Indications for Left Ventricular Aneurysmectomy

Marc Cohen, M.D., Milton Packer, M.D., and Richard Gorlin, M.D.

MORE THAN 25 years ago, C.P. Bailey performed the first successful left ventricular aneurysmectomy in a human being.\(^1\) Since then, with cardiopulmonary bypass and improved surgical techniques, aneurysmectomy has become an established procedure available to patients with ventricular aneurysms and severe symptoms.

In this paper we review the available literature concerned with elective left ventricular aneurysmectomy in the setting of chronic ischemic heart disease. We compare theoretical models and predictions with the empirical observations made by previous investigators and suggest an approach to the symptomatic patient with a left ventricular aneurysm.

Definitions

For the purpose of this discussion, an aneurysm is identified by a left ventricular angiogram as any akinetic or dyskinetic segment of myocardium.\(^2\) An akinetic segment is defined as a segment that appears to have no motion during systole, whereas a dyskinetic segment appears to bulge paradoxically during systole.\(^3\) Intraoperatively, an aneurysm is identified as a circumscribed area of scar, which is thin, often adherent to the pericardium and which may or may not bulge paradoxically during systole.\(^3\) The aneurysmal segment is easily outlined by looking for the area that puckers and collapses when the left ventricle is vented. This cineangiographic and surgical definition of ventricular aneurysm is compatible with the pathologic definition used by Schlichter et al.\(^4\) They defined an aneurysm as ‘‘a localized outpouching of the cavity of a cardiac chamber, with or without outward bulging of the external surface.’’

Historical Background

The idea that surgical exclusion of a ventricular aneurysm was a desirable goal was based on work by several investigators.\(^4\)–\(^7\) They portrayed the development of an aneurysm as a tragic sequel to acute myocardial infarction. In 1947, Murray\(^7\) successfully reduced acute mortality in a dog model of acute myocardial infarction by simple surgical exclusion of the dyskinetic infarct segment. Schlichter et al.\(^4\) reviewed the clinical course of 102 patients with autopsy-proved left ventricular aneurysms and observed a greater than 80% 5-year mortality and a high prevalence of thromboemboli. Years later, several workers drew attention to the increased burden placed on the heart by an increase in diastolic radius.\(^8\)–\(^9\)

These observations provided the impetus to search for a surgical approach to ventricular aneurysms. Beck’s pilot operation in 1944\(^10\) and Bailey’s successful aneurysmectomy in a patient with refractory congestive heart failure\(^1\) were followed in 1958 by Cooley’s first aneurysmectomy using total cardiopulmonary bypass.\(^11\) The accessibility to the aneurysm provided by cardiopulmonary bypass and the initial encouraging clinical results made left ventricular aneurysmectomy an accepted therapeutic option.

Natural History

Critical evaluation of the benefits of aneurysmectomy requires a thorough understanding of the natural history and hemodynamic characteristics of patients with ventricular aneurysms in the setting of chronic ischemic heart disease. Specifically, one must answer the following question: Is the morbidity and mortality of patients with coronary artery disease and ventricular aneurysms the result of specific liabilities conferred on them by the presence of the aneurysm? Or is the patient’s clinical course simply a function of the extent of the coronary artery disease and the amount of infarcted myocardium?

In contrast to earlier autopsy studies that showed a 5-year mortality rate of approximately 80%, Abrams et al.\(^12\) observed a 30% 5-year mortality rate in 65 patients with ventricular aneurysms at autopsy; acute myocardial infarction, not refractory heart failure, was the most common cause of death. All of these autopsy studies, however, were retrospective, estimated the age of aneurysm without the benefit of angiographic data, and limited their observations to patients who had a discrete outpouching of the ventricular cavity.

Several years later, Gorlin et al.,\(^1\) using the angiographic criteria of akinesis or dyskinesis, undertook a prospective study of 24 patients. Their observations suggested that recurrent infarction, not congestive heart failure, was the most common cause of death. Mourdjinis et al.\(^13\) conducted a prospective study of patients with radiographic evidence of ventricular aneurysm after myocardial infarction. They observed an approximately 50% 5-year mortality rate in patients with large aneurysms, and a less than 10% 5-year mortality rate in patients with small aneurysms. The majority of the deaths were attributed to recurrent infarction.

In their 10-year follow up of 601 patients who did not have open heart surgery, Proudfit et al.\(^14\) observed different survival curves for patients with different ventriculographic findings. Patients with aneurysms, i.e., ‘‘definite paradoxical bulging during systole,’’ had a 5-year mortality rate of approximately 50%; pa-

From the Division of Cardiology, Department of Medicine, Mount Sinai Medical Center, New York, New York.

