

# Pulmonary artery compression by haemorrhage from the aorta simulating pulmonary embolism

D. H. FRANKLIN and J. JACQUES

*The University Department of Medicine, The Royal Infirmary, Edinburgh and the  
Department of Pathology, Edinburgh University*

**Franklin, D. H., and Jacques, J. (1974). *Thorax*, 29, 142-144. Pulmonary artery compression by haemorrhage from the aorta simulating pulmonary embolism.** A case is presented in which pulmonary embolism was simulated by compression of the pulmonary artery by haematoma during an episode of acute bacterial endocarditis occurring 18 months after aortic valve replacement.

## CASE REPORT

A 38-year-old male clerk was admitted to hospital suffering from bacterial endocarditis. Seven years previously he had been treated for a similar episode due to *Streptococcus viridans*. Over the ensuing five years he had developed progressive aortic incompetence which ultimately required treatment by aortic valve

replacement with a fascia lata prosthesis. After his operation he remained very well, but three months before the present admission he had toothache for which he did not seek advice.

On admission he complained of fever, shivering, and stiffness in the back and joints. He was noted to have a pale, muddy complexion and petechial haemorrhages in the conjunctivae and optic fundi. A systolic murmur

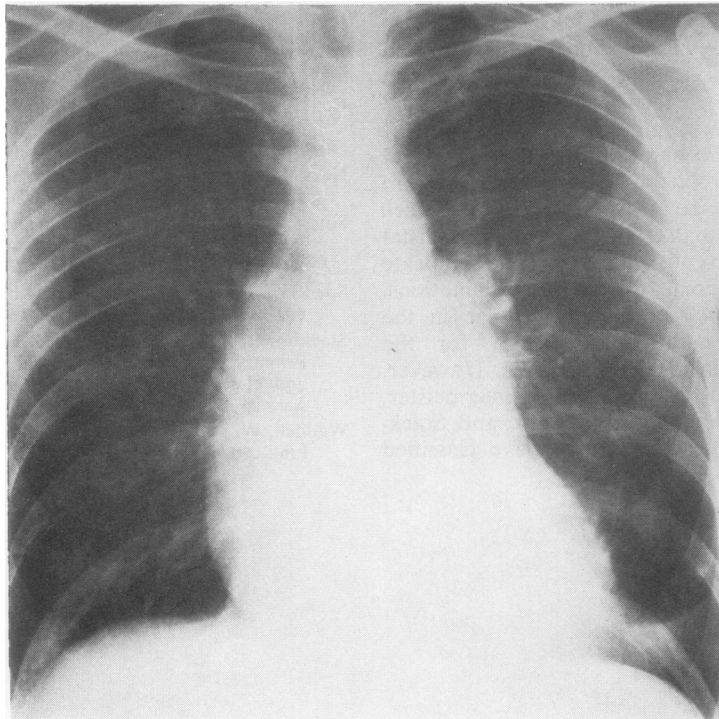


FIG. 1. Chest radiograph showing prominence of the left pulmonary artery.

audible at the cardiac apex and transmitted into the neck was accompanied by an early diastolic murmur down the left sternal edge which became progressively louder over the ensuing two weeks. Dental radiography revealed several areas of sepsis in the upper jaw. A diagnosis of bacterial endocarditis was based on these findings and five blood cultures of *Strep. viridans*. Treatment with penicillin, 8 mega-units daily, with probenecid and streptomycin, 1 g daily, produced some initial improvement. Ten days after admission he had an acute episode of dyspnoea associated with chest tightness while sitting on the commode. Clinical examination showed no signs of right heart failure. No murmurs were audible in the pulmonary area and the lung fields were clear on auscultation. A chest radiograph (Fig. 1) showed some prominence of the left pulmonary artery and the electrocardiogram showed T-wave inversion in right ventricular leads superimposed upon pre-existing first-degree heart block and left ventricular hypertrophy (Fig. 2). A diagnosis of pulmonary embolism was further supported by a macroalbumin aggregate lung scan which suggested deficient perfusion of the left lung (Fig. 3). Treatment with a heparin infusion was begun and there was no clinical deterioration but after a further five days he collapsed suddenly and died.

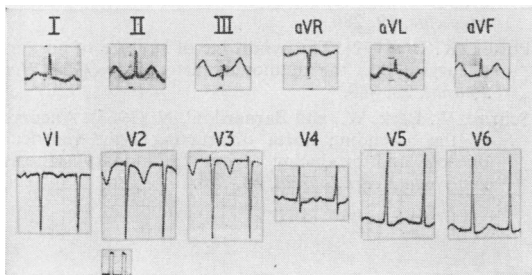


FIG. 2. ECG showing T-wave inversion in the right ventricular leads with left ventricular hypertrophy and first-degree heart block.

