Massive Thrombotic Occlusion of the Large Pulmonary Arteries


In 23 cases of massive thrombotic occlusion of the large pulmonary arteries thrombosis secondary to pulmonary embolism was the major cause, but a proportion (30 per cent) was considered to have primary thrombosis in situ. The pathologic criteria, symptoms, signs, and results of special investigations are given in detail, and the diagnosis, etiology, and precipitating factors are discussed.

PULMONARY heart disease resulting from thrombotic occlusion of the small pulmonary arteries due to repeated emboli has been recognized for some years,1-2 and the occurrence of repeated "silent" emboli to the lungs has been reported by Owen and associates.3

Massive thrombosis of the large pulmonary arteries is infrequently considered in the differential diagnosis of obscure cases of right-sided heart failure, although over 200 cases have been described in the literature. The first report apparently was that of Hélie in 1837,4 while Savacool and Charr5 collected 88 cases from the literature and added 12 of their own. Brenner6 found 4 cases in 100 unselected autopsies, and Middleton7 found 53 cases in autopsies over a period of 16 years at the Wisconsin General Hospital. In 5 of the 12 cases of widespread repeated pulmonary emboli reported by Owen and associates,8 the occlusion involved the large branches of the pulmonary artery. The condition has recently been reviewed by Magidson and Jacobson8 who have added 7 more cases.

Massive thrombotic occlusion of the main pulmonary arteries has usually been described in association with other diseases, notably pulmonary tuberculosis, carcinoma of the lung, congenital heart disease, and various forms of acquired heart disease. It may, however, occur in the absence of any other disorder, but few cases have been diagnosed in life.

The object of this paper is to present a total of 23 cases, 9 of whom were seen in life, while the remaining 14 were obtained from an examination of 7,000 autopsy records in the Department of Pathology, Postgraduate Medical School. The diagnosis was made before death in 3 of the 9 patients seen in life, and autopsy confirmation was obtained in all cases.

The diseases associated with thrombosis of the main pulmonary arteries included pulmonary disease (carcinoma of the lung, chronic bronchitis), cardiovascular disease (valvular disease, coronary artery disease, and congenital heart disease), and gastrointestinal disorders. In 4 cases no associated disease was present, although in 3 of these a generalized thromboembolic tendency of unknown etiology was manifested by repeated episodes of venous thrombosis at various sites.

The cases without associated disease will be considered first.

Case Material

Thrombosis of the Main Pulmonary Arteries without Associated Disease

Case 1. A 48-year-old woman developed progressive dyspnea on exertion, followed by syncopal attacks in 1951. In 1952, she was found to have an iron-deficiency anemia and was treated with intravenous iron. During treatment she developed signs of congestive cardiac failure, with peripheral edema, ascites, hepatic pulsation, and an elevated jugular venous pressure. There was a blowing systolic murmur over the pulmonary artery and down the left sternal border, and right ventricular enlargement. Fluoroscopy revealed large pulsating pulmonary arteries.

She subsequently developed thrombosis of the left iliofemoral vein, for which she was treated with dicumarol. Congestive cardiac failure increased and...
tricuspid incompetence developed. Thrombosis of the inferior vena cava was suspected. Treatment with digitalis, mercurial diuretics, and ion-exchange resins was without effect during any of her repeated admissions to the Central Middlesex Hospital, and on August 30, 1953, she was transferred to Hammer-smith Hospital.

On examination she was an anxious, slightly cyanosed woman with orthopnea and cold extremities. The pulse was of small volume and regular. The jugular venous pressure was raised to 8 cm. above the sternal angle with a large systolic wave, indicating tricuspid incompetence. There was extensive edema. The cardiac impulse was diffuse, tapping, and with sternal pulsation, indicating right ventricular hypertrophy. The second heart sound was normally duplicated and not accentuated. A grade 4 systolic murmur was maximal in the third and fourth left intercostal spaces. The blood pressure was 90/60 mm. Hg. The lungs were clear, and there was ascites. The liver was enlarged and pulsated in systole. The spleen was not enlarged.

Chest x-ray and fluoroscopy (fig. 1) showed a large right ventricle, considerable enlargement of both pulmonary arteries, which did not pulsate, and oligemic lung fields. An electrocardiogram showed sinus rhythm, right ventricular, and right atrial hypertrophy (fig. 2). The phonocardiogram showed a systolic murmur maximal in midsystole in the pulmonary area. Liver function tests were normal, except for albumin of 2.6 and globulin of 3.5 Gm. per cent. The blood count was normal, with a hemoglobin of 90 per cent.

On cardiac catheterization the right atrial pressure was 11/6 mm. Hg. The tracing was typical of tricuspid incompetence. The right ventricular pressure was 90/18, with a mean of 35 mm. Hg. The pulmonary artery was not entered. There was no evidence of a septal defect with left-to-right shunt. The arterial oxygen capacity was 94 per cent saturated. The cardiac output by the Fick method was 1.6 L./min. It was concluded that there were right ventricular hypertension, tricuspid incompetence, low cardiac output, and no cardiac shunt.

![Image](https://example.com/image1.png)

**Fig. 1. Case 1.** Posteroanterior roentgenogram of the chest showing enlargement of the right ventricle, main pulmonary trunk, and right main branch. The lung fields are underfilled.

![Image](https://example.com/image2.png)

**Fig. 2. Case 1.** Electrocardiogram, showing augmented P waves indicating right atrial enlargement, and qR patterns in leads V₄R and V₅, indicating right ventricular hypertrophy.
The course of the disease was punctuated by episodes of bleeding into the muscles of the thigh, accompanied by thrombocytopenia. The platelet count was 34,000/ml during the first attack. One week later the white cell count was 20,000/ml with 84 per cent polymorphonuclear cells and 1 per cent eosinophils, and the hemoglobin fell to 79 per cent. A bone marrow study showed a normal number of platelet-forming megakaryocytes, but an excess of non-platelet-forming types usually associated with thrombocytopenic purpura. The prothrombin time was normal. The initial episode of bleeding resolved slowly, concomitantly with the administration of cortisone. In spite of treatment, cardiac failure steadily increased, accompanied by several paroxysms of atrial tachycardia, followed by atrial fibrillation, and by swelling of the left arm suggestive of an axillary vein thrombosis. Death occurred on November 4, 1953.

The clinical diagnoses were congestive cardiac failure; tricuspid incompetence; thrombosis of the large pulmonary arteries, probably secondary to multiple emboli; inferior vena caval thrombosis; and episodic thrombocytopenic purpura and thrombosis of undetermined cause.

