Resection of Myocardial Aneurysms after Infarction during Temporary Cardiopulmonary Bypass

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A VENTRICULAR ANEURYSM due to coronary occlusion is not a rare pathologic entity. It was first described by Hunter\(^1\) in 1757, and Sternberg\(^2\) was the first to appreciate the concept of chronic aneurysm and reported the first cases during life. With the development of roentgenology and contrast techniques, the disease has become clinically diagnosed more and more frequently. The incidence has varied between 8 and 22 per cent of all myocardial infarcts, depending upon the criteria given by the authors.\(^3\)\(^4\) The sex incidence has been predominantly male, similar to the ratio in myocardial infarction.

The prognosis has been grave, with a mortality of 73 per cent within the first 3 years after the initial infarct and rose to 88 per cent in the next 2 years.\(^5\) This grim outlook for the left ventricular aneurysm after infarction is in sharp contrast with coronary occlusive disease without aneurysm formation. Master and co-workers\(^6\) found 50 per cent survivals 5 years following the initial infarct, and large numbers of patients were completely rehabilitated. The most frequent cause of death from left ventricular aneurysms has been congestive heart failure.\(^5\) DeCamp\(^7\) first reported a significantly reduced cardiac output in a patient with ventricular aneurysm after infarction. Unfortunately, his patient succumbed after surgery.

A high incidence of mural thrombi has been found at postmortem examination.\(^8\)\(^9\) An association of increased coagulability of the blood and coronary thrombosis has been investigated. Acceleration of the blood coagulation among patients with ventricular aneurysm has been reported with reversion to normal coagulogram following a surgical treatment.\(^10\) Sudden death due to systemic embolism has been second in frequency to congestive heart failure, being clinically observed in about 50 per cent of all the patients suffering from a ventricular aneurysm and the main cause of death in 21 per cent.\(^5\) Systemic hypertension and early ambulation\(^11\) have been emphasized as factors contributing to aneurysm formation after the initial attack.

The grave prognosis, high mortality, stormy course, and ineffective medical treatment have recently attracted more attention because successful surgical correction has become feasible with low operative mortality and a rather promising long-term course. The purpose of this study is to report our experience with chronic left ventricular aneurysm after infarct surgically treated with extracorporeal circulation. Postoperative observation now extends from 6 to 36 months after surgery.

Five patients were considered for surgery since 1958 and all were accepted for surgical treatment (table 1).

Case Reports

Case 1

W.C., a 49-year-old man, admitted to the University Hospitals in June 1958 with a diagnosis of left ventricular aneurysm, had had an acute myocardial infarction in 1952. He recovered uneventfully and had enjoyed full physical activities until several months prior to admission, when he noticed increasing precordial pulsations, occasional episodes of chest pain on effort, and progressive dyspnea.
Table 1

Summary of Five Patients with Left Ventricular Aneurysm Surgically Treated with Heart-Lung Bypass Including Follow-up

<table>
<thead>
<tr>
<th>Pts.</th>
<th>Interval infarct-aneurysm</th>
<th>Interval infarct-surgery</th>
<th>Blood pressure</th>
<th>Initial diagnosis based upon</th>
<th>Localization and size</th>
<th>Intramural clot</th>
<th>Bypass (min.)</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.C.</td>
<td>2 yr.</td>
<td>6 yr.</td>
<td>118/80</td>
<td>Radiologic findings</td>
<td>Anterior wall and apex</td>
<td>Present</td>
<td>25</td>
<td>3 yr. 4 mo.</td>
</tr>
<tr>
<td>H.V.</td>
<td>5 mo.</td>
<td>5 mo.</td>
<td>110/70</td>
<td>Electrocardiographic tracing</td>
<td>Anterior wall and apex</td>
<td>Present</td>
<td>60</td>
<td>2 yr. 6 mo.</td>
</tr>
<tr>
<td>W.R.</td>
<td>2 wk.</td>
<td>9 mo.</td>
<td>130/90</td>
<td>Radiologic findings</td>
<td>Anterior wall and apex</td>
<td>Present</td>
<td>28</td>
<td>2 yr. 3 mo.</td>
</tr>
<tr>
<td>H.A.</td>
<td>3 mo.</td>
<td>9 mo.</td>
<td>110/75</td>
<td>Radiologic findings</td>
<td>Anterior and lateral wall</td>
<td>Present</td>
<td>40</td>
<td>1 yr. 6 mo.</td>
</tr>
<tr>
<td>R.A.</td>
<td>3 mo.</td>
<td>6 mo.</td>
<td>160/95</td>
<td>Radiologic findings</td>
<td>Posterior and lateral wall</td>
<td>Present</td>
<td>30</td>
<td>1 yr.</td>
</tr>
</tbody>
</table>

