PROCEEDINGS OF THE BRITISH CARDIAC SOCIETY

The Thirty-Second Annual General Meeting of the British Cardiac Society was held at the Royal Victoria Infirmary, Newcastle-upon-Tyne, on Thursday, May 21, 1953. Chairman: Sir William Hume. The Chairman took the Chair at 9.30 a.m.; 110 members and 24 visitors were present.

PRIVATE BUSINESS

1. The minutes of the last Annual General Meeting, having been published in the Journal (14, 541, 1952) were taken as read and confirmed.

2. The balance sheet for 1952–53 was presented, having been audited and found correct by J. L. Lovibond and J. H. Wright. The credit balance on May 5, 1953, was £378 11s. 11d.

3. Professor Gustav Nylin was elected and acclaimed as an Honorary Member of the Society.

4. T. H. Crozier and G. W. Hayward were elected members of the Council in place of J. L. Lovibond and C. Bruce Perry (terms of office expired).

5. The following Associate Members were elected as Ordinary Members.
   - G. Aitken
   - D. R. Allison
   - H. A. Dewar
   - J. F. Goodwin
   - T. E. Gumpert
   - R. Hartley
   - H. E. Holling
   - F. Jackson
   - J. Macpherson
   - A. J. Thomas
   - A. J. Wilson

6. The following Associate Members were elected for a further period of three years.
   - W. H. R. Cook
   - R. Byron Evans
   - H. E. S. Pearson

7. The following new Associate Members were elected and introduced.
   - C. D. Anderson (Glasgow)
   - R. E. Benson (London)
   - C. E. Davies (Sheffield)
   - R. Hudson (London)
   - D. H. Makinson (Bangor)
   - E. Lawson McDonald (London)
   - H. Scarborough (Cardiff)
   - H. J. Wade (Manchester)
   - W. Whitaker (Sheffield)
   - A. G. W. Whitfield (Birmingham)
   - M. Zoob (London)

8. It was proposed and accepted that Rule 4 of the Society should read as follows.
   "There shall be a President of the Society who shall be nominated by the Council and elected by the Society. He shall be elected for two years and may be eligible for re-election for a further period of two years on the recommendation of the Council. He will be ex officio a member of the Council. He may preside over meetings of the Council but not at the Scientific Meetings of the Society, for which a local Chairman will be elected annually."

9. It was proposed and accepted that Rule 7 of the Society should read as follows.
   "Ordinary and Associate Members shall not exceed 225 in number. This includes up to 15 Ordinary or Associate Members who may be elected for their interest in cardiovascular surgery."

10. It was decided that the Autumn Meeting of the Society should be held in London in November, 1953, and that the programme should consist of short communications.

11. It was decided that the Annual General Meeting of the Society, in 1954, should be held at Cambridge.
12. The following Corresponding Members were proposed and elected.

**From Canada**

Dr. J. Hepburn (Toronto) 
Dr. F. A. L. Mathewson (Winnipeg) 
Dr. J. H. Palmer (Montreal) 
Dr. G. F. Strong (Vancouver)

**From Australasia**

Dr. C. R. Burns (Wellington) 
Dr. Clive Fitts (Melbourne) 
Dr. J. K. Maddox (Sydney) 
Dr. T. E. Lowe (Melbourne) 
Dr. G. Lendon (Adelaide) 
Professor F. H. Smirk (Dunedin)

**From South Africa**

Professor W. H. Craib (Port Elizabeth) 
Dr. H. L. Heimann (Johannesburg) 
Dr. M. Suzman (Johannesburg)

**SHORT COMMUNICATIONS**

**THE FREQUENCY OF DIFFERENT TYPES OF CONGENITAL HEART DISEASE**

By Maurice Campbell. From September, 1947, till the end of 1951, 1130 cases of congenital heart disease were studied and this deals with a preliminary analysis. It provided no evidence of the relative incidence of cyanotic and acyanotic disease because it is weighted by the former wanting operations, but in other particulars should be representative, except that there were relatively few children under three years of age. Three-fifths were cyanotic and two-fifths acyanotic (670 and 460). There are, no doubt, errors of diagnosis but as about 350 have had operations, and catheterization and angiocardiography have each been carried out in about 250, and the results of 120 necropsies are available, they should not be very numerous.

