RECURRENT PULMONARY EMBOLISM

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Five cases with recurrent pulmonary embolism (RPE), all having severe chronic cor pulmonale due to marked pulmonary hypertension, were reported. None of them had a history of thrombophlebitis of the legs. Dyspnea was the common symptom and signs of pulmonary hypertension were usual. Focal oligemia, cardiomegaly and plump pulmonary arteries on chest X-ray films, right axis deviation, clockwise rotation, ST-segment depression and T-wave inversion on electrocardiograms, and dilatations of the pulmonary arterial trunk, the right ventricle and the right atrium, and the posterior displacement of a small, compressed left ventricle on echocardiograms were the common findings. Gas exchange abnormalities were severe and they were considered the characteristic findings of this disease. Multiple perfusion defects were observed by a lung scan in all cases. Marked pulmonary hypertension with low cardiac output was the usual feature of RPE. Actual cutoffs or filling defects were demonstrated on a pulmonary angiogram. Deep vein of the legs were all intact and no thrombi were found. Pulmonary embolectomy was performed on one case. The importance of early diagnosis and early treatment of acute pulmonary embolism was emphasized for preventing the progress of this debilitating disease.

THERE are only a few case reports on recurrent pulmonary embolism (RPE) in Japan.¹⁻³ We experienced 5 cases of RPE between January, 1980 and March, 1982, all of which had severe chronic cor pulmonale due to marked pulmonary hypertension. We report the clinical pictures including the results of a perfusion lung scan, cardiac catheterization and pulmonary angiography, and discuss the diagnostic and therapeutic problems in this rare but serious disease.

Key Words:

Thrombophlebitis
Pulmonary embolism
Cor pulmonale
Pulmonary hypertension

CASE REPORTS

Case 1

A 69-year-old man with a past history of mild hypertension and gout was admitted to our hospital in January, 1980 because of severe dyspnea. He had been well until 1978 when he noticed mild dyspnea on exertion, and it worsened gradually. In December, 1979, he suffered from 2 attacks of dyspnea.

His pulse was 84 per minute, respiration 34 and blood pressure 124/84 mmHg. The patient was dyspneic at rest with panting respiration. The lungs were clear. The pulmonary component of the second heart sound was loud and the fourth heart sound was heard. The liver was not palpable. There was no peripheral edema. The erythrocyte sedimentation rate (ESR) was 12 mm (one hour). His chest X-ray films (Fig. 1)

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Fig.1. Chest X-ray films of 5 cases with recurrent pulmonary embolism.

Fig.2. Electrocardiograms of 5 cases with recurrent pulmonary embolism. Clockwise rotation, ST segment depressions and T wave inversions in leads II, III, aVF and in right anterior chest leads, and right axis deviation suggest the presence of right ventricular overload.

Fig. 3. Two-dimensional echocardiograms of Case 1. A: long axis view, B: short axis view, C: apical four chamber view, D: sagittal view. Marked dilatation of the right ventricle and the right atrium, and posterior displacement of the compressed, small left ventricle are evident. Right ventricular hypertrophy can be seen in B and C.

showed mild cardiomegaly with a cardiothoracic ratio (CTR) of 54%, small patchy densities in the right middle lung (RML), and focal oligemia in the RML, the right lower lung (RLL) and the left lower lung (LLL). His electrocardiogram (ECG) (Fig. 2) demonstrated a normal sinus rhythm at a rate of 82 per minute with a first degree AV block and a right axis deviation ($+107^\circ$). The R/S ratio was greater than 1 in V_1 , and smaller than 1 in V_5 . ST segments were depressed and T waves were inverted in leads II, III, aVF and V_{1-3} . An echocardiogram (Fig. 3) showed dilations of the pulmonary arterial (PA) trunk, the right ventricle (RV) and the right atrium (RA). The left ventricle (LV) was small, compressed and displaced posteriorly by the dilated RV. Arterial blood gases, while the patient was breathing room air, showed a PaO_2 of 64 mmHg,

a PaCO_2 of 31 mmHg and a pH of 7.42. A $^{99\text{m}}\text{Tc}$ -MAA perfusion lung scan demonstrated multiple perfusion defects (Fig. 4-A), and a ^{133}Xe ventilation lung scan (Fig. 4-B) showed almost normal ventilation. Right-sided cardiac catheterization disclosed a PA pressure of 92/32 (mean 54) mmHg and a cardiac index (Fick method) of 1.3 L/min/m^2 . Pulmonary angiograms (Fig. 5) demonstrated multiple pulmonary arterial occlusions.

