Suggestions are made regarding possible lines along which progress might be made towards more rapid and accurate diagnosis with a resultant improvement in treatment and results.

As has been pointed out in the text, the bulk of the routine observations are the work of the midwifery staff, past and present, to whom we express our appreciation and thanks. Dr. A. S. Simpson, divisional medical officer for Ashton-under-Lyne, has been responsible for the statistical analyses, for which we are most grateful, and Dr. Ruth FitzGerald has been of the greatest help in the preparation and revision of the text.

REFERENCES

- Baird, D. (1950). *Combined Textbook of Obstetrics and Gynaecology*, 5th ed. Livingstone, Edinburgh.
 Canon, R., Guilhem, P., and Mayer, M. (1952). *Bull. Féd. Soc. Gynéc. Obstét. franc.*, 4 (No. 1, bis), 145.
 Clayton, S. G. (1953). *Proc. roy. Soc. Med.*, 46, 91.
 Clemetson, C. A. B. (1953). *Ibid.*, 46, 94.
 Lund, C. J. (1940). *Amer. J. Obstet. Gynec.*, 40, 946.
 — (1943). *Ibid.*, 45, 636.
 McKiddie, J. M. (1949). *J. Obstet. Gynaec. Brit. Emp.*, 56, 386.
 Stander, H. J. (1941). *Williams' Textbook of Obstetrics*, New York.
 Waters, R. M., and Harris, J. W. (1931). *Curr. Res. Anesth.*, 10, 59.
 Von Winckel, F. (1903). *Handbuch der Geburtshilfe*. Bergmann, Wiesbaden.

LEFT ATRIAL TUMOUR SIMULATING MITRAL STENOSIS

BY

G. PENRHYN JONES, M.D., M.R.C.P., D.C.H.

AND

D. G. JULIAN, M.A., M.D., M.R.C.P.

Primary cardiac tumours are quite uncommon, but are now being reported with increasing frequency. On occasion surgical removal may be possible when mechanical obstruction occurs, but pre-operative diagnosis is difficult and is usually reached only at exploratory cardiomy. The case here described manifested some of the features of rheumatic mitral stenosis, but valvular disease was not detected at operation. The diagnosis of a localized cardiac tumour, usually a myxoma, producing obstruction is now more than of academic interest, and similar cases may be encountered from time to time in thoracic surgical units, where cardiomy is now a routine procedure. Anomalous signs such as were found in this case may enable a pre-operative diagnosis to be made and so facilitate radical removal in a planned operation.

Case Report

A woman of 48 was first seen in April, 1953, when she complained of gradually progressive dyspnoea of five months' duration. She had previously been healthy, having had two uneventful pregnancies, and gave no history of rheumatic infection. She had noticed increasing pallor and had a little cough with occasional slight haemoptyses. Exertional dyspnoea was severe, with considerable orthopnoea, and several attacks suggestive of acute paroxysmal dyspnoea had occurred. Fatigue was also a prominent symptom. Apart from a vague constant left submammary ache, true cardiac ischaemic pain had occurred often. There had been no syncopal attacks and no embolic accidents.

She was a frail pallid woman, showing no cyanosis, jugular engorgement, or peripheral oedema. The heart rhythm was regular and the blood pressure 120/80. The apex beat was tapping in quality with a slight thrust, and there was a pronounced left parasternal heave. The mitral first sound was accentuated and a late mitral diastolic murmur (grade 2) was audible. This varied a little with posture, being best heard in the left lateral position after exercise.

No apical systolic murmur was heard. The basal second sound was loud, but no basal diastolic murmur or clear opening snap was audible. Apart from a few coarse moist sounds over the chest, the rest of the physical examination was negative.

Cardioscopy revealed considerable enlargement of the right ventricle with little or no atrial enlargement and no visible calcification. The lung fields showed evidence of severe pulmonary hypertension, with large pulmonary arteries, peripheral vascular attenuation, and prominent basal lines B of Kerley. The radiologist (Dr. E. L. Rubin) was of the opinion that the radiological appearances were unlike those of true mitral stenosis.

Cardiac catheterization was undertaken, and the mean pulmonary artery pressure was 80 mm. Hg, and the pulmonary capillary pressure 44 mm. Hg. There was no evidence of intracardiac shunt. The electrocardiogram showed right ventricular hypertrophy pattern in the unipolar chest leads.