The acyanotic cases can be divided into five roughly equal groups—patent ductus arteriosus, 22 per cent; ventricular septal defect, 21 per cent; atrial septal defect, 18 per cent; coarctation of the aorta (7%) and aortic stenosis (11%) making 18 per cent; and pulmonary valvular stenosis, 16 per cent. The structure of the cyanotic group is quite different as 66 per cent had Fallot’s tetralogy, including about 9 per cent with pulmonary atresia; 8 per cent had transposition of the aorta and pulmonary trunk; 6 per cent had pulmonary valvular stenosis with a right to left shunt; 6 per cent had Eisenmenger’s complex; 5 per cent had tricuspid atresia; and the final 9 per cent had various anomalies including those with dextrocardia, levocardia, and Ebstein’s disease.

As expected, there was a considerable excess of males in coarctation (3 to 2) and in aortic stenosis (3 to 1), and of females in patent ductus arteriosus (2 to 1). There was no significant difference in the other acyanotic groups. Rather surprisingly this was true for atrial septal defect, but in those over 25 there was the expected preponderance of women (5 to 1).

In the cyanotic group, Fallot’s tetralogy and transposition showed male preponderance (3 to 2) and Eisenmenger’s complex female preponderance (2 to 1).

It is doubtful what conclusions can be drawn from the age incidence because children preponderate in all groups.

There is a striking contrast between the slight mortality in the acyanotic and the very heavy mortality in the cyanotic groups, quite apart from the operative deaths. In pulmonary atresia this amounted to nearly 40 per cent; in tricuspid atresia and in transposition of the main vessels to 30 per cent; in pulmonary valvular stenosis with a right to left shunt to 20 per cent; and even in Fallot’s tetralogy to 10 per cent. By contrast
the mortality was small but not insignificant in most of the acyanotic groups. It was highest in those with atrial septal defect but there were many older patients, some over 60 years of age. Simple pulmonary valvular stenosis was the group with most deaths in children.

**Patent Ductus Arteriosus: A Distinctive Syndrome associated with a Wide Short Duct**

By G. Aitken. In six of a series of 55 consecutive cases of patent ductus arteriosus a systolic thrill was palpable in the suprasternal notch, and a systolic murmur in the aortic area and in the neck vessels. These cases presented other features differentiating them from the majority. Disability was greater and all were moderately dyspnoeic on exertion; five gave a history of repeated serious respiratory illness, and two of intermittent oedema of the ankles; and haemoptysis, recurring cyanotic episodes, praecordial pain, and frequent extrasystoles had occurred in one or more of them.

All had a much wider pulse pressure than the mean value for the whole series, and all had distinct left ventricular enlargement demonstrable clinically and radiologically. Four showed the electrocardiographic changes of left ventricular hypertrophy and in two the pattern reverted to normal following operation. The characteristic basal murmur to the left of the sternum was long and harsh, occupying the whole of systole and followed by a more blowing diastolic murmur of varying length. In three cases a low-pitched apical diastolic murmur was audible. Pulmonary artery pressure was high in three of the four cases in which it was known: only in the youngest case did it approach normal. Operative closure by ligature or division of the duct was successfully performed in four cases with disappearance of the suprasternal thrill and aortic murmur, although in one case the murmur persisted for three months after operation. In all four cases, the duct was wide and short. In one the pulmonary artery pressure fell from 105 to 30 cm. saline following ligature of the duct.

At first, the presence of an aortic thrill and murmur suggested an associated aortic valvular or sub-valvular lesion. Their origin, however, is uncertain; they may arise from the patent duct or from relative aortic stenosis or distortion secondary to increased left ventricular output. We now believe that their presence in a case of patent ductus arteriosus indicates a wide short duct with, initially, a large shunt through the pulmonary circuit and that such cases are likely to become seriously incapacitated, and to develop pulmonary hypertension. Their early recognition and operative relief is important before irreversible cardiac or pulmonary vascular changes occur.