Case 2

A 56-year-old woman was admitted to our hospital in May, 1980 because of dyspnea. She had been well until 1975, when she noticed dyspnea on exertion. She was admitted to another hospital in 1977 and 1978, respectively, and was diagnosed as having ventricular septal

Fig.4-A. Perfusion lung scans of 5 cases with RPE, indicating multiple perfusion defects. Blood flow of the right lung was totally absent in Case 5.

defect. Her pulse was 84 per minute, respiration 28 and blood pressure 116/66 mmHg. She felt dyspnea on minimum exertion. Both cheeks were reddened. S_2 was widely split with loud P_2 , and a pulmonic ejection sound was heard at third left intercostal space (3LSB). A grade 3/6

holosystolic murmur, which increased in intensity during inspiration, was heard at 4LSB. Liver was palpated 4 cm below the right costal margin, and there was slight peripheral edema. The ESR was 15 mm (one hour). Her chest X-ray film (Fig. 1) showed an enlarged cardiac silhouette

Post

Fig. 4-B. Ventilation lung scan in Case 1, showing almost normal ventilation.

(CTR 63%) mainly due to the increased size of the RV. The right PA was slightly enlarged, and the RML was somewhat clear. An ECG (Fig. 2) showed a normal sinus rhythm at a rate of 86 per minute, and a right axis deviation ($+110^\circ$). The R/S ratio was greater than 1 in V_1 and less than 1 in V_5 , and ST segment depressions and T wave inversions were noted in leads II, III, aVF and V_{1-4} . An echocardiogram demonstrated dilatations of the PA trunk, the RV and the RA, and the small, compressed LV, which was displaced posteriorly. Tricuspid regurgitation of moderate degree was demonstrated by contrast echocardiography. Arterial blood gases (room air) showed a PaO_2 of 52 mmHg, a PaCO_2 of 34 mmHg and a pH of 7.48. A perfusion lung scan (Fig. 4-A) revealed multiple perfusion defects. Right-sided cardiac catheterization revealed a PA pressure of 105/27 (mean 53) mmHg, and a cardiac index of 1.3 L/min/m^2 . Pulmonary angiogram (Fig. 5) showed multiple, high-degree obstructions of the pulmonary arteries.

Case 3

A 49-year-old woman was admitted to our hospital in February, 1981 because of dyspnea. She had been well until January, 1979, when she noticed hemoptysis and right back pain. She had gradually worsening exertional dyspnea. Peripheral edema appeared in September, 1980. Her pulse was 88 per minute, respiration 22 and blood pressure 132/84 mmHg. Both cheeks were reddened. S_2 was widely split with loud P_2 , and there was pulmonic ejection sound at 3 LSB. A

grade 2/6 holosystolic murmur, which increased in intensity during inspiration, was heard at 4 LSB. The liver was palpated 4 cm below the right costal margin, and there was slight peripheral edema. The ESR was 20 mm (one hour). Her chest X-ray film (Fig. 1) showed slight cardiomegaly (CTR 53%), several small patchy densities bilaterally, a large nodular shadow ($25 \times 20 \text{ mm}$) in the RUL, and several areas of focal oligemia. An ECG (Fig. 2) showed a normal sinus rhythm at a rate of 86, a right axis deviation ($+110^\circ$) and right bundle branch block pattern. The R/S ratio was less than 1 in V_5 , and ST segment depressions and T wave inversions were noted in leads II, III, aVF and V_{1-4} . An echocardiogram showed dilatations of the PA trunk, the RV and the RA. The small LV was compressed and displaced posteriorly by the dilated RV. Arterial blood gases (room air) demonstrated a PaO_2 of 64 mmHg, a PaCO_2 of 38 mmHg and a pH of 7.38. A perfusion lung scan (Fig. 4-A) revealed multiple perfusion defects. Right-sided cardiac catheterization demonstrated a PA pressure of 99/39 (mean 60) mmHg and a cardiac index of 1.2 L/min/m^2 . Pulmonary angiogram (Fig. 5) showed multiple occlusions of the pulmonary arteries.

