Ventricular aneurysm is not so unusual an entity as might be supposed. The lesion is said to have been described first by John Hunter in 1757. For many generations, its recognition by the clinician has been considered a diagnostic feat, though this was of little help to the suffering patient. With the recent advent of open-heart surgery and its application to various forms of acquired heart disease, the recognition of ventricular aneurysm as a clinical entity has become mandatory. This acquired defect of the heart is correctable and the patient, if not actually cured, may receive significant palliation.

Surgical efforts to remove ventricular aneurysm were few and poorly rewarded until 1957 when Cooley and associates employed extracorporeal circulation with a pump-oxygenator. Since that time, successful operations for ventricular aneurysm have been reported by Lillehei and coworkers, Lam and colleagues and Effler and associates.

Etiopathologic Features

Ventricular aneurysm results from severe myocardial infarction. The patient who survives massive destruction of ventricular myocardium acquires a distinct entity that is recognizable by fluoroscopic study and by left heart catheterization with ventriculography.

Traditionally, ventricular aneurysm has been associated with syphilis, rheumatic myocarditis, tuberculosis, trauma, and other etiologic factors. From a practical standpoint, however, coronary atherosclerosis that results in myocardial infarction is the causative factor responsible for this entity. The sequence of events that leads to the development of ventricular aneurysm and its clinical impact upon the patient is easily followed.

Selective coronary arteriography (Sones' technic) has made individual evaluation of the patient with coronary arterial disease a practical procedure. This technic combined with left ventriculography is invaluable in assessing the patient who demonstrates ventricular aneurysm. Most of the patients who present ventricular aneurysm have sustained total occlusion of the anterior descending branch of the left coronary artery. In our series of cases there have been two exceptions where aneurysm has followed occlusion of the circumflex branch of the left coronary artery. The numerical preponderance of left anterior descending occlusions is self-explanatory as one considers the anatomy involved.

Acute myocardial infarction that follows occlusion of the left main coronary artery may result in virtual death of the anterior wall of the left ventricle and much of the intraventricular septum. The fact that the patient may sustain life during the weeks and months that follow the acute episode is in itself remarkable. Total destruction of the involved myocardium is attested to by direct inspection of the surgical specimen that features clot and fibrous replacement of left ventricular myocardium and the adjacent septum. The extent of the infarction and the attendant myocardial destruction are variable; much depends upon availability of collateral support from the circumflex branch of the left and right coronary arteries. In the extreme case, a ventricular aneurysm may represent a loss of almost half of the left ventricular myocardium.

Although coronary atherosclerosis usually is a diffuse disease that produces infarction in almost any area of ventricular
myocardium, ventricular aneurysm most frequently is limited to the anterior surface of the left ventricle, because this disease is determined by the patient's ability to survive the initial insult until fibrous tissue replaces the destroyed myocardium. The anterior wall of the left ventricle and the adjacent interventricular septum are free of significant attachments and conduction pathways; hence, if the heart is normal otherwise, the patient may survive the initial insult of massive myocardial infarction, and an acquired aneurysm develops. This is not the case when the infarction occurs on the posterior wall of the left ventricle. The posterior wall of the left ventricle moors the papillary muscle that supports the mitral valve, and massive infarction in

**Figure 1**: Schematic drawing of the operation for excision of left ventricular aneurysm. (A) The basic cannulations require two vena cava cannulas introduced through the right atrium, and a left-heart cannula inserted through the right superior pulmonary vein into the left atrium. This third venous cannula maintains decompression of the left heart and protects the left ventricle from overdistension if ventricular arrhythmia occurs before extracorporeal circulation is withdrawn. The arterial line is introduced into the left common femoral artery as illustrated. (B) Ventricular aneurysmectomy is performed while the patient's circulation is supported by the pump-oxygenator. The bulging aneurysm is incised directly; the fibrous wall of the ventricular aneurysm is trimmed away; and laminated clot is removed from within the left ventricle itself. It is not necessary to occlude the aorta, as the elevated position of the apex prevents air embolus. (C) Linear reconstruction of the ventricle is accomplished by a two-layer direct suture technic.
this area produces necrosis or atrophy of the papillary muscle, and impairs function of the mitral valve. The acutely acquired mitral insufficiency presents an unbearable burden to the patient with acute myocardial infarction. Therefore, the usual location of ventricular aneurysm on the anterior wall of the left ventricle is explained by the simple fact that the patient can tolerate anatomic and physiologic loss of this area.

