PROCEEDINGS OF THE BRITISH CARDIAC SOCIETY

The THIRTY-FIRST ANNUAL GENERAL MEETING of the British Cardiac Society was held at the University, Western Bank, Sheffield, on Thursday, May 29, 1952. Chairman: J. W. BROWN. The Chairman took the Chair at 9.30 a.m.; 115 members and 13 visitors were present.

PRIVATE BUSINESS

1. The minutes of the last Annual General Meeting, having been published in the Journal (**13**, 569, 1951) were taken as read and confirmed.

2. The balance sheet for 1951–52 was presented, having been audited and found correct by H. Cookson and I. G. W. Hill. The credit balance on May 5 was £179 11*s.* 2*d.*

3. John Hay was elected and acclaimed as an Honorary Member of the Society.

4. W. M. Arnott and E. Wyn Jones were elected members of the Council in place of H. Cookson and I. G. W. Hill (terms of office expired).

5. The following Associate Members were elected as *Ordinary Members:*

R. E. Bonham-Carter	R. Kauntze	W. A. Oliver
E. M. Buzzard	G. A. Kiloh	D. A. Robertson
R. Daley	G. L. S. Konstam	R. W. D. Turner
D. H. Davies	A. Leatham	
Courtenay Evans	J. W. Litchfield	

6. The following *Associate Members* were elected for a further period of three years:

J. Benn	R. Kempthorne	D. Lewes
W. Stokes	A. J. Thomas	B. G. Wells

7. The following new *Associate Members* were elected and introduced:

R. I. S. Bayliss, London	J. D. Hay, Liverpool
A. J. V. Cameron, Glasgow	B. B. Hosford, Tunbridge Wells
J. A. Cosh, Bristol	J. F. Pantridge, Belfast
A. R. R. Cumming, Hull	E. G. Wade, Manchester
K. W. Donald, Birmingham	

8. The Society discussed the report on the reconstitution of the British Cardiac Society, which had been submitted to the Council by a sub-committee set up to consider an analysis of the questionnaire that had previously been sent out to all members. The sub-committee consisted of Sir John Parkinson, Dr. Evan Bedford, Dr. Maurice Campbell (Chairman), Dr. Shirley Smith, and Dr. Samuel Oram (Secretary). After a full discussion the following new rules were agreed to:

Previous Rule 5. Ordinary Members shall be Physicians or others whose primary interest is in the practice of Cardiology or in research in this or allied subjects.

Revised version. Ordinary Members shall be Physicians on the consultant staff of a hospital or others whose primary interest is in the practice of Cardiology or in research in this or allied subjects.

Previous Rule 6. Ordinary Members shall not exceed 75 in number and not more than 5 shall be elected annually. In addition there may be 10 Ordinary Members who may be elected for their interest in Cardiovascular Surgery.

Revised version. Ordinary and Associate Members shall not exceed 225 in number. This includes up to 15 Ordinary Members who may be elected for their interest in Cardiovascular Surgery.

Previous Rule 7. Extra-Ordinary Members shall not exceed 20 in number; they may be elected annually from among the senior Ordinary Members. For this purpose the Council may nominate

not more than 4 members to be proposed at the Annual Meeting. The affirmative vote of two-thirds of the members voting shall be required for election.

Revised version. Extra-Ordinary Members shall not exceed 25 in number; they may be elected annually from among the senior Ordinary Members. The affirmative vote of two-thirds of the members voting shall be required for election.

Previous Rule 9. Associate Members shall be elected from those engaged in research in cardiovascular problems or in the practice of Cardiology. Their number shall not exceed 50, and not more than 20 (including those re-elected) shall be elected in any year. The procedure for proposal and election of Associate Members shall be the same as for Ordinary Members.

Revised version. Associate Members shall be elected from those engaged in research in cardiovascular problems or in the practice of Cardiology. Their number, added to those of the Ordinary Members, shall not exceed 225, and not more than 15 new Associate Members shall be elected in any year.

Previous Rule 11. All Ordinary and Associate Members shall become subscribers to the Journal. (The subscription is 30*s.* per annum, reduced to 25*s.* for those who are members of the B.M.A.)

Revised version. All Ordinary and Associate Members shall become subscribers to the Journal. (The subscription is 42*s.* per annum.)

