Proceedings of The British Cardiac Society

The Forty-sixth Annual General Meeting of the British Cardiac Society was held in the Institute of Clinical Science of the Queen's University, Belfast, on Thursday, May 18, 1967. The President, Shirley Smith, took the Chair at 9.00 a.m. during Private Business before handing over to the Chairman, T. H. Crozier.

Private Business
1. The Minutes of the Annual General Meeting having been published in the Journal (1967, 29, 627) were taken as read and confirmed.
2. The following alterations to the Rules of the Society were confirmed:
   Rule 22 to read—
   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 President, the Chairman, the Chairman Elect, the Chairman of the Annual Meeting, the Editors of the Journals or Assistant Editors delegated by them, the Secretary, the Assistant Secretary and the Treasurer. The Council shall have power to co-opt...
   An additional Rule to be inserted after Rule 28 as follows—
   Cardiovascular Research
   The Editors, and the Editorial Board, shall be appointed by the Council of the British Cardiac Society. The Editor of the British Medical Journal shall be a member of the Editorial Board. The Editors shall be appointed for a period not exceeding five years, and shall be eligible for reappointment. The Editor or Assistant Editor delegated by him shall, ex officio, be a member of the Council.
3. The Treasurer reported that the audited accounts for the year ended December 31, 1966, had been presented to, and accepted by, the President and Council.
   The General Fund of the Society held £1,179.19.9.
   The income in 1966 from subscriptions and interest on investments was £1,465.4.5, which represented an increase of £126.14.11 as compared with 1965.
   The expenditure in 1966 was £1,092.3.1, representing an increase of £141.1.6.
   There was a decrease in the amount of excess of income over expenditure in 1966 as compared with 1965. Excess of income over expenditure in 1966 was £373.1.4 and in 1965 £387.0.0.
   In addition to the normal expenses, the General Fund donated £200 to the New Delhi V World Congress of Cardiology, and lent £200 for expenses for the VI World Congress to be held in London in 1970. It was agreed by Council to donate £50 to the Society of Cardiological Technicians and £200 to the Congress Fund.
   There had been a slight reduction in the expenses of printing during the past year, and only a slight increase in the expenses of meeting. A considerable increase, however, had occurred in secretarial expenses from £444 in 1965 to £518.7.9 in 1966. This increase was attributable to the increase in the cost of secretarial expenses and to the amount of secretarial work entailed by the expanding activities of the Society. Every effort was being made to reduce secretarial expenses but it was felt inevitable that some increase would occur with the increased cost of living over the coming years. It was felt too early to comment on the saving which would be effected by the use of meal cards before the meetings, but it was hoped that some saving would result.
   The Treasurer felt that the financial position of the Society was satisfactory, and said that the increase in secretarial expenses would be carefully watched.
   The Congress Fund had paid out £438.14.4 on grants and secretarial expenses for the V World Congress. The cash in hand was £1,009.4.0 and the total expenses for 1966 £640.8.11.
   The Thomas Lewis Lecture Fund held a total of £1,247.7.7, of which £1,173.10.2 was in investments, £55.3.3 in current account, and a tax rebate due of £8.14.2. The lecturer for 1967 would be paid 100 guineas, leaving some surplus to pay a larger fee for the subsequent lecturer who might come from further afield.
4. D'Abreu, Goldblatt, and Lowther had tendered their resignations to the Society.
5. Shirley Smith was re-elected President of the Society.
6. Sir John McMichael was appointed President Elect.
7. The following two new Members of Council were elected in place of Hayward and Marquis:
   M. F. Oliver
   Lawson McDonald
8. The following Honorary Member was elected:
   Datey—from Corresponding Membership
9. The following Extra-Ordinary Members were elected:
   Chamberlain
   Kerley
   Peel
10. The following Corresponding Member was elected: Cyril Fortune of Australia.