Physical examination at the time of admission revealed a forceful apical pulse outside the midclavicular line. The blood pressure was 118/80 mm. Hg. The preoperative electrocardiogram showed low voltage in the standard leads with QS pattern in lead I, aV\(_1\), and chest leads V\(_3\), 4, 5; the QRS was positive in aV\(_R\). Chest films, fluoroscopy, and kymography revealed localized bulging with paradoxical movement along the left ventricular border, moderate cardiac enlargement, and mild pulmonary vascular engorgement (fig. 1A). A right ventricular angiogram confirmed the presence of a large left ventricular aneurysm involving the lateral wall and distorting the right ventricular cavity through the weakened septum (fig. 1B).

On June 5, 1958, through a transverse bilateral thoracotomy, the heart and the great vessels were exposed. A huge, left ventricular aneurysm 12 by 15 cm. in diameter, involving the anterior and apical portion of the chamber was resected during temporary heart-lung bypass for 25 minutes. The endocardial surface of the sac was lined with a layer of organized clot. The septum as well as the papillary muscles and the mitral valve were normal. He left the hospital in good condition 20 days after operation, receiving a maintenance dose of digitalis. Postoperative kymography revealed normal heart pulsations. From 1958 to August 1961 he has been working full-time as an electrical appliance repairman. In August 1961, he was admitted to the hospital because of hemoptysis. Bronchoscopy showed bloody secretions coming from the left upper lobe bronchus but roentgenograms and bronchograms were within normal limits. His general and cardiac status were most satisfactory. At this admission, over 3 years after surgery, selective left ventriculography (fig. 1C) revealed a mild enlargement of the left ventricle without evidence of ventricular aneurysm. The study ruled out also a cardiopulmonary fistula as a differential diagnostic possibility for his hemoptysis.

**Case 2**

H.V., a 47-year-old man, had an acute myocardial infarction in March 1959 with an electrocardiographic pattern suggesting a ventricular aneurysm. Following his acute episode, he remained in chronic congestive heart failure unresponsive to digitalization, diuretics, and salt restriction.

At his first admission to this hospital in June 1959, x-ray disclosed an aneurysmal bulge over the lateral superior heart border (fig. 2A). Right heart catheterization revealed normal pulmonary pressure as well as normal resting cardiac output. Because of a pleural pericardial friction rub and a sedimentation rate of 74 mm. in 1 hour, an acute process was suspected and the patient was discharged. On a second admission in August 1959 angiography showed a large left ventricular aneurysmal sac largely filled with clot (fig. 2B). The aneurysmal sac remained opacified a long time after emptying of the left ventricular cavity (fig. 2C).

At operation in August 1959 a very large left ventricular aneurysm, extending to the left lateral thoracic wall, was excised. The procedure was performed under extracorporeal circulation for 60
minutes. The sac, 14 by 12 cm. in diameter, involved the anterolateral left ventricular wall. The resected specimen weighed 527 Gm. including the aneurysmal wall as well as laminated clot (fig. 2E). The muscle and connective tissue wall of the excised sac was 0.4 cm. thick in its central portion after decorticating the clot. Sections from the edge of the aneurysm showed a few degenerating muscle cells and occasional bundles of cardiac muscle with marked chonic inflammatory reaction along the epicardial surface. The clot was partially organized and attached to a dense connective-tissue scar.