A point of special note was the development of dyspnea and syncope, before the onset of signs of peripheral venous thrombosis and the progressive reduction in pulsation of the enlarged pulmonary arteries on fluoroscopy. This observation suggested that the initial occlusions had occurred in the small pulmonary arteries and spread proximally.

At autopsy both main branches of the pulmonary artery were almost completely occluded by laminated, organizing thrombi, which met half an inch above a normal pulmonary valve (fig. 3). The right ventricle was hypertrophied, but the tricuspid valve

![FIG. 4. Case 2. Posteroanterior roentgenogram of chest showing enlargement of the right ventricle, main pulmonary trunk, and right and left main branches. The lung fields are underfilled and clear, except for an ill-defined loss of translucency in both costophrenic regions.](image1)

![Fig. 3. Case 1. Drawing of postmortem appearance of heart and pulmonary arteries. The heart is normal, apart from hypertrophy of the right ventricle. The distal portion of the main trunk of the pulmonary artery and the right and left main branches are filled with adherent thrombus. Many of the smaller arteries are also filled with thrombus, which is traversed by fibrous trabeculae. The pulmonary veins are normal.](image2)

![Fig. 5. Case 2. Postmortem appearance of the heart, pulmonary artery, and lungs. The heart is normal except for hypertrophy of the right ventricle. Both main branches of the pulmonary artery (arrows) contain adherent thrombus, which shows clear evidence of coiling in the left main branch.](image3)
was not dilated. The mitral and aortic valves were normal, as were the septa. There was no evidence of thrombosis of the caval, iliac, or femoral veins.

Antemortem thrombus extended into the peripheral branches of the pulmonary artery on both sides, being more recent on the left. No pulmonary infarcts were present. The thrombus in the main pulmonary arteries and their immediate branches was pale and had clearly been deposited in layers. In the smaller arteries the lumina were frequently broken up by fibrous strands into a number of channels, and these in their turn were occluded by recent red thrombi. Histologic examination confirmed that the thrombus in the main arteries had been deposited in layers, of quite different ages, the deeper being well organized and the superficial being relatively recent. There was no evidence of atheroma of any significance and none of arteritis.

The bronchial arteries were more prominent and dilated than normal. The lung parenchyma and bronchi were normal.

The liver was markedly congested and showed changes of early cirrhosis. The bone marrow was normal; the spleen was very congested.

Case 2. A 56-year-old woman began her illness with swelling and tenderness of the calf, followed 1 month later by dyspnea on exertion and orthopnea. Two weeks later she developed pleuritic pain on the right side, and suffered 2 hemoptyses. Death occurred suddenly, despite anticoagulant therapy.

On examination she was a dyspneic, cyanosed woman, with a small regular pulse, elevated jugular venous pressure, and edema of the legs. There was doubtful enlargement of the right ventricle, the second heart sound was normal, and there was a loud systolic murmur over the pulmonary artery. The blood pressure was 140/80 mm. Hg. There was dullness at the base of the right lung, but no clubbing of the fingers.

X-ray film of the chest showed large main pulmonary arteries and right ventricle. The lungs were clear except for an opacity at the right costophrenic angle (fig. 4). (The patient was too ill for fluoroscopy.) Electrocardiogram showed low voltage with first degree atrioventricular block, and doubtful right ventricular hypertrophy.

The clinical diagnosis was thrombosis of main pulmonary arteries, probably secondary to repeated pulmonary emboli.

At autopsy the main pulmonary artery was occluded by an adherent laminated thrombus that showed coiling (fig. 5). No underlying disease of the pulmonary artery was present. The appearances were those of embolism with secondary thrombosis, the age of the thrombus being consistent with the duration of the symptoms. The heart was normal, apart from hypertrophy of the right ventricle. Both femoral veins were thrombosed. No other disease was present.

The diagnosis was suspected in these patients be-

### Table 1.—Cases* of Thrombosis of the Large Pulmonary Arteries without Associated Disease

<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>Age</th>
<th>History</th>
<th>Clinical findings and progress</th>
<th>X-ray</th>
<th>Electrocardiogram</th>
<th>Autopsy findings</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Description</td>
</tr>
<tr>
<td>3</td>
<td>MF</td>
<td>46</td>
<td>Attacks of fever, substernal pain, hemoptysis, and dyspnea; died suddenly</td>
<td>Signs of venous thrombosis in legs; fluid in right chest; apical systolic murmur; triple rhythm; falling B.P.</td>
<td>Heart enlarged</td>
<td>Left bundle-branch block; large right atrium, ?</td>
<td>Thrombosis of main pulmonary arteries to right lower and upper lobes; dissection shows emboli embedded in local thrombus; histology shows organizing clots; multiple thromboses in leg and arm veins</td>
</tr>
<tr>
<td>4</td>
<td>HB</td>
<td>79</td>
<td>Admitted because of severe epistaxis; vomited twice; died suddenly 9 days after admission</td>
<td>No abnormal signs; B.P. 130/80; slight albuminuria</td>
<td>—</td>
<td>—</td>
<td>Thrombosis of right and left main pulmonary arteries; recognizable embolus lies in mass of secondary thrombus; histology shows severe pulmonary atheroma; clot lies over atheromatous plaques; no source for embolus</td>
</tr>
</tbody>
</table>

* Cases 1 and 2 discussed in detail in text.
**Table 2—Cases of Thrombosis of the Large Pulmonary Arteries Associated with Mitral Stenosis**