Address for correspondence: Marc Cohen, M.D., Division of Cardiology, Mount Sinai Medical Center, One Gustave L. Levy Place, New York, New York 10029.

Received August 19, 1982; revision accepted November 3, 1982.

Circulation 67, No. 4, 1983.

717
tients with a scar, i.e., a hypokinetic or akinetic segment, had a 30% 5-year mortality rate. Recent data from the Coronary Artery Surgery Study (CASS) suggest that the natural history of patients with angiographically defined aneurysms is a function of overall ventricular performance.\textsuperscript{15} Given similar degrees of left ventricular dysfunction, the presence of an aneurysm did not add to the risk of mortality. These more recent studies suggest that the natural history of patients with ventricular aneurysms is not as poor as the earlier autopsy studies suggested.

**Hemodynamics**

**Pathophysiologic Factors**

At least three factors may affect left ventricular performance and result in symptoms in patients with a left ventricular aneurysm: a decrease in left ventricular systolic function consequent to myocardial infarction and enhanced systolic wall stress, secondary mitral regurgitation, and interference with ventricular diastolic function.

Klein et al.,\textsuperscript{16} combining theoretical models and empirical observations, delineated the hemodynamic alterations observed in patients with ventricular aneurysms. Their results are supported by the observations of Feild et al.,\textsuperscript{17} who studied patients with akinetic and/or dyskinetic segments after myocardial infarction. Using angiographic variables to approximate the relative size of the akinetic or dyskinetic segment, both groups independently observed that once 17–20\% of the ventricle becomes nonfunctional, hemodynamic evidence of pump failure is unavoidable. Pump failure is defined as delivery of a lower-than-normal stroke volume in the setting of a higher-than-normal end-diastolic pressure or end-diastolic volume. Both end-diastolic pressure and volume increase in proportion to the relative size of the aneurysm. Although an increase in end-diastolic volume should enable the residual contractile segments to maximize the stroke volume for any given percent fiber shortening, these changes in end-diastolic volume, whether adaptive or not, result in an increased wall stress and, thereby, a greater impedance to ejection and may further decrease systolic shortening. In addition, enhanced wall stress may increase myocardial oxygen requirements and may provoke ischemic dysfunction in residual (nonaneurysmal) segments perfused by diseased coronary arteries; subendocardial perfusion to such segments may be additionally compromised by the increased end-diastolic pressure.\textsuperscript{18} Consequently, the patency of the coronary arteries perfusing these residual segments assumes major significance. The increased afterload and possible ischemic dysfunction of residual segments may explain the lower-than-expected stroke volumes in patients with left ventricular aneurysms.

Further compromise of forward output and exacerbation of clinical symptoms may result from mitral regurgitation. Depending on its location and on its size, an aneurysm may interfere with papillary muscle function; this may occur directly because of its location, or indirectly because of left ventricular dilation.\textsuperscript{19, 20}

Ventricular aneurysms may alter diastolic pressure-volume relationships sufficiently to cause symptoms by increasing left ventricular diastolic pressures disproportionate to the degree of left ventricular dysfunction. Most aneurysmal segments are fibrous and noncompliant regardless of size;\textsuperscript{21} in addition, an aneurysm may induce changes in adjacent segments so as to alter their diastolic properties. However, little work has been done to define such effects before or after aneurysm resection.

Alternatively, ventricular aneurysms may have additional effects on cardiac performance. Right ventricular filling and performance may be hampered by septal aneurysms and by the volume consumed by a relatively large aneurysm within the pericardial sac. Furthermore, while most patients with aneurysms have an expanded ventricular volume, some patients maintain a normal volume. In this subset of patients, wall stress reduction by aneurysmectomy may not be beneficial.

**Models of Akinesia and Dyskinesia**

The hemodynamic alterations outlined above pertain in a general sense to both akinetic and dyskinetic aneurysms. However, based on the work of Parmley et al.,\textsuperscript{22} it seems likely that most aneurysms secondary to chronic ischemic heart disease are fibrous and noncompliant, i.e., akinetic aneurysms. It is of value, therefore, to compare hemodynamic alterations in the presence of akinesia vs dyskinesia.

Tyson et al.\textsuperscript{23} compared distensible and nondistensible “aneurysmal” sacs in a dog model. They observed significant depression in left ventricular performance only in the dogs with distensible sacs. The lack of a measurable decrease in ventricular performance in the dogs who had nondistensible sacs was not proved because changes in myocardial oxygen consumption were not measured. Païrolero et al.\textsuperscript{24} did not detect any major improvement in hemodynamic values after excision of akinetic segments in their dog model.

