Acute Massive Pulmonary Embolism

Successful Surgical Treatment Using Temporary Cardiopulmonary Bypass


Insidious venous thrombosis resulting in fatal pulmonary embolism remains one of the most serious and dreaded complication in clinical practice. Despite the widespread interest in the problem of thromboembolism during the past 6 decades, many aspects of the pathogenesis, clinical courses, and therapy remain confused and obscure. De Bakey's comprehensive review 1 demonstrated that even with newer methods of prophylaxis and therapy the incidence of thromboembolism had not shown a remarkable or significant change during the past 50 years. In a collected series of 3,077,495 surgical operations, 4,567 deaths occurred from pulmonary embolism; and among 375,000 autopsy cases, 10,497 were attributable to pulmonary embolism. When these statistics were arranged chronologically into decades, no significant trends in incidence were noted, indicating that pulmonary embolism is even today a persistent and constant threat to life in patients of all types.

Trendelenburg 2 in 1908 analyzed the clinical course in 9 patients dying of massive pulmonary embolism and noted that in 7 the interval between onset of the embolism and death was 10 to 60 minutes and that in 2 deaths occurred immediately. He proposed a technique of immediate embolectomy—an operation which since his report has been generally known as the Trendelenburg operation. He attempted 3 such operations, the longest survival being 37 hours. In 1924 Kirschner 3 reported the first successful pulmonary embolectomy with survival. When the technique advocated by Trendelenburg and Kirschner was used in the United States between 1920 and 1940, results were uniformly unsuccessful. Steenburg 4 reviewed the world literature in 1958 and collected 12 reports from European countries of patients with massive (i.e., sufficiently severe to produce sustained hypotension or to impair function of vital centers) pulmonary embolism who survived pulmonary embolectomy and were discharged well from the hospital. He reported one successful embolectomy in a 64-year-old woman, operated upon at the Peter Bent Brigham Hospital, who was the first survivor of the Trendelenburg operation for massive pulmonary embolism in this country. Wieberdink 5 in 1960 recorded a successful result of the Trendelenburg operation in a patient with massive embolism, and recently Dubost 6 reported one successful case from France among 5 attempts. Allison 7 in 1960 reported a successful pulmonary embolectomy using cardiac inflow occlusion under general body hypothermia. Operation...
was performed 13 days after the initial embolus, when the patient had evidence of increased systemic venous pressure and acute cor pulmonale. During a brief period of circulatory arrest, the main pulmonary artery was opened, and emboli were removed from both pulmonary arteries, using strong suction and long forceps.

Recently, using the pump oxygenator for temporary cardiopulmonary bypass, we operated upon a patient with massive pulmonary embolism who is the first reported case of survival following pulmonary embolectomy by this method.

Report of a Case

A 37-year-old woman was first admitted to Jefferson Davis Hospital on April 4, 1961, complaining of lower abdominal pain associated with pain in her right shoulder. She was found to have a ruptured tubal pregnancy, and a total abdominal hysterectomy and right salpingo-oophorectomy was performed. Fever developed after operation but finally responded to antibiotic therapy, and she was discharged from the hospital on April 15, 1961.

The following morning, as she climbed out of a tub bath, she experienced sudden, crushing substernal and left costal pain, associated with extreme dyspnea. This was followed by 2 episodes of syncope, and she was returned to the hospital in profound vascular collapse 12 hours after the onset of symptoms.

Physical examination revealed an acutely ill, almost ashen woman whose blood pressure was not detectable. Her respirations were rapid and shallow. The neck veins were severely distended. Auscultation of the lungs revealed no abnormality. The heart was not enlarged, and no murmurs were heard. A gallop rhythm was present at the apex. The abdomen was mildly distended, and a well-healed transverse scar was present in the lower abdomen. The liver edge was palpable at the level of the umbilicus. Vaginal examination showed induration of the vaginal cuff with grossly purulent drainage. No evidence of venous disease or edema of the lower extremities was present.