11. The following Ordinary Members were elected from Associate Membership:
   - Hywel Davies
   - Epstein
   - Finlayson
   - Leather
   - Resnekov
   - D. N. Ross
   - Segel
   - Turner
   - Williams

12. The following Associate Members were elected:
   - Thomas B. Begg
     - Glasgow
   - Percy Cliffe
     - London
   - Ronald Graham Grainger
     - Sheffield
   - Abraham Guz
     - London
   - Katherine Ann Hallidie-Smith
     - London
   - Michael Christopher Joseph
     - London
   - Martin W. McNicol
     - London
   - John Richard Antony Mitchell
     - Oxford
   - Edgar Bernard Raftery
     - London
   - Robert Simon Owen Rees
     - London
   - Basil Strickland
     - London
   - Reginald Joseph Twort
     - Nottingham

   and as Associate Surgical Members—
   - William Herbert Bain
     - Glasgow
   - John W. Jackson
     - London
   - Patrick John Molloy
     - Liverpool

13. The following Overseas Members were elected:
   - Eldryd Hugh Owen Parry
     - Ethiopia
   - Peter Audaer Overend Wilson
     - Kuwait

   and the following re-elected—
   - Davidson and Harries

14. Plans were already advanced for the VI World Congress of Cardiology to be held in London from September 6 to 12, 1970, in the Festival Hall complex. Meetings had already been held with the International Society of Cardiology in Geneva. Further particulars would be sent to members as soon as possible.

15. The V European Congress of Cardiology will be held in Athens from September 8 to 14, 1968.

16. The Autumn Meeting of the Society will be held on November 2 and 3, 1967, at the Royal College of Physicians.

17. The Annual General Meeting of the Society in 1968 will be held on April 18 in Liverpool, under the Chairmanship of Wyn Jones.

18. The Autumn Meeting of the Society in 1968 will be held in London at the Royal College of Physicians, provisional dates being December 5 and 6.

19. The Third Thomas Lewis Lecture will be given by Professor Durrer of Amsterdam at the Society's Autumn Meeting on November 2, 1967. The title of the Lecture will be—"Electrical Excitation and Stimulation of the Human Heart".

Demonstrations were held at the Institute of Clinical Science.

The Society dined together at the Conway Hotel, Dunmurry, the principal guests being the Dean of the Faculty of Medicine, and the Chairman of the Belfast Hospital Management Committee. The President, Shirley Smith, proposed the health of Crozier, the Chairman of the Scientific Meeting, and Crozier replied.

Open Heart Surgery for Mitral Disease

By Julian Bath (introduced), Arthur Kitchin, Andrew Logan, and Richard Turner

Since 1950, in Edinburgh, approximately 1700 patients have been treated for mitral valvular disease by closed operation, and since 1961 approximately 200 patients by repair or replacement of the valve under direct vision.

Replacement in the earlier cases was with a Starr-Edwards valve. More recently the Hammersmith valve has been used. The mortality rate, complications, and results in approximately 1000 closed and 150 open operations are considered in the patients treated from the Cardiac Department of the Western General Hospital, Edinburgh, with special reference to replacement of the valve. The incidence of subsequent embolism (6%) after replacement has been lower than that generally reported and a possible reason for this is suggested. In about one-third of all cases there is a post-operative increase in heart size, in contrast to what is found after aortic valve replacement.

The indications for open operation are discussed in the light of these findings. Operative mortality is now considered low enough for wider application of open operations. The place of transventricular valvotomy is defined.

Systemic Hypertension in Mitral Valve Disease

By S. A. Fenton and H. C. Mulholland (introduced by J. F. Pantridge)

There is no unanimity of opinion regarding incidence of systemic hypertension in patients with rheumatic heart disease. Gray (1954) and Roseman and Wasserman (1951) found that the incidence of hypertension was not significantly greater in patients with mitral stenosis than in control groups. These findings were not in accord with those of Obeyesekere et al. (1965) who found that more than half their patients with mitral valve disease had a systemic blood pressure above normal.