Thus, we may infer that patients with dyskinetic segments are at a distinct disadvantage, and predict improvement in these patients after aneurysmectomy. The natural history and theoretical data with regard to patients with simple akinesia is insufficient to allow a confident prediction as to the benefits of aneurysmectomy.

**Indications**

**Congestive Heart Failure**

The goal of aneurysmectomy in the patient with severe congestive heart failure and an increased ventricular volume is to reduce the end-diastolic volume of the left ventricle. This would reduce regional wall stress in the residual contractile segments and minimize mitral regurgitation, and thereby improve cardiac performance. In fact, most investigators observed a distinct improvement in clinical symptoms in the majority of their patients with severe congestive heart failure who underwent aneurysmectomy.\textsuperscript{1, 5, 11, 24-25} The New York Heart Association class usually improves
from III or IV to I or II and most patients no longer use
digoxin or diuretics. Objective assessment of improve-
ment of clinical symptoms by exercise testing was per-
formed by Cullhed et al.33 They observed an im-
provement in exercise tolerance after aneurysmec-
tomy in several patients. Best results are obtained in patients
with good residual contractile function, isolated left
anterior descending disease and a discrete apical aneu-
rysms.3, 39, 43-47 Such patients have a 3-5% operative
mortality.39

In contrast, patients with abnormal residual contrac-
tile function may require heightened preload to main-
tain forward output. Decreasing end-diastolic volume
in this group of patients, by aneurysmectomy, may
compromise cardiac output. The ideal balance be-
tween the benefits derived from wall stress reduction
by decreasing end-diastolic volume and the necessity
of maintaining an adequate preload varies from person
to person. Therefore, aneurysmectomy in these pa-
tients is likely to be associated with variable results.
Indeed, increased operative mortality and less substan-
tial clinical improvement is seen with incomplete
revascularization in the setting of multivessel dis-
ease and abnormal residual contractile func-
tion.3, 24, 26, 30, 36, 39, 43, 46-49 Operative mortality in
patients with multivessel disease and impaired residual
contractile function is about 12%, with a 7-year survi-
aval of about 55%.3 Patients with refractory ventricular
tachycardia have an operative mortality rate of greater
than 15%.3

To our knowledge, no one has compared patients
with akinesis vs dyskinesis with respect to clinical
response to aneurysmectomy.

In addition to clinical variables, several groups have
also evaluated hemodynamic variables before and after
aneurysmectomy.31, 33, 35, 37, 38, 41, 42, 45, 50-52 Some in-
vestigators found a significant improvement, some no
change, and some significant deterioration in ventricu-
lar performance. The studies all suffer from at least one
of a number of major deficiencies; sample size was
small, only two were prospective, not all the patients
studied had the same operation, reevaluation was done
at different times postoperatively.

Kitamura et al.42 reported an increase in stroke vol-
ume at lower end-diastolic pressures in six of nine
patients after aneurysmectomy. Five of the six patients
who improved had dyskinetic aneurysms. The change
in mean stroke volume and mean end-diastolic pres-
sure, however, did not reach statistical significance,
and the authors used levoephase angiograms in the ma-
jority of their patients. Otterstad et al.41 observed dra-
matic decreases in left ventricular end-diastolic pres-
sure in the majority of his patients postoperatively;
however, no mention was made of changes in stroke
volume. In addition, a few patients who had progres-
sive coronary artery disease at the time of their reeval-
uation had increases in their end-diastolic pressure.
The prospective trial of Stephens et al.49 demonstrated
a significant decrease in end-diastolic pressure without
a loss in stroke volume. Of interest, they did not ob-
serve any change in exercise tolerance of their patients
postoperatively. Schonbeck et al.32 studied 13 patients
undergoing aneurysmectomy and coronary artery by-
pass surgery and observed an increase in the mean
value for stroke volume at the same end-diastolic pres-
sure. Lefemine et al.37 obtained pre- and postoperative
hemodynamic data on 10 of 50 patients who underwent
aneurysmectomy. Eight of these 10 patients had a
greater stroke volume at the same or lower end-diastol-
IC pressure. Because only one in five of their patients
was reevaluated postoperatively, bias in patient selec-
tion may have influenced their results. Schimert et al.35
observed distinct clinical improvement in three pa-
tients with akinetic aneurysms. Postoperative angi-
ography in these three patients demonstrated significant
reductions in mitral regurgitation compared with the
aneurysmectomy angiogram.

