although the effects of bundle branch block and

concomittant antiarrhythmic therapy as late potentials is unclear, some studies (33) indicate that the HRECG retain its value. However, the independent contribution of the HRECG to the results of a careful history, Holter monitor and determination of left ventricular ejection fraction has not been clarified. Three reports published in 1987 (19,52,81) partially examined their independent and combined value in identification of patients with VT. Complete data for the evaluation of all possible combination of evaluation (e.g., Holter alone, Holter plus HRECG, etc.), were not reported. However, as a group, these studies indicate that the HRECG plus measurement of left ventricular function provider good sensitivity (65 to 100%) and specificity (60-80%) for detection of patients also will develop VT. An important question is whether Holter monitoring adds useful information.

In sum, the HRECG is an important component of the evaluation of a patient with abnormal wall motion and no history suggestive of VT. The information it provides is independent of that obtained by Holter monitoring, and it is a useful complement to measures of left ventricular function.

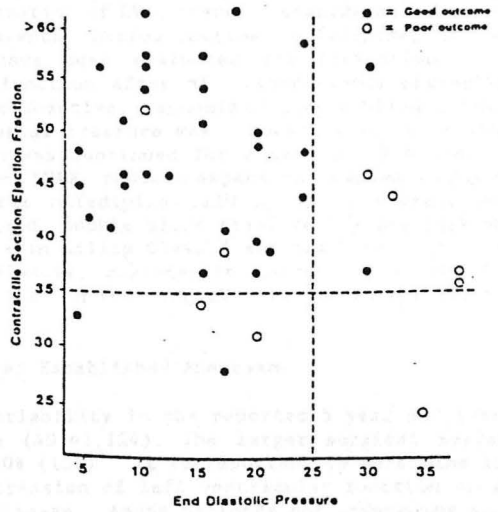

Figure 7. Relaxation between CSEF, LVEDP and surgical outcome (75).

Electrophysiology Study

Electrophysiology study may be used for two purposes in patients with LVA: first, among symptomatic patients with VT, to map the site of arrhythmia induction in the left ventricle to guide subendocardial resection, and second, to demonstrate that ventricular tachycardia may be induced and terminated by programmed electrical stimulation, i.e., that the substrate for reenty is present.

The details of programmed electrical stimulation (pacing cycle length, stimulus current, stimulus duration, etc.) vary among laboratories. Typically, stimulation is performed in the absence of antiarrhythmic drugs at two right ventricular sites. Single, double or triple premature extrastimuli are delivered. Progressively shorter cycle lengths are used until the ventricle becomes refractory or until VT is induced. In the setting of an electrophysiological study, sustained VT is defined as requiring pacing or electrical cardioversion. Identification of the origin of the arrhythmia in the left ventricle (left ventricular mapping) is useful for guiding surgical excision of the focus. However, it is not uncommon for the patient to be hemodynamically unstable during VT.

6. MEDICAL THERAPY

Prevention of Aneurysm Formation by Limiting Infarct Expansion

The current rapid evolution of therapy for MI (angioplasty and thrombolytic therapy) will alter the natural history of LVA. In addition to reperfusion therapy for prevention of formation of LVA, there is considerable interest in limiting infarct expansion. These agents, nitroglycerine, nifedipine, and angiotensin converting enzyme inhibitors have been evaluated for prevention of infarct expansion or preservation of LV function after MI. Intravenous nitroglycerin or placebo were administered in a prospective, randomized single-blind study to 310 patients with acute MI. Mean arterial pressure was reduced by at least 10%, but not below 80 mm Hg, and the infusion was continued for a mean of 39 hours. Nitroglycerin reduced infarct size, improved LVEF, reduced expansion, and improve mortality at 1 year (70). In another study oral nifedipine (120 mg/d) or placebo were administered in a prospective, randomized, double blind trial to 132 low risk patients with acute MI. All the patients were in Killip Class I and had LVEF >35%. There was no benefit of nifedipine (54). Finally, angiotensin converting enzyme inhibitors may play an important role (111,112) in reducing LV dilation in the first year after MI.

