as showing moderate generalized paroxysmal dysrythmia suggesting diffuse central nervous system disease. (About four years previously the patient had had an electroencephalogram that was entirely within normal limits.) A urological consultant believed acute tubular degeneration without obstructive uropathy to be present, and the following day an internist consultant reached the same conclusion.

Intensive supportive care was given but three days after admission the blood urea nitrogen had risen to 80 mg per 100 ml. Serum creatinine was 12 mg per 100 ml, and serum sodium and potassium 121 and 5.5 mEq per liter respectively. Excretion of phenolsulphthalein was 4 per cent in two hours. Urine sodium was reported to be 19 mEq per liter. This value rose to only 20 mEq the following day.

Four days after hospital admission, sodium and potassium levels had returned to normal, and all vital signs including blood pressure were within normal limits, but the blood urea nitrogen was 112 mg and serum creatinine 12 mg per 100 ml.

Beginning 48 hours after admission mannitol was given intravenously, 50 gm daily was used to promote diuresis,³ with salutary effect (Table 1). Administration was discontinued after four days when fluid output began exceeding intake.

Urine chlorides remained very low (20 and 26 milliequivalents per liter on the fifth and sixth days in hospital) but the sensorium cleared dramatically with diuresis. Blood pH four days after admission was 7.42 with a base excess of -7. Dialysis was not performed. Radiographs of the lungs on the fifth day showed only a small amount of pleural fluid at the left base.

Body weight declined from 210 pounds when the patient was admitted to 199 pounds when he was discharged seven days later.

Two weeks after discharge all vital signs were

TABLE 1.—Data	Showing	Diuretic	Effect	of .	Manni	tol
Intravenously, 50	gm a Dag	y (Asteris	ks Ind	icate	Days	of
	Mannitol	l Therapy)			

Date Admitted	Rody	Fluid		
	Weight (pounds)	Intake Output (milliliters)		
5-2-65		2,850	200	
5-3-65	208	3.850	625	
*5-4-65		2,700	1.600	
*5-5-65		2.675	2,385	
*5-6-65		2.940	2,660	
*5-7-65		2,390	3,800	
5-8-65	201	2.645	4,100	
5-9-65		2,100	3.425	

normal, the sensorium was clear, blood urea nitrogen was 15 mg and serum creatinine 1.1 mg per 100 ml, and results of a blood cell count and urinalysis were entirely within normal limits, as were serum, sodium, potassium and chloride. On neurological examination no abnormalities were noted.

Summary

A 57-year-old white man with acute tubular necrosis that had developed from excessive water drinking while working in a hot desert area, had alarming mental and physical symptoms.

Gratifying recovery followed conservative treatment. Administration of large amounts of mannitol intravenously—as recommended by Barry and coworkers¹—contributed to this good result.

205 Walnut Avenue, San Diego, California 92103.

REFERENCES

1. Barry, K. G., et al.: Mannitol Infusion II—The prevention of acute functional renal failure during resection of an aneurysm of the abdominal aorta, New Eng. J. Med., 264:967-971, 1961.

2. Lindsay, R. M., Linton, A. L., and Longland, C. J.: Assessment of postoperative renal function, Lancet, I:978, 8 May 1965.

3. Luke, R. G., Linton, A. L., Briggs, J. D., and Kennedy, A. C.: Mannitol therapy in acute renal failure, Lancet, I:980-982, May 1965.

4. Luppi, A. P.: Homeostatic problems in general surgery, Calif. Med., 102:412-415, June 1965.

5. Marshall, Sumner, and Lyon, R. P.: Differential renal function study, Calif. Med., 103:9-12, July 1965.

6. Nomenclature and classification of the disorders due to heat, Lancet, II:637-639, September 1964.

7. Schreiner, G. E.: Toxic nephropathy, J.A.M.A., 19:849-850, 8 March 1965.

Surgical Treatment of Massive Pulmonary Embolism

RICHARD S. MILLIGAN, M.D. PERRY A. OLSEN, M.D. GERALD J. TOOLE, M.D. NEWELL E. WOOD, M.D. San Jose

MASSIVE PULMONARY EMBOLISM is almost always a fatal disorder.^{7,10} In 1908 Trendelenburg²¹ asked "whether the diagnosis of embolism can be ascertained with sufficient accuracy and whether there