A postoperative electrocardiogram showed no significant changes, the pattern of old anterolateral infarction being unchanged. The chest x-ray 18 months after operation revealed a normal heart contour (fig. 2D). Two and a half years after operation the patient was well, was free of all cardiac symptoms or pain, and was taking no medication.

Case 3

W.R., a 52-year-old man, suffered two myocardial infarctions in May 1959 for which he was hospitalized 6 weeks and treated with digitalis and complete rest. X-rays showed a left ventricular aneurysm, and the electrocardiogram was consistent with anterior myocardial infarction. He again experienced sharp shooting anginal pains relieved by taking large amounts of nitroglycerin. After recovery, he had severe angina pectoris, dyspnea, easy fatigability, and intermittent hepatic engorgement for which he was given nitroglycerin, digitalis, a low-salt diet, and diuretics. His functional cardiac status was considered to be class III (American Heart Association Classification).

Studies on admission to the University of Minnesota Hospitals in February 1960 were consistent with the diagnosis of left ventricular aneurysm. His blood pressure at this time was 130/90 mm. Hg and his pulse rate was 88 per minute and regular. The second aortic sound was loud and split. The laboratory data were all within normal limits. An electrocardiogram revealed a QS pattern in leads I and aVL and upward QRS complex in aVR.

X-ray studies demonstrated a deformity in the left heart border with bulging, about 7 cm. in length, across the base, and 3 cm. in depth from the expected contour of the heart (fig. 3A). Kymography revealed active movement of this left ventricular pouch out of phase from the extreme lower left heart border and in phase with the aortic expansion. Forward biplane venous angiography was consistent with left ventricular aneurysm with a relatively thick aneurysm wall, which suggested some persistent clot in the sac. The communication between the pouch and the rest of the left ventricle seemed to be narrow like a diverticulum (fig. 3B). This aneurysm also remained opacified a long time after emptying of the left ventricular cavity.

On February 26, 1960, a left ventricular aneurysm was resected successfully with extracorporeal circulation. The aneurysm involved the anterior and apical portion of the ventricle and was the size of an orange; the inner part was lined by soft clot, about 1 cm. in thickness. The specimens consisted of a 6 by 4 cm. ventricular wall, 0.5 cm.

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Case 2. A, upper left. Posteroanterior chest radiography 18 months after infarction, indicating a large left ventricular aneurysm. Patient was in chronic congestive heart failure. B, upper right. Posteroanterior angiography confirming left ventricular aneurysm, largely filled by intramural clot. Radiopaque substance outlined left atrium, left ventricle, left ventricular aneurysm, and aorta. C, lower left. Note the contrast medium still opacifying the left ventricular aneurysm a long time after the functional left ventricular cavity and aorta have been flushed clear, confirming the basic hemodynamic defect of ventricular aneurysm. D, lower right. Posteroanterior chest radiograph demonstrating normal heart shadow 18 months following surgery.
thick, composed of dense collagenous tissue and a few fibroed muscle fibers.

The postoperative course was uneventful. An electrocardiogram was not significantly changed but a postoperative x-ray showed a normal heart contour. For 2\( \frac{1}{2} \) years he has remained free of symptoms, has taken Dicumarol and digitalis, but has not been restricted in diet.

Case 4

H.A., a 58-year-old man, suffered myocardial infarctions in January 1960 with a rather stormy course, with congestive heart failure, repeated atrial fibrillation, and pulmonary and systemic emboli. He was treated with bedrest, digitalis, anticoagulants, and quinidine.

Examination in October 1960 revealed a blood pressure of 110/75 mm. Hg and a regular pulse rate of 90. The heart was markedly enlarged. An electrocardiogram was consistent with an anteroseptal infarct with low voltage in the standard leads, QS pattern in leads I and aV\(_L\), upward QRS complex in aV\(_R\), and deep Q wave and ST elevation in V\(_2\) to V\(_5\). X-ray examination supported a diagnosis of left ventricular aneurysm with abnormal prominence of the left midcardiac border (fig. 4A), and paradoxical pulsations.

