Proceedings of The British Cardiac Society

The Autumn Meeting of the British Cardiac Society was held at the Royal College of Physicians, London, on Thursday and Friday, December 1 and 2, 1966. The President, Shirley Smith, took the Chair at 9.00 am. during Private Business before handing over to the Chairman, Graham Hayward. 178 members and 68 guests were present.

Private Business
1. The President reported with deep regret the deaths of Samuel A. Levine and A. R. R. Cumming.
2. The Minutes of the Annual General Meeting having published in the Journal (1966, 28, 847) were taken as read and confirmed.
3. The Treasurer reported that the balance in the Society's current account was £1,784.0.4 at October 31, 1966, of which £565 was ear-marked to pay for British Heart Journals to the B.M.A.

The deposit account stood at £100, and investments at £529.2.0.
£50 had been donated to the Society of Cardiological Technicians, who had expressed grateful thanks in a letter from the Treasurer which said that the donation was used to buy books for the library, pay for subscriptions to journals, and assist in the running of refresher courses and technical meetings. There had been a saving of £8 on the B.C.S. booklet this year. The congress fund held £310.7.1; £200 was due for refund to the British Heart Foundation in respect of saving from the donation of £2,000 given to assist members to attend the 5th World Congress of Cardiology in New Delhi. Twenty grants of £110 each, and one grant of £50, were made. Refunds from some members who attended the Congress but received help from other sources may be available.

The Thomas Lewis Lecture Fund held £1,173 in investments and £126 would be available to pay for the 1967 Lecture.

Members were reminded that the subscription for the new journal—Cardiovascular Research—was not included in the British Cardiac Society subscription, and that they would not automatically be sent the new journal.
4. The Secretary reported that the Annual General Meeting will be held on May 18, 1967 in Belfast under the Chairmanship of Crozier. Boyle will be the Local Secretary.
5. The Autumn Meeting will be held on November 2 and 3, 1967 in the Royal College of Physicians.
6. The European Congress of Cardiology will be held in Athens (provisional dates are September 8 to 14, 1968), and arrangements are being made for this to fall either immediately before or after the Afro-Asian Society of Cardiology meeting to be held in Tel-Aviv.
7. The VI World Congress of Cardiology is to be held in London in 1970 and the Chairman of the Organizing Committee will be Mounsey.
8. The President reported that he had been in contact with the Ministry of Health over the cardiological technicians' terms of employment, and that as a result of this he advised individual members to make their own approaches to the Ministry if they were in any difficulty.
9. The Secretary reported that the 6th European Congress on Ballistocardiography was to be held in London at the Royal Society of Medicine from April 2 to 5, 1967.

On the evening of December 1, the Society dined together at the Royal College of Physicians, the principal guests being Dr. Hugh Clegg and Brigadier Erelid Cardiff. The President, Shirley Smith, proposed the health of Hayward, the Chairman of the Scientific Meeting, and Hayward replied.

Return of the Electrocardiogram to Normal after Myocardial Infarction

By C. J. Burns-Cox (introduced by K. P. Ball)
The electrocardiograms of 174 men aged 34–59 who had survived 1 year after a first myocardial infarct were followed for 1–4 years. If a recurrence of the infarct occurred, or other illness developed, they were excluded from that time.
The electrocardiograms were assessed by the standards of Simonson for normal characteristics in middle-aged white men. It was found that at 1 year from the infarction 13 per cent, and at 4 years 22 per cent of electrocardiograms were normal by these defined criteria. Of those who developed abnormal Q waves, only 10 per cent had electrocardiograms reverting to normal, compared to 70 per cent of those without abnormal Q waves. The incidence of return to normal of Q and T waves was studied. There was no relation between return of the electrocardiogram to normal and the relapse rate.
This study, though having certain limitations, was thought to be useful in showing the limitation of the electrocardiogram in the diagnosis of past myocardial infarction.
SERUM FREE FATTY ACIDS AFTER ACUTE MYOCARDIAL INFARCTION

By V. A. Kurien, T. W. Greenwood (both introduced), and M. F. Oliver

Kurien and Oliver (1966) have shown that serum free fatty acids (FFA) were conspicuously raised within an hour after acute myocardial infarction and remain high for 48 to 60 hours. Similar levels were seen in unconscious patients soon after the onset of a hemiplegia. These effects were independent of dietary changes and the anxiety of admission to hospital; pain contributes to the elevation of FFA but is not the sole explanation. There was no correlation with blood glucose levels.

The serum FFA have now been studied in 101 patients who were admitted within 24 hours of the onset of symptoms to the Coronary Care Unit at Edinburgh Royal Infirmary on account of acute myocardial infarction. There were 19 deaths, and in 13 the maximum serum FFA were greater than 1100 μEq/l. (normal serum FFA = 525 ± 125 μEq/l.). Ventricular fibrillation, ventricular tachycardia, complete heart block, and atrial fibrillation all occurred with significantly greater frequency in 36 patients with FFA levels greater than 1100 μEq/l., compared with those with moderate increases (800–1099 μEq/l.). There was no correlation between serum FFA levels with serum CPK or aspartate aminotransferase levels in the 101 patients.