**Clinical Features**

The patient with ventricular aneurysm is chronically ill; he requires constant medical supervision and therapy. The degree of disability is related to the size of the aneurysm, the amount of associated paradoxic motion, and the extent of the residual coronary disease. Characteristically, this patient requires repeated periods of hospitalization for paroxysmal ventricular arrhythmia and/or congestive cardiac failure. The classic paper by Schlichter, Hellerstein, and Katz, based on a review of 102 necropsy reports, states that 75 per cent of the patients died within three years after the initial infarction, and that 83 per cent of the deaths were related to congestive cardiac failure, thromboembolic phenomenon, or recurrent infarction.

It is often stated academically that the ventricular aneurysm is prone to rupture and to produce fatal tamponade. Such an event has not occurred in the history of the Cleveland Clinic Hospital. From surgical observation, we have found that the aneurysm consists of a tough fibrous wall supported by a thickened adherent pericardium. Within the aneurysm itself there is usually a laminated blood clot. The combination of clot, fibrous sac, and reinforcing adherent pericardium makes spontaneous rupture unlikely. Perhaps the frequent observation of fatal tamponade associated with postinfarction necrosis of the ventricular wall has been considered as a rupture of ventricular aneurysm itself. Actually postinfarction tamponade is a complication of acute myocardial infarction which occurs within days or weeks after the initial occlusion. Ventricular aneurysm, as a pathologic entity, does not exist until there is actual fibrous replacement of the destroyed myocardium.

**Surgical Treatment**

*Selection of patients.* Enthusiasm, based on results, for surgical treatment of ventricular aneurysm prompts us to consider all patients with this entity as potentially suitable for surgery. The earliest patients in

![Figure 2: Roentgenograms made before and after surgical removal of left ventricular aneurysm. The striking reduction in size of the heart plus the improvement in the pulmonary vascular markings is a frequent observation after surgical treatment.](image-url)
this series are approaching the five-year postoperative point, and show promise of continued survival. Extension of life alone is not the only benefit of surgery; the rehabilitation that may follow is sometimes dramatic.

Every patient in this series has had conventional clinical study and, in addition, has undergone selective coronary arteriography and left ventriculography. Opacification of the coronary circuit is not necessary to establish the diagnosis of ventricular aneurysm, but it does help in assessing the patient as an operative risk. If the patient demonstrates diffuse atherosclerosis in the remaining coronary circuit, his chance for survival is reduced, and expected palliation obviously is limited. Occasionally, however, both the circumflex branch of the left coronary artery and the right coronary artery are relatively free of disease; knowledge of this bolsters the need for operation, as excision of the aneurysm may be tantamount to excision of that patient's coronary arterial disease. Intractable cardiac failure associated with ventricular aneurysm is not a contraindication to surgical therapy. Currently, we advise every patient with symptomatic ventricular aneurysm to have surgical treatment regardless of his cardiac functional status.

*Surgical technic.* The initial operation in this series was based on suggestions offered by Lam. Consistently satisfactory results have prompted continued use of an operative approach that is now considered obsolete by some surgeons.

In open-heart surgery today, transverse, bilateral thoracotomy is used rarely, yet this incision is particularly applicable to the problems relative to right-heart cannulation and excision of large ventricular aneurysms. Undoubtedly most of these procedures could be accomplished with the midline sternotomy approach.

The patient is operated upon in the supine position. After heparinization, routine cannulations are made; vena cava cannulas are inserted through the right atrium; the left atrial cannula enters by way of the right superior vein, and arterial line is inserted retrograde into a femoral artery. The patient's circulation is maintained on high-flow perfusion; neither hypothermia nor cardioplegia is employed.

While the patient is supported by the pump-oxygenator, the decompressed heart is raised and cushioned by soft pads to elevate the apex and to rotate the heart counterclockwise. This brings the lateral aspect of the aneurysm into optical view. It also elevates the left ventricle to the highest

![Figure 3: (Left) Preoperative. Typical appearance of ventricular aneurysm. By fluoroscopy, the paradoxical motion, which impairs left ventricular function, is readily apparent. (Right) Postaneurysmectomy. Note reduction in heart size and alteration in cardiac contour.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21420/)
point in the circulation and thus eliminates the hazard of systemic air embolus. The midzone of the aneurysm is incised and the fibrous wall is dissected away with surgical scissors. At completion of the aneurysmectomy, all clot and virtually all fibrous replacement of anterior ventricular myocardium are excised. Usually the anterior aspect of the interventricular septum has been replaced with fibrous tissue, and it may show significant paradox. This is not disturbed, as replacement of the fibrous portion of the interventricular septum by prosthetic material would increase the risk of the procedure and, in a sense, would substitute one form of prosthetic tissue for another.

