slow and imperfect after correction of the hypercalcaemia by cortisone. The present case report shows that, in spite of established renal calcinosis, marked and rapid improvement in renal function followed the use of cortisone. There was no change in the degree of renal calcinosis as determined by serial renal biopsy after one month of cortisone therapy, although renal function had returned to normal. The mechanism is therefore unlikely to be the removal of precipitated calcium from the kidney, although this may well be the end-result of continued steroid administration. We suggest that cortisone exerts its beneficial effect on the renal failure of hypercalcaemic sarcoidosis principally by correcting the hypercalcaemia itself.

Summary

A case is reported of sarcoidosis with hypercalcaemia, nephrocalcinosis, nephrolithiasis, and renal failure. Treatment with cortisone resulted in lowering of the serum calcium level to normal and improvement in renal function within one month, although serial renal biopsies showed no change in the degree of nephrocalcinosis during this time.

It is suggested that renal failure in hypercalcaemic sarcoidosis is not necessarily a reflection of irreversible nephron destruction by calcinosis, but that it may result from the high level of circulating calcium itself adversely affecting renal function. Also, the presence of nephrocalcinosis does not necessarily imply that the response to steroid therapy will be unsatisfactory.

We thank Dr. W. Freeborn, general medical superintendent of the Royal North Shore Hospital of Sydney, for allowing us to publish details of this case, and Dr. C. Graham for giving the histopathological report.

REFERENCES

- Anderson, J., Harper, C., Dent, C. H., and Philpot, G. R. (1954). *Lancet*, **2**, 720.
 Berger, K. W., and Relman, A. S. (1955). *New Engl. J. Med.*, **252**, 44.
 Davidson, C. N., Dennis, J. M., McNinch, E. R., Willson, J. K. V., and Brown, W. H. (1954). *Radiology*, **62**, 203.
 Dent, C. E., Flynn, F. V., and Nabarro, J. D. N. (1953). *Brit. med. J.*, **2**, 808.
 Elsom, K. A., Wood, F. C., and Ravdin, I. S. (1936). *Amer. J. med. Sci.*, **191**, 49.
 Gleckler, W. J. (1956). *Ann. intern. Med.*, **44**, 174.
 Henneman, P. H., Carroll, Evelyn L., and Dempsey, Eleanor, F. (1954). *J. clin. Invest.*, **33**, 941.
 — Dempsey, Eleanor F., Carroll, Evelyn L., and Albright, F. (1956). *Ibid.*, **35**, 1229.
 Klatskin, G., and Gordon, M. (1953). *Amer. J. Med.*, **15**, 484.
 Longcope, W. T., and Freiman, D. G. (1952). *Medicine (Baltimore)*, **31**, 1.
 Phillips, R. W., and Fitzpatrick, D. P. (1956). *New Engl. J. Med.*, **254**, 1216.
 Scholz, D. A., and Keating, F. R., jun. (1956). *Amer. J. Med.*, **21**, 75.
 — Power, M. H., and Dearing, W. H. (1957). *Proc. Mayo Clin.*, **32**, 182.
 Shulman, L. E., Shoenrich, E. H., and Harvey, A. McG. (1952). *Bull. Johns Hopk. Hosp.*, **91**, 371.

The term "biophysics" has hardly yet had time to acquire a fixed or generally accepted meaning. In consequence, the author of *Elements of Biophysics*, James E. Randall, Ph.D. (of Missouri University), explains that he wrote his book "to present to the student of biology and medicine the physical and quantitative concepts of basic importance to these fields." The subjects he discusses include the following: principles of measurement and statistical analysis; mechanics and kinetics; electricity, atomic structure, and radioactivity. The book has 333 pages and is published by Year Book Publishers for \$8.

COR TRIATRIATUM (STENOSIS OF THE COMMON PULMONARY VEIN)

SUCCESSFUL TREATMENT OF A CASE

BY

J. R. BELCHER, M.S., F.R.C.S.

Assistant Thoracic Surgeon

AND

WALTER SOMERVILLE, M.D., F.R.C.P.