Medical Therapy for an Established Aneurysm

There is great variability in the reported 5 year survival of medically treated patients: 10 to 70% (40,41,124). The larger surgical series also report 5 year survivals of about 70% (134). It is important to determine if LVA confers a higher risk than global depression of left ventricular function or a corresponding degree of coronary artery disease. Among patients not undergoing surgery, Proudfit et al. found that for similar coronary disease, the five year mortality of patients with dyskinesis was higher than for patients with regional akinesis or hypokinesis, suggesting that LVA adds to mortality independent of the extent of coronary disease. However, the Coronary Artery Surgery Study found that, given similar degrees of left ventricular failure, mortality was not increased by LVA (41).

In spite of the high prevalence of left ventricular thrombus in patients with LVA, the incidence of embolic events is very low (82) when CVA is defined as a dyskinetic segment. The absence of contraction of underlying myocardium probably allows the thrombus to become adherent and well-organized. Chronic oral anticoagulation is not recommended for patients with LVA unless systemic emboli have occurred. This is in contrast to recommendations regarding dilated cardiomyopathies (1,97).

7. SURGICAL THERAPY

Surgical treatment of LVA is a standard procedure which is described in an extensive literature. Recent surgical results are summarized in Tables 7-11. These reports were selected for review because they are relatively recent, and because all patients underwent some form of aneurysm reduction aneurysm reduction surgery. Frequently, coronary artery bypass surgery was performed, and in a few instances, other procedures such as mitral valve replacement were necessary. Where possible, these patients were excluded from this review. These results are summarized to identify the following features of this population: 1) the symptoms and indications for surgery, 2) preoperative left ventricular function, 3) operative mortality, 4) the risk of death due to arrhythmias in an unselected population, and 5) functional class in survivors. The studies which were reviewed are listed in Table 1.

Table 7. Selected studies of left ventricular aneurysm reduction surgery from 1980 to present. Date refers to the date of publication. nr, not reported.

Authors (Ref.)	Date	Institution	Period
Jais, et al. (65)	1980	Laennec Hospital, Paris	1968-76
Froelich, et al. (46)	1980	University of Iowa	1977-78
Harken, et al. (57)	1980	University of Pennsylvania	1974-78
Crosby, et al. (30)	1980	University of Virginia	1973-78
Jones, et al. (69)	1981	Emory University	1974-79
Rittenhouse, et al. (119)	1982	Providence Med. Center	1974-80
Brawley, et al. (16)	1983	Johns Hopkins University	1975-80
Barratt-Boyes, et al. (8)	1984	Green Lane Hospital	1969-81
Olearchyk, et al. (105)	1984	Deborah Heart & Lung Center	1971-80
Skinner, et al. (129)	1984	Mercy Hospital, Des Moines	1974-77
Novick, et al. (104)	1984	McGill University	1970-82
Keenan, et al. (73)	1985	Southampton General Hospital	1973-83
Akins (3)	1986	Massachusetts Gen. Hospital	1977-84
Faxon, et al. (40)	1986	Coronary Artery Surg. Study	1974-79
Marks, et al. (91)	1986	Ichilov Hospital, Tel Aviv	1980-84
Garan, et al. (49)	1986	Massachusetts Gen. Hospital	nr
Wright, et al. (144)	1987	Prince Henry Hospital	1967-83
Louagie, et al. (88)	1987	Montreal Heart Institute	1979-84
Miller, et al. (98)	1988	University of Pennsylvania	nr
Walker, et al. (138)	1988	Edinburgh Royal Infirmary	1973-84
Vauthey, et al. (134)	1988	Ochsner Clinic, New Orleans	1970-85

Patient Characteristics and Indications for Aneurysmectomy

The traditional indications for aneurysmectomy include angina, CHF, ventricular arrhythmias and thromboembolism. The primary indications for aneurysmectomy are summarized in Table 2. In some cases the distribution of symptoms shown in this table was approximate. Among 1651 patients, angina alone was the primary indication for surgery in about 44% of patients; CHF (either alone or in combination with angina) was the primary indication in another 40% of patients. Ventricular arrhythmias, alone

or in combination with CHF or angina, were the primary indication in about 14%. Signif-icantly, thromboembolism constituted less than 2% of the primary indication, and the majority of these patients were from 2 studies (105,119) which reported patients treated before 1980. The impact of modern two dimensional echocardiography on assessment of these patients is unknown. Little information is available regarding prior medical therapy of these patients.