It was suggested that acute anoxia depletes the myocardium of its noradrenaline stores, leading to release of adipose tissue FFA. It has yet to be determined whether increased circulating noradrenaline or excess FFA are responsible for the high incidence of serious arrhythmias and deaths. The measurement of serum FFA after acute myocardial infarction is a valuable immediate prognostic index, which may influence the decision about the length of stay of patients in coronary care units.

REFERENCE


FREE ADRENA-LINE AND NORADRENA-LINE EXCRETION IN ACUTE MYOCARDIAL INFARCTION

By Camillo Valori (introduced by J. P. Shillingford)

Free adrenaline and noradrenaline have been estimated by a spectrofluorometric technique in 24-hour urine collections in a series of patients following myocardial infarction in the acute and convalescent phase. The catecholamine values have been expressed in absolute values of μg./24 hours, and, in relation to creatine excretion, μg./mg. creatine. Corrections have been made for dopamine.

Adrenaline and noradrenaline excretion varied from normal to approximately eight times normal in different patients in the acute stage of the disease. In most patients the catecholamine excretion fell towards normal during convalescence but in a few the excretion remained high.

These results have been correlated with the clinical and hemodynamic picture.

BETA ADRENERGIC BLOCKADE IN EXPERIMENTAL MYOCARDIAL INFARCTION

By B. L. Pentecost, and W. G. Austen (introduced)

Theoretically, two benefits might derive from the use of β-adrenergic blocking agents in the treatment of myocardial infarction. First, by reducing cardiac work, the oxygen requirements of the myocardium may be similarly reduced, thus diminishing the degree of myocardial hypoxia. Secondly, these pharmacological agents have been demonstrated to have antiarrhythmic properties possibly independent of their β-adrenergic blocking action. The most apparent danger in their use appears to be myocardial depression from removal of the normal sympathetic drive.

An experiment was devised to assess the effect of prior treatment with propranolol at two dose levels on the outcome of experimental myocardial infarction in dogs. Twenty adult mongrel dogs were studied under pentothal anaesthesia, respiration being maintained with a 50 per cent oxygen in air mixture. Thoracotomy was performed and a ligature placed around the circumflex coronary artery, approximately 0·5 to 1·0 cm. from its origin. Basal measurements were then made of cardiac output, arterial blood pressure, central venous pressure, and myocardial contractility, as measured by a Brodie strain gauge bridge sewn on to the anterior aspect of the left ventricle. Arterial and central venous pressure and myocardial contractility were then monitored together with the electrocardiogram throughout the duration of the experiment. In 7 dogs, propranolol, 0·08 mg./kg. body weight, was then infused intravenously. In 6 dogs, 0·1 mg./kg. propranolol was infused intravenously. The remaining 7 dogs served as controls. Hemodynamic measurements were repeated before ligation of the circumflex artery. Among 7 control dogs, 6 died between 3 and 5 minutes following coronary artery ligation, each dog developing ventricular fibrillation. Among the 7 dogs pre-treated with propranolol, 0·08 mg./kg., only one dog died in this manner, the remaining 6 surviving the full hour of the experiment. Of the 6 dogs receiving 0·1 mg./kg. propranolol, 3 died during the course of the first 10 minutes following coronary artery ligation. The remaining 3 survived the experiment.

INFLUENCE OF PROPRANOLOL ON THE MORTALITY AND COMPLICATIONS OF ACUTE MYOCARDIAL INFARCTION

By D. E. Jewitt, R. Balcon (both introduced), and Samuel Oram

One hundred and fourteen patients with a confirmed diagnosis of acute myocardial infarction took part in a double-blind controlled trial of propranolol. The group of 56 patients received oral propranolol in a dose of 20 mg. 6-hourly for 28 days, and the control group of 58 patients was given placebo tablets. The mortality at 28 days was 23 per cent in the treated group and 24 per cent in the control group. The incidence of heart
failure, shock, hypotension, and hypotension with bradycardia was compared in the two groups. A constant monitoring system was used to detect and record arrhythmias. Ninety per cent of patients in the study developed arrhythmias. Discriminant analysis by computer was used to compare the incidence of specific types of arrhythmia in the treated and control patients.

**Relation of Hypertrophic Obstructive Cardiomyopathy to Subvalvar Mitral Incompetence**

By C. M. Oakley, E. B. Raftery and I. F. Brockington (both introduced), R. E. Steiner, and J. F. Goodwin

Hypertrophic obstructive cardiomyopathy is frequently associated with mitral incompetence which clinically tends to be dominated by features that are attributed to the cardiomyopathy. Haemodynamically, the mitral regurgitation is assigned a role subsidiary to the muscular overgrowth and left ventricular outflow gradient, as it is generally considered to be merely consequent upon distortion of subvalvar structures by the already hypertrophied muscle.