This communication deals with the blood pressure records of 1108 patients who had mitral valvotomy. The patients were followed for periods varying from 1 to 16 years. Details of the blood pressure records of patients in sinus rhythm, those in atrial fibrillation, and those with a history of embolism are given. The data obtained do not support the contention that there is a significantly greater incidence of systemic hypertension in patients with mitral valve disease.

References

A Critical Study of the Value of Direct Current Conversion of Atrial Fibrillation
By M. E. Scott and G. C. Patterson
(introduced by J. F. Pantridge)

The results of D.C. conversion of atrial fibrillation in 300 patients have been studied with particular reference to the number maintaining sinus rhythm and to the factors influencing relapse of the arrhythmia.

In view of the conflicting reports of the effect of D.C. conversion on cardiac output, serial output studies have been obtained in patients converted to sinus rhythm. Little change in output or stroke volume was noted when measurements were made 3 hours after conversion. However, when output and stroke volume were measured 3 days after conversion, a significant increase was found and a further increase was noted when the output and stroke volume were measured 1 week after conversion of atrial fibrillation to sinus rhythm. Conflicting reports of the effect of conversion on cardiac output appear to relate to the time after conversion at which measurements were made.

Long-term Results of Closure of Ventricular Septal Defects in Patients with Pulmonary Vascular Disease
By K. A. Hallidie-Smith, A. Hollman, J. A. Abbott
(introduced, H. H. Bentall, W. P. Cleland, and J. F. Goodwin)

Between 1958 and 1967, 33 children with ventricular septal defects and pulmonary vascular disease have been surgically treated at the Royal Postgraduate Medical School. Before operation these children had balanced systolic pressure in both ventricles, a left-to-right shunt, minimal or no right-to-left shunt, and a pulmonary vascular resistance of 8 units or more.

Of the 25 survivors, 22 have been reinvestigated from one to eight years after operation, 10 on 2 separate occasions. They have been assessed clinically, radiologically, haemodynamically, and by radioactive gas tests of pulmonary blood flow.

All the children have become asymptomatic and their rate of growth has increased. Nine patients have evidence of a very small residual left-to-right shunt, 5 detectable only by central sampling dye dilution curves. The pulmonary artery pressure fell at the time of closure of the defect in all our patients and the immediate post-operative level proved a reliable guide to subsequent findings. Residual pulmonary vascular disease was assessed by absolute units and by systemic-pulmonary resistance ratios and systemic artery-pulmonary artery systolic pressure ratios and judged to persist with mild or moderate severity in 15 patients. Only in one patient is there some indication that the pulmonary vascular disease is progressing. In the 12 patients in whom it was possible to carry out exercise or isoprenaline studies, 6 showed a rise in mean pulmonary artery pressure of over 10 mm. The degree of pulmonary vascular disease after operation was not related to the amount of pulmonary vascular disease before, nor was it related to the presence or absence of a residual defect. The 8 deaths were also unrelated to the severity of the pulmonary vascular disease, being attributable rather to anatomical complications and respiratory problems. There have only been 2 deaths in the last 13 patients as a result of improved surgical technique and experience.

We conclude that the improvement in pulmonary vascular disease after surgery fully warrants operation in this group of patients.

Left Ventricular Angiocardiography in Atrioventricular Defects
By Jane Somerville and Keith Jefferson

Left ventricular angiocardiograms, using fast biplane Elena roll films, have been performed in 40 patients with atrioventricular defects, and the pathological anatomy of the mitral valve at open-heart surgery correlated well with the angiocardiographic findings. It has been possible to separate those patients with adequate mitral cusp tissue who will do well with simple repair of the atrioventricular defect through a right atriotomy, from those in whom the mitral valve is grossly deficient, with short tendinous attachments.

Differentiation of the ostium primum defect from the common atrioventricular canal can be made if contrast medium is seen to pass directly from the left to the right ventricle in the lateral plane, before the right atrium is opacified from mitral regurgitation. The characteristically deformed left ventricular outflow tract is present in all forms of atrioventricular defect, and has not been seen in any other type of septal defect. Thus, left ventricular angiocardiography is reliable in differentiating atrioventricular defects from other types of atrial and ventricular septal defect which may mimic them in presentation.