Assistant Physician

Department of Cardiology, Middlesex Hospital, London

In the cor triatriatum the left atrium is divided by a septum into two chambers—postero-superior, which receives the pulmonary veins, and antero-inferior, which opens into the mitral valve and left ventricle. The septum contains one or more apertures which may be large or small, or it may be imperforate, in which case the infant is stillborn or dies in the first few months. Since the septum causes an obstruction at left atrial level, it can simulate the symptoms, radiological appearances, and haemodynamics of mitral stenosis. It is eminently suitable for surgical treatment, yet to date only three successful cases have been reported. A fourth is now presented.

Case Report

A woman aged 28 complained of progressive breathlessness, palpitation, and tiredness for four years. A "leaky valve" had been diagnosed when she was aged 12. Walking a few yards caused severe dyspnoea, but she complained little and was working as a secretary. Orthopnoea, haemoptysis, syncope, or chest pain was not present.

The rhythm was atrial fibrillation; venous pressure was raised to 4 cm. above the sternal angle, and there was an obvious systolic lift to the left of the sternum. The main auscultatory findings were a mitral pansystolic murmur

FIG. 1.—Electrocardiogram showing atrial fibrillation and right ventricular hypertrophy.

(grade 2) and a loud first sound at the mitral (grade 3) and tricuspid area (grade 3). The second sound was closely split, the pulmonary element being loud. There was no diastolic murmur and no opening snap. The blood pressure was 120/85. Other findings were normal. The electrocardiogram showed atrial fibrillation and a strong right ventricular hypertrophy pattern (Fig. 1). The heart was moderately increased in size (Fig. 2), the pulmonary artery prominent, and the left atrium slightly enlarged when viewed

in the right oblique position. The lungs showed pulmonary venous congestion with Kerley's lines at both bases. The provisional diagnosis was severe mitral stenosis. Cardiac catheterization was attempted twice, the second time in the semi-recumbent position, but on each occasion the patient

FIG. 2.—X-ray film showing cardiac enlargement, prominent pulmonary artery, and hilar vascular shadows. The aortic knob is small. Kerley's lines were present, but are not seen in this reproduction.

developed a rapid ventricular rate and pulmonary oedema before any observations were made.

At operation the anterior part of the left atrium was soft, but the posterior part, into which the pulmonary veins drained, was tense. The atrium was explored through the appendage; there was moderate mitral incompetence but no stenosis, and the valve cusps were soft and freely mobile. The tense intra-atrial septum was easily palpable and contained a calcified orifice 2 mm. across on the posterolateral aspect just in front of the pulmonary veins. The septum was divided with a Brock posterior commissure knife from side to side of the atrium. There was an immediate inrush of blood, and a fall in tension in the posterior chamber. No attempt was made to enlarge the orifice in the septum as it was so eccentrically placed.

Mean pressure readings taken at operation were as follows:

	Before		After
Pulmonary artery ..	66 mm. Hg	..	50 mm. Hg
Third atrium ..	40 "	..	8 "
Left atrium proper ..	8 "	..	8 "

Convalescence was normal. Four months later she had no symptoms. The venous pressure was now normal, but atrial fibrillation and the mitral systolic murmur persisted. Cardiac catheterization while she was lying flat gave the following results: pulmonary capillary venous (or left atrial) mean pressure, 5 mm. Hg; pulmonary artery, 35/15 (mean 24) mm. Hg; right ventricle, 35/-3 mm. Hg; cardiac output, 5.2 litres a minute; pulmonary vascular resistance, 4 units (320 dynes sec./cm.²).

Discussion

The first complete anatomical description of the cor triatriatum was given by Church (1868), although passing reference was made to it by Andral (1829). Twenty-nine cases have now been described, including the present one, 20 of these from the literature being well documented by Pedersen and Therkelsen (1954). Individual cases have been reported by Doxiadis and Emery (1953), Becu *et al.* (1955), Hartmann (1955), Lewis *et al.* (1956), Barrett and Hickie (1957), and Keith *et al.* (1958, 2 cases). Incomplete septa and fibrous bands running across the left atrium belong to a different and larger group and are not considered here. Three cases have been operated on successfully—a woman aged 21 (Vineberg and Gialloredo, 1956), a man aged 23 (Lewis *et al.*, 1956), and a girl aged 17 (Barrett and Hickie, 1957).

