Surgical Repair of a False Aneurysm of the Left Ventricle Resulting from a Separation Between the Aorta and the Heart

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SUMMARY

In the case of a 44-year-old Negro man with lupus erythematosus reported, a rapidly expanding mass had developed in the region of the left ventricle. Surgical exploration revealed a false aneurysm arising from a defect at the base of the heart. The defect is interpreted as a separation of the aorta from the heart in the region of the left coronary cusp of the aortic valve. Surgical repair is outlined and illustrated. The relationship of this defect to annular subvalvular aneurysm occurring in African Negroes is discussed.

DEFECTS between the root of the aorta and the heart below the aortic valve ordinarily present as ventricular septal defects. Edwards and Burchell1 have pointed out that the single area of the aortic root below the valve cusps which is related neither to a cardiac chamber nor to the pulmonary trunk is that segment between the aorta and the heart directly beneath the intermediate part of the left aortic cusp. Adjacent to the wall of the aortic sinus in this region is the epicardium and a defect occurring in this area would produce an epicardial hemorrhage. If contained by the epicardium or by the surrounding pericardium, false aneurysmal formation could result. Such a lesion has been encountered at The Johns Hopkins Hospital and a successful surgical repair has been performed.

Report of Case

On May 4, 1966, a 44-year-old Negro man was admitted to The Loch Raven Veterans Administration Hospital with fever and pleuritic pain. Chest roentgenograms revealed a mass at the left heart border (fig. 1). In 1945, while in India with the United States Armed Forces, the patient had developed malaise and articular pains. In 1946, areas of depigmentation were noted over his anterior chest, and a diagnosis of discoid lupus erythematosus was made. He continued to have intermittent fevers and joint pain. In 1952, a diagnosis of systemic lupus erythematosus was made. He was treated with a single course of steroids, but his condition remained essentially unchanged between 1959 and 1964. In 1964, he was hospitalized with high fever, chest pains, joint pains, and peculiar behavior. A gastric aspirate revealed acid-fast bacilli. Following admission he continued to have high fevers to 103 F and irrational behavior. An electrocardiogram demonstrated the changes of pericarditis. He was treated with steroids. Improvement occurred dramatically. In January 1966, he again developed episodes of weakness, loss of weight, and fever associated with pleuritic chest pain which persisted until admission to the Veterans Hospital.

Initial examination revealed a blood pressure of 105/68 mm Hg, a temperature of 101.8 F, and a pulse rate of 100. Lungs were clear. The heart was not enlarged and there was a grade I to II systolic murmur. Marked splaying and deep pigmentation were seen over the anterior and posterior thorax, on the skin of the face, and on the scalp. There were large areas of alopecia. Further investigation included chest fluoroscopy which demonstrated paradoxical pulsation in the region of the abnormal left ventricular contour. An intravenous angiogram was variously interpreted as showing a slight opacification in the region of the mass and as showing no opacification with dye. It was eventually decided that the patient had a tumor in the lower lobe of the left lung. On thoracotomy June 16, 1966, an intrapericardial mass was seen to move paradoxically with ventricular systole. An attempt was

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Work was supported in part by Grants HE-00226-14 and HE-00226-15 from the U. S. Public Health Service.
made to open the pericardium, but extensive pericardial fusion was encountered. The thoracic incision was closed. The patient’s postoperative course was unremarkable. He was transferred to The Johns Hopkins Hospital on June 23, 1966. Pertinent physical findings at that time included a well-healed thoracotomy scar, a blood pressure of 105/70 mm Hg, and a pulse rate of 90 with sinus rhythm. The jugular venous pressure was not elevated. The heart was not enlarged. A soft systolic murmur was present at the base and a soft third heart sound was present in this area. The chest was clear to auscultation, the edge of the liver was palpable two-finger breadths below the right costal margin, and the skin revealed large areas of scarring and depigmentation. Laboratory investigation included an electrocardiogram which showed a normal sinus rhythm with low voltage and nonspecific T-wave changes but no evidence of myocardial infarction. Chest x-rays showed a mass adjoining the left border of the heart; comparison of these films with a series taken during the previous months showed a continuous rapid increase in the size of the mass. Cardiac catheterization was performed. The pressures in the right side of the heart were slightly elevated, but no gradients were recorded on pullback across the pulmonary or tricuspid valve. The appearance time of inhaled hydrogen in the pulmonary artery was normal. The end-diastolic pressure in the left ventricle was slightly elevated and demonstrated a significant rise after the injection of contrast material. Fluoroscopy revealed that the mass along the cardiac border was pulsatile. A cineangiogram of the left ventricle with the patient in the left anterior oblique position demonstrated that the left ventricular cavity was small and emptied well. Puffs of contrast material could be seen passing posteriorly above the region of the mitral valve. Although these were interpreted at the time of the study as evidence of minimal mitral insufficiency, it became clear later that the dye was passing into the aneurysm itself. A left ventricular cineangiogram performed in the right anterior oblique position demonstrated a normal ascending aorta without evidence of aortic regurgitation. The right coronary artery and the anterior descending coronary artery filled well with dye.