Case 4

A 57-year-old woman was admitted to our hospital in July, 1981 because of dyspnea. She had been well until August, 1980, when she noticed exertional dyspnea, and it worsened gradually. In March, 1981, peripheral edema appeared. Her pulse was 82 per minute, respiration 32 and blood pressure 138/90 mmHg. S_2 was widely split with loud P_2 . A grade 1/6 holosystolic murmur, which increased in intensity during inspiration, was heard at 4 LSB. There was mild peripheral edema. The ESR was 7 mm (one hour). Her chest X-ray film (Fig. 1) showed mild cardiac enlargement (CTR 54%), and the RUL and the RML were oligemic. Her ECG (Fig. 2) demonstrated a normal sinus rhythm at a rate of 80 and a right axis deviation ($+95^\circ$). The R/S ratio in V_5 was less than 1, and ST segment depressions and T wave inversions were noted in leads II, III, aVF and V_{1-5} . An echocardiogram showed dilatations of the PA trunk, the RV and the RA, and the small LV was compressed by the dilated RV and was displaced posteriorly. Arterial blood gases (room air) revealed a PaO_2 of 54 mmHg, a PaCO_2 of 29 mmHg and a pH of 7.51. A perfusion lung scan (Fig. 4-A) demon-

Fig.5. Pulmonary angiograms of Cases 1, 2, 3 and 4, showing severe obstructions of the pulmonary arteries.

TABLE I CLINICAL FEATURES, HISTORIES AND SYMPTOMS IN FIVE CASES WITH RPE

Cases	Age	Sex	Duration of symptoms (months)	Episodes of DVT	Acute attack of dyspnea	Clinical diagnosis before admission	Symptoms				
							Dyspnea	Edema	Pleural pain	Hemoptysis	Sweats
1	69	M	25	—	+	dyspnea	+	—	+	—	—
2	56	F	76	—	—	dyspnea VSD	+	+	—	—	—
3	49	F	20	—	+	pulmonary hypertension	+	+	+	+	—
4	57	F	12	—	—	dyspnea	+	+	—	—	—
5	41	M	73	—	+	dyspnea	+	+	—	—	+

RPE = recurrent pulmonary embolism; DVT = deep vein thrombophlebitis; VSD = ventricular septal defect

strated multiple perfusion defects. Right sided cardiac catheterization showed a PA pressure of 102/28 (mean 53) mmHg and a cardiac index of 2.4 L/min/m². Pulmonary angiogram (Fig. 5) revealed multiple obstructions of the pulmonary arteries.

Case 5

A 41-year-old man was admitted to our hospital in January, 1982 because of dyspnea. He had been well until 1977, when he noticed mild exertional dyspnea. In March, 1981, his dyspnea worsened and peripheral edema appeared. He

TABLE II GAS EXCHANGE ABNORMALITIES IN FIVE CASES WITH RECURRENT PULMONARY EMBOLISM

Case	PaO ₂ (mmHg)	PaCO ₂ (mmHg)	pH	A-aDo ₂ (mmHg)	a-ETDco ₂ (mmHg)
1	64	31	7.42	49	10
2	57	32	7.43	61	11
3	64	38	7.38	47	10
4	53	34	7.45	56	11
5	39	27	7.51	79	7
mean	55	32	7.44	58	10

A-aDo₂ = alveolar-arterial O₂ tension difference; a-ETDco₂ = arterial-endtidal CO₂ tension difference