On October 28, 1960, the patient was operated upon with a pump-oxygenator bypass for 40 minutes. The entire pericardial cavity was found obliterated by adhesions. The aneurysmal sac, 11 by 6 by 0.8 cm. occupied the anterolateral portion of the left ventricle, and held at least 200 to 250 ml. A moderate amount of clot lined the endocardial surface of the sac. The aneurysm wall was composed of fibrous tissue, partly dense and hyaline and partly cellular and vascular. The thrombus was in an early stage of organization, and was fairly well adherent to the endocardial surface, but one portion contained fresh blood cells among a mass of fibrin.

The postoperative course was uncomplicated except for brief bums of atrial fibrillation during the first week after surgery. The patient left the hospital in good condition 3 weeks after surgery and did not require medical or diuretic management. The x-ray revealed a normal heart contour (fig. 4B). Ten months after surgery, he reported that he was working full time without physical or dietary restriction, and without medication. He has remained symptom free for 1\( \frac{1}{2} \) years.

Case 5

R.A., a 63-year-old man, was admitted to the University of Minnesota Heart Hospitals on March 26, 1961. For a year prior to admission he had suffered several episodes of brief, severe cardiac pain and repeated attacks of pulmonary edema.

Physical examination revealed a blood pressure of 160/95 mm. Hg and a regular pulse rate of 88 per minute. The apical impulse was to the left of the midlateral line and the second pulmonic sound was loud. The laboratory data were all within normal limits. An electrocardiogram showed an old anterolateral myocardial infarction with QS pattern in leads I, II, and aV\(_L\), low voltage in the standard leads, and negative T waves in V\(_5\) and V\(_6\). The QRS complex in aV\(_R\) was negative. Chest radiograms were consistent with left ventricular aneurysm (fig. 5A). Kymography revealed a motionless left ventricular border. Right heart catheterization showed an elevated pulmonic artery pressure of 50/25 (mean pressure of 33 mm. Hg), which rose to 60/30 with mild exercise. The mean pulmonary wedge pressure was 21 mm. and rose to 25 mm. with exercise. The cardiac index was 2.3 L. per minute and remained at 2.4 L. per minute during exercise. The venous oxygen saturation decreased during a very mild exercise test.

In April 1961, the patient was operated on with cardiopulmonary bypass. A large posterior and lateral left ventricular aneurysm was resected, 10 by 8 cm. in size, and 0.8 to 1.5 cm. thick in its central portion. The inner part of the aneurysm was found lined with clot that extended into the ventricular cavity. The myocardium in the aneurysmal wall was largely replaced with dense relatively acellular scar tissue.

The postoperative course was surprisingly benign considering his poor preoperative status, and the patient left the hospital in 2 weeks with a normal cardiac contour on radiologic examination (fig. 5B). The electrocardiogram remained unchanged. Since surgery he has been feeling very well, without symptoms, and without restriction in diet and physical activity.

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![Figure 2E](http://circ.ahajournals.org/)

**Figure 2E**

Case 2. Organized and relatively fresh clots with operative specimen removed from the left ventricular aneurysm during operation.
Eleven months after operation, he returned for right and left heart catheterization. The pulmonary artery pressure was now normal (24/8 mm. Hg, with a mean of 12). With mild exercise for 5 months, there was no rise in the pulmonary pressure (22/11 mm. Hg, mean 14) or in the left ventricular diastolic pressure (140/0.5 mm. Hg) in marked contrast to the findings before operation. Thus, the complete correction of his preoperative chronic left ventricular decompensation was confirmed. Left ventriculography at this time also showed a normal ventricular cavity.