**Operation**

No precise preoperative diagnosis was made. A clotted ventricular aneurysm or a cardiac tumor was thought to be the most likely possibility. Operation was performed on July 5, 1966. The chest was re-entered through the old left posterior lateral thoracotomy incision. Inspection revealed a pulsatile mass lying above the hilum of the lung in the region of the atrioventricular groove (fig. 2). There were extensive pericardial adhesions, presumably the

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**Figure 1**

Roentgenographic appearance of the chest. (Left) Before the second surgical procedure. The abnormal left contour of the heart silhouette is apparent. (Right) Postoperative appearance of the chest.
result of lupus pericarditis. A meticulous dissection of the pericardium was carried out. Needle puncture of the mass was performed. Aspirated blood from the mass had an oxygen saturation of 100%. The pressure recorded within the mass was 80/0 mm Hg. Cardiopulmonary bypass was accomplished by placing a venous cannula in the outflow tract of the right ventricle and an arterial catheter in the left subclavian artery. A vent was placed in the apex of the left ventricle. On bypass, the heart emptied but the mass did not. An incision was made directly into the anterior aspect of the mass and blood was aspirated from it. The mass contained organized clot obliterating about one fourth of its cavity (fig. 3). At the base of the mass there was an elliptically shaped opening measuring about 2 cm in its longest diameter. The edges of this opening were thickened fibrous tissue, and within this opening there was white fibrous tissue with two small holes measuring about 3 or 4 mm in diameter through which arterial blood was squirting under high pressure. The two defects were closed with mattress sutures of Teflon tied over pledgets of Teflon felt. A single mattress suture was used for each of the openings and, when these were drawn down over the Teflon pledgets, the flow of blood into the cavity of the aneurysm ceased, indicating that there was no longer communication with the left ventricle or with the aorta. At this point the ventricular vent was removed from the left ventricular cavity and finger exploration of the left ventricle was carried out (fig. 4). Palpation of the mitral and aortic valves revealed

Figure 2

Appearance of the false aneurysm at the time of the second surgical procedure. Catheters are inserted into the left subclavian artery and the right ventricular outflow tract for cardiopulmonary bypass. A vent is inserted into the left ventricular apex. Adherent pericardium has been dissected away from the anterolateral aspect of the heart. Pressure in the aneurysm was 80/0 mm Hg. Blood within the aneurysm had 100% oxygen saturation.
false aneurysm of left ventricle

no abnormality. With bimanual examination on both sides of the defect it was possible to identify the defect site within the left ventricular cavity; this was situated between the anterior leaflet of the mitral valve and the aortic valve. It was possible to localize the site of the disruption precisely at the base of the heart where the aorta joins the heart in the region of the intermediate portion of the left coronary cusp. The examining finger was then removed from the heart and the left ventricular vent again inserted. The larger opening at the base of the cavity of the aneurysm was then sutured. Closure was accomplished with a row of interrupted mattress sutures tied over pledgets of Teflon felt. The aneurysm wall itself was biopsied and contained only fibrous tissue with no evidence of cardiac muscle. The aneurysm wall was partially excised and the remaining portions were sutured by imbricating the upper edge over the lower edge as in the repair of an umbilical hernia. It was felt that this would reinforce the rupture site. The vent was then removed from the left ventricle, and cardiopulmonary bypass was discontinued. Cannulae were removed from the heart and from the subclavian artery. Postoperative intracardiac pressures were normal.