We have recently studied 6 patients with subvalvar mitral incompetence in whom it was exceedingly difficult to determine whether or not there was also an underlying hypertrophic cardiomyopathy, and 7 other patients with typical clinical and angiocardiographic hypertrophic obstructive cardiomyopathy who were haemodynamically similar. Although the initial difficulty in diagnosis was clinical, the diagnosis was unresolved by conventional investigation. We therefore sought further to elucidate the relationship. The left atrial and left ventricular pressures were studied by a technique which permitted recording from two and sometimes three sites within the ventricle, and the appearances of the left ventricle on angiography were compared, including the volume changes and the responses to amyl nitrite and phenylephrine.

No separation could be made, and this led us to assign a more fundamental role to mitral incompetence in hypertrophic obstructive cardiomyopathy instead of one haemodynamically subsidiary to the muscular overgrowth and left ventricular outflow gradient. A possible temporal relationship between the acquisition of non-rheumatic mitral incompetence and the development of hypertrophic obstructive cardiomyopathy is postulated, whereby mitral incompetence leads to premature left ventricular emptying, diminished residual volume, left ventricular-aortic cut-off, catecholamine excess, hypertrophy, a bigger gradient, more catecholamine stimulation, greater emptying, and further hypertrophy.

**Auscultatory Features of Hypertrophic Obstructive Cardiomyopathy**

By R. B. K. Tucker, J. B. Barlow, M. M. Zion, and W. A. Pocock (both introduced by J. F. Goodwin)

The auscultatory findings in 90 cases of hypertrophic obstructive cardiomyopathy were presented. There was a high incidence of ventricular gallop (third or fourth) sounds, left- and right-sided mid-diastolic murmurs, and abnormal splitting of the second sound. The splitting may be paradoxical (reversed) or pseudoparadoxical (partially reversed). An early diastolic murmur was present in 7 patients. Ejection or non-ejection systolic clicks were uncommon.

The phonocardiographic features of the systolic murmur were analysed, and it was shown that the times of onset, maximal accentuation, and offset of this murmur almost invariably occurred earlier in systole than those of the late systolic murmur due to mitral incompetence. The effects of posture, as well as other vasoactive manoeuvres, are useful in differentiating the murmur of hypertrophic obstructive cardiomyopathy from that of mitral incompetence.

**Quantitation of Precordial Movement in Normal Subjects and in Patients with Mitral Regurgitation**

By George C. Sutton and Ernest Craigie (both introduced by Aubrey Leatham)

This study was designed to quantify precordial displacement records, previously not attempted, and to determine whether patients with mitral regurgitation could be distinguished from normal subjects by use of such a method.

Studies were made on 45 normal people and 39 patients with mitral regurgitation. Each subject was placed in the left lateral position at 45° and a record was made of precordial displacement at the point of maximal discrete impulse, using a Hellige transducer. Without altering the sensitivity setting on the recorder, a standard impulse (X), electrically produced, was imparted to the Hellige sensing head, and the deflection produced at this setting was recorded. The heights of the "a" wave (related to atrial contraction), systolic outward movement, and rapid filling wave in each record could then be measured in terms of X. Both systolic outward movement and rapid filling wave gave abnormal measurements even in mild mitral regurgitation without radiological abnormality.

**Endomyocardial Fibrosis and Filariasis**

By I. F. Brockington, A. C. Ikeme, A. J. Willis, and F. A. I. (all introduced by F. Shillingford)

Endomyocardial fibrosis is a disease of unknown aetiology. It is associated with fever, eosinophilia, swelling of the face, and itching of the skin, suggesting allergy. Geographically it belongs in Nigeria to the equatorial forest; its distribution fits closely with that of Loa loa. These clues encouraged us to take up the French hypothesis of "filarial heart disease" and study the incidence of filariasis in endomyocardial fibrosis.

Forty-seven patients with endomyocardial fibrosis diagnosed during life, mainly by angiocardiography and right heart (Telco) catheterization, were compared with 115 controls from the forest zone, using 3 day-and-night blood samples, 4 skin snips, and an intradermal test. The incidence of filariasis in endomyocardial fibrosis was 79 per cent and in controls 46 per cent. The 11 patients with endomyocardial fibrosis who were negative...
all showed some evidence of filariasis when other tests were used. Our results suggest that endomyocardial fibrosis is associated, in Nigeria, with Loa loa. This association might be due to a shared environment, a shared antigen, or an allergic mechanism. The last explanation is favoured by the similarity of endomyocardial fibrosis to Löffler’s endocarditis.

