Proceedings of the British Cardiac Society

The Autumn Meeting of the British Cardiac Society was held at the Royal College of Physicians on Friday, November 26, 1965. The President, Shirley Smith, took the Chair at 9.0 a.m. during Private Business before handing over to the Chairman, William Evans. 175 members and 75 visitors were present.

The second Thomas Lewis Lecture was given at 5.0 p.m. by Dr. Bernard Lown on "Cardioversion of Arrhythmias".

Private Business

1. The Minutes of the Annual General Meeting, having been published in the Journal (1965, 27, 942), were taken as read and confirmed.
2. The Treasurer reported that cash in the current account stood at £1,206, deposit account at £100, and investments at £529.
3. The Congress Fund held £1,121. 9. 4d. in the current account, £400 in the deposit account, and investments still stood at £732. 18. 4d.
4. £200 was ear-marked for expenses of members attending the World Congress in New Delhi in 1966.
5. The Thomas Lewis Lecture Fund held £18 7s. 5d. in current account with investments at £1,173. 10s. 2d.
6. The Secretary reported that the Annual General Meeting would be held on April 14, 1966 in Cambridge under the Chairmanship of Cole.
7. The V World Congress of Cardiology is to be held in New Delhi from October 30 to November 5, 1966.
8. The Autumn Meeting of the Society will be held at the Royal College of Physicians on December 1-2, 1966. The Dinner will be held at the end of the first day.
9. The Annual General Meeting in 1967 will be held in Belfast on May 18.
10. The European Congress of Cardiology will be held in Athens in 1968.

After the Scientific Meeting the Society dined together at the Royal College of Physicians with Shirley Smith in the Chair. Shirley Smith proposed the health of the guests who included Sir Charles Dodds, Dr. and Mrs. Bernard Lown, and H. G. Lazell, Esq. William Evans replied.

Computers in Cardiovascular Investigation

By S. H. Taylor, A. Macdonald, R. Sapru (last two introduced), and K. W. Donald

The facility with which circulatory measurements may be made is imposing considerable restriction on the design of circulatory investigations due to the problems of measurement of the data involved. A study was, therefore, designed to explore the practical feasibility of analysing cardiovascular measurements with the aid of a transducer-computer link and digital computer.

In addition the accuracy of the three small proprietary cardiac output computers currently available has been assessed and the problems associated with their operation evaluated.

The combination of these small on-line cardiac output monitors in association with off-line data storage and analysis by digital computation offers important advantages not only in routine research cardiovascular investigations but in the monitoring of patients acutely ill with circulatory disorders.

Measurement of Right Ventricular End-systolic and End-diastolic Volumes by a Thermodilution Technique

By R. Balcon (introduced by S. Oram)

This communication is a preliminary account of experience with a new method of measuring ventricular volumes.

The history and theory of indicator dilution methods for the assessment of ventricular volumes is reviewed. The application of a thermodilution technique is described in the investigation of various cardiac abnormalities. An analysis of the findings obtained in these patients is given, and an attempt made to correlate the changes with other parameters of ventricular function such as end-diastolic pressure and stroke output.

An account is given of some of the difficulties encountered in the application of this technique and some possible solutions are suggested.

Right Ventricular Volumes Estimated by the Thermodilution Technique after Radical Correction of the Tetralogy of Fallot

By Maylene Wong (introduced) and Michael Dulake

The thermodilution technique for measuring ventricular volume was applied to subjects in whom the tetralogy of Fallot had been radically corrected.
A conventional diagnostic right heart catheterization was performed to exclude any shunt. In 9 such patients a special catheter was introduced, a rapid responding thermodilution technique at its tip being placed in the pulmonary artery and the side-hole in the right ventricle. Pressures and samples could be obtained through the latter, and 1–2 ml of cold saline were injected to obtain a staircase exponential temperature curve from the thermodilution in the pulmonary artery. Cardiac output was obtained by the cardiac method of thermodilution techniques before and after a series of thermodilution curves at rest and under the influence of infused isoprenaline.