Postoperative Course

The patient’s postoperative course was complicated by mediastinal hemorrhage resulting from the extensive pericardial and mediastinal adhesions in combination with heparinization. It was necessary to re-explore the mediastinum to control this bleeding and to evacuate mediastinal clots 8 hours following the original surgery.

Figure 3

Appearance of the internal aspect of the false aneurysm. (Left) Removal of organized thrombus which partially obliterates the aneurysm cavity. (Right) A large opening at the base of the aneurysm does not communicate with the heart. Two smaller openings communicate with the left ventricular cavity. Closure of the communicating orifices is done with Teflon sutures reinforced with Teflon felt.
Repair of the false aneurysm. (Above left) Separation sites between the aorta and the base of the heart have been repaired. The thickened epicardial rupture site at the base of the false aneurysm is closed with Teflon mattress sutures reinforced with Teflon felt. (Above right) The false sac of the aneurysm is partially excised and then the remainder is imbricated to reinforce the repair of the site of separation of the aorta from the heart. (Below) Representation of the development of a false aneurysm arising from a separation of the aorta from the heart in the region of the intermediate portion of the left coronary cusp of the aortic valve. The position of the defect was confirmed by digital exploration of the left ventricular cavity. Epicardial hematoma has ruptured into an obliterated pericardial space with formation of a false aneurysm.

Tracheotomy was performed at this time which provided the patient with ventilatory support. The patient made a slow but uneventful recovery and was discharged from the hospital on August 5, 1966. Preoperative and postoperative roentgenograms of the chest are shown in figure 1.

Comments
The pathological anatomy of deficiencies between the aortic root and the heart has
been studied by Edwards and Burchell.¹ They concluded that the essential lesion is a separation between the aortic media and the heart. This lack of fusion occurs at the annulus of the aortic valve. It is ordinarily located, therefore, above the valve. However, in certain cases, the separation between the aortic media and the heart is located below the aortic valve. Such lesions are independent of aortic sinus aneurysms and produce anatomical defects which are dependent upon the specific location below the valve cusps of the aortic valve and a relationship of the defect to the cardiac chambers. The ventricular septum is located below all the parts of the right aortic sinus and separations below the valve in this area produce a ventricular septal defect. Similarly the ventricular septum lies below the right and central portions of the posterior aortic sinus. The anterior mitral leaflet lies beneath the left part of the posterior aortic cusp and the adjacent portion of the left aortic cusp. Edwards and Burchell² stated that they are unaware of examples of separation between the aorta and the heart beneath the posterior part of the left aortic cusp. Beneath the intermediate part of the left aortic cusp there is an area in which the aortic valve is not related to a cardiac chamber or to the pulmonary trunk. Adjacent to the wall of the sinus in this region is epicardium. Included in the series of necropsy specimens studied by Edwards and Burchell is one originally seen by Dr. John Coe of Minneapolis. This specimen was from a 39-year-old woman with an aneurysm at the base of the left ventricle. The mouth of the aneurysm lay between the aortic and the mitral valves and was centered at the level of the central portion of the left aortic leaflet. It was considered that following separation between the aorta and the heart there had been a hemorrhage into the epicardium which was not fatal. It was felt that this hemorrhage organized and created a fibrous and partly calcific wall and that it was in a strict sense a false aneurysm rather than a true aneurysm of the left ventricle. In our case, intracardiac exploration at the time of surgery also located the site of aneurysm formation below the central portion of the left aortic cusp. The resulting aneurysm on pathological sectioning proved to contain no myocardial tissue and is also interpreted as a false aneurysm. It seems likely that these cases are entirely similar. The rapid expansion of the aneurysm in the present case probably resulted when the epicardial hematoma ruptured into the pericardial cavity, which had been previously obliterated by adhesive pericarditis, and thereby produced continued dissection into the pericardial space.