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From the Department of Surgery, Anesthesiology and Medicine, Santa Clara County Hospital and Medical Center, San Jose. Submitted 23 August 1965.

is sufficient time for an operation." Subsequently he attempted surgical removal of pulmonary emboli in three cases without success. In 1924 Kirschner¹³ performed the first successful Trendelenburg operation. Between then and 1961, when Cooley⁴ reported successful removal of a massive pulmonary embolus while the patient was sustained with cardiopulmonary bypass, the total of successful operations rose to only 23. Then in less than three years an additional 18 patients were salvaged with this procedure. Now, with the perfection of techniques for cardiopulmonary bypass in many medical centers, Trendelenburg's query becomes much more pertinent.

Feasibility of Pulmonary Embolectomy

Donaldson³ reviewed 283 cases of pulmonary embolus, proved at autopsy, which occurred on the Massachusetts General Hospital Surgical Services over a 30-year period. In 271 cases, the time interval could be determined. Twenty-five per cent of this group survived longer than one hour, 22 per cent longer than two hours and 17 per cent longer than six hours after onset of embolism. An analysis of 52 autopsy proven cases of pulmonary embolism by Flemma⁹ demonstrated a 55 per cent two-hour survival and a 48 per cent eight-hour survival in patients who were previously healthy. These studies certainly indicate that there is adequate time to ready cardiopulmonary bypass for pulmonary embolectomy in many patients with pulmonary embolism and underscore the need for expeditious diagnosis of this disorder.

Reports of Cases

Recent experience with successful pulmonary embolectomy in two cases at Santa Clara County Hospital serves to illustrate several features concerning the diagnosis, decision to operate, surgical problems and postoperative morbidity associated with this procedure.

CASE 1. A 29-year-old Caucasian mother of four had vaginal hysterectomy at Campbell Community Hospital, 29 October 1964. At 1 p.m. on the fourth postoperative day, she began having severe pleuritic substernal pain, pronounced dyspnea, occasional non-productive cough and tachycardia of 120. A few rhonchi were heard in the left lower lung field. A film of the chest was within normal limits. At 4 p.m. excruciating chest pain began abruptly, accompanied by vomiting and signs of shock. Pronounced cervical vein distention was noted, a widely split aortic second sound, pulsus paradoxicus, protodiastolic gallop rhythm and moderate agitation. An electrocardiogram indicated right heart strain.

The patient was then treated with metaraminol, digitalis, heparin and oxygen. She had persistent hypotension and intractable chest pain over the next few hours. Eventually norepinephrine was begun to maintain arterial blood pressure and an electrocardiogram was consistent with incomplete right bundle branch block.

The patient was admitted to Santa Clara County Hospital 2 November 1964 at 11 p.m. On physical examination the findings already mentioned were noted, plus non-tender abdomen, nontender extremities, shallow respirations, clear breath sounds and a pulse rate of 160. She was taken directly to the operating room where, after cannulation of the right femoral vein and right femoral artery under 1 per cent lidocaine anesthesia, partial cardiopulmonary bypass was begun. A disposable bag oxygenator was used and primed with Ringer's lactate and two units of heparinized ACD blood. The patient remained alert and talkative, but had a sensation of impending doom throughout these preparations. General anesthesia was then induced with halothane via endotracheal tube, and median sternotomy was done. The vena cavae were cannulated via the right atrium and total cardiopulmonary bypass at a flow rate of 3,300 ml per minute was begun.

A longitudinal main pulmonary arteriotomy was performed, and upon opening the lumen a grapelike mass was immediately extruded. Additional clots were then removed from both main pulmonary arteries and their major branches. The lungs were then milked bilaterally and more clot material was removed. The arterial incision was sutured and the bypass was discontinued after 20 minutes of total bypass time. The lumbar vena cava was then doubly ligated with 0 silk ligatures via a flank incision.

Examination in the immediate postoperative period showed normal heart sounds, no conduction defect in the electrocardiogram and no cervical vein distention. The postoperative course was complicated by a right hemothorax which developed one week following embolectomy and required decortication on the 18th postoperative day. The patient was also bothered considerably by phlebitis in the right leg. Moderate swelling of the right lower extremity persists to the present time but she has returned to her duties as a housewife.