**The Clinical and Radiological Assessment of Pulmonary Arterial Pressure in Mitral Stenosis**

By L. G. Davis, R. E. Steiner, B. D. van Leuven (introduced), and J. F. Goodwin. Published in full, Brit. Heart J., 15, 393, 1953.

**Left Atrial and Pulmonary Venous Pressures in Mitral Stenosis**


**The Value of Electrocardiography in the Assessment of Patients with Mitral Valvular Disease**

By Hugh Fraser (introduced) and Richard Turner. A study has been made of the value of electrocardiography in the assessment of patients with mitral valvular disease with particular reference to 115 consecutive patients submitted to operation. In 100, mitral stenosis was the sole or predominant defect and valvotomy was carried out. In 15 mitral incompetence was predominant and most of them were subjected to a different procedure. The cardiographic findings are compared with the clinical, radiological, catheterization and operative observations.

Severe mitral incompetence may co-exist with normal electrocardiograms even though there is considerable left ventricular enlargement clinically and radiologically. In cases of mitral stenosis even minor cardiographic changes demonstrating the presence of left ventricular hypertrophy indicate that some important complication is present, and of these mitral incompetence is the most difficult to assess.

Criteria for the cardiographic diagnosis of right ventricular hypertrophy are reviewed and difficulties discussed. In our experience any cardiographic evidence of right ventricular hypertrophy has signified not
only that mitral stenosis was the predominant defect, but that it was severe in degree, that pulmonary hypertension was usually considerable, and also that the patient was likely to derive much benefit from valvulotomy.

However, cardiographic evidence of right ventricular hypertrophy was present in only 30 per cent of the patients in whom predominant mitral stenosis was confirmed at operation although presumptive evidence for right-sided enlargement was present in the majority. Similar observations have been made on post-mortem material. It is concluded that cardiographic changes only occur when right ventricular hypertrophy is considerable. Such evidence is found more often in patients with normal rhythm than in those with auricular fibrillation. Possible reasons for this are given.

Changes in the P waves and in axis deviation and the value of right-sided V leads are analyzed. Reference is made to changes suggesting myocardial ischaemia which were recorded during paroxysms of dyspnea, to the difficulties caused by old rheumatic myocardial fibrosis and by bi-ventricular hypertrophy, and to post-operative pericarditis patterns. In some cases serial electrocardiograms recorded objective evidence of deterioration before, or of improvement after operation.

### The Left Atrial Electrokymogram in Mitral Stenosis

By P. H. Davison and R. G. Epps (introduced). Movement of the posterior border of the left atrium was recorded electrokymographically in the right anterior oblique position in 29 patients with severe mitral valve disease and 10 control subjects. The patients with mitral disease were divided into three clinical classes: pure mitral stenosis, mitral stenosis with incompetence, and pure mitral incompetence. Confirmation of the clinical diagnosis was obtained in 23 by digital palpation of the valve at operation.

It was found possible to classify the electrokymographic records of atrial movement into four main types — undulatory, ventricular systolic expansion, ventricular systolic retraction, and absence of movement.

Undulatory movement of the auricular border showed a variable pattern from case to case, and was observed in the majority of the controls and in the minority of those with mitral disease.

Ventricular systolic expansion was shown electrokymographically by a single positive wave, flat-topped or peaked and occupying the whole of ventricular systole, which represented an abrupt sustained backward movement of the posterior atrial border. This pattern was recorded in three-quarters of the patients with pure mitral stenosis, in an equal proportion of those with mitral stenosis and incompetence, and in both cases of pure incompetence. It appeared to be unrelated to the type of mitral valvular lesion, the degree of right ventricular hypertrophy, the left atrial pressure, or the left atrial pulse pressure, but occurred with greatest frequency when mitral disease was complicated by auricular fibrillation.

It has been concluded that electrokymography of the left atrial border movement provides no help in differentiating between pure mitral stenosis and mitral stenosis with incompetence.