The clinical picture based on the present case and others of comparable severity described in the literature appears in a child or young adult. The chief complaint is breathlessness; paroxysmal dyspnoea is uncommon. The rhythm is sinus or atrial fibrillation. The diagnosis of severe mitral valve disease is suggested by a mitral pansystolic murmur, a loud mitral first sound, and signs of pulmonary hypertension with or without pulmonary incompetence, and is supported by the electrocardiogram and cardiac catheterization. This diagnosis is made uncertain by the absence of a mitral diastolic murmur and opening snap and little or no left atrial enlargement radiologically. The possibility of a left atrial myxoma is usually raised. In all the cases to date, however, the diagnosis of cor triatriatum has been made only after exploratory cardiomy.

The aperture in the septum varies in size from a few millimetres to 2 cm. across, and its edges may be calcified. It is usually centrally placed, but may be close to the periphery. Sometimes two or more apertures are present. The foramen ovale is in the superior chamber and may be open or closed. The left atrial appendage is part of the inferior chamber, where the pressure is low and is therefore not seen on the straight x-ray film of the heart. The mitral valve is usually normal, but may be hypoplastic with shortened chordae (Parsons, 1950) or deformed, allowing slight regurgitation as in Vineberg and Gialloredo's case and the present case. One instance of a tricuspid valve with two fused leaflets has been reported (Barnes and Finlay, 1952). The commonest associated congenital defects are a ductus arteriosus (three cases) and Fallot's tetralogy (two cases).

The embryological derivation of the cor triatriatum has been debated for more than 70 years. Kingston Fowler's (1882) contention that the anomalous septum was the septum primum and the aperture the interatrial ostium primum was preferred by Wardrop Griffith (1902) to his own thesis of failure of amalgamation of the common pulmonary vein in the left atrium. The latter theory has come to be generally accepted to-day, and certain authors feel so strongly about it that they call the condition "congenital stenosis of the common pulmonary vein" (Edwards *et al.*, 1951).

Summary

A woman aged 28 suspected of having mitral stenosis was found at operation to have a cor triatriatum. The

anomalous intra-atrial septum was successfully divided. The pressure in the third atrium fell immediately from 40 to 8 mm. Hg. This is the fourth case in the literature to be treated successfully by surgery.

We are indebted to Dr. K. P. Ball, Central Middlesex Hospital, who referred the patient to us.

REFERENCES

- Andral, G. (1829). *Précis d'anatomie pathologique*, 2, 313. Gabon, Paris.
Barnes, C. G., and Finlay, H. V. L. (1952). *Brit. Heart J.*, 14, 283.
Barrett, N. R., and Hickie, J. B. (1957). *Thorax*, 12, 24.
Becu, L. M., Tauxe, W. N., DuShane, J. W., and Edwards, J. E. (1955). *A.M.A. Arch. Path.*, 59, 463.
Church, W. S. (1868). *Trans. path. Soc. Lond.*, 19, 188.
Doxiadis, S. A., and Emery, J. L. (1953). *J. Pediat.*, 42, 87.
Edwards, J. E., DuShane, J. W., Alcott, D. L., and Burchell, H. B. (1951). *A.M.A. Arch. Path.*, 51, 454.
Fowler, J. K. (1882). *Trans. path. Soc. Lond.*, 33, 77.
Griffith, T. Wardrop (1902). *J. Anat. Physiol.*, 37, 255.
Hartman, H. A. (1955). *A.M.A. Arch. Path.*, 59, 706.
Keith, J. D., Rowe, R. D., and Vlad, P. (1958). *Heart Disease in Infancy and Childhood*, p. 352. Macmillan, New York.
Lewis, F. J., Varco, R. L., Taufic, M., and Niazi, S. A. (1956). *Surg. Gynec. Obstet.*, 102, 713.
Parsons, C. G. (1950). *Brit. Heart J.*, 12, 327.
Pedersen, A., and Therkelsen, F. (1954). *Amer. Heart J.*, 47, 676.
Vineberg, A., and Gialloretto, O. (1956). *Canad. med. Ass. J.*, 74, 719.

normal. Electrophoretic strip, moderate increase in γ -globulin; normal pattern 10 days later. Stools: no parasites or cysts.