Laboratory studies revealed a hemoglobin level of 8.4 gm. per cent, a hematocrit of 25 per cent, and a leukocyte count of 18,200 per cubic millimeter. Urinalysis demonstrated albuminuria of 50 mg. per cent, with from 15 to 20 red blood cells and from 10 to 12 white blood cells per high-power field. The blood urea nitrogen level was 16 mg. per cent; the serum sodium level, 144 mEq/L; the serum potassium level, 5.6 mEq/L; the serum chloride level, 103 mEq/L; and the carbon dioxide combining power, 18.7 mEq/L. The electrocardiogram revealed a pattern of right bundle branch block. Roentgenographic examination of the chest was suggestive of decreased pulmonary vascular markings on the left but was otherwise not unusual.

Immediately following admission to the emergency room an intravenous infusion containing a vasopressor (phenylephrine hydrochloride) was begun. Her systolic blood pressure rose to 90 mm. Hg, and she regained consciousness. She was admitted to the hospital with a diagnosis of massive pulmonary embolus, probably secondary to pelvic thrombophlebitis. Anticoagulant therapy was instituted, using oral bis-hydrosycoumarin and both intravenous and intramuscular heparin sodium. Digiotor was administered, attaining full digitalization in a 12-hour period. Over the next 24 hours the patient's condition gradually deteriorated, requiring a progressively increasing amount of vasopressors to maintain her systolic blood pressure at 90 mm. Hg. Neck veins remained markedly distended, and she again began to complain of anterior chest pain. A pronounced gallop rhythm was present, and the basal second sound was widely split with accentuation of the pulmonary component. The liver edge was noted to have become quite tender. Further studies at this time revealed an arterial pH of 7.48, an arterial oxygen saturation of 84.9 per cent, an arterial pCO2 of 34 mm. Hg, and an end-tidal alveolar pCO2 of 27 mm. Hg.

Surgical consultation was obtained, and emergency pulmonary embolectomy was recommended. Two hours later, after preparations for cardiopulmonary bypass were completed, the patient was anesthetized with nitrous oxide, oxygen, pentothal sodium, and meperidine. At 2:00 A.M. on April 18, a median sternotomy was performed, 30 hours after admission to the hospital and 42 hours after the onset of symptoms. Immediately on entry into the pericardium, turbid fluid was encountered, which was subsequently proved on culture to be sterile.

The pulmonary artery, right ventricle, right atrium, and both venae cavae were all severely distended, and the myocardium was cyanotic and contracting weakly. Heparin sodium (1 mgm. per kilogram of body weight) was administered intravenously, the dosage approximately half the usual amount for bypass because of the previous anti-

Multiple emboli removed from both pulmonary arteries, showing many branching specimens which were lodged in secondary and tertiary divisions of vessels.

coagulant therapy. A disposable bubble-diffusion oxygenator and roller pumps were used, maintaining a flow rate of 40 ml. per kilogram of body weight per minute. The aorta was temporarily cross-clamped, and the main pulmonary artery was opened longitudinally. At first no emboli were discovered in the main pulmonary artery. Introduction of the suction tip into each pulmonary artery produced many long thrombi from both sides. The pleural spaces on each side were opened, and the lungs were compressed repeatedly, producing more thrombi with long peripheral branches. (See figure.) By repeated compression of the lungs and irrigation of the pulmonary arterial tree, numerous fragments of emboli were removed, following which pulmonary compression produced only liquid blood. After 15 minutes of total cardiopulmonary bypass the pulmonary arteriotomy was closed, and the pump oxygenator was discontinued. The patient's blood pressure was immediately maintained at 120/70 mm. Hg without vasopressors, and at no time following embolectomy were further vasopressors required. The inferior vena cava was immediately ligated through an abdominal, muscle-splitting, extraperitoneal incision in the right flank region. No thrombi were demonstrated in the
vena cava, which was deliberately opened in search of the source of the emboli. Mild fever was present for the first 3 days following pulmonary embolectomy, but convalescence was otherwise smooth and uncomplicated. Repeat arterial and alveolar gas studies revealed disappearance of the differential between the arterial and end-alveolar pCO₂. Roentgenograms of the chest one week later showed normal pulmonary vascular markings. Minimal edema of the lower extremities was noted on ambulation but was easily controlled with elastic supports. The patient was discharged from the hospital on the 14th day following operation. Subsequent right heart catheterization study has demonstrated a normal hemodynamic pattern in terms of cardiac output and pulmonary artery pressure both at rest and during exercise. When last examined 3 months after operation, the patient was asymptomatic.

