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Pulmonary Embolectomy: *

Successful Removal of a Massive Pulmonary Embolus with the Support of Cardiopulmonary Bypass— Case Report

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ALTHOUGH Trendelenburg⁷ first proposed an operative procedure to remove pulmonary emboli in 1908, few successful operations have been carried out. In 1958, Steenburg⁵ reported the first successful pulmonary embolectomy performed in the United States and summarized the world experience. Recently a successful pulmonary embolectomy was performed in this clinic supporting the patient during operation with cardiopulmonary bypass. The details of this case are described in the following report:

Case Report

H. S., a 67-year-old man, was first seen in August 1960 complaining of low back pain following an automobile accident. The diagnosis of a ruptured L₅-S₁ disc was confirmed with a myelogram during a hospital admission in Septem-

ber 1960 and conservative therapy advised. Four days following discharge he awakened with a stabbing right chest pain and subsequently developed a cough productive of a small amount of blood. He came to the emergency department and was admitted immediately. There were no signs of phlebitis and a chest roentgenogram showed a circular area of increased density in the right costophrenic angle. Serial electrocardiograms revealed only a sinus tachycardia of low voltage with a prolonged Q-T interval and an occasional premature ventricular contraction. The diagnosis of a pulmonary infarct with pneumonia was made and anticoagulants and antibiotics begun. By the third hospital day he was asymptomatic and was discharged ten days later. Coumadin was continued until November 7, 1960, when he was discharged from the clinic. On December 2, 1960, a ruptured intervertebral disc was removed and convalescence was uneventful.

On January 18, 1961, the patient suddenly developed right anterior chest pain aggravated by breathing. He returned to the hospital two days later with increasing pain and hemoptysis. He was promptly admitted and examination showed a blood pressure of 115/78, pulse 96 per minute, respiratory rate of 16 per minute, and a temperature of 37.2° C. orally. He appeared short of breath and on examination of the chest a few

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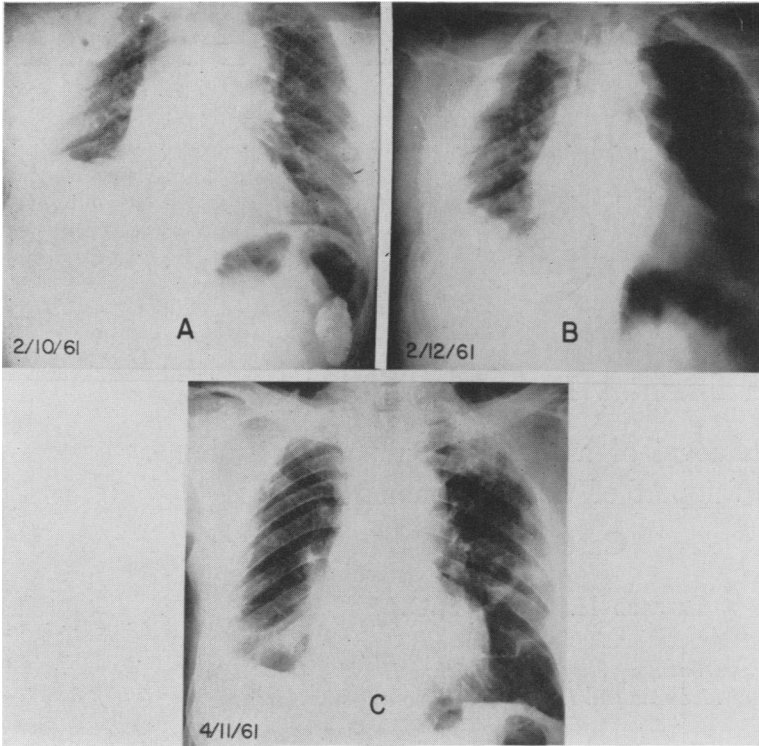


FIG. 1. A. Portable chest film on the day following right lower lobectomy. B. Portable chest film following a massive pulmonary embolism. No vascular markings are seen in the left lung field. C. Chest film two months following pulmonary embolism. Vascular markings in the left lung field are relatively normal.