With adequate extracorporeal circulatory support for the patient, the surgeon may take as much time as needed for complete excision of the aneurysm and for functional reconstruction of the left ventricle. In the majority of cases the operation is performed while the heart is beating. Because the aortic valve is competent, occlusion of the aorta is not required; thus, myocardial perfusion continues throughout the procedure. Ventricular fibrillation that occurs during aneurysmectomy is corrected, after reconstruction of the ventriculotomy, by direct-current countershock.

At first we were concerned about the extent or limits of excision, fearing critical reduction in capacity of the reconstructed left ventricle. The concern has been unwarranted; therefore, it is our belief that the entire aneurysm, regardless of its size, must be removed to achieve the desired result. There is justification for leaving a fibrous border of tissue, as this will support sutures better than will healthy myocardium. Reconstruction of the open left ventricle after aneurysmectomy is somewhat akin to closure of an opened plant pod. The line of closure is vertical from apex to base of the heart; the suture line follows the path of the obliterated anterior descending branch of the left coronary artery. Initial closure is accomplished with a continuous running suture of nonabsorbable material that in turn is reinforced by interrupted mattress sutures. Particular care is taken to get complete hemostasis, as the contracting left ventricle will eject blood through the tiniest fistula in the suture line. When the supporting pads are removed, the ventricle falls back into the now redundant pericardial sac and the suture line can no longer be in-

Figure 4: Roentgenograms taken before and after surgery, made in the oblique projection, of the last patient included in this series, a 38-year-old man who had sustained a massive myocardial infarction six months before operation and who remained incapacitated with intractable congestive cardiac failure. Postoperative film, made six weeks after surgery, shows great improvement in cardiac silhouette. Subjective improvement has been striking in this short time.
spected without rotation of the heart.

Resection of ventricular aneurysm is re-
warded by prompt improvement in cardiac
function. There is elevation of the mean
arterial pressure and vigorous contraction
of the two ventricles. Increased cardiac ef-

ciciency is apparent in the immediate post-
operative period. For this reason, the recov-
ery period may be surprisingly short as the
patient enjoys cardiac rehabilitation.

Results

Since 1959, at the Cleveland Clinic, 29
patients received surgical consultation for
ventricular aneurysm.* Twenty-eight pa-

tients underwent surgical correction for
the disease, and one patient died before surgery
was undertaken.

Of the 28 patients, one with posterior
myocardial infarction required replacement
of the mitral valve by a Starr-Edwards
prosthesis; the infarcted papillary muscle
and chordae tendineae made the mitral
valve hopelessly incompetent. This patient
survived the operation and was discharged
from the hospital, but approximately six
weeks later died of an acute myocardial in-
farction.

Twenty-six patients have survived the
initial postoperative period; two died in the
hospital—one from renal failure associated
with long-standing hydronephrosis, and
the other from uncontrolled arrhythmia. Of
the 26 survivors, three have since died; two
succumbed to another myocardial infarc-
tion (this includes the patient mentioned in
the preceding paragraph), and in one pa-
tient a mycotic aneurysm developed. As
mentioned before, the longest survivors are
approaching their fifth year since opera-
tion, and in some, the rehabilitation has
been suggestive of surgical cure rather than
of palliation.

Comment

The life expectancy of the patient who
has a ventricular aneurysm is limited. The
patient who is severely handicapped by this
entity should be assessed in regard to mere
existence as distinguished from gainful liv-
ing. Inefficient contraction of the left ven-
tricle and impaired cardiac output render
the patient a real or potential cardiac cripp-
le. The medical therapy of the patient
with ventricular aneurysm at best supports
that portion of the myocardium spared by
previous myocardial infarction, and in no
way can be considered as definitive therapy.

The place for surgery in ventricular
aneurysm is now apparent. The patient
who demonstrates postmyocardial infa-
cction aneurysm is studied as an individual.
Selective coronary arteriography and left
ventriculography are extremely helpful, but
not mandatory, in final assessment of the
patient as a surgical subject. We have
found selective coronary arteriography to
be extraordinarily helpful in preoperative
determination of surgical risk and postope-
rate prognosis.