With some reservations, owing to the several anomalous features of the case, the patient was submitted to cardiomy. This was undertaken by Mr. Ronald Edwards on January 5, 1954, when a pedunculated tumour was palpated in the left atrium, arising from the septum. This was acting in the manner of a ball-valve, plunging into the mitral orifice with each auricular systole. The tumour was friable and, in view of the danger of embolism, removal could not be undertaken; the chest was therefore closed. A more radical operation was to be attempted later under hypothermic anaesthesia, but the patient died of acute pulmonary oedema on the second post-operative day.

Necropsy.—Both lungs were oedematous, with some discrete areas of consolidation. On section the alveolar walls were thickened and the pulmonary vessels hypertrophied. Pigment-containing macrophages were present in many of the alveoli. An infarct, presumed to be a few days old, was present in the left kidney. The left thyroid lobe contained a small colloid adenoma. The right ventricle of the heart was hypertrophied and dilated, the wall being 7 mm. in thickness. The left atrium was almost completely filled with a tumour, 16 by 40 by 25 mm. in size, which was adherent to the atrial wall at the site of the

Drawing of the heart, displaying the atrial tumour entering the left ventricular cavity through the mitral orifice.

foramen ovale by a pedicle 15 mm. in diameter. The tumour projected longitudinally towards the mitral valve and the tip protruded through the valve orifice for 10 mm. The tumour was brownish in colour and resembled in many ways an ante-mortem thrombus, but also contained areas suggestive of myxomatous change. The mitral and other valves were normal.

Histology.—The tumour consisted largely of a rather structureless fibrillary basophilic material, having a mucinous appearance, in which were scattered groups and cords of rather rounded cells with eosinophilic cytoplasm. There were numerous blood-filled spaces present, many of which were lined by flattened endothelium-like cells, giving the growth in places an angiomatous appearance. A considerable amount of iron pigment was scattered throughout the tumour, particularly near its attachment. It did not invade the endothelium, but was attached to it along its surface, and in places faded imperceptibly into it. The appearances were those of the so-called pseudomyxoma of the heart.

Discussion

The benign myxoma is the commonest primary cardiac tumour, and is thought to rise from thrombosis in the atrium. Structurally it resembles the mass thrombus associated with mitral stenosis (Evans and Benson, 1948), but it may occur in the absence of valvular obstruction as in this case, and the actual mechanism of production is unknown. The site of attachment may be on either side of the atrial septum and almost invariably at the foramen ovale. When on the right side, the signs resemble those of superior vena caval obstruction, as in the case of Macoun (1949), and the clinical picture of mitral stenosis may be simulated when the tumour arises from the left side of the septum (Jensen, 1934; Bien and Ch'in, 1936; Hamilton-Paterson and Castleden, 1942; Thompson, 1944; Burnett and Davidson, 1945; Nanson and Milnes Walker, 1952).

The following points have been stressed by writers concerned with the diagnosis of this condition:

Short History of Symptoms, Followed by Inexorable Downward Progress and Failure to Respond Adequately to the Usual Lines of Treatment.—Symptoms had lasted only six weeks in one case (Houck and Bennett, 1930), two months in another (Allison and Susman, 1949), and twelve months in another (Fawcett and Ward, 1939). The tumour is presumably symptomless until it begins to engage in the A-V orifice, obstruction rapidly becoming severe, owing to the driving down of the tumour between the mitral cusps.

Pain.—This has been mentioned as a feature in most of the case histories described, both the common type of submammary ache and the less commonly encountered true anginal pain. But this symptom is relatively so common in rheumatic mitral disease that it can be of little diagnostic value.

Postural Fainting and Postural Dyspnoea.—Sudden syncopal attacks have often been referred to in case reports, but they have not occurred in all cases. Suspicion would be aroused, however, if postural fainting were a dominant symptom.

Murmurs Varying with Posture.—Several cases are reported to have shown this phenomenon, but it is such a common finding in ordinary mitral stenosis that little significance can be attached to it.

It would seem that a short history of symptoms, and fairly rapid deterioration, combined with absence of the classical radiological appearances of mitral stenosis, are the only helpful indications of the presence of a left atrial tumour.