<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>Age</th>
<th>History</th>
<th>Clinical findings and progress</th>
<th>X-ray</th>
<th>Cardiogram</th>
<th>Autopsy findings</th>
<th>Infarcts in lungs</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>F</td>
<td>59</td>
<td>Progressive dyspnea on exertion; paroxysmal dyspnea; hemoptysis</td>
<td>Signs of severe mitral stenosis; sinus rhythm with calcified mitral valve; valvotomy performed; subsequent atrial fibrillation; 5 months after operation succumbed to coronary thrombosis and multiple systemic emboli</td>
<td>LA +</td>
<td>Slight right vent. hypertrophy</td>
<td>Thrombus in left coronary artery with anteroseptal infarct; embolus in right external iliac artery. Thrombus partially occluding right main PA; heavily calcified mitral valve with tear in one commissure</td>
<td>0</td>
<td>A local thrombosis in the PA overlying a patch of atheroma (fig. 8)</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>18</td>
<td>Dyspnea on exertion; hemoptysis; jaundice; fever; stabbing pain in the chest; died suddenly</td>
<td>Signs of mitral stenosis and tricuspid incompetence; systolic murmurs at apex and tricuspid area, due to mitral and tricuspid disease respectively; rales over right lung; sinus rhythm, jaundice</td>
<td>Heart enlarged; opacity at base of right lung</td>
<td>RA +</td>
<td>Mitral and tricuspid stenosis, thrombosis of right main PA; no source for emboli found</td>
<td>1 small</td>
<td>Probably repeated pul. emboli but thrombus not coiled in PA; impossible to tell origin pathologically</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>77</td>
<td>Sudden ischemia of left foot due to arterial embolus</td>
<td>Atria’ fibrillation; gangrene of left foot; no heart failure or cardiac murmurs detected; died in coma due to cerebral vascular accident</td>
<td>—</td>
<td>Ischemic pattern</td>
<td>Occlusion of left femoral artery; m’tral stenosis; thrombosis right PA; lower lobe artery contained a thrombus, which took the shape of, and filled the artery; histology showed laminated thrombus; no source for emboli</td>
<td>—</td>
<td>Probably thrombus in situ</td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>80</td>
<td>Progressive dyspnea; confusion; old cerebral embolism; bedridden</td>
<td>Mitral stenosis; aortic incompetence; atrial fibrillation; anemia; old hemiplegia</td>
<td>—</td>
<td>—</td>
<td>Mitral stenosis; aortic incompetence; small myxoma of left atrium; adher nt laminated organized thrombus partially occluding right PA; also, recent pulmonary embolus with infarct in right upper lobe; thrombosis of the femoral vein</td>
<td>+</td>
<td>Thrombus secondary to embolism</td>
</tr>
</tbody>
</table>

* Cases 5 and 6 discussed in detail in text.
LA—left atrium; PA—pulmonary artery; RV—right ventricle.
cause of progressive right ventricular failure associated with signs of peripheral venous thrombosis. The pulmonary systolic murmur and normal pulmonary component of the second heart sound suggested obstruction to the pulmonary arteries near the pulmonary valve, rather than in the small branches, when signs of pulmonary hypertension might have been expected. Virtual disappearance of pulmonary artery pulsation in case 1, strongly favored the main branches as the site of obstruction. Progressive diminution in pulsation has been noted in cases of atrial septal defect with thrombosis of the main pulmonary arteries by Canada and co-workers. In case 1, the original finding of marked pulsation of the main pulmonary arteries, after signs of cardiac failure had appeared, suggests that retrograde thrombosis may have followed multiple emboli in the small branches.

The remaining 2 cases without associated diseases (case 3 and case 4) were not seen in life by the authors, and their clinical findings are therefore very briefly summarized in Table 1.

Thrombosis of the Main Pulmonary Arteries Associated with Mitral Stenosis

Mitral stenosis was the most frequent single disease associated with pulmonary artery thrombosis in the series, and was present in 6 of the total of 23 patients. Five of these 6 patients were seen during life, but the diagnosis of thrombosis of the pulmonary arteries was not made. The symptoms and signs attributable to mitral valve disease overshadowed any referable to pulmonary artery thrombosis in all except 3 patients, who, in retrospect, might have been correctly diagnosed in life. Two of these 3 patients are described in detail.

Case 5. A woman, aged 55, complained of progressive dyspnea on exertion for 11 years. More recently subternal pain on exertion, relieved by rest, was noticed, and repeated hemoptyses occurred for 4 weeks. She also had a cough with sputum. On examination she was an orthopneic woman with a malar flush. The arterial pulse was of small volume, and there was an augmented 'a' wave in the jugular venous pulse. The diffuse tapping cardiac impulse suggested right ventricular hypertrophy. The second heart sound was split with marked accentuation of the pulmonary element, and there was a very faint pulmonary diastolic murmur. Triple rhythm and a soft diastolic murmur were heard at the apex. The blood pressure was 110/60 mm. Hg. There was an effusion at the base of the right lung and slight clubbing of the fingers. The abdomen was normal and there was no peripheral edema.

The hemoglobin was 99 per cent, the white cell count was 18,000, and the platelet count was 350,000/ml.

Fluoroscopy and roentgenogram of the chest (fig. 6) showed enlargement of the right ventricle. The main pulmonary arteries were grossly enlarged and pulsating slightly. The right main branch was irregular in contour. The left atrium was enlarged, and there was a right pleural effusion. There was a rounded shadow in the right lower midzone. An electrocardiogram showed right ventricular hypertrophy.

On cardiac catheterization, the right ventricular pressure was 70/10 mm. Hg and the pulmonary artery pressure was 74/52 mm. Hg. The cardiac output was 2 L./min. An angiocardiogram showed a large right ventricle and very large pulmonary arteries. No shunt was visualized. The shadow in the right midzone appeared to become denser, suggesting pulmonary artery origin. There was poor contrast and poor filling of the distal pulmonary arteries.

The clinical course was marked by repeated pulmonary infarcts and increasing cardiac failure. The patient improved sufficiently to be discharged from hospital, but was readmitted in acute circulatory failure, with an impalpable pulse, high venous pressure, and rapid atrial fibrillation; death occurred within 24 hours.

The clinical diagnosis was pulmonary hypertension, probably due to mitral stenosis.

At autopsy there was tight mitral stenosis. The distal half of the main pulmonary artery contained laminated antemortem thrombus extending down the right and left main branches. No thrombosis was found in the femoral or abdominal veins. There were multiple pulmonary infarcts.

On histologic examination, in both main pulmonary arteries, the thrombus lay over severe atheromas and was deposited in successive layers, the deepest being well organized. It was thought to be a thrombus in situ.

In this patient the flow through the mitral valve must have been so reduced as to prevent almost completely the production of the typical murmurs. The low cardiac output would favor stasis and thrombosis in the pulmonary arteries, especially in the presence of local disease and severe pulmonary hypertension. Poor pulsation of the main pulmonary arteries is unusual with severe pulmonary hypertension; if this had been associated with the abnormal contour of the right main pulmonary artery and signs of repeated pulmonary infarcts, the correct diagnosis might have been made.