Among the many forms of acquired
heart disease now amenable to surgical
treatment, ventricular aneurysm may offer
the greatest yield in rehabilitation of the
patient with the least amount of surgical
effort. The surgical approach is simple; a
transverse, bilateral thoracotomy is per-
formed by the authors, but undoubtedly the
midline sternotomy approach could be
used. In the majority of instances, recovery
is prompt and gratifying. Every patient
who is known to have ventricular aneurysm
should receive consideration for elective
cardiac surgery.

Summary

Post-infarction ventricular aneurysm im-
pairs cardiac output. Fibrous replacement
of destroyed ventricular myocardium re-
sults from significant coronary occlusion. If
the involved area is of sufficient size, an
aneurysm forms and in many cases para-
diic motion is demonstrated by radio-
graphic technics. The functional impair-
ment produced by ventricular aneurysm
may render the patient a cardiac cripple
by: (a) left-heart failure, (b) paroxysmal
ventricular arrhythmia and, (c) embolic
accidents.

* An additional 11 patients have undergone sur-
gical treatment for ventricular aneurysm (be-
tween July 1, 1964 and February 5, 1965) at the
Cleveland Clinic Hospital. There was no
death in this group.
The great majority of ventricular aneurysms are found on the anterior wall of the left ventricle and the anterior aspect of the interventricular septum. Massive infarction of the posterior left ventricle involves the papillary muscles and chordae tendinae of the mitral valve; rarely will the patient survive the combined insult of massive myocardial infarction and acute mitral insufficiency.

In the authors' opinion, every patient with ventricular aneurysm is a subject for surgical treatment. Medical therapy at best can only prepare the patient for definitive therapy. Removal of the aneurysm and reconstruction of the left ventricle can be accomplished in the majority of cases with extracorporeal circulation. The authors' technics and clinical experience are presented.

**Résumé**

L'anévrisme ventriculaire consécutif à un infarctus amoindrit le débit cardiaque. Le remplacement du myocarde ventriculaire détruit par du tissu fibreux résulte d'une occlusion corona- rienne significative. Si la zone intéressée a une taille suffisante, un anévrisme se forme, et dans de nombreux cas une expansion systolique est provoquée par les techniques radiologiques. L'amoidrissement fonctionnel produit par un anévrisme ventriculaire peut rendre le patient un infirme cardiaque du fait: (a) d'une défaillance cardiaque; (b) d'une arythmie paroxystique ventriculaire, et (c) d'accidents emboliques.

La grande majorité des anévrismes ventriculaires se trouve sur la paroi antérieure du ventricule gauche et sur la partie antérieure du septum inter-ventriculaire. L'infarctus massif de la paroi postérieure du ventricule gauche englobe les muscles papillaires et les cordages de la valvule mitrale; rarement le malade survivra à l'assaut combiné d'un infarctus myocardique massif et d'une insuffisance mitrale aiguë.

D'après l'auteur, chaque malade ayant un anévrisme ventriculaire est un candidat pour une intervention chirurgicale. Le traitement médical peut au maximum préparer le malade pour une thérapeutique définitive. L'ablation de l'anévrisme et la reconstruction du ventricule gauche peut être faite dans la majorité des cas avec une circulation extra-corpselle. L'auteur présente la technique et son expérience clinique.

**Zusammenfassung**

Kammeraneurismen nach Herzinfarkt beeinträchtigen das Herzschlagvolumen. Der Ersatz des zersägerten Kammermyokards durch fibröses Gewebe ist das Ergebnis eines einschneidenden Coronarverschlusses. Hat der befallene Bereich eine genügende Größe, bildet sich ein Aneurisma, und es läßt sich in vielen Fällen durch entsprechende Strahlentherapie eine paradox Bewegung nachweisen. Die funktionelle Schädigung, die durch das Kammerwandaneurisma entsteht, vermag aus dem Patienten einen Herzkrüppel zu machen und zwar (a) infolge Versagens des linken Herzens, (b) infolge paroxysmaler ventrikulärer Arrhythmie und (c) infolge embolischer Zwischenfälle.

Die Mehrzahl der Ventrikelanerismen findet man an der Vorderwand der linken Kammer und der Vorderfront des Kammereptums. Eine massive Infarzierung der rückwärtigen Anteile des linken Ventrikels betrifft auch die Papillarmuskeln und die Chordae tendineae der Mitralklappe; nur ausnahmsweise dürfte der Patient eine kombinierte Schädigung dank eines massiven Myokardinfarkts und einer akuten Mitralinsutizien überleben.


**References**


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