**THE PACEMAKER “HEART SOUND”**

By Alan Harris

In some patients with artificial pacemakers we have noticed an extra sound, always earlier, and sometimes louder, than the usual heart sounds, and it has been found to occur both with epicardial or endocardial systems (Nager et al., 1965). This extra sound can be heard, and more often recorded, 7 msec. after the pacemaker impulse is recorded on the electrocardiogram. In addition, an early outward systolic movement can be recorded in the apex cardiomap, which coincides with the extra heart sound. Nager et al. (1965) concluded that the extra sound was possibly of intracardiac origin related to premature contraction of the heart muscle underlying the electrode. This would imply that heart muscle could behave in an abnormal way in respect to its known electro-mechanical interval, and contravenes the “All or None law” (Bowditch, 1871). In view of the physiological importance of these findings, the pacemaker heart sound has been further investigated.

Six patients with chronic heart block and either an endocardial or epicardial electrode system of pacing have been studied by external phonocardiography, apex cardiography and, in addition, one patient by intracardiac phonocardiography. The results of this investigation showed that the pacemaker extra sound was not due to premature contraction of ventricular muscle but was produced by intercostal muscle contraction resulting from spread of current from the myocardial electrode to the intercostal nerves adjacent to the electrode.

**REFERENCES**


**LEFT VENTRICULAR VOLUME IN AORTIC STENOSIS BY A THERMO-DILUTION AND A RADIOMICROGRAPHIC METHOD**

By James Fleming (introduced), and John Hamer

Full assessment of the behaviour of the left ventricle requires a knowledge of changes in both left ventricular pressure and volume. Estimates of the volume of the left ventricle can be obtained by a thermodilution method and by angiocardiology. In order to assess left ventricular performance in aortic stenosis as a preliminary to valve replacement, we have measured left ventricular volume by each method in 20 patients.

A transseptal catheter was placed in the left ventricle and a thermocouple passed through a catheter in the ascending aorta till the tip protruded immediately above the aortic valve. Cold saline was injected into the left ventricle and thermodilution curves showing a stepwise washout of the indicator were obtained. Cardiac output was measured by indicator dilution. Angiocardiograms were taken on an Elema-Schonander roll film changer at 4 per second, and left ventricular volumes were measured on appropriate films, assuming that the chamber was an ellipsoid.

There was good agreement between the left ventricular end-diastolic volumes measured by the two techniques. Although many patients had a greater left ventricular end-diastolic volume than normal, a large volume was not necessarily associated with a high left ventricular end-diastolic pressure.

We conclude that left ventricular volume measurements are of value in assessing the degree of left ventricular failure in patients with aortic stenosis.

**REVERSIBILITY OF POSTSTENOTIC DILATATION IN THE FEMORAL ARTERIES OF DOGS**

By Margot R. Roach (introduced by Peter Harris)

Previous work (Roach, 1963) showed that poststenotic dilatation would develop in the femoral and carotid arteries of adult dogs within a few days of production of moderate stenosis which caused distal turbulence (indicated by the presence of a thrill and bruit). The vibrations produced by the turbulence weakened the wall (probably the elastin primarily) so that the artery became more distensible. The present experiments were carried out to determine whether the dilatation would disappear after removal of the stenosis. The femoral arteries of adult dogs were stenosed as previously. Each artery was painted with thorium dioxide so that daily x-ray films would indicate changes in diameter. After 10 days to 8 months, the bands were removed. Radiographs showed that in all 11 cases, when removal of the band was associated with disappearance of the murmur, the dilatation disappeared within 8 hours, and the distensibility curve returned to normal. In two arteries where the murmur persisted, though the band was removed, the dilatation increased slightly. In 7 sham-operated controls, where the band was not removed, the dilatation persisted unchanged. There was no evidence of progressive constriction in dogs studied for several months after band removal.

**REFERENCE**


**LEFT VENTRICULAR FUNCTION IMMEDIATELY AFTER HOMOGRAPH REPLACEMENT OF THE AORTIC VALVE**

By Richard Fordham (introduced), and Leon Resnekov

The haemodynamic effects of rapid changes in the mean venous filling pressure of the heart were studied in 7 patients in the immediate post-operative phase, following homograft replacement of the aortic valve for aortic stenosis or aortic incompetence. Left atrial, right atrial, and pulmonary and systemic arterial
pressures were measured during the control period and following rapid serial transfusions of blood, which increased the right atrial pressure by a mean value of 160 per cent above the control values. Arteriovenous oxygen difference, cardiac output, left ventricular stroke work index, and total peripheral resistance were measured. By comparing the relation between the stroke work of the left ventricle and the mean left atrial pressure, left ventricular function curves were obtained for each patient. It was shown that though the left ventricular function was depressed at the time of study, the ventricle was capable of increasing its stroke work index in response to the stimulation of a high venous filling pressure. It was deduced that a mean right atrial pressure of not less than 10 cm. of water (with reference to the anterior axillary line) should be maintained in the immediate post-operative phase to obtain adequate myocardial function.