Forward stroke volume (FSV) was obtained from the cardiac output.

\[
\text{End-diastolic volume (EDV)} = \frac{\text{FSV}}{1 - \frac{T_n + 1}{T_n}},
\]

where \( T_n + 1 \) and \( T_n \) are differences between the baseline pulmonary artery temperature and those at beats \( T_n + 1 \) and \( T_n \) on the thermodilution curve.

\[
\text{FSV/EDV} = 1 - \frac{T_n + 1}{T_n}.
\]

End-systolic volume = EDV - FSV.

The data were shown and the advantages and limitations of the techniques discussed.

**Experimental results and clinical application of paired stimulation of the heart**

By Edgar Sowton, Leon Resnekov, Peter Lord, and John Norman (the last two introduced)

The application of closely coupled pairs of electrical stimuli to the heart provides the most potent inotropic intervention known at present, and also offers a possible method of controlling intractable arrhythmias. The first stimulus of each pair provokes a ventricular contraction, but the second stimulus is timed to cause an electrical depolarization without any appreciable mechanical activity, so that there are 2 electrical events for each ventricular contraction.

In 13 dogs, paired stimulation invariably slowed the heart below its sinus rate, and also slowed tachycardias of sinus, ventricular, or supraventricular origin; the maximum slowing was by just under 50 per cent. Where the arrhythmia had resulted in a low cardiac output, paired stimulation restored aortic flow to normal.

In dogs with severe heart failure paired stimulation resulted in a fall in LV end-diastolic pressure, an increase in LV DP/DT and LV peak systolic pressure, and a dramatic increase in aortic blood flow. The effects were reversible and highly repeatable, and stimulation at the same rate with single stimuli produced no beneficial effects.

The technique has been applied clinically in 3 patients with severe intractable arrhythmias. In one case, clinical and haemodynamic benefit was obtained for 48 hours. Since the second stimulus of the pair falls near the vulnerable period, there is a risk of inducing ventricular fibrillation, and this occurred in 1 patient.

**Elective circulatory arrest by artificial pacemaker**

By J. M. Small, S. C. F. Stephenson, T. V. Campkin, D. J. S. McI advise (all introduced), and P. H. Davison

During operations on 12 patients with cerebral aneurysm, the circulation was profoundly reduced or arrested by an electrode catheter in the right ventricle, which was used to pace the heart at rates up to 240 a minute. Electrocardiographic, aortic, and right atrial pressures were monitored. The patients were cooled to 31–32°C. Under these conditions the cardiac output and systemic blood pressure were very sensitive to the pacing rate. Above 120 a minute there was reduction of both. At rates of 160 or more, systolic pressures below 30 mm Hg were maintained for periods up to 15 minutes without loss of control of cardiac rhythm. With faster rates and systolic pressures below 20 mm Hg, ventricular fibrillation usually occurred within 1 to 2 minutes. Reversion by D.C. shock and external cardiac massage was then used. Elective arrest with ventricular fibrillation has been maintained for periods up to 64 minutes. There has been no morbidity or mortality to date. The method offers a useful alternative to inflow occlusion.

**Myocardial threshold and impedance in complete heart block: methods for assessing pacemaker function and malfunction**

By R. D. Judge, T. A. Preston, D. L. Bowers, and J. D. Morris (all introduced by E. Sowton)

Analysis of 120 pacemaker implants disclosed that the major causes for failure were (1) lead fracture, (2) battery failure, (3) exit block. Fortunately, component failure has not been a problem. This experience emphasized the need for developing indirect methods of monitoring myocardial threshold and impedance to aid in exactly identifying malfunction and anticipating failure before they manifested themselves clinically.