rales were found over the right upper lobe area; no friction rub was heard. There was no edema or other signs of phlebitis in the extremities. The venous pressure in the right antecubital fossa was 45 mm. saline. The hematocrit was 37 per cent

and white cell count 10,500 cells per cubic millimeter. The chest roentgenogram showed two densities in the right lower lobe and an electrocardiogram showed the findings of slight right heart strain. Anticoagulants were administered

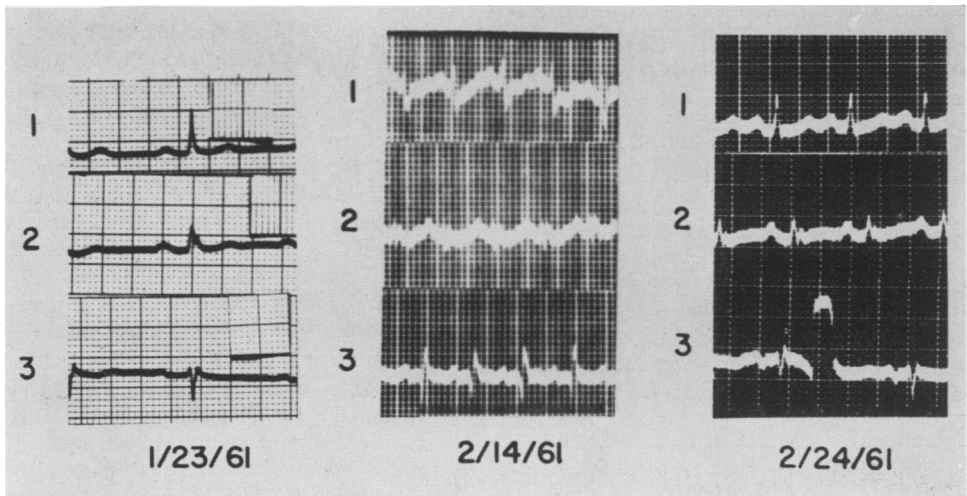


FIG. 2. Electrocardiograms, three standard limb leads. Typical electrocardiographic signs of pulmonary embolism can be seen in the record of 2/14/61. In this record there is a deep S₃ and a tall R₃. The ST segments are elevated in lead 3 and depressed in lead 1. These changes are not present in the record of 1/23/61 and had disappeared by 2/24/61.

and during the next two weeks the symptoms disappeared. However, because of the persistence of the densities in the right lower lobe, an exploratory thoracotomy was performed on February 9, 1961 to exclude cancer. Three separate infiltrations were found in the lower lobe. With a tentative diagnosis of cancer, lobectomy was performed. Histological examination of these areas showed them to be organizing infarcts.

The postoperative course was uneventful for three days until on February 12, 1961, at 6:30 a.m. he suddenly became faint and severely dyspneic. The pulse was 160 per minute, respirations 44 per minute, and blood pressure 122/70 mm. Hg. Bilateral basilar rales were heard. There were no signs of phlebitis. The venous pressure was 90 mm. saline. A portable chest roentgenogram (Fig. 1B) showed increased radiolucency of the left lung field as compared to the right and an electrocardiogram showed a marked shift in the axis to the right (Fig. 2).

Treatment with digitalis and anticoagulants was started immediately, but during the next five days a tachycardia of 120 to 140 per minute persisted and on several occasions vasopressors were required for hypotension. He remained dyspneic and restless with cold clammy extremities. The venous pressure ranged between 140 and 150 mm. saline. His clinical course was interpreted as being due to a massive pulmonary embolism and thus a pulmonary embolectomy was undertaken on February 17, 1961.