Left ventricular angiocardiography appears, from this study, to be essential for the diagnosis of the different types of atrioventricular defect, and in the assessment of the mitral valve in all patients with atrioventricular defects who are being considered for operation.

Relationship Between Metabolic Acidosis and Cardiac Arrhythmias in Acute Myocardial Infarction
By R. Anderson (introduced), Frances Gardner, M. Honey, Isabel M. Noble, and D. J. Woodgate
(introduced)

Twenty-one patients with clinical and electrocardiographic evidence of recent myocardial infarction and raised serum enzymes were studied. The heart rate and rhythm were monitored for at least 72 hours, and arterial samples were obtained on admission, 24 hours later, and at the onset of any arrhythmia. The arterial blood pH, Pco₂, bicarbonate, and base deficit were measured using the Astrup micro-electrode technique.

Where metabolic acidosis was present on admission there was an increase in the incidence of cardiac arrhythmias. Nine patients had a base deficit of more than 2.5 mEq/l on admission; all 9 were hypotensive at the time, with cold extremities. Eight of the 9 patients
developed arrhythmias in the following 3 days, while in the 12 patients with normal acid-base states, 7 developed arrhythmias over the same period. About half the arterial samples, 13 of 24, obtained at the time of an arrhythmia showed a significant base deficit. These patients were also hypotensive and/or had pulmonary congestion at the time.

These results suggest that where metabolic acidosis occurs at the time of an arrhythmia it is probably a result of the circulatory inefficiency due to the arrhythmia rather than the cause of the arrhythmia itself.

**Circulatory Effects of Intravenous Heroin in Patients with Acute Myocardial Infarction**

By H. R. Macdonald, H. A. Rees, A. L. Muir, D. M. Lawrie, J. Burton (all introduced), and K. W. Donald

Morphine is the most widely used drug for the relief of pain in myocardial infarction. Shillingford and his colleagues have shown that morphine may have adverse circulatory effects (Thomas et al., 1965). Heroin (diamorphine), a more potent analgesic, has been suggested as an alternative. The present study was designed to assess the clinical and circulatory effects of this drug in patients with acute myocardial infarction admitted to the Coronary Care Unit of the Edinburgh Royal Infirmary. Studies were made in three patients during acute pain and in the remainder within 48 hours of the onset of pain. Heart rate, cardiac output, systemic arterial, pulmonary arterial, and right atrial pressures were measured in 6 patients, in the supine position. The changes produced by this drug were small and of no practical significance.

These preliminary findings suggest that serious consideration should be given to using heroin in place of morphine for the relief of pain in myocardial infarction.

**Reference**


**Inhomogeneity of Myocardial Blood Flow as Detected by Washout of Inert Gases**

By D. G. Greene, F. J. Klocke, R. C. Koberstein, D. Rosing, H. L. Falsetti, and I. L. Bunnell (all introduced by Sir George Pickering)

An unusually sensitive gas chromatograph (Physiologist, 1966, 9, 211) was used to follow coronary venous-arterial differences of hydrogen after 20-minute periods of breathing hydrogen-oxygen-air mixtures, or to follow coronary venous hydrogen after left ventricular infusions of hydrogenated saline for 3-4 minutes. Semilogarithmic plots of the washout curves were non-linear below 10 per cent of initial values, a region previously difficult to explore by less sensitive techniques, suggesting inhomogeneity of myocardial perfusion. The deviations were greater in 11 patients with arteriographically-proven coronary artery disease than in 4 patients with clinically and arteriographically normal coronary circulations. Similar studies in closed-chest dogs before and after embolic occlusion of part of the left coronary arterial bed showed striking alterations in washout pattern, with great slowing of the rate of decay after embolization. Additional data in both patients and dogs suggest that short saturation periods (30 seconds) may fail to demonstrate inhomogeneous flow and that even 10-minute periods of breathing hydrogen may not be sufficient for disclosing the full extent of inhomogeneity of perfusion. Simultaneous washout curves with mixtures of inert gases differing in solubility and diffusivity suggest that gas exchange between blood and tissue may sometimes be limited by diffusion as well as perfusion.