Previous Rule 21. The business of the Society shall be conducted by a Council which shall arrange the programme of each meeting. The Council shall consist of eight Ordinary Members and, in addition, the Chairman, the Chairman-Elect, the Editors of the Journal, and the Secretary. Two of the eight Ordinary Members shall retire annually in rotation and shall not be eligible for re-election for a period of one year. The Society shall elect two new members to serve in their place, and the Council may submit names for the vacancies.

Revised version. The business of the Society shall be conducted by a Council which shall arrange the programme of each meeting. The Council shall consist of eight Ordinary Members and, in addition, the Chairman, the Chairman-Elect, the Editor of the Journal or one of the Associate Editors delegated by the Editor to act as his deputy, and the Secretary. Two of the eight Ordinary Members shall retire annually in rotation and shall not be eligible for re-election for a period of one year. The Society shall elect two new members to serve in their place, and the Council may submit names for the vacancies.

Finally, an *additional rule* of the Society was agreed, namely, "There shall be a President of the Society who shall be elected by the Council."*

9. It was agreed to raise the *annual subscription* to two pounds for Ordinary Members and one pound, ten shillings for Associate Members. This alteration necessitates modifying Rule 22 of the Society which will now read: "The subscription shall be fixed by the Council and shall become payable on the first day of March. For Ordinary Members it is two pounds and for Associate Members, one pound, ten shillings. Failure to pay the subscription due within two years shall be considered equivalent to resignation."

SHORT COMMUNICATIONS

A STUDY OF CORONARY AND MYOCARDIAL LESIONS

P. J. D. SNOW (*introduced*), A. MORGAN JONES and K. DABER (*introduced*). The coronary injection technique of Schlesinger (*Amer. Heart J.*, **15**, 528, 1938), combined with the serial slice method of myocardial examination has been used to examine 180 hearts. The first 25 cases of coronary disease have been analysed, 42 occlusions and 59 infarcts being present. This contrasts with the results of Blumgart, Schlesinger, and Davis (*Amer. Heart J.*, **1**, 19, 1940), who in 30 cases found 53 main-vessel occlusions and only 16 infarcts. Incomplete myocardial examination is the probable explanation for the small number of infarcts found.

In 10 cases a single occlusion resulted in an infarct that subsequently extended on one or more occasions without further occlusion. In another 3 cases a single occlusion was responsible for two anatomically separate infarcts. In all 13 cases the additional infarcts occurred in zones rendered ischæmic by the original occlusion. The period during which further infarction is most likely to occur is up to eight weeks.

Of 42 occlusions, all but 4 were followed by infarction, and in each of these 4, the occlusion was one of two in the same vessel.

All 18 old occlusions found had recanalized, and their functional significance is shown by the fact that re-occlusion in five was followed by fresh infarction in each case.

* The exact method of his election is being discussed further by the Council.

The rate of revascularization of infarcts was studied by the filling of the infarct with injection medium. This was complete in eight weeks, thus agreeing well with the traditional period of rest and with the time during which further infarction is liable to occur.

Electrocardiographic assessment of Posterior Cardiac Infarction

By Cornelio Papp and K. Shirley Smith. In a consecutive series of 100 patients with posterior cardiac infarction assessed according to severity of the attack, 22 per cent were slight, 21 per cent moderate, and 57 per cent severe. This was in contrast to anterior cardiac infarction where this incidence was 42 23, and 35 per cent respectively. Greater gravity in posterior infarction can be explained by the frequency of arrhythmias and by the tendency of some slight posterior infarctions to become severe, a development exceptional in slight anterior infarction.

In slight posterior cardiac infarction the following electrocardiographic patterns were shown: absent pathological Q waves in about half the cases; R–T and T changes of the subacute type, e.g. bowed R–T with isoelectric take-off and deep inversion of T; electrocardiographic restoration in about a quarter of the cases. The severe cases in contrast exhibited pathological Q waves in almost every instance, an acute pattern, e.g. high R–T take-off and monophasic T wave in the great majority, and arrhythmias in more than one-third of the cases. In the moderate group the incidence of these signs was transitional, except that no arrhythmias were observed. There was no mortality in the slight and moderate group during the first two months. The mortality in the severe group of treated and untreated cases was 33 per cent.

The diagnostic difficulties in slight posterior cardiac infarction were solved by effort test; IIIR and aVFR leads. Recent bipolar leads advised for posterior infarction proved disappointing. No diagnostic problems have arisen in the moderate and severe cases. The absence of secondary T wave changes in anterior chest leads proved an important point in the diagnosis of antero-posterior cardiac infaction.