Table 8. Primary indications for aneurysmectomy. Abbreviations: TE, thrombo-embolism; VT-VF, fraction of patients for whom ventricular arrhythmias were the primary indication. In some cases TE + misc. included combined primary complaints not reported separately; nr, not reported.

angina	x			x	x	x			
CHF		x		x		x	x		
arrhythmia			x		x	x	x		
TE + misc.								x	(VT-VF)
ref.									
65	7	28	14	0	0	21	0	0	0.50
46	1	2	0	11	0	1	0	0	0.07
57	nr	nr	nr	nr	nr	nr	nr	nr	0.49
30	44	20	1	0	0	0	0	1	0.02
69	56	1	0	17	0	0	0	0	0.00
119	50	9	2	31	0	0	0	12	0.02
16	25	48	6	0	0	3	0	2	0.11
8	54	30	0	39	11	11	0	0	0.15
105	149	24	19	29	10	5	3	5	0.15
129	nr	nr	nr	nr	nr	nr	nr	nr	0.22
104	35	11	0	21	0	0	0	0	0.00
73	23	58	1	17	0	0	0	1	0.01
3	42	23	13	22	0	0	0	0	0.13
40	nr	nr	nr	nr	nr	nr	nr	nr	nr
91	15	0	5	9	4	0	4	0	0.35
49	0	0	35	0	0	0	0	0	1.00
144	108	46	0	0	0	0	0	0	0.00
88	0	15	0	22	0	9	0	3	0.18
98	10	0	40	0	0	0	0	0	0.80
138	4	0	0	28	3	0	0	0	0.09
134	105	102	4	0	11	0	0	0	0.07
fraction	0.44	0.25	0.08	0.15	0.02	0.03	0.01	0.01	

The age, sex and functional classification of patients treated surgically are similar in most series (Table 9). Most patients (83%) are male, and the average age in 18 studies ranged from 51 to 59 years. Among the 1620 patients for whom functional class was reported, about 72% were in New York Heart Association functional class III to V on medical therapy.

Table 9. Summary of same patient characteristics of patients undergoing aneurysmectomy.

					Number of patients in (preoperative) functional class	
ref.	# pts	# m	m/(m+f)	age (years)	I-II	III-V
65	70	59	0.84	55	3	24
46	15	nr	nr	59	0	15
57	101	nr	nr	nr	nr	nr
30	66	55	0.83	51	5	61
69	74	64	0.86	56	19	55
119	104	91	0.88	57	22	82
16	84	72	0.86	53	36	48
8	145	122	0.84	54	39	106
105	244	187	0.77	54	39	205
129	41	39	0.95	52	nr	nr
104	67	49	0.73	53	3	64
73	100	83	0.83	56	nr	nr
3	100	83	0.83	58	16	84
40	238	192	0.81	nr	119	119
91	37	32	0.86	58	nr	nr
49	35	31	0.89	nr	nr	nr
144	154	116	0.75	42	42	112
88	49	42	0.86	55	13	36
98	50	41	0.82	59	nr	nr
138	35	29	0.83	53	8	27
134	246	203	0.83	57	87	131

Preoperative Evaluation

Modern techniques of evaluation of ventricular tachycardia and its surgical treatment evolved in the late 1970s. Hence, in most surgical reports the most important preoperative studies were coronary angiography, left ventriculography and hemodynamic measurements. In most cases (ca. 90%) significant CAD and a prior myocardial infarction were documented. The results of left ventriculography and hemodynamics are summarized in Table 3.