**Collateral Blood Flow in Experimental Myocardial Infarction. Early Changes by a New Method Using Xenon**

By J. Russell Rees, V. J. Redding, and P. Cliffe

The measurement of collateral flow in experimental animals has always involved thoracotomy, and usually coronary arterial ligation. Because such procedures may affect the results, we have developed a method whereby infarction and collateral flow measurements are achieved without disturbing the heart or its nerve supply.

After measurement of myocardial blood flow in the undisturbed dog's heart, myocardial infarction is produced by wedging a modified Courand catheter in a large branch of the circumflex artery under radiographic control. Collateral blood flow is then measured by Xenon clearance, using an external scintillation counter, after solutions of isotope have been delivered to the ischemic myocardium through the lumen of the obstructing catheter. Of 11 dogs, 7 survived this procedure. Mean flow was reduced to 35 per cent of control flow rate. At 1-2 hours flow rates had risen to 43 per cent but thereafter fell gradually to 28 per cent of control rates 6 hours after infarction. This fluctuation was not related to arterial blood pressure. In a further group of animals infarcts were caused by tying the anterior descending artery and clearance rates were similar. Neurone blockade with bretylium tosylate or local injection of procaine did not increase collateral flow.

**Pulmonary Function in Myocardial Infarction**

By B. J. Kirby (introduced by K. P. Ball) and M. W. McNicol

Extending previous work (McNicoll et al., 1965), we have examined gas transfer in myocardial infarction. In a group of 50 patients, hypoxemia was found in 46 (92%). Alveolar oxygen was low in all patients; in 29 the right heart was also catheterized and mixed venous blood oxygen tension difference of collapse was due to impairment of oxygen transfer in the lung. The physiological dead space and physiological shunt were much increased, indicating that the abnormality in gas transfer was due to ventilation—blood flow imbalance. Cardiac output
as measured by the direct Fick method was low, though right atrial pressure was commonly raised. The combination of reduction in oxygen content of the mixed venous blood and shunting in the lungs produced the arterial hypoxaemia. The changes in cardiopulmonary function were most severe in patients with clinical signs of cardiac failure.

REFERENCE

EFFECT OF POSTURE ON CARDIAC OUTPUT DURING THE LAST SIX WEEKS OF PREGNANCY
By D. McC. Boyle, and C. H. Mulholland and G. C. Patterson (both introduced)

Many studies are now available of the hemodynamics in pregnancy. A constant finding in these studies is that the cardiac output rises to a peak in mid pregnancy and after 28 weeks tends to fall to a lower level. No adequate explanation has been offered for this fall in cardiac output. It may be significant that all the measurements of cardiac output were undertaken with the patient in the supine position. It is therefore possible that compression of the inferior vena cava by the enlarging pregnant uterus leads to a fall in cardiac output when the patient is placed in the supine position.

This communication records the results of measuring the cardiac output by the Fick method in both the supine and the left lateral position in the later weeks of pregnancy. Results show that the cardiac output is greater in the lateral position.

The fall in cardiac output in the later weeks of pregnancy may be less than is usually thought. This possibility has important implications in the management of the pregnant patient.

Hemodynamic Effects of Alcohol in Patients with Coronary Heart Disease
By Neville Conway (introduced), and Edgar Sowton

Eight patients with coronary heart disease who were all used to moderate amounts of alcohol were investigated. Aortic and pulmonary arterial pressures, electrocardiogram, and cardiac output were recorded with the patients supine, erect at rest, and during exercise on a bicycle ergometer at work loads of up to 350 kpm/min. Each subject was then given 0.5 g./kg. body weight of absolute alcohol diluted in water, and blood alcohol levels were measured for one hour during which time the resting measurements were repeated. At the end of this time a second exercise study was carried out.