**THE BALLISTOCARDIOGRAM AND LEFT VENTRICULAR EJECTION IN THE DOG**

By D. C. Deuchar, A. Guz, M. I. M. Noble, D. Trenchard, and P. J. Winter

*The last four introduced*

Ballistocardiograms have been recorded from dogs simultaneously with aortic root blood flow obtained from previously implanted electromagnetic flowmeters. The ballistocardiograph consisted of a horizontal metal cylinder suspended by long wires; its movement was detected by an electromagnetic velocity transducer whose output was differentiated to yield an acceleration record. The dogs were previously prepared with implanted catheters so that atrial and arterial pressures could be recorded. The blood flow signal was differentiated to yield acceleration and integrated to give the stroke volume.

The effects of respiration, tachycardia, and arrhythmia had seriously disturbing effects on the ballistocardiograms, but excellent records similar to those described by other authors were obtained from a conscious dog.

Cardioactive substances in doses too small to affect the circulation when given intravenously were injected through a catheter implanted in a coronary artery. Changes in the peak aortic blood acceleration and flow resulting from these manoeuvres occurring without changes in rate or stroke volume were reflected in concordant changes in the HIJ deflections of the ballistocardiogram. Furthermore, the sensitivity of the ballistocardiogram to change was greater than that of the flowmeter. Changes in peak aortic blood acceleration and peak flow rate correlated closely with known changes in myocardial contractility (Noble, Trenchard, and Guz, 1966).

This study provides direct confirmation of a relation between myocardial contractility and the ballistocardiogram.

**REFERENCE**


**CARDIO-PULMONARY STUDIES IN CHEYNE-STOKES RESPIRATION FOLLOWING MYOCARDIAL INFARCTION**

By D. C. Fluck, J. M. B. Hughes, P. A. Valentine (*all introduced*), and J. P. D. Mounsey

Serial measurements of pulmonary arterial pressure, systemic arterial pressure, right atrial pressure, cardiac output, and pulmonary artery occlusion pressure were made in a patient who developed Cheyne-Stokes respiration for three weeks following myocardial infarction. Results showed that the pulmonary arterial pressure, right atrial pressure, and systemic arterial pressure rose during the hyperpnoeic and fell during the apnoeic phase. The cardiac output also changed in a phasic manner. Simulation of Cheyne-Stokes breathing by voluntary hyperpnoea resulted in similar but less marked changes. Variations of PaO2, PaCO2, and pH showed a lag in relation to the hyperpnoeic and apnoeic phases, as previously described. The administration of oxygen did not abolish Cheyne-Stokes respiration or the changes in the haemodynamic picture associated with this.

**AN INCREASE IN HEART RATE IN RESPONSE TO STIMULATION OF RECEPTORS IN THE LEFT ATRIUM**

By R. J. Linden, and J. R. Ledsome and C. Kidd (*the last two introduced*)

The response to stimulation of left atrial receptors has been the subject of many conflicting reports. In the present investigation the response to such stimulation was consistently an increase in heart rate.

In anaesthetized dogs left atrial receptors were stimulated in one of two ways: (1) by inflating small balloons placed in some pulmonary vein-atrial junctions, and (2) by distending a small isolated pouch of the left atrium containing the receptors. Inflation of the balloons or distension of the pouch always resulted in an increase in heart rate. Action potentials from afferent fibres in the cervical vagus nerve were recorded during inflation of the balloons and distension of the pouch. Records indicated that receptors, proved *post mortem* to be left atrial, were viable, and also showed that the stimuli applied resulted in discharges from the receptors which were within the physiological range.

Possible reflex pathways for this increase in heart rate were examined by division of the vagus nerves at appropriate points in the thorax and the neck, by section of one or both ansae subclavie, and by use of a β-blocking agent to prevent efferent sympathetic nerve activity to the heart. It was shown that the response of an increase in heart rate was reflex, and that the afferent pathway lay in the vagus nerves and the efferent pathway was solely in the sympathetic nerves to the heart.

**FLOWMETER STUDIES OF INTERNAL MAMMARY ARTERY FUNCTION AFTER IMPLANTATION INTO THE LEFT VENTRICULAR MYOCARDIUM**

By J. L. Provan (*introduced by A. Hollman*)

A sine wave electromagnetic flowmeter was used to study internal mammary artery flow in 30 dogs. In 10
animals blood flow was measured before dissection of the artery and again immediately after implantation into the myocardium. In 20 dogs gradual myocardial ischemia was produced by the simultaneous application of an aneroid constrictor to the origin of the anterior descending coronary artery; implant flow was then measured 2 to 36 weeks later. Measurements were made in the resting state, during anoxia, and after administration of amyl nitrite, noradrenaline, isoprenaline, and aramine. Changes in systemic blood pressure and femoral flow were also recorded. Immediately after implantation, 5 ml blood entered the myocardium per minute. Implant flow took 6 weeks to increase after implantation but continued to increase up to 30 weeks after implant. At rest, flows up to 49 ml. a minute into the myocardium were recorded in older implants. The flow pattern in the implant resembled that in a coronary rather than a systemic artery. Implant flow paralleled increases in coronary flow following anoxia or drug administration, though the changes in flow were less than in a normal coronary artery. Changes in implant flow under these circumstances were independent of systemic flow and frequently increased while systemic flow decreased.