Table 10. Summary of preoperative left ventricular function. The right anterior oblique ventriculogram was analyzed to determine the location of the aneurysm (septal aneurysms could not be identified). Abbreviations: AA, anteroapical; akin?, was an akinetic segment considered an aneurysm; AP, apical; def?, was an explicit definition of aneurysm provided; EDVI, end diastolic volume index; EF, ejection fraction; PI, posterior or inferior or both; L, lateral or posterolateral; LVEDP, left ventricular end diastolic pressure.

	definition			LV function+			Aneurysm location			
ref.	def?	akin?	(frac)	EF	EDVI	LVEDP	AA	AP	PI	L
65	y	y	(0.17)	nr	nr	nr	nr	nr	nr	nr
46	y	y	(0.17)	0.27	135	18	9	5	1	1
57	n	nr		nr	nr	nr	nr	nr	nr	nr
30	n	nr		nr	nr	nr	52	0	14	0
69	y	y		0.35	nr	nr	31	24	6	13
119	n	nr		0.37	nr	18	101	0	0	3
16	n	nr		nr	nr	nr	*	*	*	*
8	y	y		*	*	*	101	37	5	2
105	y	y	(0.40)	0.40	111	16	158	52	21	13
129	n	nr		nr	nr	23	37	0	4	0
104	y	n		nr	nr	*	52	12	3	0
73	y	y		nr	nr	19	82	0	14	4
3	n	nr		0.37	nr	19	93	0	7	0
40	y	y		nr	nr	nr	nr	nr	nr	nr
91	n	nr		0.36	nr	21	35	0	2	0
49	y	y		0.28	nr	nr	25	0	5	5
144	y	y		nr	nr	nr	nr	nr	nr	nr
88	y	y		0.29	121	25	34	2	13	0
98	y	n		0.26	nr	19	50	0	0	0
138	y	y		nr	nr	24	9	22	4	0
134	y	n		0.36	nr	18	141	0	14	63

+ EF, normal >50%; EDVI, normal range approximately 55-90 ml/m^2; LVEDP, normal < 12 mm Hg.
* complete data in original report

The descriptions of left ventricular function are difficult to compare since: 1) Many reports (33%) do not explicitly define criteria for the diagnosis of aneurysm. Some series explicitly exclude patients with akinesis, but others include a significant number of patients with akinesis. Only a few include explicit descriptions of aneurysm size. 2) The majority of reports do not include a description of standard measurements of global left ventricular function such as ejection fraction, ventricular volumes, and ventricular pressures. 3) The patient

populations may not be comparable. For example, some reports include a small number of patients with additional procedures such as mitral valve replacement; other reports focus exclusively on patients with ventricular arrhythmias or 3 vessel coronary artery disease.

Surgical Considerations

Most patients undergoing aneurysmectomy will have CABG, and the usual procedures (median sternotomy, saphenous vein harvesting, etc.) will be followed. During the procedure, manipulation of the left ventricle (e.g., dissection of pericardial adhesions) is minimized until the aorta is cross clamped. This should minimize dislodging thrombi into the systemic circulation. Generally, cardiopulmonary bypass is initiated without using a left ventricular vent. After the patient and heart are cooled to 25°C, the aneurysm is opened and debris removed. The entire aneurysm is resected, leaving a rim of scar tissue to anchor closure. Endocardial mapping, endocardial resection and other procedures directed at arrhythmias are performed at this point. The ventricle is closed, air is evacuated, and bypass grafts are placed in the usual manner.

Alternatives to aneurysm resection and repair have been suggested. Plication refers to pinching the aneurysm at its base and excluding it from communication with the left ventricle. Although the procedure has been widely used (Table 5), specific indications for this procedure are unclear. Another approach is patch repair of the ventricle which leaves an akinetic segment to replace the aneurysm. Magovern and colleagues have reported results with patch myoplasty (89,90,93). In addition to these investigational techniques, Jatene has suggested that careful attention to preserving normal left ventricular geometry will improve post operative left ventricular performance (66).

As an alternative to subendocardial resection for ventricular arrhythmias, other procedures have been suggested and are ivestigational. Guairdon (56) suggested non-directed encircling ventriculotomy in 1978 (before publication of subendocardial resection results), but the procedure has not become widely accepted. Cryoablation and laser irradiation to destroy abnormal tissue are also practiced (132). Extensive blind subendocardial excision has been advocated for patients who cannot be mapped (80).

Complete coronary revascularization is essential. Vauthey, et al. and Walker et al. recommend preserving, if possible, the aneurysm related artery and performing bypass grafting of that artery (134,138).

