

Proceedings of The British Cardiac Society

THE FORTY-EIGHTH ANNUAL GENERAL MEETING of the British Cardiac Society was held in the Natural Philosophy Lecture Theatre of King's College, Aberdeen, on Thursday, April 10, 1969. The President, SIR JOHN MCMICHAEL, took the Chair at 9.00 a.m. during Private Business before handing over to the Chairman, DAVID SHORT.

PRIVATE BUSINESS

1. The deaths were announced with regret of G. E. S. Ward and D. H. Davies.
2. The Minutes of the Annual General Meeting having been published in the *Journal* (1968, **30**, 869) were taken as read and confirmed.
3. The Treasurer reported that the investments of the General Fund remained at £529.2.0 and that income from investments had increased by £40, due to the larger amount held on deposit. The income from members' subscriptions had increased by £89 in the last year. The excess of income over expenditure was £673 in 1968, but the income of the Society would remain static after the maximum number of members allowed by the present rules had been elected in 1970-71. The excess of income over expenditure was necessary to allow for increased expenses and capital growth. No immediate increase in subscription was anticipated unless unexpected expenses arose.
The Congress Fund investments were £732.18.4, with £900 in the Congress Deposit Account and £84.17.7 in cash.
The Thomas Lewis Lecture Fund held £1173.10.2 in investments which brought in an annual income of £60.17.0. There was £58.6.5 in cash at bank at the present time.
The Treasurer showed a chart illustrating the expenses of the Society over the last seven years, which showed a fall in the past year except for an increase in stationery and general expenses due mainly to reordering of stationery in bulk.
4. The following resignations were accepted with regret:
Brock, Cooke, Malley, Thompson.
5. The following two new *Members of Council* were elected:
Counihan and Daley.
6. The following *Honorary Members* were elected:
Brock, Holmes Sellors, Shirley Smith.

7. The following *Extra-Ordinary Member* was elected:
John Towers.

8. The following *Corresponding Members* were elected:

B. G. Barratt-Boyes	New Zealand
Bernard Lown	Boston, U.S.A.
Rolf Rokseth	Norway

9. The following *Ordinary Members* were elected from Associate Membership:

Boyle	Braimbridge
J. P. H. Davies	Dollery
Dulake	Hay
Keen	Linden
MacKenzie	Miller
Norman	F. G. M. Ross
David B. Shaw	Sleight
Donald R. Smith	Desmond G. Taylor

and as new members:

G. S. Dawes	Oxford
Keith Ross (SM)	London
E. J. M. Weaver (SM)	London

10. The following *Associate Members* were elected:

Raphael Balcon	London
David Charles Fluck	London
Derek G. Gibson	London
David Edward Jewitt	London
Alasdair Colin Frame	
Kenmure	Glasgow
Benjamin Wyly Lassers	Edinburgh
Thomas I. McBride	Glasgow
Alastair H. McDonald	London
Hamish Ronald	
Macdonald	Huddersfield
Shahbudin Rahimtoola	London
Robert Gray Shanks	Belfast
Shyam Pratap Singh	Birmingham
George Christopher	
Sutton	London

and as *Associate Surgical Members*:

David I. Hamilton	Liverpool
Tom Hywel Lewis	
Rosser	Penarth, Glam.

11. The following *Overseas Members* were elected:

Hywel Davies	Colorado, U.S.A.
Fulton	Nairobi, Kenya
Resnekov	Chicago, U.S.A.

and the following re-elected:

Davidson	Harries
Parry	Shaper
Somers	Wilson

12. Goodwin (Deputy Chairman of the Organizing Committee) reported that the brochure of the VI World Congress of Cardiology (which would be held at the Festival Hall) would be issued in September 1969. All papers must be submitted through the Congress Office to the British Cardiac Society, and abstracts with the form of submission must reach the Congress Office by January 1, 1970. The deadline for registration for the Congress was May 1, 1970.

The official languages would be English, French, and Spanish.