At rest the maximum changes were noted about 45 minutes after alcohol, when the average blood level was approximately 50 mg./100 ml. Aortic pressure fell progressively, and in one subject dropped from 150/90 to 85/50 mm. Hg. In two subjects T waves became flat or inverted. Cardiac output was slightly reduced in the supine position but unchanged in the erect position. Tension time index and left ventricular work fell to a minimum at 45 minutes.

On effort, aortic pressure and left ventricular work were reduced at all exercise levels. The cardiac output tended to be lower but this was not significant. No alteration in effort tolerance was found in patients with angina.

Observations on Lung Mast Cells and Serotonin in Rats with Pulmonary Hypertension Produced by Ingestion of Crotalaria Spectabilis Seeds
By Donald Heath, and Michael Kay, Dean Gillund, and Neville Crawford (introduced)

Weanling albino Wistar rats fed on a diet containing 0.1 per cent of ground Crotalaria spectabilis seeds develop pulmonary hypertension. This is associated with hypertensive pulmonary vascular disease characterized by medial hypertrophy, and sometimes by necrotizing arteritis, in the small pulmonary arteries. There is gross increase in thickness of the pulmonary trunk. Rats allowed to die show exudative changes and a pronounced proliferation of mast cells in the lungs. Mast cells are known to contain serotonin as well as the better known heparin.

Nevertheless, the lungs of test animals do not show an increased content of serotonin. Platelet-bound serotonin is also virtually identical in both test and control animals. There is, however, an unequivocally raised level of free plasma serotonin in the test rats. The injection of serotonin intravenously into one rat brought about an acute rise in pulmonary arterial pressure.

These results suggest that the production of pulmonary hypertension by the oral ingestion of such seeds acts through the medium of increased free plasma serotonin.

Alternating Aberration
By H. J. L. Marriott (introduced by J. F. Goodwin)

Aberrant ventricular conduction is the last important stronghold of arrhythmic uncertainty. Because of its common operation, we can seldom identify ectopic ventricular rhythms with any degree of assurance.

Aberration usually behaves logically—developing as the ventricular cycle shortens because the earlier impulse then falls within the relative refractory period of one of the bundle branches, usually the right. On the other hand, there are times when conduction apparently defies logic—as when aberration appears only when the cycle lengths or persists even after the cycle has resumed a comfortable length following a bout of tachycardia.

An unusual and at first inexplicable form of ventricular aberration is sometimes seen with atrial bigeminy: the premature beats show alternating forms of aberration related to alternating coupling intervals. Such “alternating aberration” implies that one bundle, while unable to conduct after a longer interval, is able to conduct after a shorter rest. This seeming paradox can be explained by assuming either that there is an inverse relation between the absolute and relative refractory periods.
of the two bundle branches, or that there is a supernormal phase of conduction recovery during the absolute refractory period of one bundle.

**Effects of the Dextro Isomer (+) of Propranolol on Sinus Rate and Cardiac Arrhythmias**

By G. Howitt, E. G. Wade, and M. Husaini, W. F. W. E. Logan, and R. Shanks (introduced)

Experimental work has shown that (+) propranolol has only slight beta blocking activity compared with laevo (-) propranolol, but an equivalent quinidine-like action; the effect against digitalis-induced arrhythmias in dogs is confined to (+) propranolol.

(+ ) Propranolol was given intravenously in doses up to 20 mg. to 57 subjects, 41 of whom had cardiac arrhythmias. Most were also given propranolol. (1) In 6 normal volunteers exercise tachycardia was reduced by propranolol but not by larger doses of (+) propranolol. (2) (+) Propranolol had slight or no effect on the resting sinus rate or on the ventricular rate in patients with atrial fibrillation or flutter compared with smaller doses of propranolol. (3) In ectopic arrhythmias-prefarct beats of tachycardia both (+) propranolol and proprano- lol usually had either no effect or approximately equiv-alent effects. (4) In 4 of 5 patients with arrhythmias due to digitalis overdosage the arrhythmias were abolished or reduced by small doses of (+ ) propranolol.