Our thanks are due to Dr. W. Sutton and Dr. Ronald Ellis, to Mr. Ronald Edwards, and to Miss Duckworth for the drawing.

REFERENCES

- Allison, D. R., and Susman, W. (1949). *Lancet*, 2, 11.
 Bien, C. W., and Ch'in, K. Y. (1936). *Chin. med. J.* (Suppl. 1), p. 64.
 Burnett, W., and Davidson, J. L. (1945). *Brit. Heart J.*, 7, 180.
 Evans, W., and Benson, R. (1948). *Ibid.*, 10, 39.
 Fawcett, R. E. M., and Ward, E. M. (1939). *Ibid.*, 1, 249.
 Hamilton-Paterson, J. L., and Castleden, L. I. M. (1942). *Ibid.*, 4, 103.
 Houck, G. H., and Bennett, G. A. (1930). *Amer. Heart J.*, 5, 787.
 Jensen, C. R. (1934). *Northw. Med. (Seattle)*, 33, 394.
 Macoun, S. J. R. (1949). *Thorax*, 4, 39.
 Nanson, E. M., and Walker, R. M. (1952). *Ibid.*, 7, 263.
 Thompson, R. B. (1944). *Brit. Heart J.*, 6, 23.

Medical Memoranda

Perforation of Intestinal Tuberculous Ulcer

Perforation of a tuberculous intestinal ulcer is thought to be very rare. The report of a case by Douglas (1953) has stimulated me to add the following case to the few already recorded.

CASE REPORT

A man aged 25 was brought to me with a history of diffuse abdominal pain of four days' duration. The pain had started around the umbilicus and had then become diffuse. It had not increased in severity since it started. The patient had vomited several times on the first day. On examination he looked emaciated, with pale conjunctivae, and had a toxic appearance, a dry furred tongue, and oedema of both feet. His pulse was 125 and of poor volume. His abdomen was moderately distended, and there was a generalized tenderness, especially marked in the right iliac fossa. The abdomen was rigid, particularly in the right iliac fossa and on the right side. On screening the abdomen a large quantity of gas was noted under the right leaflet of the diaphragm. A perforation was diagnosed. It was labelled "? duodenal ulcer perforation, ? an ambulant typhoid with intestinal perforation" (such perforations being very common in India).

Operation.—On opening the abdomen thin watery and slightly purulent fluid welled out. A duodenal perforation was searched for and excluded. The distal part of the small gut was carefully examined and in its distal 2-3 feet (60-90 cm.) many peritoneal tubercles were found in groups, and one of the groups showed a tiny perforation. The perforation was closed in the usual way.

COMMENT

Although perforation of a tuberculous ulcer of the small intestine is thought to be very rare, Brown and Sampson (1926) say that it is not so very uncommon. They estimate the frequency of such perforations at 3.7%.

R. G. TALWALKAR, F.R.C.S.,
Bombay, India.

REFERENCES

- Brown, L., and Sampson, H. L. (1926). *Intestinal Tuberculosis*. Baillière, Tindall and Cox, London.
 Douglas, K. M. (1953). *British Medical Journal*, 2, 922.

Pectus Excavatum

The term "pectus excavatum" describes the funnel-shaped depression of the front of the chest caused by backward displacement of the lower sternum. In the past various aetiological factors were thought to be the cause of the deformity, but it is now attributed to congenital shortening of the anterior part of the diaphragm (Brodkin, 1949), the deformity becoming apparent as growth proceeds. If severe it may cause secondary mechanical effects on the respiratory and cardiovascular systems, but these are generally mild and most patients are free from organic symptoms. By contrast, functional disturbances often give rise to symptoms (*British Medical Journal*, 1952), and attempts to preclude them by operative correction of the deformity in childhood have been advocated in America by Dörner (1950), Lester (1950), and others, and have recently been reported in Great Britain by Chin and Adler (1954). In the following case the diagnosis of pectus excavatum was followed by the prolonged incapacitation of a symptomless patient.

CASE REPORT

At a routine medical examination a symptomless school-boy aged 12 was found to have a cardiac murmur and pectus excavatum. His family doctor confirmed these findings and referred him to a cardiologist, who believed the murmur was not of organic significance, but on detecting albuminuria he referred him for investigation to a general hospital, where a chest radiograph suggested tuberculosis, and he was