CASE 2. A 54-year-old Caucasian man, a painter, was first seen at Santa Clara County Hospital in December 1964 with complaint of sudden onset of numbness, parasthesias and cyanosis of the fingers of the left hand. After extensive diagnostic study of this neurovascular disorder, left cervical sympathectomy was done 18 January 1965 through a right thoracotomy in the third intercostal space. After moderate ambulation on the second postoperative day, the patient had pain in the left side of the chest. On examination this was difficult to distinguish from wound pain. An electrocardiogram suggested minor ischemic changes. The following morning the patient was apprehensive but his blood pressure was stable and the pulse rate was under 100. At 1:15 p.m. on the third postoperative day, pronounced dyspnea and signs of shock developed. Although the patient had no significant chest pain, he had a sensation of pressure on his chest.

Blood pressure and pulse were briefly unobtainable; but soon a pressure of 56/0 mm of mercury developed with a pulsus paradoxicus and the patient remained responsive at all times. His skin was decidedly violaceous. On auscultation a protodiastolic gallop rhythm with a fixed split of the second heart sound was noted. An electrocardiogram taken at this time indicated complete right bundle branch block, which had not been present on a tracing 24 hours before. The circulation time was greater than one minute.

An attempt to document the diagnosis of pulmonary embolus was made in the radiology department with bilateral cephalic vein infusion of large amounts of sodium diatrizoate. Films taken from 45 to 90 seconds after infusion showed no dye material in the superior vena cava. The patient was taken immediately to the operating room, where preparations for cardiopulmonary bypass had been made. Under local 1 per cent lidocaine anesthesia, cannula was put into the right femoral artery and a large plastic cannula was placed in the right internal jugular vein. Partial bypass was established at a flow rate of 2,000 ml per minute. A few minutes later, the patient lost all evidence of pulsatile flow and the systemic arterial systolic pressure was 50 mm of mercury while the venous pressure was 20 mm of water. The pump flow was gradually increased to 3,000 ml per minute and general anesthesia with halothane via endotracheal tube was begun. At this instant, the systolic arterial pressure was 25 mm of mercury. As soon as the sternum was opened and the right atrium cannulated, the systolic arterial pressure rose to 80 mm of mercury, pulsatile flow returned and the vital signs stabilized.

Total bypass then was begun and a longitudinal incision was made in the main pulmonary artery after cross-clamping of the trunk. The lumen of the right pulmonary artery was found to be totally occluded with clotted material. An embolus 18 cm long and 1 to 1.5 cm in diameter was removed in one piece. Several other small clots were removed. No clotted material was found in the left main pulmonary artery. Both lungs were milked and peripheral clots were recovered only from the right side. Bypass was discontinued approximately one hour after it was begun.

During the closure of the thoracic wound and ligation of the abdominal vena cava, the patient had two episodes of atrial flutter with block. The heart reverted to normal sinus rhythm spontaneously.

On physical examination in the immediate postoperative period, heart sounds were normal, there was total reversion of the right bundle branch block to normal sinus rhythm, and neck vein distention disappeared. The patient was hampered in the postoperative period with edema of both legs but there was no evidence at any time of acute thrombophlebitis. Anticoagulant therapy was begun on the seventh postoperative day. When last examined the patient could tolerate moderate exercise without leg edema.

Discussion

Because of the limited survival time in patients with pulmonary embolism, an attempt should be made to establish necessary criteria for a confident diagnosis on clinical grounds alone, lest valuable time be lost.

The diagnostic features in the cases reported here included:

- Abrupt development of clinical shock.
- Evidence on physical examination of right ventricular overload and dilatation.
- Systemic venous congestion without signs of pulmonary venous congestion.
- An electrocardiogram characteristic of acute right heart strain.

- No previous significant heart disease.
- Pulsus paradoxicus.

The sudden development of tachycardia and hypotension with peripheral signs of shock occurred in both cases. In the first case, sustained use of vasopressors became necessary, but eventually these were to no avail. In the second case, a vasopressor was used for the first hour and then adequate blood pressure was maintained without drugs in the interval before operation. Signs of pronounced peripheral vasoconstriction persisted in both cases, but both patients remained amazingly alert until induction of general anesthesia.

