important to exercise considerable care in excluding a complete rupture of the oesophagus, a grave condition in which surgery usually offers the only hope of survival. Thus patients with a presumptive diagnosis of incomplete rupture must be repeatedly examined to exclude surgical emphysema and signs to the left side of the chest, and a plain x-ray film of the chest should be taken to further exclude these stigmata of complete rupture.

The radiological findings in the present case were sufficiently clear-cut to dispel any doubts regarding the diagnosis of an incomplete and intramural rupture of the oesophagus. The small extraluminal collection remained in close apposition to the intraluminal barium, and the unusual band-like vertical filling defects in the middle and lower thirds suggested a severe reaction process, or, just possibly, haematoma formation in the walls. In this connexion it is of interest that a fatal case of intramural oesophageal dissection starting at a mucosal laceration has recently been described by Thompson *et al.* (1967); x-ray studies in this patient showed a "double-barrelled" oesophagus due to detachment of the mucosa and submucosa from the muscular wall. Band-like vertical filling defects of the oesophagus have also been recorded by Brombart (1961, 1967), who considered them to be due to oesophagitis.

Gastro-oesophageal reflux noted in the present case suggests that an underlying oesophagitis may have facilitated the rupture (Mackler, 1952). In any event the precipitating factor in the rupture was the forced attempts at swallowing initiated by spasm or a bolus of food impacted in the lower oesophagus. Though severe vomiting is the most common cause of complete rupture, occasional cases of the latter may occur as a result of forced swallowing of an impacted bolus of food (Truelove and Reynell, 1963; Conte, 1966).

The site of the complete rupture in the present case conforms to that in which clinical and experimental complete rupture of the oesophagus occurs. The fact that the incomplete rupture involved only the mucosal layer may be construed as being at variance with the experimental findings of Mackler (1952) and Lion-Cachet (1963). These workers, using different inflation and bursting techniques, found that the muscle layer of the oesophagus ruptures earlier than does

the mucosal layer. Lion-Cachet, however, noted that the musculature of the lower end of the oesophagus and the stomach have the capacity to distend beyond the limits of their respective mucosal linings.

Incomplete rupture of the oesophagus should not be confused with the Mallory-Weiss syndrome, a condition characterized by brisk haematemesis due to vomiting-induced mucosal tears of the cardia of the stomach (Mallory and Weiss, 1929). The long axes of these mucosal tears are in line with the oesophagus, and, rarely, they may extend from the cardia into the lower end of the oesophagus. Clinically the accent in this syndrome is on bleeding and not on pain.

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Right Atrial Myxoma with Right-to-left Interatrial Shunt and Polycythaemia

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This paper describes a case of right atrial myxoma in which the correct clinical diagnosis led to successful surgical removal and in which polycythaemia was associated with arterial desaturation due to a right-to-left shunt at atrial level.

CASE REPORT

The patient, a 45-year-old married woman, gave a history of progressive effort dyspnoea for 18 months, "tightness in the chest" on effort for six months, and anorexia and nausea for three months. There was no history of rheumatic fever. There had been one episode of near syncope without neurological sequelae three months before admission.

On examination she was afebrile and the positive physical signs were confined to the cardiovascular system. There was central cyanosis and the jugular venous pressure was raised to the angle of the jaw. The wave form of the venous pulse showed a giant "a" wave which was easily palpable; in fact, some observers mistook it for the carotid pulse. On auscultation a soft systolic murmur and a soft diastolic murmur with presystolic accentuation were heard.

These murmurs were maximal at the left sternal edge and their intensity increased on inspiration. There was no clinical evidence of pulmonary hypertension.

Investigations.—Blood: Hb 124% (18 g./100 ml.), R.B.C. 6,200,000/cu. mm., platelets 184,000/cu. mm., W.B.C. 9,000/cu. mm., E.S.R. 1 mm./1 hour (Westergren), total serum proteins 7.1 g./100 ml., albumin 4.1 g., globulin 3.0 g. Electrophoresis showed a slight increase in alpha-2 globulin fraction.

E.C.G.: sinus rhythm; P-R 0.16 sec.; P wave in standard leads I and III was tall and peaked, measuring 3 mm.

Chest x-ray examination showed normal heart size, no enlargement of right or left atrium, and lung fields normal.