Description of the Surgical Procedure

Following endotracheal anesthesia, the patients are placed in a supine position and the heart is exposed through a transverse bilateral thoracotomy, transecting the sternum. The fifth or sixth rib on the left and the fourth rib on the right are resected. Next, the mammary artery is cannulated for continuous arterial pressure recording. The pericardium is widely opened at the atrial level (where it is free) and left intact temporarily over the left ventricle where it is adherent. Venous blood is returned to the oxygenator by cannulae in both venae cavae via the right atrium in the standard manner and the arterial return to the patient is through a cannula in the femoral artery. The heart-lung bypass was achieved with a bubble helix-type oxygenator and Sigma motor occlusive pumps. The patients are heparinized with 3 mg. per Kg. of body weight counteracted at the end of the procedure by 4 1/2 mg. per Kg. of Polybrene. Following institution of the total bypass, the adherent pericardium is gently dissected from the sac. Proper visualization of the left descending and left circumflex coronary arteries is important. Next, the left ventricle is opened through the sac at its apex (fig. 6A). A large sucker, not connected with the extracorporeal circuit, is introduced into the ventricular cavity to remove loosely attached clots. Next the left ventricular incision is extended to the junction of the sac with functional myocardium, and the left ventricular cavity is carefully inspected with observation of papillary muscle and mitral and aortic valves (fig. 6B). The aneurysmal sac is then completely resected circumferentially.

Avoiding injury of coronary arteries at this step is important. The ventricular cavity in all cases has been closed with interrupted mattress sutures of silk (no. 1 or 2) tied over long strips of compressed polyvinyl sponge and then reinforced by an additional over-and-over running silk suture (fig. 6C and D).

The heart was allowed to beat throughout the procedure, and the aorta was not cross-clamped. In addition to a continuous arterial blood pressure, electrocardiographic and electroencephalographic activities are recorded throughout the procedure. The chest is closed in the usual manner with a tube in each pleural cavity. With this technique of ventricular closure, no difficulty has been experienced from such problems as hemorrhage or aneurysm reformation.
Discussion

In reviewing the entire literature available to us on the surgically treated cases of aneurysms after infarction, we found 53 patients. The first patient was reported by Sauerbruch in 1931. A 29-year-old man thought to have a mediastinal tumor was explored, and a right ventricular aneurysm was accidentally incised. Sauerbruch successfully resected the sac, while controlling the bleeding with a finger inserted into the small neck of the aneurysm. The etiology of the aneurysmal formation remained unknown, and the patient was well 3 1/2 months after surgery.

In 1944, Beck first attacked surgically a clinically diagnosed left ventricular postinfarction aneurysm. He used a free transplant of fascia lata over the left ventricular dome without resection in order to support and prevent further extension of the aneurysm. The patient died 3 weeks later from empyema. Since then many approaches have been proposed, basically of two types: external reinforcement and resection with ventriculoplasty. All together, techies similar to Beck’s have been applied in 24 patients with use of omentum, skin, intercostal muscle, pectoralis muscle, and diaphragm to support the aneurysm wall, to prevent its further expansion, and hopefully to stimulate development of collateral circulation into the ischemic fibrotic sac. No long-term results have been reported in this group. With present knowledge of the pathophysiology of ventricular aneurysms it is doubtful that the basic course of the disease was significantly altered by this palliative approach although the procedure is apparently still popular in Russia.

However, the initial mortality was low (8 per cent) in this group (table 2).

The curative technique (table 3) has consisted of excision of the sac, as successfully performed first by Bailey in 1954 with a closed

![Figure 4](http://circ.ahajournals.org/)

*Figure 4
Case 4. A, left. Preoperative posteroanterior chest radiograph showing abnormal prominence of the left midcardiac contour as well as moderate pulmonary vascular engorge-
ment. B, right. Posteroanterior chest radiograph 3 weeks postoperatively.