The presence of a heave and a protodiastolic gallop at the lower left sternal border reflected the dilatation and overload of the right ventricle. In both cases, the second heart sound was found widely split and fixed with respirations and the sound of pulmonic valve closure was prominent.² At this time the electrocardiogram in both cases demonstrated a right bundle branch block. The electrical delay in right ventricular activation as well as mechanical prolongation of the right ventricular systolic ejection were the likely explanations for the wide separation found between aortic and pulmonic closures, but the fixation of the interval with the respiratory cycle requires further explanation.¹² Perhaps the extreme distention of the right ventricle in diastole made the chamber non-compliant and it was unable to change significantly in end diastolic volume with the fluctuation of intrapleural pressure. Hence, the usual increased filling of the right ventricle during inspiration did not occur. In both cases this chamber was observed at operation to be tense and distended during diastole. In both cases also the abnormal second heart sound reverted to normal shortly after removal of the pulmonary emboli.

Abnormal cervical vein distention was quite obvious in both cases. The patient in Case 1 was decidedly orthopneic but pulmonary rales did not develop. The other patient was quite comfortable in the supine position throughout his preoperative course and had clear breath sounds. As McGinn¹⁶ noted in his series of autopsy-proven cases, the patient with a massive embolism has pronounced systemic venous congestion in the absence of pulmonary congestion.

Electrocardiograms were helpful in the diagnosis in both cases. In the first tracings made after development of symptoms, a right ventricular conduction delay was found. In Case 1 this was classified as an incomplete right bundle branch block for the QRS duration measured 0.10 seconds. The QRS axis was 90° in the frontal plane and a slurred monophasic R wave was present in lead VI. An electrocardiogram taken one week after embolectomy showed a normal QRS duration of .08 second, an rS pattern in lead VI and a QRS axis of 60°. In the second case, a complete right bundle branch block was found at the onset of shock. This was noted to return to normal within two hours after pulmonary embolectomy was completed.

Further confidence in the clinical diagnosis was afforded by the fact that neither patient had a history of any previous cardiac disease. This information aided in distinguishing the acute episode from chronic pulmonary hypertension with severe right heart failure.

The extreme usefulness of the disposable bag oxygenator in these two cases is impressive. Although acid citrate dextrose blood was obtained by the time of operation, it would not have been possible to obtain enough heparinized blood to permit the use of the disc oxygenator. The remarkable hemodynamic improvement in these patients in the immediate postoperative period is gratifying. As Cooley and Cross⁵ have emphasized, the value of placing these patients on supportive partial bypass before the induction of general anesthesia seems indisputable. This seems to have facilitated the successful outcome of operation in the second case. Cannulation of the internal jugular vein is performed as easily as femoral vein cannulation and avoids postoperative problems related to groin phlebitis. Clamping the pulmonary trunk eliminates coronary return flow and the necessity of encircling the vena cavae. If a large catheter were placed in the internal jugular vein it might be possible to avoid caval cannulation via the right atrium. Thus attention could be directed to the pulmonary artery immediately after sternotomy.

The experience in the first case with postoperative hemothorax, requiring decortication on the 18th postoperative day, seems to support the advisability of inferior vena cava ligation as an adjunctive procedure. Thus, if it is necessary to discontinue anticoagulants during the postoperative period because of bleeding, the patient has the protection of caval ligation.

Angiograms with a centrally placed catheter

were not utilized in these cases but this is an essential step in the diagnosis of uncertain cases. Radio-active pulmonary scans¹⁸ and determination of arterial-alveolar carbon dioxide gradients have been utilized in the diagnosis of pulmonary embolism but these measures do not seem necessary in cases of massive embolism.

Summary

Two cases of massive pulmonary embolism occurring in postoperative patients have been presented. It is believed that emergency pulmonary embolectomy with the use of cardiopulmonary bypass prevented death in both cases.

1700 McHenry Village Way, Modesto, California 95350 (Milligan).

REFERENCES

1. Beall, A. C., Jr., Cooley, D. A., and DeBakey, M. E.: Surgical management of pulmonary embolism, Dis. of the Chest, 47:382, 1965.

2. Boyle, J., and Little, R. C.: Study of hemodynamic factors which alter the sequence of the second heart sound, Amer. Heart J., 68:91, 1964.

3. Cooley, D. A., and Beall, A. C., Jr.: Surgical treatment of acute massive pulmonary embolism using temporary cardiopulmonary by-pass, Dis. of the Chest, 41: 102, 1962.

4. Cooley, D. A., Beall, A. C., Jr., and Alexander, J. K.: Acute massive pulmonary embolism—successful surgical treatment using temporary cardiopulmonary bypass, J.A.M.A., 177:283, 1961.