An electrocardiogram on admission suggested a recent posterior infarction, with elevated ST segment in standard leads II and III, and Q waves in leads II, III, and AVF (on

Serial electrocardiograms

Medical Memoranda

Myocardial Infarction in a Young Adult

In the last two decades it has become apparent that coronary artery disease does occur in young adults. Newman (1946) published a series of 50 cases, the youngest aged 20. Poe (1947) showed that in 280 unselected necropsies on American Service personnel between the ages of 18 and 40, coronary thrombosis was the cause of death in 3.2%.

CASE REPORT

A well-developed athletic young man aged 19, on holiday from Germany, attended the casualty department on July 27, 1957, having been awakened early that morning by a constant gripping substernal pain, radiating to both shoulders and made worse by deep breathing. He had no cough, no sputum, no haemoptysis, and no dyspnoea. The pain lasted for two hours. The previous day he had felt unwell after strenuous exercise. His past history included several attacks of tonsillitis, the last occurring several weeks previously. A routine medical examination, including an electrocardiogram the year before, had been normal. His mother had angina pectoris, and his father had had a myocardial infarct.

On examination, his temperature was 99° F. (37.2° C.). His tonsils were enlarged and inflamed, and large tender tonsillar glands were palpable. His pulse rate was 64, and regular. His blood pressure was 110/50, and there were no cardiac murmurs or signs of cardiac failure. The other systems were normal.

Investigations.—Haemoglobin, 99%; W.B.C., 5,000 (polymorphs 51%, eosinophils 5%)→8,000 (polymorphs 67%, eosinophils 12%)→5,000 (polymorphs 54%, eosinophils 4%). E.S.R., 13→6 mm. (Wintrobe). W.R. and Kahn test negative. Chest x-ray examination normal. Serum transaminase, 29 units; two weeks later it was 12 units. C-reactive protein, negative. Blood cholesterol, 174 mg./100 ml. Throat swab showed *Streptococcus viridans*. 120 units of antistreptolysin per ml. No L.E. cells. Urine

inspiration). Chest leads were normal. Serial records showed that T-wave inversion developed in lead II four days after admission (see Fig.). This was not present in the final tracing. Elevation of the ST segment settled over six weeks. Q waves remained in leads II, III, and AVF.

The patient had a second attack of substernal pain lasting an hour, 48 hours later, and ran a low-grade pyrexia for two weeks. Otherwise his convalescence was uneventful. His blood pressure rose to 140/80. After 10 days he developed an apical systolic murmur and a split A 2, which were still present on his discharge on September 19.

COMMENT

This case shows various features which have been observed in previous series. Coronary artery disease in young people is very much commoner in men than in women (30:1), and occurs in a mesomorphic type of build (Gertler *et al.*, 1951). A family history of ischaemic heart disease can be demonstrated four times more frequently than in a control group. The attack is often related to effort, and in 50% of the cases there are premonitory feelings, which at the time or in retrospect can be attributed to myocardial ischaemia (Yater *et al.*, 1948). This patient complained that the pain increased on breathing deeply, although there was no pleural or pericardial rub, a symptom which was noted in 10% of the cases of Yater *et al.*

We thank Dr. H. V. Morlock, Dr. F. V. Gardner, and Dr. E. M. Spence for their help and encouragement, and Dr. H. V. Morlock for permission to report this case.

ANN L. HUNTER, M.B., B.S., B.Sc.,
House-physician.

JOHN N. INSALL, M.B., B.Chir.,
Casualty Officer.

Hampstead General Hospital, London.

REFERENCES

- Gertler, M. M., Garn, S. M., and White, P. D. (1951). *J. Amer. med. Ass.*, 147, 621.
Newman, M. (1946). *Lancet*, 2, 409.
Poe, W. D. (1947). *Amer. Heart J.*, 33, 76.
Yater, W. M., Traum, A. H., Brown, W. G., Fitzgerald, R. P., Geisler, M. A., and Wilcox, B. B. (1948). *Ibid.*, 36, 334.