In five patients slight cardiac infarction was followed by increasingly severe angina; two of these developed severe posterior cardiac infarction within a fortnight, and one of them died. Early anti-coagulant treatment delayed the severe attack by months in one, while in the other two it prevented it. All five showed the electrocardiographic signs of posterior subendocardial infarction consisting in sagging or flat R–T depression in leads II, III and VF. Effort test caused temporary subepicardial ischæmia and this result was considered an important indication for prompt anticoagulant treatment.

The Heparin-Retarded Coagulation Time in Coronary Occlusion

By A. A. F. Peel. To be published in full, *Brit. Heart J.*, 1953.

Ischæmic Heart Disease and Peripheral Occlusive Arterial Disease

By Lawson McDonald. This paper gives the results of a study made during the past two years of patients with occlusive arterial disease, either resulting in ischæmic heart disease or affecting the blood supply to the legs. An attempt is made to estimate how often the two conditions occur together.

Eighty-seven patients presenting with intermittent claudication due to occlusive arterial disease have been examined clinically, and also by arteriography, tonoscillography after exercise, cardioscopy, and unipolar lead electrocardiography. Nearly half of these patients had evidence of ischæmic heart disease. Fifty patients presenting with angina pectoris due to occlusive coronary arterial disease have also been examined; in about a quarter evidence was found of occlusive arterial disease affecting the blood supply to the legs.

The coincidence of ischæmic heart disease and occlusive arterial disease affecting the legs is shown to be high. Although the clinical association of these two conditions, manifested by angina pectoris and intermittent claudication, is well recognized, there is scanty information regarding the frequency of their coincidence. The findings in these cases are particularly analysed with regard to sex, blood pressure, the age of onset and the duration of symptoms; the three groups are compared. In deciding upon treatment, either medical or surgical, for these patients, the importance of a knowledge of the coexistence of the other condition is noted.

The Diagnosis of Phæochromocytoma

By W. S. Peart (*introduced*) and J. W. Litchfield. The various methods of diagnosis are considered with reference to their use in 5 patients. The common features of pallor, palpitations, headache, vomiting,

breathlessness, and hypertension are stressed, while attention is drawn to some rarer symptoms and signs, such as swelling of the thyroid and A-V dissociation. Although the hypertension is commonly paroxysmal, three cases with fluctuating sustained hypertension were seen. The type of case with maintained steady hypertension is probably rare. There were two cases with malignant hypertension as judged by retinal changes, though no fibrinoid arteriolar necroses were discovered in the suprarenals. Localization of the tumours when not palpable has been accomplished by repeated soft tissue radiographs or pyelography. The blood flow through the hand was low in all except one of these patients, even when the blood pressure was normal, and this suggests that the pressor substances were released continually. Pharmacological methods of diagnosis, involving the use of stimulating drugs like histamine or antagonist drugs like benzodioxane, have been misleading in cases reported elsewhere, and two false negative tests with benzodioxane occurred here. The best method was the estimation of the urinary excretion of adrenaline and nor-adrenaline described by Euler and Engel. An increased excretion was found in all our cases.

Rapid Methods of Assessing Heart Failure

By E. P. Sharpey-Schafer. Postural changes in peripheral blood flow and arterial pulse pressure showed conspicuous differences in normal subjects and subjects with heart failure. Similar differences in the circulation have been found during and following the Valsalva manœuvre and the mechanisms involved have been investigated. The Valsalva manœuvre may be used as a simple clinical test.

Cardiac Output in Man

By J. McMichael. A film of this was shown at the end of the morning session.

Clinical Value of the A Wave of the Jugular Pulse

By Paul Wood. A film of this was shown at the opening of the afternoon session.

Pulmonary Artery and Pulmonary Capillary Pressures in Mitral Stenosis, and their Response to Exercise

By F. Jackson. The resting mean pulmonary artery, pulmonary capillary, and right ventricular pressures were measured by means of saline manometer in 50 cases of mitral stenosis.

The relation between the pulmonary artery and capillary pressures was in simple proportion at the lower levels, but the arterial pressure rose disproportionately when the capillary pressure exceeded 30–35 cm. saline, as described by Dexter *et al.* (*J. Clin. Invest.*, **29**, 602, 1950) and by H. E. Holling (*Guy's Hosp. Gaz.*, **65**, 271, 1951). The mean pulmonary artery and right ventricular pressures showed a linear relationship except at high levels. Clinical estimation of the severity of pulmonary hypertension based on the symptoms, signs, and radiological and electrocardiographic findings correlated reasonably well with the measured pressures.