5. Cross, F. A., and Mowlem, A.: Pulmonary embolectomy utilizing cardiopulmonary bypass, SG&O, 117:71, 1963.

6. Davis, W. C.: Immediate diagnosis of pulmonary embolus, Amer. Surgeon, 30:291, 1964.

7. DeBakey, M. D.: Critical evaluation of the problem of thromboembolism, SG&O, 98:1, 1954.

8. Donaldson, G. A., Williams, C., Scannel, C., and Shaw, R. S.: A reappraisal of the application of the Trendelenburg operation to massive total embolism, New Eng. J. Med., 268:171, 1964.

9. Flemma, R. J., Young, W. G., Jr., Wallace, A., Whalen, R. E., and Freese, J.: Feasibility of pulmonary embolectomy, Circulation, 30:234, 1965.

10. Gorham, L. W.: A study of pulmonary embolism, Arch. Int. Med., 108:8, 1961.

11. Hampson, J., Milne, A. C., and Small, W. P.: The surgical treatment of pulmonary embolism, Lancet, London, 2:402, 1961.

12. Hyman, A. L., Myers, W. D., and Meyer, A.: The effect of acute pulmonary embolus upon cardiopulmonary hemodynamics, Amer. Heart J., 67:313, 1964.

13. Kirschner, M.: Ein Duch die Trendelenburgsche Operation Geheilter Foll von Embolic der Art. Plmonalis, Arch. Klin. Chir., 133:312, 1924.

14. Levy, J. F., and Keltner, R. M., Jr.: Improved technic of cardiopulmonary bypass for emergency pulmonary embolectomy, Amer. J. Surg., 109:148, 1965.

15. Mach, I., Beall, A. C., Jr., Griffith, G. C., Rosenberg, D. M. L., and Sampson, J. J.: Salvaging the patient with acute massive pulmonary embolism, Dis. of the Chest, 42:584, 1962.

16. McGinn, S., and White, P. D.: Acute corpulmonale resulting from pulmonary embolism—its clinical recognition, J.A.M.A., 104:1473, 1935.

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17. Palumbo, L. T.: New surgical approach for upper thoracic sympathectomy, Arch. of Surg., 76:807, 1958.

18. Sabiston, D. L., Jr., and Wagner, H. N. Jr.: The diagnosis of pulmonary embolism by radioisotope scanning, Ann. Surg., 160:575, 1964.

19. Steenburg, R. W., Warren, R., Wilson, R. E., and Rudolf, L. E.: A new look at pulmonary embolectomy, SG&O, 107:214, 1958.

20. Sasahara, A. A., Stein, M., and Littman, D.: Pulmonary angiography in the diagnosis of thromboembolic disease, New Eng. J. Med., 270:1075, 1964.

21. Trendelenburg, F.: Ueber die Operative Behandlung der Emboli der Lengenarterie, Arch. Klin. Chir., 86:686, 1908 (Translation in Ann. of Surg., 48:772, 1908).

Primary Amyloidosis With Death Due to Progressive Hypotension

THOMAS N. CAMPBELL, M.D. RALPH GOLDMAN, M.D. Los Angeles

RENAL FAILURE without significant hypertension is known as a characteristic of amyloid renal disease. Yet severe, incapacitating hypotension, progressing to become a significant contributory cause of death, has been infrequently recognized as a complication of primary systemic amyloidosis. This report is of a patient in whom that occurred. A similar case was described previously by Schneckloth and Page.⁶

Report of a Case

The patient, a 48-year-old Caucasian housewife, was put in hospital in April 1962, following sudden onset of colicky abdominal pain. The blood pressure was 110/60 mm of mercury and the pulse rate 100. Muscle guarding precluded adequate abdominal examination. Four plus proteinuria was noted. An x-ray film of the abdomen showed enlargement of the liver, with the lateral tip at the right iliac crest. There were two radiopaque densities to the right of the second lumbar interspace. An intravenous pyelogram showed equal bilateral excretion at five minutes. Partial obstruction at the right uretero-pelvic junction was seen.

From the Department of Medicine, University of California, Los Angeles, Center for the Health Sciences.

This work was supported in part by the University Medical Research Foundation, Los Angeles, and the United States Public Health Service grant HE 07852. Submitted 10 August 1965.