The circuit design of one unit has made possible the development of a simple and safe method of determining (1) threshold, (2) myocardial resistance, (3) electrode capacitance, (4) battery level. All parameter measurements are made externally (without surgery) by analysing various electromagnetic signals emitted by the pacemaker. Preliminary studies in dogs have shown the following: that the methods are safe; that it is possible to predict energy values from pulse signal analysis; that increases in rate cause increases in threshold. As expected, threshold proved to be a function of total pulse energy. Using calibrated pacemakers, it was possible to measure myocardial resistance and electrode capacitance following human implantation. In a series of 20 patients, capacitance varied insignificantly, while resistance increased gradually during the immediate post-operative period and then became stable. Myocardial impedance did not always vary directly with threshold changes. These studies not only demonstrate a simple, practical method of monitoring pacemaker function, but also promise a means of studying the electrophysiological effects of cardiac drugs in the future.
ATRIAL FUNCTION FOLLOWING CARDIOVERSION

By W. F. W. E. Logan, D. J. Rowlands (both introduced), and G. Howitt

Left and right atrial pressure pulses have been recorded from patients with atrial fibrillation of various aetiologies. In some instances, simultaneous ventricular traces have been recorded. Similar records have been taken immediately after D.C. conversion to sinus rhythm. Comparison of the records taken before and after the rhythm change has revealed a disparity between the effect of cardioversion on the electrical activity and its effect upon the mechanical activity of the atria. In all cases, normal atrial excitation was restored, as evidenced by the development of clearly discernible P waves with a normal P–R interval and with a regular ventricular rate. In most cases these changes in the electrocardiograph have been accompanied by the development of 'a' waves in the right atrial pressure trace. However, in a very significant proportion of cases of rheumatic valve disease no such 'a' waves were developed in the left atrial pressure trace. The possible mechanism and significance of these findings are discussed.

NON-RHEUMATIC MITRAL INCOMPETENCE

By E. B. Raftery (introduced), C. M. Oakley, and J. F. Goodwin

Non-rheumatic causes of mitral incompetence include left atrial tumour, subannular ventricular aneurysm, and cardiomyopathy, as well as endocardial cushion defects and other congenital anomalies involving the mitral valve. Recently the clinical syndrome associated with acquired disease of mitral chordae or papillary muscles has become recognized, and in the past two years 15 such patients have been seen at Hammersmith Hospital.

The clinical features included sudden onset of dyspnoea or pulmonary oedema with development of an apical systolic murmur conducted to the base and mimicking aortic stenosis. Sinus rhythm was the rule and the heart tended to be only slightly enlarged. The left atrial pressure pulse characteristically showed giant 'v' waves which reflected torrential retrograde flow into a normal-sized left atrium.

It was not possible to make a distinction between ruptured chordae and papillary muscle dysfunction on clinical or angiographic grounds, but evidence of subendocardial cardiac infarction indicated failure of papillary muscle function to be the cause in four patients. Two patients with ruptured chordae had recently had bacterial endocarditis, and one patient had papillary muscle invasion by sarcoid granuloma. The etiology was unknown in the others, and a familial incidence was not found.

As pulmonary oedema may result from overfilling of an undilated pulmonary venous reservoir rather than from left ventricular failure, these patients can present a particularly urgent but low risk surgical problem. Four patients were operated upon and the mitral valve successfully replaced.

SELECTIVE CINEFLUOROSCOPIC STUDIES OF PULMONARY CIRCULATORY DISORDERS*

By M. J. Raphael (introduced) and R. E. Steiner

We report some results in our investigation of pathological pulmonary circulation in heart disease using the Chrispin and Steiner (1964) technique of selective pulmonary cine-angiography. Out of 60 patients studied, we have selected the examinations illustrating the changes in circulation in 8 patients with varying degrees of pulmonary hypertension secondary to acquired valvular heart disease and in 6 patients with intracardiac shunts of varying sizes.

In patients with raised left atrial pressures and varying degrees of pulmonary hypertension, the reduced lower lobe blood flow is manifested by impaired onward movement of the contrast medium with each heart beat and delayed appearance time of contrast in the pulmonary veins.