Comment

Diagnosis of acute pulmonary embolism may be difficult, particularly in the absence of hemoptysis. Although operative risk is minimized by early intervention in such patients, tragic consequences might result from an ill-advised thoracotomy if the diagnosis were incorrect. In this case accentuation of the second heart sound associated with pulmonary valve closure, atrial gallop rhythm, persistent hypertension, and elevated peripheral venous pressure indicated pulmonary arterial hypertension and acute cor pulmonale in a patient with no previous signs of cardiopulmonary involvement. Confirmatory evidence for pulmonary embolism as the cause of these changes was obtained, utilizing the method of Robin et al.8 for demonstration of increased ventilatory dead space accompanying embolization. The method is based on the premise that occlusion of a major pulmonary arterial branch eliminates gas exchange in the corresponding segment of lung although alveolar ventilation remains unchanged. In a well-ventilated but nonperfused lung segment the composition of the alveolar air tends to approach that of inspired air, in which the partial pressure of carbon dioxide is nil. Mixture of such air during expiration with that from the remaining lung segments, where nearly normal ventilation perfusion relationships are maintained, results in a reduced mean alveolar carbon dioxide tension as measured at end-expiration. Meanwhile, arterial carbon dioxide tension tends to remain at a relatively normal level, regulated as it is by the function of the unaffected lung segments. Thus, a difference between the arterial and the alveolar carbon dioxide tensions should be considered significant if the alveolar value is a minimum of 4 mm. Hg less than the arterial. For various reasons, the method is not a sensitive one, and quantitative estimates of the calculated amount of dead-space ventilation based on these data is usually less than the actual value. In this instance, for example, the calculated amount of lung involved was only about 20 per cent of the total, although the development of pulmonary hypertension and cor pulmonale as well as the operative findings indicated that a much greater percentage of the pulmonary vascular bed was obstructed. Nevertheless, the finding of a difference of 7 mm. Hg between arterial and alveolar carbon dioxide tension demonstrated an appreciable degree of dead-space ventilation in this patient and was considered to be diagnostic of pulmonary embolism. The interval between the initial embolic episode and actual operation in our patient was 42 hours, during most of which period she received constant circulatory support from intravenous vasopressors. Although many patients die instantly following massive pulmonary embolism, it is not unusual for patients to survive one or more hours before death occurs from pulmonary vascular obstruction or visceral reflex mechanisms. An aggressive attitude toward surgical treatment of these cases should permit the saving of many patients who otherwise are doomed. As a rule, recognition of the potentially fatal cases of pulmonary embolism is not difficult, and for these patients immediate plans should be made for operation using the pump bypass. The decision to operate in our patient was made at 12:00 midnight, and the pump oxygenator and fresh heparinized blood were in readiness at 2:00 A.M. Improvements in methods of blood procurement and storage for the extracorporeal circuit or development of methods of bypass not requiring the use of fresh heparinized whole blood should simplify the preparation necessary for total cardiopulmonary bypass. For example, priming the extracorporeal circuit with blood substitutes such as dextran or using citrated blood or plasma would simplify use of the pump oxygenator. Thus, the extracorporeal unit may be immediately available to the dying patient, eliminating a crucial delay in surgical treatment. The generally poor record obtained in the past with the Trendelenburg operation could be attributed to several factors. Notable among these were use of inadequate anesthetic techniques. Often local anesthesia was used without endotracheal insufflation of the lungs, and the combination of poor oxygenation of blood and reduced cardiac function produced irreparable damage of vital organs. Complicated incisions were employed, with resecting of costal cartilages and attempts to remain in an extrapleural plane to avoid the problem of an open pleural space and pulmonary collapse. Inadequate surgical exposure was obtained, thus preventing an effective attack upon the pathologic lesion. Finally, the limitations of time placed upon the Trendelenburg operation by attempting embolectomy during brief periods of circulatory arrest made adequate removal of the emboli impossible. Pulmonary emboli seldom remain free in the main pulmonary arteries; they quickly lodge in the periphery of the vascular bed where secondary and tertiary branches become occluded by distal thrombosis. Therefore, dislodgement of these thrombi
cannot be accomplished effectively in a matter of seconds or even a few minutes. Death occurred in some instances from retained embolus or thrombus when the removal of clots was incomplete or from new emboli occurring following operation.