Goodwin outlined the provisional programme of the Congress. Morning scientific activities would be in the main hall and would be devoted to plenary sessions covering recent advances in important subjects. In the afternoon there would be simultaneous discussions in different theatres in the form of round table discussions and symposia. Following these there would be free communications, up to a total of 120 in number. The morning plenary sessions would be published in full in the *British Heart Journal*, and the abstracts of the round table discussions and free communications in *Cardiovascular Research*.

The opening ceremony would be on Sunday, September 6 at the Festival Hall at 9.0 p.m., and there would be an official reception and concerts at the Festival Hall on the Monday evening and receptions at Royal Colleges and at Embassies on other evenings.

The brochure would contain the full provisional programme, including the Ladies Programme.

All inquires should be addressed to:

VI World Congress of Cardiology,
The Conference Centre,
43, Charles Street,
Mayfair,
London, W.1.

Goodwin concluded by saying that time now did not permit a full description of the activities planned, but that he would be happy to discuss details with anyone who wished during the day.

13. The Autumn Meeting of the Society would be held on November 13-14, 1969, at the Royal College of Physicians, London.
14. The date of the Annual General Meeting in 1970 had not yet been agreed; it might possibly be a joint meeting with the Dutch Society in Amsterdam, but if not it would be held in London, probably on April 9, immediately before the meeting of the Association of Physicians.
15. As the VI World Congress of Cardiology was being held in London in September 1970, there would be no Autumn Meeting of the Society that year.

Demonstrations were held at the Natural Philosophy building.

The Society dined together at the Staff Refectory, King's College, the principal guests being the Vice-Chancellor and Principal of the University, the Dean of the Faculty of Medicine, the Chairman of the North Eastern (Scotland) Regional Hospital Board, the former Chairman of the Board—Lady Baird, and Professor Helen Taussig. The President, Sir John McMichael, welcomed the guests and expressed appreciation of Aberdeen's scientific eminence and hospitality. Short thanked the local organizers.

DECREASE IN BAROREFLEX SENSITIVITY WITH INCREASING ARTERIAL PRESSURE AND WITH INCREASING AGE

By B. Gribbin, T. G. Pickering (*both introduced*), and P. Sleight

We have previously described to the Society a method for measuring baroreflex sensitivity in man. A transient rise in arterial pressure is produced by the intravenous injection of 25-150 μ g. phenylephrine which evokes a reflex bradycardia lasting approximately 30 sec. The systolic pressure of each beat during this rise is then correlated with the pulse interval of the beat following; a regression line is calculated, the slope of which is a measure of the blood pressure—heart rate reflex in m.sec./mm. Hg. This procedure has been used to study baroreflex sensitivity in 55 subjects aged 19 to 66 years, 20 of whom had raised arterial blood pressure (arbitrarily defined as a resting mean arterial pressure > 100 mm. Hg).

The results show decreasing sensitivity of the reflex, both with increasing mean arterial pressure and also with increasing age. Matched for age there was still a significant decrease in sensitivity in hypertensive compared with normal subjects.

These findings appear to parallel what is known of baroreceptor function in experimental hypertensive animals, and also with vessel wall changes with increasing age and hypertension in man. It is possible that the decrease in baroreflex sensitivity seen in the hypertensive subjects represents accelerated age changes.

DIETARY SUGAR INTAKE AND ISCHAEMIC HEART DISEASE*

By T. B. Begg and M. F. Oliver

The dietary intake of sucrose was claimed by Yudkin and Roddy (1964) to be of considerable importance in the pathogenesis of ischaemic heart disease. We have examined this claim, using the same method of dietary history-taking. Forty-nine men who had recently suffered their first myocardial infarct were compared with 94 men of similar age and weight, who had no overt vascular disease and suffered from various medical and surgical disorders.

* This work was undertaken as part of a study planned by the Medical Research Council Working Party on the Relationship between Dietary Sugar Intake and Arterial Disease.