From these results it appears likely that the effect of propranolol on the sinus and A-V nodes is via beta-blockade but its effects in ectopic arrhythmias and in digitalis toxicity are through other mechanisms.

**Effect of Propranolol on Left Ventricular Work in Aortic Stenosis**

By John Hamer, and James Fleming (introduced)

The suggestion that propranolol relieves angina by reducing the work done by the left ventricular muscle has been investigated by the thermodilution method at left heart catheterization in 12 patients with severe aortic stenosis. A Brockenbrough catheter was placed in the left ventricle by the transseptal approach, and a No. 6 Gensini catheter was passed percutaneously from the femoral artery to the ascending aorta. A thermocouple wire was inserted in the aortic catheter till it just protruded at the tip, and thermodilution curves were obtained by injecting 5 to 10 ml. of cold saline into the left ventricle. Cardiac output was measured by the indicator dilution method using a Gilford cuvette and the dynamic method of calibration. The procedure was repeated after the injection of 5 mg. propranolol. In 6 patients the study was carried out after 2 mg. atropine had been given.

Propranolol produced a slight fall in left ventricular systolic pressure. Heart rate and cardiac output fell and systole was prolonged. The end-diastolic pressure in the left ventricle tended to rise. Left ventricular volume was generally increased and there was no significant change in force time per minute which was selected as the most appropriate measure of left ventricular work. However, the mean velocity of contraction of the left ventricular muscle was considerably reduced.

The reduced velocity of contraction of the left ventricular muscle is probably, at least in part, due to the slower heart rate. Although conventional indices of left ventricular work are unchanged, a reduced velocity of contraction may be associated with a lower myocardial oxygen consumption, and so with a beneficial effect in angina pectoris.

**Prediction of Left Ventricular Diastolic Pressures in Aortic Stenosis from the Apex Cardiogram**

By James Fleming (introduced), and John Hamer

In 23 patients with left ventricular outflow tract obstruction, the left atrial and left ventricular pressures have been measured by transseptal cardiac catheterization, and compared with the apex cardiogram. The presystolic wave in the apex cardiogram was normally large in aortic stenosis at valve level, subvalvar level, and in hypertrophic obstructive cardiomyopathy, but the size of this wave did not reflect the size of orifice of the stenotic left ventricular outflow tract nor did it correlate well with the magnitude of the left atrial systolic pressure wave in individual patients.

The height of the third sound point was found to correlate well with the left ventricular end-diastolic pressure, and we suggest that the apex cardiogram may be used as a simple bedside technique to predict the left ventricular end-diastolic pressure in left ventricular out-flow tract obstruction.

The presystolic wave and the third sound point on the apex cardiogram are influenced by the diastolic compliance of the left ventricle. The place of the apex cardiogram in the evaluation of left ventricular function in aortic stenosis is discussed.

**Cardiac Output during Internal Cardiac Compression in Dogs**

By R. J. McFarland and P. F. Binnion (both introduced by Evan Fletcher)

Ventricular fibrillation was produced by the topical application of aconitine solution to the epicardial surface of the right ventricle of anesthetized dogs. Cardiac output was determined by the dye dilution technique using indocyanine green; during the insertion of the dye curve, arterial blood pressure and an electrocardiogram were simultaneously recorded. During sinus rhythm the mean cardiac output was 2.15 l./min., while during cardiac compression it was 0.44 l./min. (average change in cardiac output, 77%). The arterial pressure pulse during cardiac compression was peaked with a reasonable systolic level. On comparison with the arterial pressure pulse during sinus rhythm, it appears that transmission of the compressing force during cardiac massage contributed to the rise in systolic pressure which did not, therefore, appear to bear with flow. There seems to be little relation between cardiac output and systolic arterial pressure during cardiac compression.