### The Left Auricular Appendage in Mitral Stenosis


### Circulation in Aortic Stenosis

By R. Gorlin, I. K. R. McMillan, M. B. Matthews, and W. E. Medd, (introduced by R. Daley). Nine patients with clinical evidence of aortic stenosis (eight of whom had an aortic diastolic murmur) were studied at rest by cardiac catheterization; observations were made in seven of these during exercise. (Only one patient had a mitral diastolic murmur; post mortem the mitral valve admitted two fingers and was competent). Cardiac outputs were measured by the Fick method. Pulmonary artery, pulmonary capillary, right atrial and brachial artery pressures were recorded, both phasic and mean. Pulmonary capillary diastolic pressure was used as an index of left ventricular diastolic pressure. The left ventricle and stenosed aortic valve of two patients who subsequently came to necropsy were perfused in the pump system described by McMillan, (McMillan et al. (1952), Brit Heart J., 14, 42). The stenotic and regurgitant valve areas were measured from motion pictures taken during perfusion under simulated in vivo conditions.

Three observations were considered to be of importance.

1. Prolonged systolic upstroke, as compared with total systole, in the brachial artery tracing.
2. Increased amplitude of the "a" wave in the pulmonary capillary tracing.
3. Increased amplitude of the "a" wave in the right atrial tracing.
Pulmonary "capillary" diastolic pressure was normal at rest in four patients; in all it was abnormally high on exercise. In four patients the right atrial pressure was increased at rest; in all it rose on exercise. In the two patients* in whom the valve areas were measured total cardiac output and left ventricular systolic mean pressures were calculated from hydraulic equations. From the data, total work of the left ventricle and its relationship to diastolic filling pressure could be calculated. These data were presented.

A short film showed the movements of the aortic valves which were measured at necropsy.

CARDIAC CALCIFICATION

By D. Pyke and C. Symons (both introduced by J. McMichael). In a random survey of 400 men over the age of sixty, 27 cases of cardiac calcification were detected by X-ray screening. A film was shown composed of cineradiographs of calcified aortic and mitral valves, of calcified left and right coronary arteries, and of calcification of the pericardium, showing the position and movement of these structures during life. The patients with aortic and mitral valvular calcification had the clinical signs of valve disease, but the patients with coronary artery calcification showed neither clinical nor cardiographic signs of heart disease.

THE CARDIAC OUTPUT DURING EXERCISE AND RECOVERY IN NORMAL SUBJECTS AND IN PATIENTS WITH RHEUMATIC HEART DISEASE

By K. W. Donald, J. Bishop (introduced), and O. L. Wade. A technique whereby the cardiac output can be estimated from minute to minute during changing states is described. The behaviour of the cardiac output during exercise and recovery has been studied in normal subjects and in patients with rheumatic heart disease at different levels of work. The achievement or otherwise of a relatively steady state during short periods of exercise has been particularly investigated. The minute to minute changes caused by exercise in mixed venous and arterial blood saturation, in oxygen uptake, and in ventilation and ventilatory equivalent in different patients with mitral valvular disease are described and discussed.

THE PULMONARY EARLY SYSTOLIC CLICK

By Aubrey Leatham and Louis Vogelpoel (introduced). We have investigated 50 patients in whom we heard an early systolic sound in the pulmonary area. This sound was of click-like or snapping quality and louder in expiration; occurring soon after the first heart sound it gave the impression of wide splitting of the first sound but was louder in the second and third left intercostal spaces rather than the mitral and tricuspid areas. The presence of this sound in early systole was confirmed by phonocardiograms in every case.

Of the 50 patients showing this physical sign on auscultation, 44 had pulmonary hypertension, either primary (3) or associated with a septal defect (24), with transposition of the great vessels (1), or with mitral stenosis (16). All but three of these 44 cases had obvious enlargement of the pulmonary trunk. Of the remaining 6, 2 had septal defects with dilatation of the pulmonary trunk but without pulmonary hypertension, 2 had slight pulmonary valve stenosis with dilatation of the pulmonary trunk, one had a faint pulmonary diastolic murmur and slight dilatation of the pulmonary trunk, and in the single remaining patient there was no evidence of heart disease. We conclude that the pulmonary early systolic click is a sign of dilatation of the pulmonary trunk especially when due to pulmonary hypertension.