A CRITICAL ANALYSIS OF 50 COMPLICATIONS FOLLOWING THE ELECTROCONVERSION OF CARDIAC ARRHYTHMIAS IN 220 PATIENTS

By Leon Resnekov and Lawson McDonald

The electroconversion of supraventricular and ventricular arrhythmias was attempted in 220 patients of whom 180 had atrial fibrillation, 24 had atrial flutter, 9 had atrial tachycardia, and 7 had ventricular tachycardia. Sinus rhythm was established in 193 (88%). Immediate complications occurred in 32 patients (14.5%). Increased serum levels of lactic dehydrogenase or aspartate aminotransferase (SGOT) occurred in 20, and pulmonary oedema, or significant radiographic increase in the size of the heart, in 7. Hypotension, that persisted for up to three hours following the treatment, was recorded in 7, and T wave inversion or changes of cardiac infarction in 5 patients. In 1 patient, persistent multifocal ventricular ectopic beats were shown. Systemic emboli occurred in 2 patients and pulmonary embolism in 1, despite anticoagulant treatment. In 3 patients, third heart sounds and atrial sounds became audible following the shock. Ventricular fibrillation leading to death occurred 36 hours after the shock in 1 patient, and after 48 hours in another; late deaths with changing heart rhythms occurred in 2. Complications were multiple in 12 of the 32 patients. Evidence of myocardial damage following direct current shock was related to higher levels of energy used; it was particularly frequent in patients in whom the myocardium was already diseased.

The significance of these complications was reviewed and their occurrence related to the selection of patients for electroconversion.

BLOOD QUINIDINE MEASUREMENTS IN THE MANAGEMENT OF D.C. DEFIBRILLATION

By N. H. Stentiford, K. H. Chee, R. Saynor (all introduced), and D. Verel

A group of 23 patients with atrial fibrillation was admitted to hospital for assessment and attempted D.C. defibrillation. They were subdivided approximately alternately into two subgroups, one of which received quinidine and the other no quinidine. Two patients converted to sinus rhythm with quinidine alone and a third failed to attend for follow-up, leaving 10 patients of comparable age in each group. Quinidine was given in a long-acting form (quinidine bisulphate) and in a dosage adjusted according to serum levels estimated fluorimetrically.

At this preliminary stage of the trial, throughout which an attempt was made to maintain constant serum levels, it was found that maintenance of quinidine therapy was both difficult and complicated by frequent minor toxic effects. No apparent difference was observed either in the success of cardioversion or in the duration of subsequent sinus rhythm. Admission was prolonged in the quinidine group by the time taken to achieve a satisfactory blood quinidine level.

DETECTION OF IMPENDING FAILURE OF IMPLANTED PACEMAKERS

By Edgar Sowton

Various out-patient tests have been evaluated and applied to the detection of faulty implanted pacemakers before clinical symptoms appear.

Evaluation. Pacing stimuli of increasing amplitude were applied to the hearts of patients in complete heart block via transvenous electrode catheters and the stimulus deflections recorded from limb-lead electrocardiographic electrodes. The amplitude of the stimulus deflection was shown to be a good index of the amplitude of the pacing stimulus applied to the heart. The waveform of the pacing stimulus as recorded from the limbs was photographed from the screen of a calibrated oscilloscope and analysed, measurements of impulse duration and voltage being made directly. The ratio of the voltage at 0·5 msec. to the peak initial voltage was used as an index of the rate of fall of the waveform—the decay ratio. Changes in decay ratio were experimentally correlated with changes in applied pacing voltage and load impedance.

Clinical application. All patients with implanted units attended regularly at a pacemaking clinic and at each visit measurements of pacing rate, limb lead stimulus amplitude, and decay ratio were made. These values were compared with controls obtained before discharge from hospital.

Pacemakers were electively replaced on the following indications: (i) rate change of ± 5 beats/min.; (ii) change in impulse duration of 15 per cent; (iii) fall in stimulus amplitude of 20 per cent; and (iv) change in decay ratio of 25 per cent.

Special tests. New pacemakers are now being used which contain a magnetically actuated reed switch;
when the special magnet is placed on the skin overlying the unit the output is temporarily reduced by 30-50 per cent. If pacing continues, the pacemaker has a reserve of at least this degree: if pacing ceases, the unit is replaced.

**Results.** In the National Heart Hospital pacemaker clinic, covering 30 patients, five units were electively replaced during the past year; these were subsequently shown to be faulty. There were no emergency admissions due to pacemaker failure during this time.