In patients with small intracardiac shunts and vessels of normal size, the angiographic studies follow the normal pattern. When the shunt is large and associated with distended pulmonary vessels, the onward movement of contrast medium in the pulmonary artery with each heart beat is accentuated. Unless the shunt is very large, the period of diastolic stasis of contrast medium is maintained as in normal patients. This suggests that pulsatile arterial flow is still maintained even in the presence of large shunts. Contrast very rapidly reaches the enlarged veins and fails to show pulsatile fluctuations in density, thus suggesting absence of pulsatile venous flow.

The disappearance of pulsatile flow in the pulmonary veins in patients with large shunts is surprising, in view of the accentuated pulmonary arterial pulsation. This paradox might be explained by the excessive venous distension producing a volume damping effect.

EXPERIENCE WITH BYPASS SURGERY IN CORONARY ARTERIAL DISEASE COMPLICATING AORTIC VALVAR DISEASE

By Dennis Boyle (introduced), Lawson McDonald, Leon Resnekov, and Donald Ross

In 66 patients with aortic valvar disease, who underwent bypass surgery, 20 were found to have concomitant coronary arterial disease, as evidenced by macroscopical abnormalities of the coronary arteries and infarctions of the left ventricular wall. The mortality rate, following surgery, was similar in patients who had coronary arterial disease and in those who had not. Symptoms, clinical signs, and electrocardiographic findings were unhelpful in the precise diagnosis of additional coronary arterial disease, and will be discussed in relation to radiographic findings and other special investigations. With aortic valvar disease, in which surgery is indicated, the presence of additional coronary arterial disease is not considered to be a contra-indication to operation.

* To be published in full in a future issue of this Journal.
FATE OF THE HUMAN AORTIC VALVE HOMOGRAFT
By R. Hudson
[Published in full in Brit. Heart J., 1966, 28, 291.]

OBSTRUCTED TOTAL ANOMALOUS PULMONARY VENOUS DRAINAGE
By Jane Somerville and Alan Chrispin (introduced)
In a consecutive series of 80 patients with total anomalous pulmonary venous drainage, 18 (22%) were found to have obstruction of the common pulmonary venous channel; patients with cor triatriatum were excluded. The site of narrowing was in the ascending pulmonary venous trunk before it joined the left innominate in 7 patients, descending venous trunk entering the portal system in 7 patients, and at other sites in 4. The age of the patients ranged from 11 days to 5 months with equal numbers of males and females. 50 per cent had symptoms in the first month of life. They presented a recognizable clinical syndrome with laboured respiration, small hearts, signs of pulmonary hypertension, and intense pulmonary edema. Seven patients had continuous murmurs, but in the majority murmurs were unimpressive, so that the underlying heart disease was frequently unrecognized. The diagnostic features on the chest radiographs and angiograms are discussed in detail.

DIFFERENTIAL HYPERCAPNIA IN THE EISENMENGER DUCTUS IN RELATION TO THE DYSNIAEA OF CYANOTIC CONGENITAL HEART DISEASE
By Hywel Davies and Nikos Gazetopoulos (introduced)
Wood pointed out in 1958 that patients with an Eisenmenger ductus were significantly less disabled than those with pulmonary-systemic communications at other levels. This we consider to be due not so much to the presence of differential cyanosis but rather to differential hypercapnia, the blood to the vital centres of the head and neck being not only relatively rich in oxygen but low in carbon dioxide content. The results of exercise studies which demonstrate this are presented, and their significance is discussed in the context of the determinants of disability in patients with cyanotic congenital heart disease.

SUPRAVALVAR AORTIC STENOSIS
By G. F. Gearty (introduced by R. E. Steen)
Obstruction to left ventricular outflow may occur at subvalvar, valvar, or supravalvar levels. Thus aortic stenosis, though usually valvar, is not always so, and pressure gradients alone may afford inadequate preoperative assessment. What features then suggest the possibility of obstruction other than at valvar level? How are such cases best investigated, particularly in children?
One aspect of this problem is illustrated by the recognition, in recent years, of the syndrome of peculiar facies, mental retardation, and supravalvar aortic stenosis. This communication outlines findings in 4 such patients.