Mitral valve repair or replacement is occasionally necessary. The frequency and severity of associated mitral valve disease is not well documented, but aneurysm involving the inferior or lateral walls of the left ventricle (i.e., at the insertion of the papillary muscles) may be particularly prone to significant regurgitation.

In 1985 the United States Food and Drug Administration approved the automatic implantable cardioverter-defibrillator (AICD) for survivors of cardiac arrest or patients with ventricular tachycardia which cannot be controlled by medical therapy. The availability of this device is important since it may be used in patients whose ventricular arrhythmias are not controlled by surgery. In this case, recurrent intractable arrhythmias may be managed by a subcostal insertion of the patches. The

effect of patches on graft patency is unknown. Rarely, constrictive pericarditis has been reported and implant infection occurs in about 2% (142)

Results of Surgery: Mortality and Functional Class

Among the 20 studies which reported the surgical procedure, plication was used in 10, and the majority had simultaneous CABG. Among 2005 patients, 208 died in the first 30 days after surgery or were considered operative deaths, for an overall mortality rate of 10%. In the CASS study, operative mortality was about 8% (40). Both probably overestimate current rates because 1) significant recent improvements in technique, and 2) some studies reported very high risk patients.

Table 11. Operation, mortality, and late functional class. Surgical procedure refers to the number of patients undergoing aneurysmectomy (An), plication (Pl), coronary artery bypass surgery (CABG) and the use of directed endocardial mapping (D?). Arrhythmia deaths refers to the number of early deaths (ED, within 30 days of surgery or attributed to surgery) and late deaths (LD) which were attributed to ventricular arrhythmias.

Surgical ref.	procedure An	Pl	CABG	(frac)	D?	#ED	mort. (frac)	arrhythmia deaths ED	LD	#LD	death due to arrhythmia	Late FC I-II
65	70	0	31	0.44	n	10	0.143	0	0	5	0.00	0.93
46	11	4	13	0.87	n	0	0.000	0	0	1	0.00	0.53
57	101	0	nr	0.00	y	14	0.139	6	nr	nr	0.43	nr
30	66	0	62	0.94	n	5	0.076	0	3	4	0.33	0.95
69	42	32	72	0.97	n	2	0.027	nr	nr	4	nr	*
119	104	0	104	1.00	n	8	0.077	1	2	14	0.14	0.95
16	nr	nr	nr	nr	n	16	0.190	5	7	14	0.40	*
8	138	7	113	0.78	n	22	0.152	6	8	44	0.21	0.87
105	208	36	218	0.89	n	26	0.107	2	nr	43	0.03	0.83
129	41	0	37	0.90	n	5	0.122	0	2	15	0.10	nr
104	58	9	56	0.84	n	6	0.090	1	4	14	0.25	0.80
73	97	3	60	0.60	n	7	0.070	2	*	*	0.29	0.82
3	100	0	97	0.97	n	2	0.020	2	1	13	0.20	*
40	238	0	208	0.87	n	21	0.088	*	+	*	nr	*
91	37	0	24	0.65	n	11	0.297	nr	nr	nr	nr	nr
49	35	0	15	0.43	y	6	0.171	0	0	0	0.00	0.68
144	111	43	89	0.58	n	19	0.123	nr	nr	nr	nr	0.96
88	43	6	38	0.78	n	4	0.082	0	2	11	0.13	*
98	50	0	nr	nr	y	nr	0.000	nr	nr	nr	nr	nr
138	28	7	35	1.00	n	6	0.171	1	2	9	0.20	nr
134	181	65	246	1.00	n	18	0.073	1	5	56	0.08	nr

* details in study

\+ 30% incidence of late sudden death

Several factors may influence surgical mortality. 1) Poor function of the residual myocardium (manifest either as severe heart failure or estimated EF of the residual myocardium) is associated with higher operative mortality (77). 2) The extent of revascularization may also play a role, although this is controversial (105,138). A higher risk has also been attributed to the use of plication (105). Since thrombus is present in 50% of aneurysms, open resection is theoretically safer. 4) Cold crystalloid cardioplegia may improve myocardial protection, although randomized studies in aneurysmectomy have not been performed. 5) Anterior aneurysm associated with septal aneurysm suggests a large volume of involve myocardium, and aneurysmectomy may be contraindicated in these patients (102).

