Pulmonary Artery Aneurysms

Report of a Case Treated by Surgical Intervention

By Brian Blades, M.D., William Ford, M.D., and Patrick Clark, M.D.

The successful surgical treatment of an aneurysm of the left main pulmonary artery is reported. It appears that, after ligation of the main pulmonary artery, it is desirable not to remove the lung but preserve it as a space occupying organ which will retain some function. Literature on the subject of pulmonary aneurysms is reviewed.

Pulmonary artery aneurysms are relatively rare lesions. One hundred and fifty-two cases have been found in world literature. The purpose of this communication is to review briefly available information on the subject and to report a case in which successful surgical treatment has been achieved.

Boyd and McGavack, in 1939, reported 111 cases of pulmonary aneurysm proved at necropsy. In 1941, Deterling and Clagett added 36 cases in their excellent paper on this subject. Five more authenticated cases reported since 1947 may be added to the list. In 1946, Thompson and Gerstl recorded a case which was not included in the bibliography of Deterling and Clagett. Since 1947, Dotter and Steinberg have reported two cases and Lillian has had one case. Available evidence indicates that one aneurysm of the pulmonary artery may be found in approximately 14,000 necropsies.

Jules, Breslin and others have described the symptoms of pulmonary aneurysm which include dyspnea, cough and hemoptysis. These manifestations of the disease may occur early. Chest pain, cyanosis, edema, ascites and clubbing of the fingers are late manifestations. Tachycardia is sometimes evident with associated polycythemia. Roentgenograms of the chest often reveal a well delineated hilar shadow and hypertrophy of the right heart may be found. Pezzi's sign of hilar dance caused by transmission of an expansile pulsation may be observed. The lesion can usually be delineated by angiocardiography.

Aneurysms of the pulmonary artery occur in a younger age group than aortic aneurysms. The sex incidence is about equal. Aneurysms of the left pulmonary artery are more common than those on the right.

The etiology of pulmonary artery aneurysms is not understood, but it might be safe to assume that conditions which raise the pulmonary arterial pressure may cause dilatation of the vessels. Important contributory effects, therefore, might result from a patent ductus arteriosus, mitral stenosis, interauricular septal defects and other congenital anomalies. Deterling and Clagett concluded that pulmonary aneurysms have a congenital origin in about 47 per cent of the cases they studied. Boyd and McGavack were of the same opinion.

Miscellaneous etiologic factors which have been considered include syphilis, arteriosclerosis, trauma, subacute bacterial endocarditis, tuberculosis and atheroma. Earlier writers on the subject believed that syphilis was a probable etiologic factor in about one-third of the cases. This conclusion might be questioned.

It is important to note that about one-third of the cases of pulmonary aneurysm die from sudden rupture of the lesion. It is apparent, therefore, that this condition deserves prompt and adequate surgical treatment if possible. A notable exception is a patient described by Mason in 1938, who has been observed for 17 years and has suffered no ill effects from the lesion.

Case History

A 59-year-old white man, a welder by trade, was admitted to the George Washington University Hospital on Sept. 27, 1949. His only complaint was that seven weeks before admission he coughed and...
blood was apparent, but never in great amounts. There were no complaints of chest pain, dyspnea, fatigue or anorexia.

The past history revealed that the patient had had typhoid fever at the age of 10, which was complicated by pneumonia. He also had been troubled with attacks of acute sinusitis in the past. The family history was significant in that one brother had had pulmonary tuberculosis.

Examination through the bronchoscope revealed a normal tracheobronchial tree. Bronchial washings were negative for acid fast bacilli but, on one occasion, the Papanicolaou test revealed cells which were considered suspicious of tumor. All other washings were negative. An electrocardiogram demonstrated slight right ventricular strain.

Immediately after these examinations the patient stopped coughing up blood-streaked sputum and returned to his home. Hemoptysis recurred, however, the following month and again in February of 1950 when he had a severe attack of hemoptysis and it is estimated that he coughed up about 200 cc. of blood. During the same period he developed dyspnea on exertion.

**Fig. 1.** Frontal projection demonstrating a prominent left hilar shadow with density extending upward.

The patient was a well developed, well nourished white man. Temperature, pulse and respiratory rates were normal. The blood pressure was 150/90. The anteroposterior depth of the chest wall was increased. Breath sounds were somewhat distant and there was some question of early emphysema.

Examinations of the blood and urine were within normal limits and repeated serologic tests for syphilis were negative.

Roentgen examination of the chest on Sept. 28, 1949, revealed a prominent shadow at the level of the left hilum with the density extending somewhat upward. The diaphragms were flat and the roentgen appearance of the chest suggested early emphysema (figs. 1 and 2). Visualization of the tracheobronchial tree with lipiodol was not helpful.
Roentgen examination of the chest at that time revealed enlargement of the shadow at the left hilum. Examinations with the bronchoscope were again negative, but a suspicious Papanicolaou stain was obtained.

During this period of study, the possibility of an aneurysm of the left pulmonary artery was considered because of the odd configuration of the shadow. It must be admitted, however, that this possibility was dismissed in favor of the diagnosis of bronchiogenic carcinoma and angiocardiography was not done.