In view of the short history of effort intolerance and one episode of near syncope, and the clinical features, a diagnosis of subacute tricuspid valve obstruction was made. It was thought that the obstruction was probably due to a tumour, ? right atrial myxoma. Emergency admission into hospital was arranged and cardiac catheterization was performed the next day. There was gross increase in the right atrial pressure, primarily due to an "a" wave measuring 21 mm. Hg. The right atrial systolic pressure was higher than the right ventricular systolic pressure, and the pressure in the right atrium exceeded that in the right ventricle at all times except during peak ventricular systole (Fig. 1). The right ventricular, pulmonary arterial, and indirect left atrial (wedge) pressures were normal. There was systemic arterial desaturation (81%) and the cardiac index was 1.6 l./min./sq. m. Angiocardiography showed a large filling defect in the right atrium (Fig. 2). On cine-angiocardiography this filling defect was seen to prolapse through

the tricuspid valve during ventricular diastole. Contrast medium was also seen to pass from the right atrium into the left atrium, demonstrating a right-to-left shunt via an interatrial communication.

At operation the next day with total cardiopulmonary bypass a pear-shaped tumour measuring 7 by 5 cm. was removed from the right atrium. The tumour was attached by a short pedicle to the

FIG. 1.—Right atrial and ventricular pressure.

FIG. 2.—Angiocardiogram showing large filling defect in right atrium.

region of the foramen ovale and filled almost the whole of the right atrium, prolapsing through the tricuspid valve. There was a defect in the foramen ovale, and this was closed. During the operation the arterial oxygen saturation before the institution of cardiopulmonary bypass was 70%. After removal of the tumour and closure of the foramen ovale the arterial oxygen saturation rose to 98%. The patient made an uneventful recovery from the operation. Histological examination showed the tumour to be a myxoma. Two weeks after surgery the haemoglobin was 99% (14.4 g./100 ml.).

DISCUSSION

Morrissey *et al.* (1963) reviewed the 16 reported cases of right atrial myxoma diagnosed before death and added two cases from their own experience. Surgical removal was successful in 10 of these 18 cases. Since that time there have been further reports of the successful diagnosis and surgical removal of right atrial myxomata (Barlow *et al.*, 1962; Catt *et al.*, 1962; Sannerstedt *et al.*, 1962; Brandfonbrener *et al.*, 1963; Bross, 1965; Currey *et al.*, 1967; Sanyal *et al.*, 1967). Some degree of polycythaemia has been reported in five cases (Macoun, 1949; Bahnson and Newman, 1953; Alessandri *et al.*, 1960; Levinson and Kincaid, 1961; Siggillino *et al.*, 1963) and slight polycythaemia was also a feature of our case.

Siggillino *et al.* (1963) suggested that prolonged tissue anoxia due to a reduced systemic circulation may have resulted in polycythaemia, while Levinson and Kincaid (1961) speculated that impairment of pulmonary function from small pulmonary emboli might have been the stimulus, though there was no evidence of impaired pulmonary function in their case when studied postoperatively. A third possible explanation for polycythaemia in some cases might be the stimulus to red cell production resulting from arterial oxygen desaturation due to a right-to-left shunt through a patent foramen ovale. Such right-to-left shunting has been shown to be present in three of the reported cases (Hanlon, 1957; Coates and Drake, 1958; Sanyal *et al.*, 1967) with arterial oxygen saturations of 76%, 68%, and 56% respectively.

At the time of cardiac catheterization the present patient had an arterial oxygen saturation of 81%, and the passage of a small quantity of contrast medium from right atrium to left atrium demonstrated the presence of a right-to-left shunt at atrial level. At the time of surgery arterial oxygen saturation was 70% and rose to 98% immediately following removal of the myxoma with reduction in right atrial pressure and closure of the foramen ovale. Though cyanosis is often reported, it cannot have been due to intracardiac shunting in the two cases with polycythaemia reported by Bahnson and Newman (1953) and Siggillino *et al.* (1963), since at necropsy both these patients were reported to have a closed foramen ovale. Certainly where the foramen ovale is patent (20% of adults) right-to-left shunting is to be expected in view of the high right atrial pressures found in the presence of a right atrial myxoma. Cyanosis which varied with the patient's position was reported by Sanyal *et al.* (1967), and the present patient became markedly cyanosed on effort.

These cases of right atrial myxoma with a patent foramen ovale and reversed interatrial shunt must carry a risk of systemic embolism from blood clot or a fragment of the tumour becoming detached and entering the systemic circulation via the patent foramen ovale. Therefore in view of the risks of sudden death due to complete obstruction of the tricuspid valve, or to serious complications arising from systemic embolism, we consider that any case in which the clinical diagnosis of a right atrial myxoma is made should be treated as an emergency. Right heart catheterization with selective angiography should be performed as soon as possible, and if the diagnosis is confirmed, operation for removal of the tumour and closure of the patent foramen ovale should be carried out without delay by means of a total cardiopulmonary bypass.

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