Table 2
Plastic Palliative Procedures in Surgical Treatment of Left Ventricular Aneurysm Used by Different Authors

<table>
<thead>
<tr>
<th>Name</th>
<th>No. pts.</th>
<th>Year</th>
<th>Method</th>
<th>Operative mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beck</td>
<td>1</td>
<td>1944</td>
<td>Fascia alta</td>
<td>1</td>
</tr>
<tr>
<td>Neidner</td>
<td>2</td>
<td>1955</td>
<td>Skin</td>
<td></td>
</tr>
<tr>
<td>Spacek</td>
<td>4</td>
<td>1956</td>
<td>Intercostal muscle</td>
<td>1*</td>
</tr>
<tr>
<td>D’Allaine</td>
<td>1</td>
<td>1956</td>
<td>Pectoralis muscle</td>
<td></td>
</tr>
<tr>
<td>Petrovsky</td>
<td>6</td>
<td>1959</td>
<td>3-Diaphragm</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2-Transsutting</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1-Pericardium</td>
<td></td>
</tr>
<tr>
<td>Martinova</td>
<td>10</td>
<td>1960</td>
<td>8-Diaphragm</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2-Transsutting</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>24</td>
<td></td>
<td></td>
<td>2</td>
</tr>
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</table>

*Late mortality (7 months).
technic. Later, in 1958, he added an additional six patients in whom the same procedure was done with an operative mortality of 14 percent. He described a special clamp for the resection of ventricular aneurysms and a flush-out technic to avoid the dangers of systemic embolism during surgery. One of his six patients succumbed on the operating table, however, as a result of peripheral embolism. Following Bailey's first report, a few additional resections of ventricular aneurysms were reported with use of similar techinies (table 3). DeCamp, Neidner, and Kalesov described their own special clamps for resection of ventricular aneurysm. Of interest was the technic used by Kalesov of resecting the aneurysm without bypass and closing the ventriculotomy by tantalum clips and a suture apparatus rather quickly while the ascending aorta was clamped. Cooley reported two patients operated upon in 1958 with successful resection of the left ventricular postinfarction aneurysm with use of the heart-lung machine. Between 1958 and 1960 another 12 resections during extracorporeal circulation were reported, all without operative mortality. The five patients herein reported make a total of 17 without operative mortality. Neptune advocated induced fibrillatory cardiac arrest for this lesion.

Schlichter et al. mentioned a forceful cardiac impulse in contrast to a weak peripheral pulse as a suggestive clinical sign of ventricular aneurysm. Two of our patients had an increased cardiac impulse, but the peripheral pulse was normal. Only one patient had a systolic murmur that appeared after the initial infarct. Likoff stressed the presence of gallop rhythm associated with a dull first heart sound and the appearance of a systolic or diastolic murmur after the myocardial infarct as helpful clinical signs of left ventricular aneurysm. A patient sustaining ventricular infarction and showing a friction rub in the immediate postinfarct period has a greater tendency to develop left ventricular aneurysm, probably because pericarditis suggests a full thickness involvement of the ventricular wall. One of our patients had symptoms of pericarditis after the myocardial infarct.
Goldberger\(^3\) has reported that the electrocardiographic tracing is characteristic of a ventricular aneurysm. He thought the upward QRS complex in aV\(_r\) lead was pathognomonic of ventricular aneurysm. Further, he considered that the absence of this pattern ruled out ventricular aneurysm. In our five patients the electrocardiogram was consistent with old myocardial infarction characterized by a QS pattern in leads I and aV\(_L\). Low voltage and deep Q waves over the precordial leads were present in two of the patients. An upward QRS complex in aV\(_r\) lead (Goldberger's sign for aneurysm) was found in four of our five patients; in one tracing, case 5, this sign was absent. This patient was the only one with a posterior wall aneurysm, all others being anterolateral. In case 2, the electrocardiogram suggested a developing aneurysm prior to its radiologic appearance. Laake\(^3\) found the persistence of the electrocardiographic pattern of acute myocardial infarction for several months to be very suggestive of aneurysmal formation. The upward QRS complex in aV\(_r\) is certainly additional support for the early diagnosis of ventricular aneurysm.