**CAUSE OF DEATH IN PATIENTS WITH CHRONIC HEART BLOCK AND ARTIFICIAL PACEMAKERS**

By Alan Harris, and David Redwood, Michael Davies, and Geoffrey Davies (all introduced)

The cause of death in 26 patients treated with endocardial (19), or epicardial (7) systems of pacing have been analysed. The indications for pacing were Stokes-Adams attacks in 24 patients and chronic heart failure in 2 others. The patients were paced for an average of 13-6 months before death occurred (range, minutes to 54 months). Necropsy included a special study of the conducting tissue by serial sectioning at 6μ intervals, and multiple blocks have been examined from all valves and chambers. The coronary arteries were studied by a simple injection technique and serial blocks. The pacemaker systems were examined for faults in the units, electrodes, and connecting wires. The results were divided into 3 groups.

**Group 1:** Eleven patients died as a direct result of faulty technique in positioning the endocardial electrode, resulting in intermittent pacing (6), septicemia (2), unit failure (2), and a fractured electrode wire (1).

**Group 2:** Ventricular fibrillation resulted in death in 9 patients whose pacing system was probably functioning correctly. Factors included occurrence of unstable cardiac rhythms in competition with the pacemaker, changes in the threshold (power) for pacing, and the underlying myocardial pathology.

**Group 3:** Two patients in extremis died during installation of a pacing system. One patient died from carcinomatosis unrelated to his heart block, one from thrombosis of the aorta, and two patients died without any detectable cause.

**THE AETIOLOGY OF BUNDLE-BRANCH BLOCK**

By Risteard Mulcahy, and Noel Hickey (introduced)

One hundred and sixty cases of bundle-branch block have been studied over a period of five years, including 100 patients with left bundle-branch block and 60 with right bundle-branch block. Only patients with a QRS complex of 0-12 sec. or more were included in the study. Details of age, sex, and aetiological background were reviewed.

Coronary heart disease and hypertensive heart disease, alone or in combination, were highly associated with both forms of bundle-branch block. An unusually high incidence of chronic respiratory disease was noted in patients with right bundle-branch block. The mechanism of this association was considered. Catheter studies on some of these patients showed no evidence of pulmonary hypertension. A number of less common causes of bundle-branch block were noted, and the multifactorial origin of these conduction defects was emphasized. Current views on the pathogenesis of bundle-branch block were briefly reviewed.

The clinical and aetiological backgrounds of patients with bundle-branch block differed in many respects from those of patients with classical coronary heart disease.

**LEFT HEART CATHETERIZATION BY DIRECT VENTRICULAR PUNCTURE**

By J. B. McGuinness, H. Gardner (introduced), and T. Semple

We have explored and modified a technique developed by Levy and Lillehei (1964). A teflon needle catheter is introduced by direct puncture of the left ventricle. Pressure records from this chamber as well as from the aorta and left atrium may be obtained. A No. 7 Gensini catheter is then introduced by Seldinger technique allowing ventriculography and aortography to be performed. The method was evaluated in dogs, using various sizes of catheter and varying numbers of cardiac punctures, the effects of which were examined post mortem. In addition, several dogs had the procedure performed with the chest opened, when the needle, guide wire, and catheter could be seen in situ in the actively beating heart.

With this experience in animals, and the knowledge of the possible hazards of the technique, we have investigated 45 patients. Observations on the value of this technique, together with the complications which we had encountered, were presented.

**REFERENCE**


**ASSESSMENT OF AORTIC VALVE STENOSIS BY LEFT VENTRICULAR PUNCTURE AND LEFT ATRIAL CATHETERIZATION**

By D. Verel and D. G. Taylor

In three patients, measurement of the aortic valve gradient in aortic stenosis by left ventricular puncture and by another technique, at different times, resulted in a difference in the measured gradient. In view of this discrepancy, a series of patients was investigated simultaneously by catheterization of the left ventricle, using the modified transseptal technique, and by left ventricular puncture. Preliminary results have shown good agreement between the ventricular pressure measured by a catheter passed through the mitral valve and by a needle passed through the ventricular wall.
EXPERIENCE WITH A SEPTAL PUNCTURE TECHNIQUE WHICH ALLOWS THE RECORDING OF INTRACARDIAC PHONOCARDIOGRAMS FROM THE LEFT SIDE OF THE HEART
By D. Mendel

Using the technique described by Forman, Laurens, and Servelle (1962), the Telco micromanometer has been passed to the left atrium and ventricle via the punctured atrial septum. In these chambers artefact-free pressure tracings can be recorded from the micromanometer, and the murmurs of aortic incompetence and mitral stenosis and incompetence can be recorded simultaneously with the pressures in the two chambers. This has allowed a number of difficult small points about these murmurs to be resolved. The pressure on both sides of the mitral valve can be measured simultaneously, using the micromanometer together with the side-hole of the catheter and an external manometer.

This technique has been helpful in distinguishing the systolic murmurs of mitral and tricuspid incompetence, the systolic murmurs of mitral incompetence and aortic stenosis, and the diastolic murmur of patent ductus arteriosus from that of aortic incompetence in a patient who was wrongly thought to have a patent ductus arteriosus.