Case 6. A woman, aged 27 years, complained of dyspnea on exertion for 7 years; 3 weeks before admission the dyspnea increased and was accompanied by peripheral edema and cough with purulent sputum. There were several attacks of acute dyspnea and syncope. On examination there were signs of mitral stenosis, with right ventricular hypertrophy. The pulmonary component of the second heart sound was accentuated. The jugular venous pressure was 4 to 5 cm. above the sternal angle. An early diastolic murmur of either pulmonary or aortic incompetence
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and a fourth heart sound were heard. The pulse was regular and of normal volume, and the blood pressure was 120/65 mm Hg. There were peripheral edema and hepatomegaly. Bronchial breathing was audible at the base of the right lung, and scattered rales were heard throughout both lungs. Roentgenogram of the chest and fluoroscopy showed a lack of marked pulsation of the main pulmonary arteries. There was enlargement of the right atrium and ventricle, pulmonary artery, and left atrium. An electrocardiogram showed right ventricular and atrial hypertrophy, with first degree atrioventricular block.

There was steady deterioration and death in congestive heart failure.

At autopsy there was mitral stenosis and aortic incompetence, with a thrombus in the right atrium. The right ventricle was hypertrophied. The lungs showed edema, bronchitis, and pulmonary infarcts. There was adherent thrombus in the right main pulmonary artery. The smaller arteries showed hyperplasia of the internal elastic lamina, fibrinoid necrosis, acute arteritis, and local thrombosis (fig. 7). There was no source for emboli other than possibly the right atrium and the lesion was considered to be a thrombus in situ in the pulmonary artery, overlying severe vascular disease.

FIG. 6. Case 5. Posteroanterior roentgenogram of the chest showing enlargement of the right ventricle and enormous distention of the main and left main pulmonary arteries. The right main pulmonary artery is also greatly enlarged, and its contour is irregular. There is a large effusion in the right costophrenic angle, overlying a pulmonary infarct.

FIG. 7. Case 6. Oblique section of a small pulmonary artery. On one side is a plaque of atheroma (arrow), and over this, filling the lumen, is antemortem thrombus, apparently formed in situ. (X 90. Weigert's elastic stain and van Giesen's stain.)
The rapidly progressive deterioration and congestive heart failure, coupled with the episodes of acute dyspnea and fainting, in this case might have suggested the diagnosis.

The details of the other cases of mitral stenosis are outlined in table 2 (cases 7–10). The signs of thrombosis of the pulmonary arteries were obscured in cases 7, 9, and 10, by other complications of the mitral valve disease, but might have been suspected in case 8, since there were episodes suggestive of repeated pulmonary infarction.

Four of the 6 patients with mitral stenosis had apparently developed a primary thrombosis in situ, and this aspect of pulmonary artery thrombosis is considered later.

**Over-All Findings**

**Thrombosis of the Main Pulmonary Arteries in Association with Other Diseases**

These cases are outlined in table 3. With the exception of patients no. 20 and 22, who were seen in life, the remainder were studied only retrospectively from case notes and autopsy reports. In many patients any signs or symptoms attributable to pulmonary artery thrombosis were completely overshadowed by some other clinical catastrophe, such as a cerebral vascular accident.

**Clinical Picture**

**Symptoms.** The commonest was dyspnea; symptoms compatible with pulmonary infarction (pleural pain or hemoptysis) were present in 11 of the 23 patients. Fainting was a striking symptom in 3 cases. In most cases, however, the symptoms of the associated or underlying disease overshadowed any due to the pulmonary lesion itself. Several patients died in coma; while in the group with mitral disease, many of the symptoms could be accounted for by the valvular lesion.

**Physical Signs.** The commonest sign was hypertrophy of the right ventricle, but in a number of these patients it was due to mitral stenosis. The same was true of accentuation of the pulmonary element of the second heart sound and of abnormalities in the central venous pulse. Two patients had a systolic murmur in the pulmonary area, and neither had any cardiac lesion other than right ventricular hypertrophy. The diagnostic value of the pulmonary systolic murmur and normal second heart sound in these patients is discussed later. The arterial pulse was small in volume in 5 patients, 1 of whom had mitral disease. Congestive cardiac failure was noted in 10 patients, but there were signs suggesting peripheral cardiac failure in only 3, while 4 patients had no signs referable to disease of the heart or lungs.

**Mode of Death.** This was gradual in 18 and sudden in 5.

**Radiology.** Adequate chest films were available in 14 patients. Right ventricular and pulmonary artery enlargement was a constant feature and the lung fields appeared underfilled in 3 patients, (1 of whom, however, had pulmonary atresia). Restricted pulsation of the main arteries was striking in 2 patients, but in many cases fluoroscopy was not possible. In 1 patient with mitral stenosis (case 5), an irregular enlargement of the right pulmonary artery due to the thrombus partly obscured by a large pleural effusion was seen (fig. 6).

The important radiologic signs were underfilled lung fields and enlarged, poorly pulsating main pulmonary arteries.

**Electrocardiography.** Recent, adequate electrocardiograms were available in 12 patients: 8 showed right ventricular hypertrophy (4 gross and 4 moderate), and 4 showed right atrial hypertrophy. There was no evidence of enlargement of the right heart in 4 patients.

**Pathologic Findings in Twenty-three Cases of Massive Thrombosis of the Large Pulmonary Arteries**

Thrombosis was considered to be secondary to embolism in the majority of the cases, but appeared to have arisen in situ in a minority.

In making this differentiation we have relied mainly on the naked-eye appearances of the dissected specimen, supplemented by the histologic appearances. One source of error that we cannot exclude is the initiation of thrombosis by a small, undetected embolus. Since we are concerned here with thrombosis in the large pulmonary arteries where only massive emboli are likely to lodge, we believe that this source of error is not great.

Table 4 sets out the criteria we have used in the differentiation of primary from secondary
### Table 3.—Thrombosis of the Large Pulmonary Arteries Associated with Other Disease