In contrast to these encouraging results, other inves-
tigators observed mixed results.31, 33, 35, 45, 50-52 Cullhed
et al.33 reviewed 14 patients postoperatively, of whom
seven had adequate pre- and postoperative hemody-
namic evaluation. Four of the seven patients had an
increase in stroke volume at a lower end-diastolic pres-
sure, while three of the seven experienced a deteriora-
tion in left ventricular performance. Sesto and his col-
leagues51 studied six patients with isolated left anterior
descending disease and anteroapical aneurysm 6-10
months after aneurysmectomy; they observed no sig-
nificant changes in either left ventricular end-diastolic
pressure or ejection fraction. Given the significant de-
crease in end-diastolic volume after aneurysmectomy,
the absence of any change in the ejection fraction
implies a decrease in stroke volume. The decrease in
end-diastolic volume without a concurrent decrease in
end-diastolic pressure suggests a shift in the pressure-
volume curve to the left. However, neither stroke vol-
ume nor ventricular compliance were measured direct-
ly. In a prospective study, Froehlich et al.42 measured
ejection fraction by isotope ventriculogram in 15 pa-
tients before and after aneurysmectomy and bypass
surgery. Despite dramatic improvement in New York
Heart Association functional class, no change in mean
ejection fraction was observed. However, almost one-
third of the patients had a normal preoperative end-
diastolic pressure and at operation were found to have
small aneurysms. It is difficult, therefore, to make
inferences based on this small patient sample.

Refractory Ventricular Tachycardia

Four years after Bailey's first successful aneurys-
mectomy for control of congestive heart failure, Couch44
reported the first successful aneurysmectomy for control of disabling ventricular tachycardia. Subse-
quent reports55-59 suggested that simple aneurysmec-
tomy was a reliable final alternative for controlling
refractory malignant arrhythmias.

Overshadowing the optimism was the significant
operative mortality — usually greater than 15% — and
the fact that although patient's symptoms improved,
ectopy and ventricular tachycardia were still observed
on monitoring.3, 38-41 Sami et al.60 using postoperative
Holter monitoring, observed numerous multiform
beats in seven of eight patients and subclinical ventricular tachycardia in three of eight patients who had undergone aneurysmectomy for disabling recurrent arrhythmias. Since simple aneurysmectomy left behind a rim of scar for anchoring sutures, did not involve surgical manipulation of a septal scar, and did not include excision of the border zone of the aneurysms, some investigators felt that different surgical techniques might result in more reliable abolition of arrhythmias.62-64

Based on the hypothesis that reentry was the mechanism for recurrent sustained ventricular tachycardia, several approaches were developed that were designed to interrupt the reentry circuit.62-66 Giraudon et al.63 advanced the technique of encircling ventriculotomy, which presumably interrupts any circuit between the aneurysm and its border zone and the rest of the ventricle. Other investigators attempted to interrupt the reentry circuit by pinpointing the area of earliest activation during iatrogenically stimulated ventricular tachycardia and then resecting it.65, 66

Initially, Gallagher et al.62 used epicardial mapping to pinpoint the area of earliest activation. More recently, Josephson et al.64 developed a body of evidence suggesting that endocardial mapping identifies the area of earliest activation more reliably, and that this area is usually in the border zone of the aneurysm. They also suggested that endocardial excision, as opposed to full-thickness resection, is adequate in preventing recurrent arrhythmias.64, 67, 68 Wiener et al.69 used endocardial mapping during sinus rhythm to direct the endocardial resection. The use of endocardial mapping and resection has, for the first time, allowed exclusion of areas of abnormal electrical activity located in the septum.

The vast majority of patients studied had recurrent sustained ventricular tachycardia. Too few patients with ventricular fibrillation or recurrent nonsustained ventricular tachycardia have been studied to allow conclusions about these subsets. In addition, aneurysmectomy for refractory malignant arrhythmias within 1 month of an acute myocardial infarction has such a high mortality — 60% — that it should be considered a heroic intervention.61

Recurrent Thromboembolism

Early autopsy studies of patients with a left ventricular aneurysm suggested that there was a high prevalence of mural thrombi (greater than 50%) and an alarmingly high incidence of peripheral thromboembolism (greater than 50%).6, 7 Several years later, however, Abrams et al.12 suggested that thromboembolism was not so common and urged caution in suggesting aneurysmectomy to prevent thromboembolism.

Despite the high prevalence of mural thrombi, all current investigators observe clinical episodes of thromboembolism with a frequency of approximately 5%.5, 70, 71 In addition, most of those who sustained a systemic embolism were not taking anticoagulants. Therefore, the anticoagulant therapy should be evaluated before committing patients with mural thrombus to open heart surgery. It is conceivable that a rare patient with a thrombus tenuously anchored by a thin pedicle may benefit from prophylactic surgery.