THE EFFECT OF EXERCISE IN HYPERTENSION AND THE MODIFICATION OF THIS EFFECT BY HEXAMETHONIUM

By P. B. S. Fowler and Guz (introduced by K. Shirley Smith). In hypertension, the effect of exercise on the blood pressure has not been elucidated. It has been assumed, but never proved, that sympathetic nervous cuts out the peaks of a fluctuating pressure even when there is little change in the resting figures.

It was, therefore, decided to study the effect of exercise on the blood pressure. Hypertensive patients have had their blood pressure recorded during and after exercise on an ergometer. The effect of exercise on the pressure is found to be modified by hexamethonium bromide. Similar observations have been made on patients who have had the operation of sympathectomy and on controls.

* The catheter findings in one patient were provided by the Guy's Hospital team to whom we are grateful for permission to use them.
MYOCARDIAL FIBROSIS OF UNKNOWN CAUSE

By W. G. A. Swan, and B. E. Tomlinson (introduced). Three cases of fibrosis of the myocardium of unexplained aetiology have come to autopsy in 1952. One died suddenly and there was no history of antecedent ill health. The remaining two were under observation for heart failure and were investigated before death. One of these patients had a father who died of unexplained heart failure; his electrocardiogram, taken in 1935, closely resembled that of his son taken in 1952. Otherwise there was no clear history of familial heart disease and none of Friedreich's ataxia.

All the three hearts examined showed severe and diffuse myocardial fibrosis without any inflammatory reaction. The appearances closely resembled those described by Evans in 1948. The coronary arteries were all healthy and there was no valvular disease. In the patients seen before death there were low blood pressures and general cardiac enlargement: the electrocardiograms showed low QRS voltage and low abnormal T waves. On the basis of similarity to these two patients, the diagnosis of myocardial fibrosis of unknown cause has been made tentatively in two other cases.

WOLFF-PARKINSON-WHITE SYNDROME WITH PAROXYSMAL VENTRICULAR TACHYCARDIA

By D. Robertson. Two cases of Wolff-Parkinson-White Syndrome with paroxysmal ventricular tachycardia are recorded. This is a rare combination.

One, a man, who was seen in an attack fifteen years ago, is now fit and well and has had no major attack during the past ten years. No structural heart disease is evident.

The other, a woman, was watched for four years while her attacks of paroxysmal tachycardia became progressively worse and more frequent; she eventually died, of ventricular fibrillation (in 1951). Partial post mortem was performed, including histology of the heart (by Prof. Hewer, at Bristol), and no accessory auriculo-ventricular muscular connection was apparent.

ARRHYTHMIAS FOLLOWING CARDIAC INFARCTION

By H. E. S. Pearson. In a series of 567 cases of cardiac infarction there were 202 instances of disturbance of rhythm in 144 patients. Of these 50 subjects showed more than one variety of arrhythmia and 7 more than two varieties.

The blood supplies to the sinu-atrial node, the A-V node, and the bundle are derived mainly from the larger circumflex vessel but the comparative rarity of severe ischemic damage to these structures gives clinical confirmation of the presence of important subsidiary channels and a rich anastomosis. Complete functional destruction has always corresponded with lesions of both coronary arteries and a high mortality. Auricular flutter has occurred 7 times in similar circumstances.

Auricular fibrillation and the tachycardias were common but showed no relationship to the site of the infarct. They occurred generally in the more severe case. Multiple, and especially multifocal ectopic beats were associated with a mortality that was disproportionate to the apparent initial severity of the lesion, 18 of the 78 patients dying suddenly and unexpectedly.

Disturbances of rhythm after infarction are readily influenced by drugs, but the changing nature of the underlying cellular pathology makes the response to drug treatment variable and a dangerous ventricular excitability is often present.

THE PLASMA LIPIDS IN CORONARY ARTERY DISEASE