<table>
<thead>
<tr>
<th>No.</th>
<th>Sex/age</th>
<th>Associated disease</th>
<th>History</th>
<th>Clinical picture</th>
<th>X-ray</th>
<th>Electrocardiogram</th>
<th>Autopsy findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>11 HD</td>
<td>F 72</td>
<td>Chronic bronchitis; aortic stenosis</td>
<td>Cough and dyspnea many years; hemoptysis; progressive congestive heart failure</td>
<td>Edema; signs of aortic stenosis; consolidation right base</td>
<td>LV +; fibrosis right lung</td>
<td>Aortic stenosis; thrombosis of main PA's; thrombosis of thigh veins</td>
<td>+ Impossible to tell nature of clot in PA</td>
</tr>
<tr>
<td>12 SOL</td>
<td>F 66</td>
<td>Syphilitic arteritis; coronary artery disease</td>
<td>1 year vertigo and syncope; intractable vomiting; sudden death</td>
<td>Cyanosis; small pulse; JVP + 2 cm.; heart sounds faint</td>
<td>—</td>
<td>—</td>
<td>Thrombosis at bifurcation of PA; syphilitic aortitis; coronary artery atheroma; no source for embolus found</td>
</tr>
<tr>
<td>13 JK</td>
<td>M 48</td>
<td>Diaphragmatic hernia</td>
<td>Dyspnea on exertion; epigastric pain; vomiting</td>
<td>Severe anemia due to bleeding from gastrointestinal tract; heart and lungs normal; BP 110/85; became dyspneic and cyanosed after repair of hernia; died fairly rapidly</td>
<td>LV +</td>
<td>—</td>
<td>Thrombosis of main PA; branches of 1st and 2nd divisions had fibrous cords around them and many were occluded by old fibrosis; no source for embolus found</td>
</tr>
<tr>
<td>14 FJ</td>
<td>F 83</td>
<td>Chronic bronchitis; cerebral thrombosis or embolus</td>
<td>Admitted in coma; no previous history except “dizziness”</td>
<td>Left hemiplegia; atrial fibrillation; BP 130/70; died in coma</td>
<td>—</td>
<td>—</td>
<td>Thrombosis main PA; cerebral thrombosis; lobar pneumonia; no source for embolus found</td>
</tr>
<tr>
<td>15 ER</td>
<td>F 74</td>
<td>Ca of bronchus; diverticulitis</td>
<td>Cough; hemoptysis; dyspnea; gradual deterioration; diarrhea; died slowly</td>
<td>Collapse left lower lobe, no cardiac lesion; JVP not raised</td>
<td>Collapse LLL</td>
<td>Nothing definite</td>
<td>Ca of bronchus LLL; thrombosis right main PA; no source for emboli</td>
</tr>
<tr>
<td>16 AR</td>
<td>F 60</td>
<td>Ca of pancreas</td>
<td>Abdominal pain; jaundice; laparotomy disclosed Ca of pancreas</td>
<td>Abdominal mass; jaundice; heart and lungs normal</td>
<td>—</td>
<td>—</td>
<td>Ca of pancreas; thrombosis right main PA; no source for emboli found</td>
</tr>
<tr>
<td>No.</td>
<td>Age</td>
<td>Sex</td>
<td>Cause of Death</td>
<td>Cause of Embolism</td>
<td>Symptoms</td>
<td>Diagnosis</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>17</td>
<td>M</td>
<td>58</td>
<td>Ca of stomach</td>
<td>Thrombosis</td>
<td>Died 6 days after gastrojejunostomy; mass in abdomen; heart and lungs normal; deteriorated steadily after operation</td>
<td>Thrombosis right and left PA's; no source for emboli; Ca. ventriculi with secondary deposits; histology shows small PA full of organizing clot; fibrin layers could have been formed locally</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>F</td>
<td>75</td>
<td>Ca of bronchus; SVC thrombosis</td>
<td>Could be emboli or thrombosis; source for embolism in SVC</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>F</td>
<td>60</td>
<td>Myxedema; myocardial infarction; Kimmelstiel-Wilson's disease</td>
<td>Impossible to tell whether previous thrombosis or embolism</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>M</td>
<td>54</td>
<td>Intestinal obstruction; uremia; pylonephritis</td>
<td>Almost certainly thrombosis in situ</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>M</td>
<td>8</td>
<td>Pul. atresia; cerebral venous thrombosis</td>
<td>Clearly thrombosis in situ in a stagnant vascular channel in a polyeystemic infant</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Causes and Symptoms**
- **Ca of stomach**: Died 6 days after gastrojejunostomy; mass in abdomen; heart and lungs normal; deteriorated steadily after operation.
- **Ca of bronchus; SVC thrombosis**: Loss of weight; cough; swelling of face.
- **Myxedema; myocardial infarction; Kimmelstiel-Wilson's disease**: Dyspnea on exertion; chest pain due to previous cardiac infarct.
- **Intestinal obstruction; uremia; pylonephritis**: Abdominal pain and vomiting; operation for intestinal hernia; subsequent hematuria, hemoptysis, dyspnea; died in coma.

**Diagnoses**
- **Thrombosis right and left PA's**: no source for emboli; Ca. ventriculi with secondary deposits; histology shows small PA full of organizing clot; fibrin layers could have been formed locally.
- **Could be emboli or thrombosis**: source for embolism in SVC.
- **Myocardial infarct; pyelonephritis; thrombosis right PA; peripheral veins not examined**: Posterior cardiac infarct? low potassium state.
- **Thrombosis of right main pulmonary artery**: old thrombus in the main PA propagated clot to lower lobe; upper & middle lobes showed edema; lower lobe infarcted; histology shows organized clot with fibroblasts, sinusoids, trace of collagen; no source for emboli in leg veins.

**Notes**
- 17: Possibly thrombosis.
- 18: Could be emboli or thrombosis; source for embolism in SVC.
- 19: Impossible to tell whether previous thrombosis or embolism.
- 20: Almost certainly thrombosis in situ.
<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>Associated disease</th>
<th>History</th>
<th>Clinical picture</th>
<th>X-ray</th>
<th>Electrocardiogram</th>
<th>Autopsy findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>22</td>
<td>M</td>
<td>Acute gastro-enteritis</td>
<td>Malaise, restlessness, thirst 4 days; brought in dead</td>
<td>—</td>
<td>—</td>
<td>Recent thrombus in PA to RLL; vessels normal</td>
<td></td>
</tr>
<tr>
<td>M1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0 Presumably thrombosis in situ in marasmic infant</td>
</tr>
<tr>
<td>23</td>
<td>M</td>
<td>Ca of lung; chronic bronchitis, emphysema; cor pulmonale</td>
<td>Progressive cough, dyspnea and loss of weight; 4 weeks before death, pain in left calf; syncope; attacks; right pleural pain; hemoptysis, dyspnea</td>
<td>Cyanosed; congestive r. ventricular failure</td>
<td>Opacity right upper zone; enlarged PA's and RV; diffuse mottling both lungs</td>
<td>RV ++ RA + Ca of upper lobe of right lung. RV and RA enlarged; adherent clot in main branches of PA; source of embolus not looked for; (limited autopsy)</td>
<td></td>
</tr>
</tbody>
</table>

Ca—carcinoma; JVP—jugular venous pressure; LL—left lower lobe; LV—left ventricle; RA—right atrium; RLL—right lower lobe; RV—right ventricle; SVC—superior vena cava; VSD—ventricular septal defect.
thrombosis. A source for an embolism in peripheral veins is of only limited value in the differential diagnosis, since it may sometimes be difficult or impossible to find in manifest cases of embolism.6

Using these criteria, we consider that 7 of the 23 cases had an initial pulmonary thrombosis in situ, 6 had definite initial pulmonary emboli, and in 10 the nature of the initial incident was uncertain. It seems highly probable that these last were in reality due to emboli. Five of the 7 cases with primary thrombosis had pulmonary arterial disease or other local pathology. The histologic differences between primary embolism and thrombosis are shown in figures 8 and 9. No single 1 of the above criteria, however, is alone decisive in differential diagnosis, and figure 10 illustrates this point. In this case the lamination of the thrombus implies that the upper layers have been deposited locally, but the lack of local arterial disease and the presence of a source for emboli in the femoral veins suggest that the thrombus probably originated from an embolus.