visited a physician and was found to have a cardiac murmur and cardiomegaly. Diuretics were given, but dyspnea did not improve. In January, 1982, he suffered from sudden attacks of dyspnea and was admitted to a hospital. He was transferred to our hospital 11 days later. He had no chest pain or hemoptysis. He had a past history of pulmonary tuberculosis 9 years before. His pulse was 114 per minute, respiration 32 and blood pressure 112/70 mmHg. He was very dyspneic with a pale face. His lips and nails were cyanotic. P₂ was accentuated and S₃ was heard. A grade 2/6 regurgitant systolic murmur was heard at 4 LSB. The liver was palpated 2 cm below the right costal margin. There was mild peripheral edema. The ESR was 18 mm (one hour). His chest X-ray film (Fig. 1) revealed that the entire right lung field was hyperlucent and the left lung was hypervascular. There was mild cardiomegaly. His ECG (Fig. 2) showed sinus tachycardia at a rate of 118 and a mean electric axis of +110°. There was prominent R waves in V₁, and the R/S ratio in V₅ was less than 1. ST segment depressions and T wave inversions were noted in leads II, III, aVF and V₁₋₃. An echocardiogram showed the dilatation of the PA trunk, the RV and the RA. Arterial blood gases (room air) demonstrated a PaO₂ of 39 mmHg, a PaCO₂ of 27 mmHg and a pH of 7.51. A perfusion lung scan (Fig. 4-A) revealed that the blood flow to the right lung was totally absent, and several areas of perfusion defects were also observed in the left lung. According to right-sided cardiac catheterization, a PA pressure was 75/29 (mean 37) mmHg and a cardiac index was 1.3 L/min/m². Pulmonary angiogram was not performed because of his dopamine-dependent hypotension which had persisted since the day of his admission. On February 5, pulmonary embolectomy was performed. Preparation of the right pulmonary artery was quite difficult because

of thickening and adhesion of the pleura probably due to old pleuritis. Dense thrombi in various stages, from white to brown in color, were found in the right pulmonary artery, and most of them were removed. However, it was found postoperatively that blood flow to the right lung was absent again probably due to rethrombosis of this artery. The patient is now bedridden with oxygen inhalation.

DISCUSSION

The incidence of acute pulmonary embolism (APE) in Japan was once said to be small and far less frequent as compared with Western countries.⁴ However, APE is now believed to be increasing as the life style, especially the food habits, becomes modernized.⁵

Emboli, mostly due to thrombi, lyse rapidly and usually leave no sequelae.⁶ However, since the report of Ljungdahl,⁷ recurrent attacks of pulmonary embolism (recurrent pulmonary embolism, RPE⁸) are known, and patients with RPE eventually develop severe chronic cor pulmonale due to significant obstructions of the pulmonary vascular beds.

The incidence of RPE is unknown not only in Japan but in Western countries.⁸ Owen⁹ has reported 12 cases of RPE among their 8,000 autopsy cases during a period of 20 years. Wilhemsen et al.¹⁰ and Fleischner⁸ described their cases with RPE and called attention to this rare but serious condition. In Japan, Takezawa et al.,¹ Yamada,² and Ohtsuka et al.³ have reported cases of RPE. We experienced 5 cases of RPE between January, 1980 and March, 1982 in our hospital.

Table I shows the summary of clinical features, histories and symptoms in our 5 cases. It is interesting that none of them had suffered from deep vein thrombophlebitis. We examined their

deep veins by contrast and radioisotope venography, but could not find any abnormalities.

Davison et al.¹¹ have divided the patients of RPE into 2 groups. The first group consisted of patients who had an obvious attack of pulmonary embolism, and Cases 1, 3 and 5 of ours belonged to this group. The second were the patients without any gross pulmonary events (silent pulmonary embolism¹¹), and Cases 2 and 4 belonged to this group.

Clinical diagnosis before admission was mostly "dyspnea of unknown etiology", and none of our cases had been suspected as having pulmonary embolism. This reflects some difficulties in diagnosing RPE.

All our patients had dyspnea as a subjective symptom. Longstanding dyspnea is the outstanding symptom of RPE and one should always consider RPE as a possible cause of chronic dyspnea of unknown etiology.

Tachypnea and signs of pulmonary hypertension (loud P_2 and pulmonary ejection sound) were the common physical findings in our cases. The presence of tricuspid regurgitation in 4 cases reflects severe pressure overload of the right ventricle.

Focal oligemia, cardiomegaly due to a dilated RV, plump pulmonary arteries, and a prominent left second arc were the common chest X-ray abnormalities. It is interesting that signs of acute pulmonary infarction, such as consolidation, high diaphragm, pleural effusion and atelectasis, were not seen in our cases, although patchy scars representing old pulmonary infarction were frequent. Right axis deviation (greater than $+90^\circ$), clockwise rotation (transitional zone at V_5), ST segment depressions and T wave inversions were the common ECG manifestations of RPE. Right ventricular hypertrophy (R/S in V_1 greater than 1), and P pulmonale (2.5 mm or more in leads II and/or III) were also seen frequently. They all reflect RV overload caused by persistent pulmonary hypertension.

The lack of any specific laboratory tests for acute pulmonary embolism is now well known,¹² and we also could not find any.

