Paradoxical embolism associated with patent foramen ovale

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Summary
Two patients are described in whom paradoxical embolism was diagnosed during life. One patient with cerebral embolism died and the other, with peripheral embolization, survived after treatment with streptokinase. Thrombolytic therapy has not previously been described in the treatment of patients with paradoxical embolism.

Case 2
The second patient was a 46-year-old woman who had a hysterectomy for fibroids. Three days postoperatively she experienced the sudden onset of dyspnœa which progressed over the next week. On the day of her re-admission to hospital, she suddenly developed numbness and coldness of the lower limbs associated with tingling and weakness, which immediately followed a bout of coughing. On admission to hospital she was pale and dyspnoeic with a pulse of 120/min and a BP of 190/90 mmHg. The JVP was elevated and there was a gallop rhythm but no murmurs. Pulses were absent from the middle of the abdomen downwards and the legs were cold. The ECG showed S1, Q3, T3 changes and the blood gases Po2 of 8:1 kPa (62:3 mmHg) Pco2 of 3:4 kPa (26:2 mmHg) and pH of 7:52 on 28% oxygen. The chest X-ray was normal. Pulmonary embolism and paradoxical embolism of her lower abdominal aorta were diagnosed and she was treated with streptokinase. Within 24 hr her dyspnoea lessened and the lower limb pulses reappeared. Cardiac catheterization carried out 3 months later confirmed the presence of a patent foramen ovale.

Discussion
Post-mortem evidence (Thompson and Evans, 1930) suggests that up to 6% of the population may have a sufficiently large foramen ovale to transmit a paradoxical embolus. Both of these patients confirm the association, evident from previously reported cases (Thompson and Evans, 1930; Padula and Camishion, 1968; Meister et al., 1972; Cheng, 1976; Laughlin and Mandal, 1977), between pulmonary embolism and paradoxical embolization when the cardiac lesion is a patent foramen ovale.
This association is probably due to increased right atrial pressure secondary to pulmonary embolism which leads to opening of the patent foramen ovale. Some of those earlier reports (Meister et al., 1972; Cheng, 1976; Laughlin and Mandal, 1977) suggest that the treatment of paradoxical embolism should be immediate ligation or plication of the inferior vena cava to prevent further emboli. However, this recommendation has always been based on small series of patients with marked heterogeneity and has never been compared with medical treatment within the same series. Caval interruption is theoretically attractive but in a series of patients with pulmonary embolism who were treated in this fashion there was a 4% peri-operative mortality with a 2-6% incidence of recurrent pulmonary embolism (Nabseth and Moran, 1965). This compares with a 2-3% mortality from recurrent pulmonary embolism in a series of 126 patients, reported in 2 papers (Hall, Sutton and Kerr, 1977; Sutton, Hall and Kerr, 1977), who were treated with streptokinase, heparin or pulmonary embolectomy with subsequent warfarin. If patients survive the acute event of pulmonary embolism, their chance of long-term survival diminishes markedly in the presence of pre-existing cardio-respiratory disease (Moran, Criscitiello and Callow, 1969; Paraskos et al., 1973) irrespective of the initial form of treatment.

The use of streptokinase in peripheral paradoxical embolism is logical in attempting to lyse clot in deep veins, pulmonary artery and aorta but has not been previously described. However, such peripheral embolization is unusual and paradoxical embolism more commonly affects the cerebral vessels (Laughlin and Mandal, 1977). In this situation, streptokinase might be dangerous with an increased risk of haemorrhage into the infarcted cerebrum.

Anticoagulation of patients with cerebral embolism has been much debated, but the use of heparin and warfarin is thought to be safe so long as routine precautions are taken (Marshall, 1976). In a recent review (Easton and Sherman, 1980), it was suggested that any patient with a cerebral embolism from a cardiac source should be anticoagulated immediately provided that there was no blood in the CSF or haematoma on CT brain scan.

Patients with paradoxical embolism suffer the same risks as those with a cardiac source of embolization and in both groups recurrent embolism is a major problem.

Accordingly, the management of all patients with paradoxical embolism should include immediate anticoagulation with heparin and subsequent oral anticoagulation. The use of thrombolytic therapy may be useful in the very occasional patient in whom major systemic arteries are obstructed but in whom there is no evidence of cerebral embolism. The value of various interruption operations, which in themselves may carry a significant mortality and morbidity remains unproved.

References


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