**Pulmonary Infarction.** This was frequent, but not invariable, and occurred in 4 of the

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**Table 4.—Criteria for the Differentiation of Primary and Secondary Thrombosis**

<table>
<thead>
<tr>
<th>Primary Thrombosis</th>
<th>Secondary Thrombosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Local disease of pulmonary artery.</td>
<td>No local disease present.</td>
</tr>
<tr>
<td>Thrombus is a cast of the vessel.</td>
<td>The embolus is usually coiled.</td>
</tr>
<tr>
<td>There is often deposition in episodes producing lamination of the clot in layers roughly parallel to the arterial wall. (No source for the embolus can be found).</td>
<td>The source of the embolus is often found and corresponds to the shape of the clot in the pulmonary artery.</td>
</tr>
</tbody>
</table>

---

**Fig. 8. Case 7.** Section at bifurcation of lower lobe pulmonary artery. There is a plaque of atheroma on the lower side of the artery, 1, covered by a layer of partly organized thrombus, 2. This in turn is covered by a layer of more recent thrombus, 3. (X 9. Hematoxylin and eosin stain.)
7 cases with primary thrombosis and in 10 of the 16 remaining cases, in which the thrombus was certainly or probably secondary to embolism.

Discussion

Our cases confirm the reports in the literature that thrombotic occlusion of the main pulmonary arteries may occur as a complication of a number of diseases, especially of the heart and lungs, and to a lesser extent, of the gastrointestinal tract, but may occasionally be unassociated with any other disease. It is clear that the occlusion may develop gradually, although it may be very difficult to determine the exact age of the thrombus.

Symptoms. The symptoms of thrombotic occlusion are insidious, and in general are those of right heart failure and low cardiac output. In Middleton's 73 cases, dyspnea was present in 90 per cent, hemoptysis in 62 per cent, apprehension in 30 per cent, cough in 24 per cent, and substernal pain in 24 per cent. Savacool and Charr 6 listed the principal symptoms in their own 12 cases as dyspnea, pain in the chest or epigastrium, and mental confusion. The last symptom was striking, and presumably due to cerebral ischemia. Brenner 6 stressed dyspnea as a constant feature in his cases of thrombosis of the main pulmonary arteries.

Fig. 9. Section of a pulmonary artery branch containing an embolus that appears as 2 separate pieces. Note the absence of arterial disease. (× 9. Hematoxylin and eosin stain.)

Fig. 10. Case 10. Section of a large pulmonary artery showing local thrombosis occurring in episodes. The first 3 layers of thrombus (1, 2, and 3) have undergone partial organization from their edges, as can be seen by the dark lines of fibrous tissue growing in toward the middle. The topmost layer of recent thrombosis shows Zahn's lines. Note the absence of disease of the underlying arterial wall. (× 5.5. Weigert's elastic and Van Giesen's stains.)
artery. Carroll\textsuperscript{10} mentioned dyspnea in all his 5 cases and syncope on exertion in 1. Pleuritic pain and hemoptysis are likely to occur with infarction, but are often absent. Bryson\textsuperscript{11} considered that half the cases had an acute onset of symptoms and thought that hemoptysis might arise from bronchopulmonary anastomoses. Substernal pain and syncope are presumably due to diminished coronary and cerebral blood flow, respectively. Abdominal pain may be caused by hepatic engorgement due to tricuspid incompetence. Sudden death has often been reported.

Our own cases showed a similar symptomatology, especially sudden dyspnea, fainting, and pulmonary infarction, although mental confusion was not a striking feature, and in several cases there were no symptoms attributable to the thrombosis. Sudden onset and sudden death were not common.

Physical Signs. Cyanosis was present in 68 per cent of Middleton's 53 cases. It is presumably peripheral in nature and is due to a low cardiac output, except when there is a right-to-left shunt, which would be intensified by the occlusion of the pulmonary arteries.\textsuperscript{9} Signs of tricuspid incompetence, peripheral edema, hepatic engorgement, and increased jugular venous pressure, together indicating congestive failure of the right ventricle, were reported by Carroll\textsuperscript{10} and by Hanelin and Eyler.\textsuperscript{12} Signs of pulmonary hypertension (enlarged right ventricle and accentuated pulmonary element of the second heart sound) have also been frequently mentioned. Two of the 7 cases described by Keating and co-workers\textsuperscript{19} had a lone apical systolic murmur, 2 had diastolic murmurs over the pulmonary artery, which in 1 was associated with an apical systolic murmur; but 3 had no murmurs at all. Carroll's\textsuperscript{3} 5 cases all had systolic murmurs in the region of the pulmonary artery, as had Middleton's\textsuperscript{7} case. Savacool and Charr\textsuperscript{4} stress the small pulse, low blood pressure, and restlessness as signs of low cardiac output, but these probably do not occur until the main pulmonary arteries are substantially occluded. Gibbon and associates\textsuperscript{14} in experimental studies on dogs, found that the systemic blood pressure did not fall until 60 per cent occlusion of more than 75 per cent of the pulmonary arteries caused death.

The physical signs in our own cases were often difficult to evaluate, since the associated or underlying disease tended to mask them. However, the 3 cases with repeated thromboembolic episodes of unknown cause presented the features of right-sided heart failure with low output, a systolic murmur over the pulmonary artery or at the apex, a normal or soft pulmonary component of the second heart sound, and enlargement of the right ventricle. Accentuation of the pulmonary component of the second heart sound suggesting pulmonary hypertension was found only in patients in whom pulmonary hypertension was present before the occurrence of the thrombosis. It is probable that a thrombus in the main pulmonary arteries extending down close to the valve could be associated with a low pressure in the pulmonary artery and, hence, a normal or soft pulmonary component of the second heart sound, which was in fact present in 2 of our patients.