In 9 patients re-catheterized between 3 and 12 months after valvulotomy the resting pulmonary artery and pulmonary capillary pressures were lower than before operation. In very severe cases, however, the artery pressure did not fall commensurately with the capillary pressure, possibly because of secondary vascular changes in the lungs.

Exercise produced a substantial rise in both pulmonary artery and capillary pressures followed by a slow fall to the resting level, even in the slighter grades of stenosis, compared with a small rise and rapid fall in controls without pulmonary hypertension. The actual increase in pressure was not a reliable index of the degree of stenosis, and the rate of the subsequent fall seemed to be a better guide.

Comparison with the artery and capillary pressures in pulmonary hypertension from other causes shows that the resting *pulmonary capillary* pressure relates more closely to the degree of mitral stenosis than does the *pulmonary artery* pressure, and it probably affords a better measure of the success of operation.

Observations on Pulmonary Circulatory Physiology in Mitral Valve Disease

By Geoffrey Wade, Lars Werkö, and Harald Eliasch (*introduced by Crighton Bramwell*). Using the basic technique of cardiac catheterization 26 cases of mitral disease were studied at rest and after a period of graded work. The measurements made in each case were the cardiac output, the pulmonary

arterial and capillary pressures, the pulmonary arteriolar resistance, and the cardio-pulmonary blood volume.

Results. It was found that there was an inverse relation between the cardiac output and the pulmonary capillary pressure and that a rise in this pressure on effort was generally accompanied by an increase in the pulmonary blood volume. A similar inverse relation existed between the output and the pulmonary arterial pressure but this was curvilinear owing to the frequent occurrence, in the more severe cases, of an increase in the degree of pulmonary arteriolar resistance leading to higher pulmonary arterial pressures. In these " high resistance " cases there was a smaller increase in the pulmonary blood volume in relation to the degree of pulmonary arterial pressure rise. The effect of high arteriolar resistance, which by shielding the capillaries from the output of the right ventricle may act as a useful barrier, is demonstrated. The modifying effects of the development of right heart failure on the pressure-flow pattern is also pointed out.

The nature of the increase in pulmonary resistance is discussed and its rapid reversability after successful mitral valvulotomy is shown.

The Oesophageal Pulse in the Diagnosis of Mitral Incompetence

By Max Zoob (*introduced by Frances Gardner*). Incompetence of the mitral valve is expected to produce abnormal filling of the left auricle and consequently abnormal movements of the contiguous portion of the œsophagus.

A study of the œsophageal pulse has, therefore, been made in the hope of facilitating the diagnosis of mitral incompetence. A duodenal tube was adapted to permit the simultaneous registration of the œsophageal pulse and electrocardiogram. The tube could be passed through the nose with little disturbance to the patient. The position of the recording tip—with reference to the left auricle—was determined from the contour of the P wave of the œsophageal electrocardiogram as well as by fluoroscopy. A conventional phonocardiogram was recorded simultaneously as an additional reference tracing.

Thirty-five control subjects in whom there was no suspicion of mitral valve disease were studied. These included patients with hypertensive heart disease or aortic valve disease and normal subjects. All showed essentially similar records. At auricular levels, a, c, and v waves were found analogous to those of jugular phlebogram. The summit of the v wave never preceded the second heart sound. Nine subjects thought to have innocent apical systolic murmurs showed similar curves.

Six patients thought to have pure mitral incompetence were studied. All showed striking abnormalities of the œsophageal pulse at auricular levels. The upstroke of the v wave was abnormally abrupt and its summit occurred before the second heart sound. This curve was thought to be due to abnormal auricular filling consequent upon mitral incompetence.

Fourteen of thirty-nine patients with mitral stenosis showed somewhat similar abnormalities. As, however, the curves were not identical with those found in pure mitral incompetence and as many of the patients showed no clinical or radiological evidence of incompetence, other modes of production of such abnormal curves are considered.

It is therefore concluded, that although an abnormal auricular filling wave cannot at present be used as evidence of incompetence in cases of mitral stenosis, it nevertheless provides evidence of mitral valve disease in subjects without diastolic murmurs.