The facial characteristics are described and illustrated. The facile good-humoured personality with low intelligence quotient is emphasized. The clinical findings are listed, stressing inequality of radial pulses and blood pressure, the localization of the systolic thrill and murmur, and the absence of ejection click or diastolic murmur. The radiological features are illustrated, particularly the absence of post-stenotic dilatation of the aorta. The electrocardiographic findings are noted, especially the occurrence of "strain" patterns in the presence of a dilated high-pressure coronary circulation. Findings at right heart catheterization showed that two children had pulmonary stenosis, one at valve level and the other peripherally.

The methods of assessment of the left heart and aorta, which differed in each case, are outlined, illustrated, and contrasted.
Our first case was diagnosed at thoracotomy following percutaneous left ventricular and brachial artery punctures only.
Our second child had percutaneous pressure measurements and right ventricular angiography, which provided an adequate outline of the left heart.
Our third child had transseptal left heart catheter studies and angiography from the left atrium.
Our fourth patient had retrograde arterial catheterization following arteriotomy and angiography from the supravalvar chamber.
Finally, important negative points in this series are mentioned—the absence of familial cases, the absence of chromosomal abnormality, and the absence of historical or clinical sequelae of hypercalcæmia.

A NEW PLATELET DEFECT IN PATIENTS WITH ISCHAEMIC DEFECT IN PATIENTS WITH ISCHAEMIC HEART DISEASE
By Colin Bray (introduced) and Lawson McDonald
In a study of platelet aggregation in patients with ischemic heart disease, using Born's technique, some patients showed a qualitative difference in the pattern of response to adenosine diphosphate and adenosine triphosphate. Normal initial aggregation and disaggregation of the platelets occurred, but these were followed by a totally abnormal secondary aggregation of the platelets, within two minutes of the initial response, which progressed to a massive degree. In vivo such massive aggregation could be the starting point of thrombus formation. On repeated examination of the same patients this secondary aggregation was a constant feature. It never occurred in healthy subjects. The patients who showed this phenomenon were comparatively young, had low levels of plasma cholesterol, normal levels of plasma uric acid, and were not deficient in lipoprotein lipase. This previously undescribed defect of the platelets in patients with ischemic heart disease will be related to a possible metabolic abnormality and to thrombus formation.
Brady Cardia in Acute Myocardial Infarction

By D. C. Fluck, E. Olsen (both introduced), and J. P. D. Mounsey

Brady cardia due either to sudden marked sinus brady cardia or complete heart block has been observed in 14 out of 36 consecutive patients admitted to the Intensive Coronary Care Unit at the Hammersmith Hospital. All had an acute myocardial infarction and all had continuous electrocardiographic monitoring.

The supraventricular brady cardia group consisted of 7 patients, who had episodes during the first 48 hours of marked sinus brady cardia with a ventricular rate between 35 and 55 a minute and an accompanying fall in blood pressure and signs of a vasovagal attack. These attacks were successfully treated by immediately raising the legs and giving atropine intravenously. Patients prone to these attacks formed a clinically recognizable group on admission, all having posterior infarction, a tendency to sinus brady cardia (50–60/min.), and severe cardiac pain requiring repeated analgesics.

The ventricular brady cardia group consisted of 7 patients with complete heart block, 6 of whom came to necropsy. In 3, this was a short pre-terminal rhythm in a dying heart, whereas in the other 4 the heart block was more prolonged and associated with Stokes-Adams attacks. All 7 patients died. Immediate internal cardiac pacing at the onset of heart block temporarily restored sinus rhythm in one patient with clinical improvement. The indications for cardiac pacing in heart block developing during acute myocardial infarction are discussed.