Chronic aneurysms develop as a result of scar formation that has replaced the necrotic postinfarct myocardium. Murray\(^3\), while experimenting with acute coronary thrombosis in dogs, observed the immediate development of an "expansion chamber" in the area involved by infarct. The infarcted left ventricular portion, undergoing acute inflammatory process on one side and exposed to a high left ventricular pressure on the other, dilates and forms the noncontractile fibrotic sac.

Hemodynamically the aneurysm dilates paradoxically with each systole, thus depleting the stroke volume of the heart by a substantial amount when the sac is large or contractility of the residual ventricle is poor. Furthermore, these deleterious hemodynamic changes tend to progress with time rather than to stabilize, inasmuch as the larger the sac the greater is the volume wasted into it and the greater is the tension on the already weakened wall. Lumish and Likoff\(^3\) also stressed the additional burden upon the remaining left ventricle by compensatory increases in stroke volume and the heart rate, all of which lead progressively to hyper-

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**Table 3**

<table>
<thead>
<tr>
<th>Name</th>
<th>No. pts.</th>
<th>Year</th>
<th>Method</th>
<th>Operative mortality</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sauerbruch</td>
<td>1</td>
<td>1931</td>
<td>Excision(^*)</td>
<td></td>
<td>3 mo.</td>
</tr>
<tr>
<td>Bailey</td>
<td>1</td>
<td>1954</td>
<td>Excision(^\dagger)</td>
<td></td>
<td>3 yr.</td>
</tr>
<tr>
<td>Neidner</td>
<td>1</td>
<td>1955</td>
<td>Excision(^\ddagger)</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>DeCamp</td>
<td>1</td>
<td>1956</td>
<td>Excision(^\ddagger)</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Bailey</td>
<td>6</td>
<td>1958</td>
<td>Excision(^\ddagger)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stoyanov</td>
<td>1</td>
<td>1958</td>
<td>Excision(^\ddagger)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cooley</td>
<td>2</td>
<td>1958</td>
<td>Excision(^\ddagger)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cooley</td>
<td>4</td>
<td>1959</td>
<td>Excision(^\ddagger)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Martinova</td>
<td>2</td>
<td>1960</td>
<td>Excision(^\ddagger)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sullivan</td>
<td>1</td>
<td>1960</td>
<td>Excision(^\ddagger)</td>
<td></td>
<td></td>
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<tr>
<td>Kolesov</td>
<td>1</td>
<td>1960</td>
<td>Excision(^\ddagger)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kay</td>
<td>1</td>
<td>1960</td>
<td>Excision(^\ddagger)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Templeton</td>
<td>3</td>
<td>1960</td>
<td>Excision(^\ddagger)</td>
<td></td>
<td></td>
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<tr>
<td>Neptune</td>
<td>1</td>
<td>1960</td>
<td>Excision(^\ddagger)</td>
<td></td>
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<tr>
<td>Glen</td>
<td>1</td>
<td>1960</td>
<td>Excision(^\ddagger)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lillehei</td>
<td>5</td>
<td>1961</td>
<td>Excision(^\ddagger)</td>
<td></td>
<td>6 mo.—3 yr.</td>
</tr>
<tr>
<td>Total</td>
<td>32</td>
<td></td>
<td></td>
<td></td>
<td>2</td>
</tr>
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\(^*\)Unknown etiology—right ventricular aneurysm.
\(^\dagger\)Atrial aneurysm.
\(^\ddagger\)During heart-lung bypass.
Resection of Myocardial Aneurysms

Figure 6
Schematic drawing of technic for resection of ventricular aneurysm. A. Initial opening of aneurysm at its apex followed by clot removal and careful inspection of ventricle interior to locate important structures such as the papillary muscles and chordae. B. Radical resection of the ventricular sac and intramural clot after identification of the junction of fibrous sac with the remaining functional myocardium. C. Ventriculoplasty. The entire row of full thickness mattress sutures of silk are preplaced and then tied over compressed polystyrene sponge strips. D. As a second layer reinforcement, a continuous over-and-over silk suture is placed.

trophy, dilatation, and finally congestive heart failure.