Radiologic Signs. The radiologic signs have been reviewed by Hanelin and Eyler.\textsuperscript{12} The main pulmonary arteries were enlarged, while the peripheral branches were small; the lung fields appeared clear, with the exception of shadows cast by pulmonary infarcts. Keating and associates\textsuperscript{13} found right ventricular and pulmonary arterial enlargement and clear lung fields in all their 7 cases. Absence of pulsation of the main pulmonary arteries (which had previously pulsed strongly) was noted by Canada and co-workers\textsuperscript{9} in 1 of their 3 cases with thrombosed pulmonary arteries, but comment has not been previously made on this point. Absence of pulsation in an enlarged right main branch is unusual, and was helpful in diagnosis in 1 of our cases. But it should be remembered that enlargement of the main pulmonary artery and left main branch may be associated with poor pulsation in cases of pulmonary stenosis.

Adequate radiology in many of our cases was not possible, but in 2 patients (cases 1 and 2) a combination of clear lung fields and enlarged pulmonary arteries was seen. In a
third case (case 5) the contour of the enlarged right pulmonary artery was irregular, being quite unlike the smooth convex margin usually seen when this vessel is enlarged.

The Electrocardiogram. As might be expected, the electrocardiogram has been reported to show hypertrophy of the right ventricle, and did so in many of our patients.

Diagnosis. The diagnosis of pulmonary artery thrombosis should be considered whenever a patient presents unexplained congestive cardiac failure, dyspnea, fainting, repeated episodes of pulmonary infarction, or pain in the chest. If the clinical picture includes evidence of peripheral venous thrombosis, a systolic murmur over the pulmonary artery, and a normal or soft component of the second heart sound the diagnosis becomes more probable. If, in addition, x-ray films of the chest show enlargement of the right ventricle and pulmonary arteries (without significant pulsation of the latter) and clear lung fields or shadows suggesting infarction, together with electrocardiographic evidence of right ventricular hypertrophy, the diagnosis becomes almost certain.

Cardiac catheterization is of value in excluding other lesions, such as septal defects and pulmonary stenosis, but contributes little positive information other than confirming right ventricular hypertension. Exclusion of pulmonary stenosis may be difficult owing to the difficulty of passing the catheter into a thrombosed main pulmonary artery.

Differentiation from mitral stenosis may also be difficult, especially as pulmonary artery thrombosis may complicate this condition, and the typical murmurs may be difficult to hear in patients with severe pulmonary hypertension. The characteristic radiologic vascular changes seen in mitral disease, and enlargement of the left atrium, should assist in making the differential diagnosis. Absence of a history or clinical evidence of lung disease should exclude a diagnosis of chronic anoxic cor pulmonale. Differentiation from idiopathic pulmonary hypertension may be difficult, and it is possible that pulmonary artery thrombosis might complicate this condition. Obscure forms of chronic pulmonary fibrosis would be likely to show characteristic radiologic changes in the lungs, which are absent in pulmonary artery thrombosis.

Angiocardiography should be the most satisfactory diagnostic method, but was not found to be of real value in case 5, while the other cases in which the condition was suspected were too ill for the investigation to be carried out. With improved and safer technics of angiography, it might be performed if the diagnosis is suspected.

Treatment. Lack of success has probably been due to failure of early diagnosis. Prolonged anticoagulant therapy offers the only hope of preventing extension of the thrombus. If the thrombosis is secondary to repeated pulmonary emboli from thrombosis of the leg veins that cannot be controlled by anticoagulants, ligation of the inferior vena cava might have to be considered.

Genesis of the Thrombosis and Associated Disease

It is often considered that thrombosis in the pulmonary arterial tree is always secondary to embolism. Brenner considered that primary thrombosis did sometimes occur, especially in association with disease of the pulmonary arteries, and our own findings of local pulmonary artery disease in 5 of 7 cases with primary thrombosis support this view. Differentiation between primary thrombosis and that secondary to embolism may be impossible, however. While a recent embolus is easy to recognize because the clot is not a cast of the artery and is usually coiled, after a few days further thrombus is deposited around it, tending to obscure the original outline. Where a clot does not fully obstruct an artery, endothelium rapidly grows over it, while organization from the arterial wall fixes it in position. By contrast, a local thrombus fits the vessel and is often laid down in layers at different times. Glynn and Knowles considered this laminating to be good evidence of primary thrombosis, although Brenner maintained that laminated thrombi from peripheral veins may be dislodged and carried to the pulmonary artery, so that the phenomenon might be found in thrombi secondary to embolism. In our view, however, the presence of a clot laminated in planes
roughly parallel to the wall of the artery, fitting the vessel, and overlying local arterial disease is virtually diagnostic of primary thrombosis in situ.

A number of different forms of intrathoracic disease have been described in association with thrombosis of the large pulmonary arteries, particularly congenital heart disease and pulmonary tuberculosis. Mitral stenosis has not featured prominently.

Savacool and Charro reported 12 cases associated with pulmonary tuberculosis. They were convinced that the thrombi arose in situ in view of their size and shape, their firm adherence to the intima, invasion by strands of connective tissue from the intima, and the deposition of fibrin platelets and other blood constituents. The primary site of thrombosis appeared to be the termination of the small branches of the main pulmonary artery, which were often embedded in fibrous tissue or tuberculous consolidation. Furthermore, they noted that the right pulmonary artery was involved more frequently than the left, which they attributed to the fact that the right pulmonary artery is compressed between the vein and bronchus and is crossed by the aortic arch, while the left pulmonary artery is relatively free. Gordon and Perl described pulmonary arterial thrombosis in a child with patent ductus arteriosus, pulmonary stenosis, and subacute bacterial endocarditis, and Whiphamp described an infant of 7 months, with patent ductus arteriosus and a thrombus in the pulmonary artery that projected through the valve into the right ventricle. Dickinson described occlusion of the main trunk and both main branches of the pulmonary artery in a boy aged 4½ years who had pulmonary stenosis and a ventricular septal defect. Canada reported 3 cases of atrioseptal defect with pulmonary artery thrombosis. Widespread focal thrombosis in the small pulmonary vessels has been described by Rich in cases of the tetralogy of Fallot, while in 1 of our 23 cases widespread thrombosis, including the main pulmonary arteries, occurred in association with partial pulmonary atresia and a large ventricular septal defect.