Valvulotomy in Congenital Mitral Stenosis

By A. L. d'Abreu and Clifford Parsons. Congenital mitral stenosis is of two types. The first probably results from unequal division of the common atrio-ventricular canal and the second is due to an abnormality, possibly endocarditis, occurring later in fœtal life. Surgery would be impossible in the first type as there is always hypoplasia of the left ventricle and aorta. In the second variety hypoplasia is not necessarily severe, myocardial involvement is slight and obstruction at the thickened, narrow mitral valve is the essential cause of disability and invites surgical relief.

Our patient was referred because of repeated attacks of bronchitis, pneumonia, and congestive heart failure; she had had no illness suggestive of acute rheumatism. Her eldest brother had a patent ductus arteriosus but no member of the family had had rheumatic fever. Her mother's pregnancy had been normal. The new born infant was said to have signs of congenital heart disease and when we first examined her at the age of three and a half she had classical mitral stenosis. Investigations showed large P waves and right ventricular preponderance; congested vessels, slight general cardiac enlargement, but not particularly the left auricle on fluoroscopy; a patent ductus arteriosus with reversed flow on angiocardio-

graphy; and evidence of an intact septum, and raised pressure and oxygen content in the pulmonary artery on cardiac catheterization.

The child's condition deteriorated in the next year. With increasing frequency her feet were observed to be bluer than her hands.

At the age of four and a half her thorax was explored. Temporary occlusion of the ductus did not abolish the apical murmurs nor reduce the pulmonary artery pressure (145 cm. of saline). The distended left auricle was opened and the mitral valve felt as a hard fibrotic diaphragm, with a central aperture but no commissures. With great difficulty it was dilated until the index finger passed easily, but splitting of the valve could not be achieved. After operation there was temporary improvement, but within two weeks pulmonary hypertension was again sufficient to cause persistent slight cyanosis of the lower half of the body.

It is concluded that, owing to the nature of the valve deformity in congenital mitral stenosis, digital dilatation is unlikely to relieve the obstruction.

The Anatomy of Pulmonary Stenosis

By J. W. A. Duckworth (*introduced*) and R. M. Marquis. The developmental changes occurring in the bulbo-ventricular region of the human heart from the fifth to the eighth week of intra-uterine life are compared with the anatomical features found in congenital malformations of the heart in order to show the probable time of action of the factors that result in such defects.

The relationship of these defects to dextroposition of the aorta, pulmonary stenosis, and patent inter-ventricular foramen is discussed.

The principal sites of the stenosis included under the general heading of pulmonary stenosis are (1) the infundibulo-ventricular junction, (2) the infundibulum, (3) the pulmonary valves, and (4) the trunk of the pulmonary artery. More than one type may occur in the same patient and they may or may not be associated with other cardiac abnormalities.

Once stenosis has been established the resulting changes in the anatomy of the adult heart are noted and shown to depend chiefly on the degree of stenosis and the presence or absence of other cardiac abnormalities.

Patent Ductus Arteriosus with Pulmonary Hypertension

By J. A. Cosh. Three patients with patent ductus arteriosus are described in whom the pulmonary artery pressure was approximately equal to the aortic pressure. None had a continuous murmur, but the diagnosis in all was proved by the passage of a cardiac catheter along the ductus into the aorta. In two, the usual blood flow appeared to be from pulmonary artery into aorta. In the third, the flow was from aorta into pulmonary artery, but there was in addition coarctation of the aorta; this was confirmed by thoracotomy with lung biopsy. In none was the ductus ligated.

The clinical diagnosis and the relationship to simple patency of the ductus arteriosus are discussed.

Effort Syncope in Primary Pulmonary Hypertension and Fallot's Tetralogy

By Shiela Howarth and J. B. Lowe. Blood pressure records have been obtained in cases of primary pulmonary hypertension and Fallot's tetralogy, in which exercise produced loss of consciousness. The fall in systemic blood pressure leading to syncope occurred after exercise and was gradual and not precipitous. In one case of Fallot's tetralogy, loss of consciousness occurred five minutes after the end of exercise. Sinus bradycardia occurred when the blood pressure had fallen to low levels.

One case of primary pulmonary hypertension was exercised during cardiac catheterization and lost consciousness while lying supine on the couch. Pulmonary arterial pressure and blood pressure rose during feet exercise. At the end of exercise, the systemic blood pressure fell, the pulse pressure in the right ventricle decreased, and the diastolic pressure in the ventricle rose. These results are compatible with acute failure of the right ventricle from increased stress.