The histologic specimen of the resected aneurysm is usually represented by dense collagenous or hyaline connective tissue throughout the wall. Occasionally one can find few fibers of muscle tissue surrounded by scar and foci of mononuclear or macrophagic cells, remnants of the acute inflammatory stage. The pericardial cavity has usually been found to be obliterated by dense fibrous adhesions, depending upon the time from the initial infarct and its excision.

Intramural clots have been found almost uniformly within ventricular aneurysms and were present in all five of our group. Accelerated coagulability in patients after acute myocardial infarction, a severely damaged endocardium, "stasis" of the blood in the sac, observed in two of our patients by angiography, are all factors known to facilitate clot formation.

Angiography, while probably not necessary for diagnosis in the usual case, was found most helpful for visualizing the actual size of the sac, its location, and the amount of clot formation in the sac. An additional finding of considerable interest was the observation in some of our patients of radiopaque solution remaining in the sac long after the contractile left ventricle and even the aorta had been flushed clear of the radiopaque media, adding further confirmation of the basic hemodynamic defect.

We believe there is no place today for any of the plastic supportive procedures in

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the surgical treatment of left ventricular aneurysms. Excision under bypass technic has great advantages over the simple excision by a closed method in terms of safety and curability. More accurate visualization of the actual point of origin of the sac can be obtained as well as of functioning coronary arteries. Other advantages include inspection of the ventricular cavity to obviate damage to the mitral valve, complete excision of the sac, much less possibility of operative embolism, extraction of the intramural clots even when they extend beyond the aneurysmal edges, and finally resection of the less common posterior wall aneurysms.

The question has been raised whether so large a ventricular aneurysm might be encountered that its complete excision might leave a left ventricle too small to be compatible with life. In reality this problem is probably not one of much practical significance, since an aneurysm of this magnitude would probably not permit survival long enough to come to surgical treatment.

The apparent low operative risk for a major surgical procedure in patients with severe coronary arteriosclerosis and a ventricular aneurysm is believed to be a reflection of the fact that a ventricular aneurysm is a very serious hemodynamic defect. Patients able to tolerate this burden for even a short interval undoubtedly have a reasonably satisfactory residual myocardium and have developed satisfactory intercoronary anastomoses, and thus are immediately and significantly improved by surgical removal of this parasitic burden upon their circulation.

Summary

The report consists of five patients with chronic postinfarct left ventricular aneurysms. The age distribution was between 47 and 63 years. The time lapse between the initial infarct and the appearance of the left ventricular aneurysm was 2 weeks to 2 years. The time interval between the initial infarct and surgery was 5 months to 6 years. Less than adequate bedrest following the infarct, multiple infarcts, and systemic hypertension were other factors present in this group. Right heart catheterization was performed in two patients and showed reduced cardiac output and elevated pulmonary pressures in one. The electrocardiograms were consistent with extensive old myocardial infarction and ventricular aneurysm. The diagnosis was suggested by electrocardiograms and roentgenography, supported by kymography, and confirmed by angiography.

The world literature on surgically treated patients with ventricular aneurysms is briefly reviewed with emphasis on the varied surgical approaches to the problem in the past. The method of complete excision with ventriculoplasty during cardiopulmonary bypass was used in all our patients during the past 4 years. The anatomic distribution of the ventricular aneurysm as well as the histopathology of the resected specimen is described.

All patients survived the operation, and were observed from 6 months to 40 months after operation. There have been no late deaths to date, and all patients have been rehabilitated. Postoperative catheterization studies have confirmed the clinical improvements observed.

More attention should be given to this relatively common complication after infarct because excision under temporary heart-lung bypass is a safe and feasible approach.

Addendum

One patient, case 1, died suddenly 3 1/2 years after operation.

References

RESSECTION OF MYOCARDIAL ANEURYSMS


Resection of Myocardial Aneurysms after Infarction during Temporary Cardiopulmonary Bypass
C. WALTON LILLEHEI, MORRIS J. LEVY, RICHARD A. DEWALL and HERBERT E. WARDEN

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