**PATHOLOGY** At necropsy, the main findings related to the heart and lungs. Each pleural cavity contained about 200 ml of straw-coloured fluid and there were loose fibrous adhesions around the left lung. The pericardial sac was largely obliterated by fibrous adhesions which surrounded the transverse sinus of the pericardium. The transverse sinus itself was greatly distended by organizing blood clot and more recent extensive haemorrhage into it had formed a 6 cm diameter haematoma which pushed the left pulmonary artery upwards and anteriorly, severely narrowing it. The left atrium was pushed posteriorly and compressed (Fig. 4). The haemorrhage arose from the aorta where a 2 mm diameter defect had developed in relation to one of the sutures holding the fascia lata valve in position. The upper aspect of the valve itself was unremarkable, but the ventricular surface

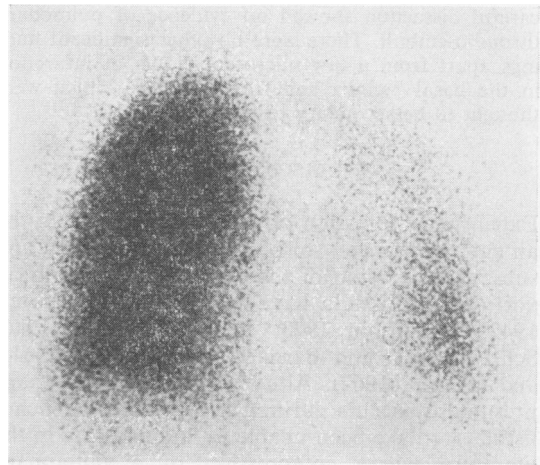


FIG. 3. Macroalbumin aggregate lung scan suggesting impaired perfusion of the left lung.

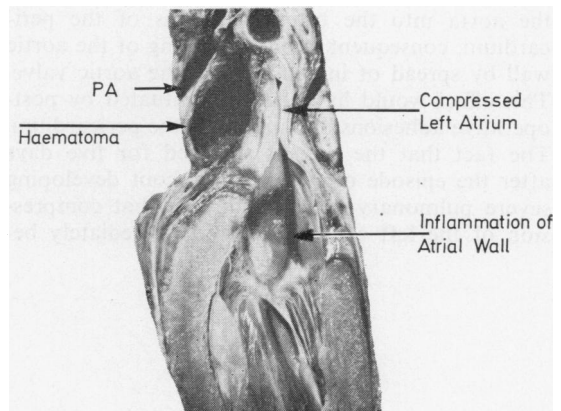


FIG. 4. Sagittal section through the left ventricle and left atrium showing inflammation of the atrial wall above the aortic cusp of the mitral valve, and a haematoma compressing the left atrium and distorting the left pulmonary artery.

of the cusps and the adjacent portion of the atrial wall were covered by friable bacterial vegetations. Inflammation could be seen extending from this area through the atrial wall above the mitral valve (Fig. 4). Histology from this region and the walls of the haematoma showed acute and chronic inflammation with patchy necrosis of atrial muscle fibres. Apart from moderate left ventricular hypertrophy and dilatation and mild right ventricular dilatation, no other cardiac lesion was seen. The lungs were congested and oedematous (right 850 g; left 700 g), but

careful dissection showed no evidence of pulmonary thrombo-emboli. There were no other significant findings apart from a few microscopic foci of infarction in the basal ganglia and frontal cortex, which were thought to be secondary to embolism.

#### DISCUSSION

Extrinsic compression of the pulmonary artery by an enlarging aneurysm of the ascending aorta and subsequent erosion of the vessel walls to form an aorto-arterial fistula have been described (Porter, 1942; Nicholson, 1943; Brill and Jones, 1946; Schrire, Beck, and Barnard, 1963; Gough, Gold, and Gibson, 1967). Although some haemorrhage presumably occurs during the formation of such a fistula, we have been unable to find any case in the literature where compression of a pulmonary artery has resulted directly from leakage from the aorta.

In the case described above, it would appear that the clinical features were produced by compression of the left pulmonary artery by haemorrhage from the aorta into the transverse sinus of the pericardium, consequent upon weakening of the aortic wall by spread of infection from the aortic valve. This effect would have been accentuated by post-operative adhesions binding down the pericardium. The fact that the patient survived for five days after the episode of dyspnoea without developing severe pulmonary oedema suggests that compression of the left atrium occurred immediately be-

fore death, probably as the result of further haemorrhage into this confined space. This theory is supported by the presence of organizing clot together with evidence of recent haemorrhage in the haematoma found at necropsy.

This case is presented to illustrate an unusual mechanism of pulmonary artery compression which may closely simulate pulmonary embolism.

We are indebted to Dr. R. M. Marquis and Dr. D. G. Julian for permission to publish details of the case and to Dr. M. D. Sumerling of the Department of Diagnostic Radiology, The Royal Infirmary, Edinburgh, for providing the lung scan.

#### REFERENCES

- Brill, I. C. and Jones, R. S. (1946). The syndrome of compression of the pulmonary artery by a syphilitic aortic aneurysm with or without arterio-arterial communication. *Annals of Internal Medicine*, **24**, 111.
- Gough, J. H., Gold, R. G., and Gibson, R. V. (1967). Acquired pulmonary stenosis and pulmonary artery compression. *Thorax*, **22**, 358.
- Nicholson, R. E. (1943). Syndrome of rupture of an aortic aneurysm into the pulmonary artery; review of the literature with report of two cases. *Annals of Internal Medicine*, **19**, 286.
- Porter, W. B. (1942). The syndrome of rupture of an aortic aneurysm into the pulmonary artery. *American Heart Journal*, **23**, 468.
- Schrire, V., Beck, W., and Barnard, C. N. (1963). Aneurysm of the ascending aorta obstructing right ventricular outflow and producing severe "pulmonary stenosis". *American Heart Journal*, **65**, 396.