Gas exchange abnormalities of a severe degree were seen in all 5 cases (Table II). The pathogenesis of hypoxemia and increased alveolar-arterial oxygen tension difference ($A-aDO_2$) in pulmonary embolism are not known clearly, but shunt or shunt-like effects caused by microatelectasis of the involved areas of the lung are suspected as a cause.¹³ $A-aDO_2$ in our cases were

calculated assuming P_{Aco_2} to be equal to $Paco_2$. Thus actual $A-aDO_2$ may be higher. Hypocapnia and increased arterial-endtidal CO_2 tension difference ($a-ETDCO_2$), which reflects increased dead space ventilation, were observed in all, and these are well known sequelae of pulmonary embolism.¹⁴ These severe arterial blood gas abnormalities are the characteristic features of RPE and clues to a correct diagnosis.

The usefulness of a perfusion lung scan in diagnosing pulmonary embolism is well established,¹⁵ and multiple perfusion defects were observed in all our cases. One can diagnose pulmonary embolism with certainty in cases with the clinical features mentioned above when perfusion defects are shown by a lung scan.

Severe pulmonary hypertension and marked low cardiac indices were demonstrated in all cases. The pulmonary to systemic peak systolic pressure ratio (Pp/Ps) and the resistance ratio (Rp/Rs) were consequently very high. This severe pulmonary hypertension was caused by significant obstructions of the pulmonary vascular beds due to recurrent embolism,¹⁶ and marked low cardiac indices were the result of right ventricular failure. Dyspnea, as a subjective symptom, is mainly due to hyperventilation caused by neurohumoral abnormalities.¹⁷ However, decreased oxygen delivery due to low cardiac output and low arterial oxygen content may also be responsible for severe exertional dyspnea in our cases.

Definite diagnosis can only be made using pulmonary angiography. Dalen et al.¹⁸ have reported that intraluminal filling defects, cutoff of arteries, areas of oligemia and asymmetry of flow were the common findings of pulmonary angiography in acute pulmonary embolism. They said that especially intraluminal filling defects and actual cutoffs were the characteristic features of pulmonary embolism. The pulmonary angiograms in our cases all demonstrated these 2 findings.

Pulmonary arterial involvement is known to occur also in Takayasu's disease.¹⁹ However, their differential diagnosis is usually not difficult, because obstructive lesions of systemic artery is usually found in Takayasu's disease. Congenital heart diseases, such as atrial septal defect, ventricular septal defect and patent ductus arteriosus with pulmonary hypertension, or primary pulmonary hypertension should be differentiated from RPE.⁸ An accurate diagnosis, however, is not difficult if one suspects RPE and looks for the

characteristic clinical features of this disease.

Treatment of RPE is difficult. Medical therapy such as digitalis or diuretics are effective for overt right heart failure, but is of limited value. We administered urokinase in Case 1 without any beneficial effect, and we think that thrombolytic therapy may not be indicated for RPE, because the thrombo-emboli which we find are usually old and organized. Prevention of a recurrence is important in RPE; anticoagulant therapy, usually with warfarin, is indicated.

Surgical intervention, such as a ligation of the inferior vena cava (IVC) or an insertion of an umbrella into IVC²⁰ are effective in some cases, but clinical experiences of these are few in Japan. There are several reports on successful radical embolectomy in RPE²¹⁻²³. We did this procedure in Case 5, but the result was not satisfactory. There are several clinical problems in radical embolectomy. For successful performance of this procedure, the site of the obstruction must be proximal, and patency of the pulmonary arteries distal to the site of obstruction should be preconfirmed²². However, we think this surgical approach may be very helpful in some selected cases.

The prognosis of RPE is poor²⁴. Cases 1 and 5 in the present study are now bedridden with oxygen inhalation, and the remaining cases have severe chronic cor pulmonale with limited daily activity. It is clear that early diagnosis and treatment of acute pulmonary embolism is mandatory in preventing this debilitating disease.