Over the past 5 years the 49 infarct patients had taken an average of 10.2 g. sucrose per cup of tea or coffee, and 77.1 g. sucrose per day in this form; the corresponding intakes for the 94 control subjects were 11.2 g. per cup and 71.0 g. per day; the differences are not significant. The total daily sucrose intake was 163.3 g. for the infarct patients and 145.9 g. for controls; the difference is not significant.

The relation between occupational activity and sucrose intake was examined.

Men with infarcts tended to smoke more cigarettes, but the difference was not significant. Nor was any relation noted between smoking habits and sugar intake.

We are unable to confirm that men with myocardial infarcts have habitually consumed more sucrose than other men.

REFERENCE

Yudkin, J., and Roddy, J. (1964). *Lancet*, 2, 6.

COMPUTER INTERPRETATION OF ELECTROCARDIOGRAMS

By Peter W. Macfarlane, A. R. Lorimer (*both introduced*), and T. D. V. Lawrie

This paper reports the techniques used and results obtained in the computer analysis of electrocardiograms from over 1000 patients. Twelve-lead electrocardiograms were recorded in the ward or cardiograph department on magnetic tape in groups of 3 leads simultaneously. Each cardiogram was then replayed to a digital computer for conversion to numerical format. In each group, analysis was begun by locating the QRS complex which is always characterized by the maximum rate of change of amplitudes. Thereafter, the P and T waves were approximately identified by a similar technique from the data preceding and following the QRS complex. Accurate determination of the amplitude and duration of P, QRS, and T waves was then made by a study of each lead individually. This method, which is also suitable for 3-lead vectorcardiograms, simplifies the logic of wave recognition. A second computer programme was then used to interpret these measurements on the basis of diagnostic criteria agreed on by a panel of cardiologists. Detailed analysis of arrhythmias was not included. It is possible to provide the clinician with a tracing supplemented by a computer print-out detailing all wave amplitudes and durations together with an interpretation.

LEFT VENTRICULAR FUNCTION IN ISCHAEMIC HEART DISEASE

By E. B. Raftery, D. Banks (*introduced*), and S. Oram

Left ventricular function has been assessed in patients with ischaemic heart disease by means of pressure measurements, angiograms, and volume studies of that chamber. The results have been correlated with the clinical condition and the angiographic appearances of the coronary arteries. Left ventricular aneurysm and areas of scarring could be readily identified and corre-

lated well with the coronary arteriogram. The left ventricular end-diastolic pressure did not correlate with the coronary arteriogram, nor with ventricular function as assessed by volume changes. A better correlation was shown between the volume studies and the angiograms, but there were discrepancies in some patients. It is concluded that myocardial function is not invariably impaired in the presence of severe coronary artery disease, and may be affected more by patchy scarring than by large scars which result from obstruction of major vessels.

This presentation was illustrated by a short film.

CIRCULATORY EFFECTS OF BODY ACCELERATION GIVEN SYNCHRONOUSLY WITH THE HEARTBEAT

By A. C. Arntzenius, J. Koops, F. Rodrigo, and H. Elsbach (*all introduced by H. A. Snellen*)

The displacement curve of a ballistocardiogram shows that the sudden motion of blood from the left ventricle into the ascending aorta produces a recoil of the body in the opposite direction. It can be reasoned from this that the reverse should also be possible. This means that a sudden caudal body displacement should cause blood to be moved from the left ventricle into the aorta.

Two separate series of experiments were performed in a study of the significance of this effect. The first experiment employed a hydrodynamic model of the systemic circulation. With rapid displacements, given at the onset of "ventricular contraction", water flow through the tubing increased from 1.9 to 3.0 l./min., and the mean "arterial" pressure increased from 48 to 82 mm. Hg. The second series of experiments used anaesthetized piglets. Measurements of cardiovascular function were made during 2-3 min. periods of tailwards directed body acceleration given synchronously with the heartbeats (BASH). When the circulation of the anaesthetized animals was normal, cardiac output during BASH did not alter. Mean left ventricular pressure however dropped. If the circulatory state of the animals was damaged by anoxia, thereby depressing cardiac output and mean left ventricular pressure to a very low level, both of these rose considerably during body acceleration given synchronously with the heart beat.