In 1956, Barnard and Brink² reported necropsy studies on two Bantu patients with unique abnormalities of the heart. In both these cases, an additional cardiac chamber was noted which communicated with the heart through a small opening in the region of the annulus of the mitral valve. The additional chamber of the heart included myocardial tissue and was interpreted by the authors as a supernumerary chamber. In 1962, Abrahams and his associates³ reported 12 cases of an unusual form of left ventricular aneurysm occurring in Negroes that was thought to be congenital in origin. All of these lesions were found in Nigerian patients. The aneurysms were situated immediately beneath the aortic and mitral valves and extended around and in the substance of the fibrous ring from which the valves arise. Of seven patients studied postmortem, four had aneurysms with an ostium at the region of the mitral annulus and were probably quite similar to the two cases reported earlier by Barnard and Brink. Three of the autopsy specimens revealed an aneurysm originating below the aortic annulus fibrosis. All of the cases reported in this series produced insufficiencies of the involved valves. Abrahams and his associates cited 17 additional published cases of unusual ventricular aneurysms which they considered similar to the lesion they described. A variety of etiological factors were considered and the authors concluded that the aneurysms originated as herniae through a congenital weakness in the ventricular wall in the region of the atrial ventricular groove. Surgical repair
of the aneurysm was undertaken in two of the cases reported from Nigeria (cases 6 and 7) with unsuccessful results. Chesler and associates added an additional six cases encountered in the South African Bantu at Baragwanath Hospital. In all but one of their cases the lesions were associated with the mitral valve, producing an annular submural left ventricular aneurysm. In all cases, there was evidence of either aortic or mitral insufficiency produced by the adjacent aneurysm. Surgical cure of one aneurysm in Chesler's series was undertaken with the aid of hypothermic arrest (case 1) but was unsuccessful. The authors pointed out the many difficulties associated with the surgical treatment of such aneurysms. These include the location of the orifice through an external approach, the obscuring of the coronary circulation by pericardial adhesions, and the distortion of the associated cardiac valves. Jacobs and Elliott, and Lurie discussed the problem of left ventricular aneurysms of obscure origin occurring in African patients. Brink and Barnard, and Higginson and Keeley have each described an isolated case of an unusual aneurysm which may be similar to the submural type described by Abrahams and associates. In 1963, Schrire and Barnard described the first successful repair of an aneurysm of undetermined etiology occurring in an adult Bantu. This is probably an example of a submural aneurysm. The operation was carried out under cardiopulmonary bypass and involved closure of the ostium in the ventricular wall and resection of the aneurysmal sac.

The patient described in this report is a Negro and the relationship of aneurysms described in African patients is worth some consideration. Abrahams and associates have provided the most complete discussion of the anatomic pathology in African patients. Their description of the subaortic type of aneurysms does not fit well with the pathology encountered in our patient since the currently reported aneurysm was essentially a false aneurysm and did not extend circumferentially in the annulus of the aortic valve. In addition, there was no associated valve insufficiency, which has been a common finding in all cases of annular subaortic aneurysms. The present case did not arise from the submural position and there is no relationship to annular submural aneurysms or to the one previously successfully treated by surgery. It seems unlikely, therefore, that this case represents another example of the disease encountered in South African Bantu.

The etiology of the present case is unknown. The majority of aneurysms arising from the left ventricle are secondary to atherosclerosis of the coronary arteries. Syphilis is usually cited as the next most common cause. Less frequently cited causes include mycotic infection, polyarteritis nodosa, rheumatic myocarditis, and trauma. This patient had both documented tuberculosis and systemic lupus erythematosus. It cannot be proved whether either of these processes produced a weakness in the region of the junction of the aortic root and the heart. It seems most likely that in this case, as in most cases of deficiencies between the aortic root and the heart, the pathological process arose from an inherent weakness of the structures in this area and that the previous pericarditis prevented a fatal cardiac tamponade at the time epicardial hemorrhage and disruption occurred.

Addendum

Since this report was submitted, Waldhausen and associates have reported the successful repair of a submural annular aneurysm of the left ventricle in a 5-year-old Caucasian which occurred 15 months after severe precordial trauma. This remarkable case is probably anatomically similar to the case treated by Schrire and Barnard, although the etiology of the two lesions may be different. It is anatomically different from the present case.

References
FALSE ANEURYSM OF LEFT VENTRICLE

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A Cycle of Life in Language, Too.

The means of reviving a language lie in the heart of the poet and upon his lips and between his fingers. The poet is the mediator between the creative power and the people. He is the wire that transmits the news of the world of spirit to the world of research. The poet is the father and mother of the language, which goes wherever he goes. When he dies, it remains prostrate over his grave, weeping and forlorn, until another poet comes to uplift it.—Spiritual Sayings of Kahlil Gibran: Edited by Anthony Rizcallah Ferris. New York, Citadel Press, 1962, p. 48.
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Circulation. 1967;35:954-961
doi: 10.1161/01.CIR.35.5.954

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