Nuclear imaging and two-dimensional echocardiography may become useful in identifying patients with aneurysms and large mural thrombus, and thereby allow prospective evaluation of their natural history.72, 73

Refractory Angina

Aneurysmectomy alone has been suggested as an alternative for the patient with one-vessel disease, an aneurysm and refractory angina. Loop et al.3 observed relief of symptoms for almost 5 years after aneurysmectomy alone in 32 of 46 patients presenting with apical aneurysms and isolated left anterior descending occlusion. Recurrent ischemia secondary to coronary artery occlusion was considered unlikely as the cause for their anginal syndrome. Lee et al.74 described a detailed case history of such a patient with a large dysskinetic aneurysm. Improved hemodynamics might explain the improvement. However, given the well known placebo effect of surgery, it is impossible to evaluate critically the role of aneurysmectomy for refractory chest pain.75, 76

Cardiac Rupture

We are aware of only one published report that describes the rupture of a true ventricular aneurysm at least 1 month after acute myocardial infarction.77 In contrast, patients with false ventricular aneurysms after myocardial infarction are at a significant risk for sudden death from cardiac rupture.78 It is of historical interest that the first operation performed on a ventricular aneurysm was done with the intent of preventing cardiac rupture.10

Guidelines to Therapy for Ventricular Aneurysm

We have formulated a current approach to patients with ventricular aneurysms secondary to ischemic heart disease who have refractory symptoms. In view of the significant operative mortality, which ranges from 3% to 50%, patients should be considered for aneurysmectomy only after maximal medical therapy has failed.

Aneurysmectomy must be supplemented with complete revascularization of any diseased and graftable coronary arteries. Operative mortality and long-term survival are critically dependent on complete revascularization. Much of the symptomatic relief attributed to aneurysmectomy may be partially or completely the result of successful coronary artery bypass grafting.

The majority of patients with refractory congestive heart failure experience relief of symptoms. Patients with discrete dysskinetic aneurysms are more likely to improve symptomatically and to have objective hemodynamic improvement than patients with akinetic aneurysms.

Current investigations suggest that patients with refractory ventricular tachycardia are more likely to experience relief from disabling symptoms by undergoing electrophysiologically guided endocardial resection or encircling ventriculotomy rather than simple aneurysmectomy.
Careful dissection and aneurysmectomy can safely remove mural thrombi, but the low frequency of clinically apparent emboli makes aneurysmectomy appropriate only after consideration of anticoagulant therapy. Most patients with aneurysms have akinetic aneurysms and the majority of patients currently considered for aneurysmectomy are suffering from severe congestive heart failure. Therefore, the main issue remains unanswered: Does removal of an akinetic aneurysm improve overall myocardial performance? The patient with a large akinetic aneurysm and good residual function usually improves with aneurysmectomy. The patient with an akinetic aneurysm and poor residual function is probably a candidate for wall stress reduction using systemic vasodilators. Appropriate vasodilators in this type of patient would permit stress reduction without compromising preload. 79, 80

Currently, any open heart surgery includes coronary artery bypass if necessary. Therefore, apart from the patient with an isolated occlusion of the left anterior descending artery with anteropapical aneurysm, it will be impossible to evaluate the effect of aneurysmectomy alone on left ventricular performance because of the potential benefits of coronary artery bypass grafting on symptoms and on left ventricular function. Multicenter trials could accumulate enough prospective data on patients who have combined procedures. However, it would probably be very difficult to recruit patients with severe heart failure for a controlled trial offering coronary bypass and aneurysmectomy in one treatment arm and coronary artery bypass alone in the other arm.

Acknowledgment

The authors express their appreciation to Dr. R.S. Litwak, Chief of Cardiothoracic Surgery, for his critical review of the manuscript and to Ruby S. Gordon for her help in preparing the manuscript.

References

52. Kitamura S: Magnitude, time course, and mechanisms of functional alterations after excision of chronic left ventricular aneurysm or large scarred myocardium following myocardial infarction. Jpn Assoc Thorac Surg 24: 1343, 1976
66. Wellens HJJ, Lie KL, Durrer D: Further observations on ventricular tachycardia as studied by electrical stimulation of the heart: chronic recurrent ventricular tachycardia and ventricular tachycardia during acute myocardial infarction. Circulation 49: 647, 1974
77. Betsch WF: Cardiac aneurysm with spontaneous rupture. Am Heart J 30: 567, 1945
Indications for left ventricular aneurysmectomy.
M Cohen, M Packer and R Gorlin

Circulation. 1983;67:717-722
doi: 10.1161/01.CIR.67.4.717

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1983 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/67/4/717.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/