Pulmonary Embolism, Pulmonary Angiography,
Pulmonary Embolectomy*  
Report of a Case
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Pulmonary embolism ranks high on the list of causes of sudden death. Waggenspack has stated that in reported series of "sudden deaths," 5 per cent are usually attributed to pulmonary emboli.

The incidence of pulmonary embolism found at necropsy has been reported to range from 2.8 per cent to 25.7 per cent. As a cause of postoperative death the incidence has been reported from 0.11 per cent to 5 per cent.

Although many patients succumb to pulmonary embolism within a period of minutes after the clinical manifestations become apparent, some patients survive for prolonged periods before dying, even from massive pulmonary infarction. Rosenberg, Pearce and McNulty in a review of 92 fatal cases of massive pulmonary embolism reported that 12 patients survived one hour or longer after their initial symptoms of embolism. Patients surviving for such periods of time, but ultimately dying of the disease, obviously might be considered candidates for pulmonary embolectomy.

From the time Tredelenburg proposed the operation of pulmonary embolectomy in 1908 until 1961, there had been approximately 23 successful cases reported. Since 1961, the successes of Sharp and Cooley, et al. in performing pulmonary embolectomy with the aid of extracorporeal circulation have stimulated surgeons to renew the direct surgical attack on this highly fatal disease. A recent review lists nine successful cases reported between 1962 and 1963.

The decision to undertake pulmonary embolectomy is often difficult because of the difficulty of diagnosing massive pulmonary embolus. Although many diagnostic measures are available, any surgeon who operates upon a patient with suspected pulmonary embolus will feel some doubts which will be fully relieved only when he is able to actually remove blood clots from the pulmonary artery.

The following case report is presented as an example of the value of preoperative pulmonary angiography as a diagnostic tool, and also to illustrate that by removal of massive pulmonary emboli, the major and minor arterial vessels are restored to patency.

CASE REPORT

Mrs. I. D., a 58-year-old white housewife, was admitted to the Louisiana State University Surgical Service at Charity Hospital of Louisiana, in New Orleans, February 3, 1964. She gave a history of upper abdominal pain, belching, flatulence, fatty food intolerance and occasional nausea for the last four years. She had previously been seen in the outpatient clinic and workup had revealed chronic cholelithiasis. She was admitted for elective cholecystectomy.

Review of systems and past history were contributory in that she had been treated in vascular clinic in 1961 for varicose veins and superficial thrombophlebitis of the right leg.

Physical examination on admission revealed a well-developed, obese, white woman in no distress. Blood pressure was 140/90, respiration 16/minute, pulse 80/minute, temperature 98.4°.

The lungs were clear and resonant to percussion and auscultation. Examination of the heart revealed no clinical enlargement, normal sinus rhythm, A2 louder than P2, no murmurs or thrills. There was epigastric and right upper quadrant tenderness without rigidity or palpable

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masses. Bilateral varicosities of the greater and lesser saphenous systems were noted, but no edema, calf tenderness or evidence of stasis. Otherwise, the physical examination was within normal limits.

Laboratory examinations included complete blood count, fasting blood sugar, urinalysis, blood urea nitrogen, CO₂, chlorides, serum proteins, serum bilirubin, SGOT, and thymol turbidity. All were within normal limits as were roentgenograms of the chest and electrocardiogram. A gall-bladder visualization performed as an outpatient on November 19, 1963 was reported to show numerous radiopaque calculi.

On February 5, 1964, under general anesthesia, cholecystectomy was performed. No untoward events occurred during the surgical procedure.

She tolerated surgery well and her postoperative course was uneventful until approximately noon of February 10, 1964 when while sitting in a chair she suddenly complained of pain in both sides of her neck, weakness, and feeling faint. Within a matter of minutes she became comatose, cyanotic and exhibited minimal dyspnea. Her pulse was weak and thready at 120 to 130 per minute. Blood pressure was unobtainable until an intravenous metaraminol (Aramine) drip was instituted, by which means blood pressure was maintained at a palpable level of 80 mm. systolic. Heart tones were faint, but P₂ was noted to be louder than A₂. Breath sounds were normal bilaterally. Examination of the extremities for evidence of venous thrombosis was entirely normal. A portable chest roentgenogram (Fig. 1B) showed decreased pulmonary vascularity of the entire right lung and left lower lobe. In comparison with her preoperative film (Fig. 1A), the proximal pulmonary artery appeared dilated and the right pulmonary artery appeared to terminate abruptly at the level of the right upper lobe artery takeoff. An electrocardiogram revealed T₂ and T₃ low, TV₂ and V₃ diphasic, depressed ST segment with diphasic T in lead I, deep S in V₆. These changes were felt, by a cardiology consultant, to be consistent with right ventricular strain. SGOT was 58, hematocrit 42 per cent, white blood count 15,950 with 90 per cent segmented neutrophiles, 9 per cent lymphocytes and 1 per cent monocytes.