Metabolic Problems of Cardiac Resuscitation

By K. P. Ball

In 21 patients with cardiac arrest due to acute myocardial infarction arterial blood gas analysis was carried out. The patients were part of a series of 49 patients with myocardial infarction in whom resuscitation was attempted. Oxygen saturation, carbon dioxide tension, and pH were measured; from these measurements, plasma bicarbonate and oxygen tension were derived.

Arterial oxygenation was fairly good in these patients, but they were being ventilated with pure oxygen, and the alveolar arterial oxygen tension gradient was high, indicating severe abnormality of pulmonary function. As arterial oxygenation was reasonably good, the metabolic acidosis from anaerobic metabolism must be due to poor tissue perfusion. Large arterio-venous differences were found in those patients in whom these were measured.

Acidosis was present in 18 of the patients; in 8 it was "respiratory," in 5 "metabolic", and in 5 "mixed respiratory and metabolic". Serum lactic levels were raised in all patients in whom it was measured.

Respiratory acidosis due to underventilation was surprisingly frequent and severe. Correction of metabolic acidosis by sodium bicarbonate will only be achieved when hyperventilation is produced. Our results suggest that even when this is appreciated ventilation will not be adequate unless great care is taken to ensure it.

Prophylactic Value of Propranolol (Inderal) in Angina Pectoris

By R. Rabkin, D. Stables, N. W. Levin (all introduced), and M. M. Suzman

In a double-blind trial, 20 patients were given 50 mg. of propranolol 4 times daily and placebo, each for 2 periods of 2 weeks, according to one of 4 randomized sequences (ABBA, BABAB, ABAB, BAAB). Of these 20 patients, 4 were withdrawn from the trial (2 defected, 1 kept inaccurate records, 1 died of myocardial infarction). Of the remaining 16 patients, 6 showed no significant preference, 10 had considerably fewer attacks on propranolol and of these 2 were totally relieved. The average reduction in the number of attacks on propranolol was 44 per cent. The Wilcoxon test for symmetry showed a value of t = 130 (p = 0.001) in favour of the drug. A carry-over effect was not demonstrated.

A separate group of 20 patients was treated on a single-blind basis, varying the dosage and the duration of drug and placebo medication according to the response in each individual patient: this group was observed for periods up to 18 months. In 15 there was unequivocal improvement in the frequency and severity of the anginal attacks, whereas 5 derived no significant benefit. On propranolol therapy, 2 patients died suddenly (one in ventricular fibrillation immediately after mild exercise), 2 developed acute myocardial infarction, and one had left ventricular failure.

Serial laboratory investigations in both trials revealed no deleterious effects on the hepatic, renal, and haemopoietic systems, and untoward side-effects were minimal.

Cardiac Impulse in Ventricular Aneurysm

By A. Mourdjinis, E. Olsen, J. Taubman (all introduced), and J. P. D. Mounsey

The "paradoxical impulse" of ventricular aneurysm was studied in 7 patients with proven ventricular aneurysm following cardiac infarction, using the technique of impulse cardiography. In addition, the detailed movements of the underlying aneurysmal wall were examined in the cineradiograph and angiocardiogram. Three patients were investigated before and after surgical removal of the aneurysm. Necropsies were performed in 2 patients and operation specimens examined in 4.

The aneurysmal wall is composed of collagen and fails to take part in concentric contraction of the left ventricular myocardium during ventricular systole. This failure of retraction of the aneurysmal wall was seen in the cineradiograph and angiocardiogram. It was also reflected in the impulse cardiogram, in the area overlying the aneurysm, where a large sustained outward movement was prolonged throughout ventricular systole, replacing the normal inward movement in the latter half of systole. After successful surgical removal of the aneurysm, the cardiac impulse reverted to a more normal pattern.

The sustained impulse of ventricular aneurysm must be distinguished clinically from that due to ventricular hypertrophy associated with ischemic heart disease, and the diagnostic value of this sign in conjunction with clinical, electrocardiographic, and radiological data is discussed.