The first differential of the left ventricular pressure can be measured at the same time. The technique also allows the passage of a number 8 side-hole angiocatheter into the left ventricle for angiography without the necessity for arteriotomy.

REFERENCE

HæMODYNA MI C AND PROGNOSIS IN VENTRICAL SEPTAL DEFECT WITH AORTIC INCOMPETENCE
By K. A. Hallidie-Smith (introduced), C. M. Oakley, and J. F. Goodwin

Twenty-three patients with ventricular septal defect and aortic incompetence have been studied. Eighteen have been operated on, an incidence of 8 per cent among 206 surgically corrected ventricular septal defects at the Postgraduate Medical School of London.

The defects were large, and four were supracristal. Aortic incompetence was due to a deformed and usually prolapsed right coronary cusp. Four patients had infundibular obstruction requiring resection. Angiocardiography showed characteristic features, the appearance of the affected cusp being valuable in differentiating the condition from sinus of Valsalva fistula with aortic incompetence. The shunt through the ventricular septal defect from the aorta occurred mainly in diastole, the prolapsed aortic cusp partly occluding the septal defect in systole, thus restricting the systolic shunt and sometimes narrowing the outflow tract of the right ventricle. Aortic incompetence appeared to be acquired in all patients and to be rapidly progressive in a minority, though it was not possible to decide which patients would show this trend. Aortic incompetence was only directly attributable to infective endocarditis in one patient, though the over-all incidence of infective endocarditis was much higher (30%) than in uncomplicated ventricular septal defect.

Complete surgical correction of the defects proved difficult and all patients had some residual aortic incompetence, whether or not the valve was repaired in addition to the ventricular septal defect. There were four deaths, all in patients with severe aortic incompetence.

These results suggest that early surgery is advisable for patients with ventricular septal defect and aortic incompetence.

SOME HÆMODYNA MI C AND BIOCHEMICAL RESPONSES TO EXTRACORPOREAL CIRCULATION
By W. H. Bain, H. I. A. Nisbet, A. C. Forrester (all introduced), and W. A. Mackey

This communication described measurements of electrolytes, arterial PO₂, and peripheral vascular resistance during cardiopulmonary bypass, using unconventionally high flow rates, haemodilution, and ACD blood, with 2 types of oxygenator. They showed little change in the electrolyte composition of the blood with haemodilution, that the urine volume was greatly increased by haemodilution, and that this exerted a protective role. The consequences of large urine volume on potassium balance were described. The relation between pump output and pulmonary vascular resistance during bypass was discussed and data were presented to show a linear relation between pulmonary vascular resistance and oxygen tension in the arterial blood during bypass. The oxygenating capacity of two types of pump-oxygenators was discussed.

CREATION OF ATRIAL SEPTAL DEFECTS BY BALLOON CATHETER IN BABIES WITH TRANSPOSITION OF THE GREAT VESSELS
By Hamish Watson

Many babies with transposition of the great vessels have a circulation that is almost incompatible with extrauterine life, and develop heart failure within a few days of birth. Those who survive long enough for planned surgery are therefore a self-selected group and palliative operations in the neonatal period, though now presenting no great technical difficulty, carry a high mortality because of post-operative biochemical and respiratory problems.

Recent results of corrective surgery and follow-up haemodynamic studies on those who have had redirection of venous return are so encouraging that clearly a greater effort must be made to keep more babies alive long enough to benefit from such treatment. The creation of an atrial septal defect without a thoracotomy has much to commend it, and successful attempts to do this with a balloon catheter suggest that it may be life-saving in the newborn.

This preliminary report was illustrated by a biplane cine-angiographic film showing the interatrial
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septum being ruptured by a contrast-filled balloon catheter in a 2-week-old baby who was in gross and progressive congestive heart failure.

THE NATURAL HISTORY OF CONGENITAL AORTIC STENOSIS

By Maurice Campbell

As more operations for aortic stenosis are carried out, knowledge of its natural history becomes more difficult to acquire. These remarks are based on 77 patients with valvar or subvalvar (one-fifth) aortic stenosis. Only one-quarter were female.

Some patients were first seen in the 1930s, but most in 1947–57. All but 6 of those still living then were reviewed in 1958, and some later up to 1966 through the kindness of my colleagues. The follow-up was terminated by death in 19 patients and by aortic valvotomy in 21 whether these last did well or badly or died. Including all these, the average follow up lasted 9 years, a total of 693 patient-years, and 7 were followed for between 20 and 32 years.

Excluding the heavy mortality (25 ± 5%) in the first year of life, there was not much difference between the first three decades. In each, about 44 per cent were very well, 26 per cent felt well but had unfavourable signs, 22 per cent had serious signs and symptoms, and 8 per cent died. In the 4th decade, the numbers in the first group were halved and those in the last two groups were doubled.

From 40 onwards, only 10 per cent were still leading normal lives and very few maintain this till 60. Probably I have made the outlook at this stage rather worse by excluding several of those who started only with bicuspid aortic valves and developed aortic stenosis later.