Thrombosis of the large pulmonary arteries has been reported in children in the absence of congenital heart disease. Gehrt found 7 cases in 600 autopsies, 5 of which followed measles. Gunther described thrombosis in a pair of identical twins, one of whom died of gastroenteritis and the other after mastoidectomy. Since children rarely develop pulmonary embolism, these cases provide evidence in favor of primary thrombosis, and there was 1 such in our series.

Primary thrombosis might be expected to occur in association with disease of the pulmonary arteries, such as atherosclerosis, which may be found in chronic lung disease and in congenital and acquired heart disease. Where disease of the arterial wall is combined with a low cardiac output, a sluggish pulmonary blood flow, and enlargement of the main pulmonary arteries, thrombosis in situ is particularly favored (fig. 8).

Disease of the pulmonary arteries is an important complication of mitral valve disease where atherosclerosis may involve the large branches, as in our cases 5 and 7. Furthermore, pulmonary hypertension is common, and the cardiac output is often reduced in severe cases. The summation of these 3 factors would be conducive to primary thrombosis.

It is not surprising, therefore, that 19 of 88 cases of pulmonary artery thrombosis collected by Savacool and Charro occurred in association with mitral disease, and the incidence in our series is of the same order (6 out of 23 cases). An additional case of thrombosis of the right pulmonary artery in a patient with mitral stenosis, thought to have occurred following cardiac catheterization, has recently been reported by Nightingale and Williams. A further factor favoring thrombosis is the tendency to peripheral venous thrombosis and pulmonary embolism from peripheral thrombosis in mitral disease. This has recently been stressed by Wood in cases with a low cardiac output and very high pulmonary vascular resistance. It is possible, however, that some of the organic occlusive lesions in the smaller arteries, which are not infrequently found in mitral stenosis, might be originally thrombotic or embolic in nature. Thrombosis of the small
pulmonary arteries, together with arteritis, was a feature in our case 6 (fig. 7).

Pulmonary Infarction in Association with Thrombosis of the Pulmonary Arteries

Pulmonary infarction is not an invariable feature in cases of thrombus of the large pulmonary arteries. Of 7 cases reported by Keating and co-workers,19 infarcts were present in only 2, in both of which previous pulmonary emboli had occurred. Infarcts were absent in Pou and Charr's 6 cases, and in 4 of Brenner's 8 cases. Fowler 29 found no infarction in a single case of thrombosis of the right and left main pulmonary arteries and explained its absence by the presence of anastomoses between the bronchial and pulmonary arteries. In this connection, the observation of Carroll 10 is of interest. At thoracotomy in 1 of his cases, the left pulmonary artery was found to be occluded, but aspiration distal to the block yielded oxygenated blood, which must have been due to retrograde bronchial flow. Gradual occlusion of the main arteries would allow time for these anastomoses to enlarge. The absence of infarction does not, however, permit distinction between primary and secondary thrombosis, since progressive thrombosis on the site of an embolus would create the same condition. A survey of the literature suggests that infarcts were more common in cases in which there was a definite source for, or history of, pulmonary embolism, than in those thought to have primary thrombosis of the pulmonary arteries.

In our own series, 14 of 23 cases had pulmonary infarcts that occurred with equal frequency in association with primary and secondary thrombosis. In 1 patient (case 5) the bronchial arteries were enlarged.

Summary

A series of 23 cases of thrombotic occlusion of the large pulmonary arteries, not due to acute massive embolism, has been presented. The clinical, radiologic, electrocardiographic, and pathologic features have been described, together with the results of special investigations in certain cases. It is concluded that thrombotic occlusion of the large pulmonary arteries can occur insidiously and present a characteristic picture of progressive right ventricular failure, the special features of which are outlined in detail. In most cases the thrombosis was secondary to embolism, but in 7 it was considered to have arisen primarily in the pulmonary artery. Six of the 23 cases had mitral stenosis, which appeared to favor primary thrombosis of the large pulmonary arteries. The etiology, diagnosis, differential diagnosis, and possible treatment have been discussed, and it is suggested that the diagnosis should be considered in any patient with obscure right ventricular failure, attacks of syncope, acute dyspnea, or chest pain; and especially if there have been episodes suggestive of pulmonary infarction. Patients with mitral stenosis who deteriorate for no apparent reason and show alteration in the second heart sound and decrease in the pulsation of the pulmonary arteries, should also be suspected of having developed a massive pulmonary artery thrombosis.

Acknowledgment

We are grateful to Dr. J. Edmunds for permission to use his notes on case 1 and to Dr. C. S. Treip for his postmortem findings. We wish to thank Professor J. McMichael for permission to publish cases 5 and 7 and Dr. J. Shillingford for his help and advice. Miss R. Klein, Mr. Knowlden, and Mr. Booker provided the photographs. The drawing was executed by Miss D. M. Baker.

Summario in Interlingua

Es presentate un serie de 23 casos de occlusion thrombotic del grande arterias pulmonar, non causate per acute embolismos massive. Es describite le aspectos clinic, radiologic, electrocardiographic, e pathologic, insimul con le resultatos de investigationes special interprente in certe casos. Le autores conclude que occlusion thrombotic del grande arterias pulmonar pote occurrer insidiosemente e que illo presenta alora un tableau caracteristic de progressive disfallimento dextero-ventricular. Le aspectos special de illo es delineate in detalio. In le majoritate del casos le thrombose eseva secundari a embolismo. In 7 casos, del altere latere, illo pareva haber occurrute primarimente in le arteria pulmonar. Sex de 23
casos hubiera stenosis mitral, lo que parecía soportar la interpretación de trombosis primaria en la grada arterias pulmonar. Es discutido el etiología, la diagnóstico, el diagnóstico diferencial, e las formas posibles de tratamiento. Es sugerido que el diagnóstico de occlusión trombótica de la grada arterias pulmonar debe esforzarse en considerar en omne paciente con obser disfarrimento dexteroventricular, atacces de syncope, dysnea, o dolores thoracic, especialmente en los presencia de un historia de episodios que sugieren infarimento pulmonar. El uso de un desvelimiento de masiva trombosis del grada arterias pulmonar es etiam justificata en pacientes con stenosis mitral que se deteriora sin causa apparente e qui mostra alteration en le secunde sono cardiac insinul con reduction del pulsiun in le arterias pulmonar.

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Massive Thrombotic Occlusion of the Large Pulmonary Arteries
K. P. BALL, J. F. GOODWIN and C. V. HARRISON

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