An important observation from Table 5 is the frequency of death attributed to ventricular arrhythmias in a population which was not selected for preoperative ventricular arrhythmias (i.e., excluding the studies by Harken, Garan and Miller). Other studies (40,69,73,91,105,144) were excluded because of the number of late deaths which could be attributed to arrhythmias were not reported. Of the remaing studies, there were 102 early deaths and 200 late deaths. Death was sudden or attributed to arrhythmias in 53 or about 17%. The mechanism of late post operative sudden death is unknown. The similarity in the rate of sudden death among patients treatd with aneurysmectomy compared to bypass alone in CASS suggests that the risk of sudden death is not due to aneurysmectomy itself and is consistent with the notion that the substrate for VE is present in many patients. However, potential benefits of aneurysmectomy may be balanced by inadequate revascularization, new myocardial injury due to inadequated protection, etc.

Results of Surgery: Left Ventricular Function

Several groups have evaluated left ventricular function and exercise capacity before and after aneurysmectomy (46,72,109,131). These studies evaluated most patients at the same time postoperatively. Results are mixed. Froelich et al. (46) found no change in radionuclide ventriculographic EF, although functional class improved with resection. Many of these patients had small aneurysm and normal LVEDP prior to surgery. Stephens et al. (131) could not show improved functional class in their patients. Palatianos (109) found that patients with NYHA Class IV heart failure showed small improvements in EF and hemodynamic, but no improvement if heart failure were not present. The most thorough study by Kawachi et al (72) included complete right and left heart catheterization including measurement of myocardial oxygen consumption before and after surgery. Results are summarized in Table 12. In thin small groups of patients aneurysmectomy plus CABG had dramatic hemodynamic benefits.

Table 12. Influence of aneurysmectomy on left ventricular performance (72).

myocardial oxygen consumption (ml/min)	17.2 ± 7.3	10.5 ± 3.4	<0.05
heart rate (beats/min)	67 ± 12	74 ± 13	<0.05
stroke volume index (ml/m^2)	34 ± 6	41 ± 11	ns
ejection fraction	0.31 ± 0.08	0.49 ± 0.11	<0.01
EDVI (ml/m^2)	166 ± 39	120 ± 36	<0.01
EDP (mm Hg)	18 ± 8	14 ± 7	ns

8. SUMMARY

Segmental left ventricular function abnormalities in patients with coronary artery disease represents a continuous spectrum of abnormalities; LVA represents an extreme. In spite of the extensive literature on surgical treatment of LVA, it is often difficult to compare studies because of differences (or lack) of definition of LVA. At a minimum, we should distinguish between an akinetic and dyskinetic aneurysm.

A significant aneurysm involves 20% or more of the left ventricular surface area. Experimental data suggest that reaction of akinetic region will not improve LV function, and clinical studies show a clear relation between a good surgical result and good function of the uninvolved myocardium. In sum, a patient should probably be considered for aneurysmectomy only if 1) the aneurysm is large (at least 20% or more of the left ventricle); 2) the aneurysm is dyskinetic; and 3) function of residual myocardium is good.

The traditional indications for aneurysmectomy should be re-evaluated.

Arterial emboli are rare in patients with dyskinetic LVA. These patients are likely to have associated vascular disease which will confuse evaluation. Little information is available on patients treated by aneurysmectomy for embolic disease. Therefore, aneurysmectomy should be reserved for a very small fraction of patients who have failed anticoagulant therapy after emboli which appear to originate in the ventricle. In general, arterial emboli should not be an indication for aneurysmectomy.

Intractable ventricular arrhythmias should not be considered an indication for aneurysmectomy. These patients should undergo directed subendocardial resection; aneurysm resection is incidental to the primary procedure.

Angina should not be considered a primary indication for aneurysmectomy since the majority of patients have significant coronary disease and will benefit from bypass surgery alone. A possible exception might be severe angina with minimal coronary disease associated with a large, discrete, dyskinetic aneurysm.

Medically refractory congestive heart failure is the only syndrome for which there is reasonable clinical and physiological evidence for benefit due to aneurysmectomy.

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