The anesthesia used was sodium pentothal, nitrous oxide, and succinyl choline. During the induction of anesthesia on two occasions the patient became pulseless and cyanotic but responded to external cardiac massage and vasopressors. A median sternotomy was performed and after heparinization with 3.0 mg. of heparin kg./body weight, the vena cavae and right femoral artery were cannulated. No abnormality could be felt in the pulmonary arteries. Cardiopulmonary bypass using a screen oxygenator was begun and a longitudinal incision made in the main pulmonary artery. With the index finger in the left pulmonary artery a soft thrombus was felt totally obstructing the vessel. Stone forceps and an open tip suction were used to remove the thrombus in several fragments. The right pulmonary artery was explored in a similar fashion removing a few small clots. The incision in the pulmonary artery was closed and bypass which totalled 35 minutes was stopped.

After the heparin was neutralized with Polybrine, the median sternotomy incision was extended to the umbilicus and the duodenum reflected to expose the vena cava. It was plicated distal to the renal veins with three mattress sutures

of 2-0 arterial silk to prevent further pulmonary emboli, after which the incision was closed.

During the immediate postoperative period his dyspnea markedly improved and his blood pressure remained stable at 120/80 mm. Hg. The chest roentgenogram showed a return of vascular markings in the left lung field and the electrocardiogram showed a shift of the axis back to normal. To aid in the elimination of secretions a tracheotomy was performed on the second postoperative day. On the fifteenth postoperative day he developed tenderness and swelling of the left thigh which responded to conservative measures. A tension abscess formed in the left lingular segment of the lung which responded to the insertion of a chest catheter and the use of suction. He was discharged on April 1, 1961, and when last seen on June 20, 1961 had remained well.

Discussion

The occurrence of a massive pulmonary embolus may be suggested by the sudden onset of circulatory collapse with hypotension, shortness of breath, sweating, tachycardia, cyanosis, and frequently death. However most of the typical clinical signs and symptoms associated with pulmonary embolism such as chest pain and hemoptysis are present only if pulmonary infarction develops.¹ To prove a pulmonary embolism is difficult. The electrocardiogram is very helpful if an acute shift of the heart axis to the right occurs. However there is disagreement among cardiologists as to what findings in the electrocardiogram, if any, should be considered diagnostic. It would seem that the portable chest roentgenogram is the simplest definitive study.² In this case absence of the left pulmonary vascular markings was felt diagnostic of an embolus obstructing the left pulmonary artery. In lesser degrees of obstruction, the findings on chest roentgenogram are more difficult to evaluate. Stoney and Adams have successfully localized pulmonary emboli in animals using a single film angiography technic but no experiences have been reported in man.⁶

Although few patients survive a massive pulmonary embolism, the possibility of pulmonary embolectomy using cardiopulmo-

nary bypass should be considered in each case. This year two patients survived emboli for four and seven days respectively which when seen at postmortem examination had almost totally obstructed both main pulmonary arteries. Most attempted embolectomies have died with irreversible ventricular fibrillation secondary to hypoxic myocardial damage.⁸ This often occurs during the induction of anesthesia or before the embolus can be removed. This hazard may be aborted by cannulation of the jugular vein and femoral artery under local anesthesia, so partial cardiopulmonary bypass can be started before general anesthesia is begun. Our patient on two occasions during the induction of anesthesia had an episode of severe hypotension, one of which required external cardiac massage for resuscitation.³

Following embolectomy the inferior vena cava was plicated beneath the renal veins to prevent any emboli from the lower extremities.⁴ No excessive bleeding was noticed even though the patient had been heparinized during cardiopulmonary bypass. In the postoperative period and following discharge no further emboli have occurred.

Summary

A case is reported in which a massive pulmonary embolus was removed while the patient was supported with total cardiopulmonary bypass. The diagnosis was established preoperatively by the obliteration of the pulmonary vascular markings of one lung on a chest roentgenogram. Following embolectomy the vena cava was plicated to prevent further emboli. This patient has remained well following discharge without further episodes of emboli. Since patients

with a massive pulmonary embolism are such poor operative risks, circulatory support by partial cardiopulmonary bypass preoperatively and during the induction of general anesthesia is suggested. As soon as possible, total cardiopulmonary bypass should be used.

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