The patient was thought to have suffered a massive pulmonary embolus. Because of absence of chest pain or dyspnea, however, some skepticism was justified and it was elected to perform pulmonary angiography. This was performed under local anesthesia of 1 per cent procaine. The left external jugular vein was exposed and cannulated with a No. 14F polyethylene tube, which was passed blindly into the right atrium. The position of the intracardiac catheter was verified by x-ray and 50 ml. of 80 per cent sodium iothalamate (Angiocronray) was injected by hand through the catheter. Multiple x-ray exposures were taken at a rate of four films per second for four seconds. The presence of massive bilateral pulmonary artery occlusion was demonstrated (Fig. 2). The intracardiac catheter was withdrawn to the superior vena cava to be used for monitoring the central venous pressure and the patient was removed to the operating room.

**FIG. 1A**: Admission chest roentgenogram. Note normal pulmonary vascularity. **FIG. 1B**: Chest roentgenogram immediately following onset of symptoms of pulmonary embolism. The only visible pulmonary vascular markings are those to left upper lobe.
Under endotracheal anesthesia, a midline sternotomy incision was made at 6:40 p.m., some seven hours after the onset of symptoms. The patient was heparinized and placed on total cardiopulmonary bypass with vena cava cannulations performed via the right atrial appendage and oxygenated blood returned from the pump oxygenator through the right femoral artery. A disposable plastic bag bubble oxygenator was used. The circuit was primed with 500 ml. 10 per cent low molecular weight dextran in normal saline (Rheomacrodex) and 1000 ml. of 10 per cent low molecular weight dextran in 5 per cent dextrose (Rheomacrodex) buffered with 44.4 mEq of sodium bicarbonate. After cardiopulmonary bypass was established, the main pulmonary artery was incised from just above the pulmonary valve to its bifurcation. Both pleural cavities were opened and the lungs were individually submitted to gentle manual compression. This maneuver, combined with intraluminal suction resulted in removal of large and small branching clots bilaterally. Compression and suction were continued until free bleeding was seen to be coming from the distal right and left pulmonary arteries. The pulmonary arteriotomy was then closed with continuous 0000 black silk sutures and cardiopulmonary bypass discontinued. Heart action remained excellent throughout the entire period of bypass and satisfactory arterial and venous pressures were maintained after bypass.

During the above procedure, a second surgeon exposed the right and left common, superficial and deep femoral veins. These veins were exposed immediately after discontinuing cardiopulmonary bypass. Free bleeding was obtained from the right common and deep femoral veins, but not from the right superficial femoral vein. The right superficial femoral vein was ligated. There was obvious thrombus present in the left common and superficial femoral vein. The left common femoral vein was opened and, utilizing a glass suction rod, the clot was aspirated from the common femoral vein until free bleeding occurred from above. Clots were also aspirated from the deep and superficial left femoral veins until scanty back-bleeding occurred. The left femoral vein was ligated.

Cardiopulmonary bypass cannulae were removed, protamine administered, the femoral arteriotomy closed and all incisions closed. The patient was sent to the recovery room in good condition.

The first 24 hours postoperatively were marked by several hypotensive episodes which responded to vasopressors. The remainder of her postoperative course was essentially uneventful.

A pulmonary angiogram was taken on March 2, 1964, 21 days following surgery. No evidence
of pulmonary arterial obstruction was seen, even in the smaller pulmonary vessels (Fig. 4).

She was discharged on March 30, 1964. On April 3, 1964, she was readmitted with severe thrombophlebitis of the left leg. This responded satisfactorily to bed rest, anticoagulants and antibiotics. She was discharged on April 30, 1964.

DISCUSSION

The x-ray film of the chest in this particular patient (Fig. 1B) was quite typical of a massive pulmonary embolus without infarction. The paucity of pulmonary vascular markings of the entire right lung and of the left lower lung field, with a dilated proximal pulmonary artery, is characteristic. An electrocardiogram showing the development of right ventricular strain pattern lent added support to the diagnosis. The absence of chest pain or dyspnea, however, led several observers to be openly skeptical of the diagnosis. The obvious pulmonary artery occlusion revealed by pulmonary angiography resolved all doubt and made surgical intervention mandatory.

Although the risk of pulmonary angiography cannot be denied, several authors have demonstrated the usefulness of the procedure.1-3 A comparison of Fig. 2, the angiogram, and Fig. 3, a photograph of the angiogram with actual pulmonary blood clots superimposed, demonstrates the high degree of accuracy with which the location and the extent of the vascular occlusion can be predicted. The angiogram also demonstrates that the major portion of the pulmonary circulation can be blocked and a patient still survive for several hours and successfully undergo pulmonary embolectomy. In this case, only the left upper lobe and a very small branch in the middle lobe are seen to be open. Figure 4 an angiogram performed on the 21st postoperative day, demonstrates excellent circulation to both lung fields. It is possible that some small clots may have remained in the peripheral lung vessels following embolectomy and that lysis of these clots occurred. It would appear, however, that 21 days would be a rather short interval for actual recanalization of the obstructed pulmonary vessels to have occurred. The time necessary for complete organization of an embolus is usually thought to be a month to six weeks.4 It is our feeling that repeated compression of the lungs and aspiration of clots from within the pulmonary arteries actually served to remove even small clots. Figure 3 demonstrates many small branching thrombi and a number of smaller clots were lost by way of the suction machine at the operating table.

REFERENCES