Preoperative studies of the blood, urine and proteins were normal. The bleeding, clotting and prothrombin times were normal. The vital capacity was 72 per cent of the expected measurement and another electrocardiogram revealed evidence of right ventricular strain.

Operation: The left chest was entered through a posterolateral incision at the level of the fifth rib.

Fig. 2. Lateral view showing the midthoracic lesion.

Fig. 3. Drawing of posterior-anterior view showing relative size, shape and position of aneurysm.
After the lung was exposed, a few lymph nodes could be seen and palpated at the level of the hilum but no mass was palpable in the lung itself. Anterior displacement of the lung revealed an aneurysm of the left pulmonary artery (fig. 3). The lesion was elliptical in shape and measured approximately 7 cm. in length and was about 5 cm. in depth at its center. The origin of the sac was about 2 cm. from the main trunk of the left pulmonary artery and extended well below the artery to the dorsal division.

The lung was not removed. The reasons for leaving the organ after ligation of the left pulmonary artery will be discussed later.

On the first day after operation the patient's temperature was 40°C and remained at rather high levels for four days and finally fell to normal on the eleventh day after the operation. During this period there was no evidence of a wound infection, infection of the urine or other sources of temperature elevation outside of the chest itself. The pulse rate

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

**Fig. 4.** Frontal projection taken one week after operation demonstrating generalized density in the left chest.

of the lower lobe ending at the level of the artery to the basal division.

Since the origin of the aneurysm was at the level of the upper lobe arteries, it became apparent that these vessels could not be saved. Accordingly, the left pulmonary artery was ligated and divided proximal to the aneurysm and each branch of the artery arising from the aneurysm was ligated. The lesion was tightly adherent to the left lower lobe bronchus and it appeared that erosion had occurred at this level. Since the aneurysm was completely excluded from the pulmonary circulation, the sac was not removed.

was also elevated and varied from 130 to 140 for three days. On the tenth postoperative day the pulse rate had stabilized at 90. Early examinations with the electrocardiogram revealed the right ventricular strain, which had been evident before the operation, and sinus tachycardia. Roentgenograms of the chest taken at daily intervals demonstrated a gradually increasing density over the entire left lung field. After a week the density in the apex of the lung began to clear (figs. 4 and 5).

Before operation the volume of packed red cells was 48 mm. but by March 6, ten days after surgical intervention, the packed red cells had fallen to 37
mm. in spite of transfusions of 1000 cc. of whole blood. It seems unlikely that this sharp reduction resulted from the deterioration of transfused red cells. The leukocyte count was 12,850 on the first postoperative day and quickly fell to normal limits. Repeated attempts at thoracentesis secured only small amounts of blood-stained fluid which was sterile. It seemed apparent, therefore, that the haziness over the left chest was not entirely the result of accumulated pleural fluid.

FIG. 5. Frontal projection taken two weeks after operation demonstrating clearing in left apex.

The patient left the hospital on the eleventh postoperative day and has been seen at frequent intervals as an out patient. Three weeks after the operation a roentgenogram of the chest showed significant clearing of the opacity which was noted from the time of operation. On this same date an electrocardiogram was obtained and did not show right ventricular strain. The patient's temperature was normal and he had a full and regular pulse in the neighborhood of 96 per minute. His appetite was good. His only complaint was slight dyspnea on exertion. Four weeks after the operation the patient had begun to gain weight, was afebrile, and his only complaint continued to be slight dyspnea after exercise.

**Comment**

A decision was made in our case not to remove the lung after ligation of the artery. The patient appeared to have moderate em-
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has employed pulmonary artery ligation as a preliminary to total pneumonectomy in poor risk patients.

Janton, Redondo and Scott\textsuperscript{11} have studied a patient in whom accidental ligation of the left pulmonary artery was done. They found that carbon dioxide and oxygen values were 3.74 per cent and 19.14 per cent, respectively, as compared to 1.65 per cent and 10.15 per cent for the other side. The observations of Roche and his co-workers\textsuperscript{13} in a similar case in which the left pulmonary artery had been ligated to control hemorrhage during the performance of an operation on a patent ductus, were in general agreement with those of Janton and his co-workers. They also found that the right pulmonary artery and right heart chambers had normal pressures when determined by cardiac catheterization.

Bloomer and Harrison,\textsuperscript{12} in experiments on animals, concluded that gas exchange after pulmonary artery ligation is the result of blood flow in anastomoses between the pulmonary artery and the pulmonary capillary bed. They employed bronchiospirometry, blood oxygen determinations and cardiac output studies in their experiment.

**CONCLUSION**

Since accumulated evidence indicates that approximately one-third of the patients with aneurysms of the pulmonary artery die of sudden severe hemoptysis from rupture of the aneurysm, it becomes apparent that these lesions should be treated by surgical intervention whenever a positive or tentative diagnosis has been made.

Experimental evidence and recent experience in the treatment of a case of pulmonary artery aneurysm indicates that it is not necessary to remove the lung after ligation of the main pulmonary artery and, moreover, it is probably desirable to leave the organ in the body as living, space filling tissue which retains some of its function.

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Pulmonary Artery Aneurysms: Report of a Case Treated by Surgical Intervention
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Circulation. 1950;2:565-571
doi: 10.1161/01.CIR.2.4.565

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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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:
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