REFERENCES

1. TAKEZAWA H, KODAMA M: Diagnosis and natural history of pulmonary embolism. *Jap Med J* 2740: 3, 1976 (in Japanese)
2. YAMADA T: Pulmonary embolism: Surgical aspects. *J Adult Dis* 9: 69, 1979 (in Japanese)
3. OHTSUKA M, SATO N, TSUTSUMI M, TANAKA M: A case of multiple pulmonary embolism. *Jap J Torac Dis* 16: 799, 1978 (in Japanese)
4. HIRST AE, GORE I, TANAKA K, SAMUEL I, KRISHTMUKTI I: Myocardial infarction and pulmonary embolism. *Arch Pathol* 80: 365, 1965
5. HASEGAWA A, WATANABE N, NAGATA H, MURAO M: Epidemiology of pulmonary thromboembolism and infarction. *Jap J Torac Dis* 35: 35, 1976 (in Japanese)
6. PARASKO JA, ADELSTEIN SJ, SMITH RE, RICKMAN FD, GROSSMAN W, DEXTER L, DALEN JE: Late prognosis of acute pulmonary embolism. *N Engl J Med* 289: 55, 1973
7. LJUNGDAHL M: Gibt es eine chronische Embolisierung der Lungenarterie? *Deutsche Arch klin Med* 160: 1, 1928
8. FLEISCHNER FG: Recurrent pulmonary embolism and cor pulmonale. *N Engl J Med* 276: 1213, 1967
9. OWEN WR, THOMAS WA, CASTLEMAN B, BLAND E: Unrecognized emboli to the lung with subsequent cor pulmonale. *N Engl J Med* 249: 919, 1953
10. WILHELMSEN L, SELANDER S, SODERHOLM B, PAULIN S, VANAUSKAS E, WERKO L: Recurrent pulmonary embolism. *Medicine* 42: 335, 1963
11. DAVISON PH, ARMITAGE GH, MCILVEEN DJS: Chronic cor pulmonale due to silent pulmonary embolism. *Lancet* i: 224, 1956
12. SASAHARA A: Current problems in pulmonary embolism. *Prog Cardiovasc Dis* 17: 161, 1974
13. WILSON JE, PIERCE AK, JOHNSON RL Jr, WINGA ER, HARRELL WR, CURRY GC, MULLINS CB: Hypoxemia in pulmonary embolism: A clinical study. *J Clin Invest* 50: 481, 1971
14. FISHER SR, DURANCEAU A, FLOYD RD, WOLFE WG: Comparative changes in ventilatory dead space following micro and massive pulmonary emboli. *J Surg Res* 29: 195, 1976
15. WAGNER HN Jr, STRAUSS HW: Radioactive tracers in the differential diagnosis of pulmonary embolism. *Prog Cardiovasc Dis* 17: 271, 1975
16. BRAUNWALD E: Heart Disease. WB Saunders Co, Philadelphia, 1980, p 1673
17. HARASAWA M, UMEDA H, YOSHIMURA M: Dyspnea. Nanzando, Tokyo, 1971, p4 (in Japanese)
18. DALEN JE, BROOKS HL, JOHNSON LW, MEISTER SG, SZUCS MM, DEXTER L: Pulmonary angiography in acute pulmonary embolism: Indications, techniques and results in 367 patients. *Am Heart J* 81: 175, 1971
19. NUMANO H, ISOHISA I, MASHIMO S, NUMANO H, MAEZAWA H: Pulmonary functions in Takayasu's disease. *J Jap Soc Intern Med* 66: 86, 1977 (in Japanese)
20. MOBIN UDDIN K, UTIEY JR, BRYANT LR: The inferior vena cava umbrella filter. *Prog Cardiovasc Dis* 17: 391, 1975
21. SABISTON DC Jr, WOLFE WG, OLDHAM HN Jr, WECHSLER AS, CRAWFORD FA Jr, JONES KW, JONES RH: Surgical management of chronic pulmonary embolism. *Ann Surg* 185: 699, 1977
22. CABROL C, CABROL A, ACAR J, DANDJBAKHCH I, GUIRAUDON G, LAUGHLIN L, MATTEI MF, GODEAU P, GRONDIN P: Surgical correction of chronic postembolic obstructions of the pulmonary arteries. *J Torac Cardiovasc Surg* 76: 620, 1978
23. DAILY PO, JOHNSTON GG, SIMMONS CJ, MOSER KM: Surgical management of chronic pulmonary embolism. *J Torac Cardiovasc Surg* 79: 523, 1980
24. Case records of the Massachusetts General Hospital (Case 38-1981). *N Engl J Med* 305: 685, 1981