On the basis of our experience with one successful case of pulmonary embolectomy where temporary cardiopulmonary bypass was used, several recommendations are justified which may lead to salvage of a higher percentage of cases through surgical treatment. Upon making the diagnosis of massive pulmonary embolism in a patient, the physician should immediately begin supportive measures, including oxygen inhalation and intravenous vasopressor substances. Preparations must be started for the bypass procedure, reducing the usual time required for procurement of blood. Priming the pump oxygenator with dextran solution would be an acceptable expedient with a gravely ill patient. Anesthesia should be induced, utilizing skeletal muscle relaxants to prevent the patient from straining, and raising the intrathoracic pressure during tracheal intubation. Shallow anesthesia is advisable, utilizing cyclopropane or halothane supplemented by muscle relaxants. A median sternotomy incision should be employed, opening both pleural spaces. Insertion of vena cava cannulae for venous outflow and a femoral arterial catheter for arterial return should be done without unnecessary manipulations of the heart, pulmonary artery, or lungs. Once cardiopulmonary bypass is started, adequate perfusion and oxygenation of vital centers provides optimum conditions for complete and unhurried removal of pulmonary emboli and thrombi. An adequate longitudinal incision is made in the main pulmonary artery, and thrombi are removed from both lungs with a suction and forceps. Repeated manual compression of the lungs is an important adjunct in dislodging peripheral clots lodged in secondary and tertiary branches of the pulmonary vascular tree. After the pulmonary arteries are completely emptied of thrombi, the pulmonary arteriography is repaired with a continuous suture. The patient's condition should improve immediately, although continued mechanical support of circulation may be necessary for 10 or 15 minutes after closure of the arteriography. Ligation of the inferior vena cava may be performed concurrently if the source of emboli is suspected to be the iliac vessels. Under other circumstances, ligation of iliac or femoral veins may suffice.

Summary

Massive pulmonary embolism remains a serious problem, the incidence and treatment of which have not been altered appreciably during recent years. Pulmonary embolectomy by the technique proposed by Trendelenburg in 1908 has not provided an effective means of dealing with gravely ill patients.

A successful pulmonary embolectomy in a 37-year-old woman was performed, utilizing temporary cardiopulmonary bypass. Large branching emboli and thrombi were removed from both pulmonary arteries, permitting an uncomplicated and complete recovery. This case is the first reported successful case using this technique, which provides the conditions necessary for safe conduct of operation with deliberate and complete removal of thrombi. Ligation of the inferior vena cava was performed concomitantly in this patient, who had recently undergone hysterectomy and salpingectomy for ruptured tubal pregnancy. On the basis of this experience, emergency surgery may be recommended for salvage of patients with acute massive pulmonary embolism.

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Supported in part by the Houston Heart Association, the Thibodaux Foundation, and the United States Public Health Service.

References