USE OF VIDEOTAPES IN TEACHING OF CARDIOLOGY

By A. C. F. Kenmure, R. D. Kennedy, T. N. Cowie, G. O. B. Thomson (*the last three introduced*), and A. J. V. Cameron

Television is now being increasingly used as a teaching aid in many disciplines. This communication describes some of its applications in cardiology teaching at both undergraduate and postgraduate levels. Cardiology is particularly suited to this medium, since visual and auditory clinical signs can be shown along with graphic recordings. If facilities are available, preparation of videotapes is relatively easy and a library of

teaching material can soon be obtained. Moreover, with modern standardized equipment, tapes can readily be exchanged between teaching centres.

Excerpts from three different categories of videotape were presented. In the first, from a series of tapes on auscultation, the murmur of aortic stenosis was heard and visualized simultaneously. Secondly, cannon waves in the neck were shown with the electrocardiogram superimposed. Finally, the technique of cardioversion was shown. Repeated objective testing of students has shown that a televised presentation can be as effective as the orthodox approach. The advantages and disadvantages of videotapes were discussed, and it was concluded that television could be a valuable addition to current teaching methods.

COMPARISON OF DRUGS USED IN MANAGEMENT OF ARRHYTHMIAS DEVELOPING AFTER ACUTE MYOCARDIAL INFARCTION

By D. E. Jewitt, C. J. Mercer, P. J. Hubner
(*the last two introduced*), and J. P. Shillingford

The haemodynamic effects and anti-arrhythmic properties of intravenous diphenylhydantoin (250 mg.), procainamide (4 mg./kg. body weight), I.C.I. 50172 (5 and 20 mg.), and d-propranolol (40 mg.) have been compared directly with those of intravenous lignocaine (2 mg./kg. body weight) in patients with acute myocardial infarction.

While lignocaine and d-propranolol were without serious haemodynamic effects in this dosage, 20 mg. I.C.I. 50172 did cause a rate-dependent fall in cardiac output and central aortic pressure. Both procainamide and diphenylhydantoin were associated with greater falls in aortic pressure and cardiac output than lignocaine.

In patients with frequent ventricular extrasystoles, lignocaine was generally more effective than procainamide or diphenylhydantoin; d-propranolol was relatively ineffective. Ventricular arrhythmias resistant to lignocaine were suppressed on several occasions by diphenylhydantoin. I.C.I. 50172 appears to be of value in slowing the ventricular rate or reverting sustained tachycardias, particularly those of supraventricular origin, against which lignocaine is ineffective. We found d-propranolol to be without significant clinical value in patients with sustained tachycardias after infarction.

ANTI-ARRHYTHMIC ACTIVITY IN RELATION TO CALCIUM BINDING BY SARCOPLASMIC RETICULUM

By Elliot Shinebourne and Roger White
(*both introduced by John Hamer*)

The effect of local anaesthetics and beta-adrenergic blocking agents on the calcium uptake of the isolated sarcoplasmic reticulum has been investigated. Radioactive calcium was used to determine the rate of calcium uptake by sarcoplasmic reticulum obtained from dog myocardium by differential centrifugation.

Myocardial contractility is thought to depend on the myofibrillar calcium concentration. The rate at which calcium is removed from actomyosin complexes by the

sarcoplasmic reticulum determines the rate of relaxation. An increase in the store of calcium in the sarcoplasmic reticulum may augment the force and velocity of contraction.

Propranolol and quinidine, which depress myocardial contractility, significantly decreased the calcium uptake of the sarcoplasmic reticulum, while in equimolar and equipotent doses lignocaine and amethocaine, which do not alter contractility, had no effect.

All 4 anti-arrhythmic agents are local anaesthetics and alter membrane depolarization, but only those with beta-blocking activity depress the calcium uptake of the sarcoplasmic reticulum. The findings indicate that the anti-arrhythmic activity and depression of myocardial contractility produced by these drugs are due to different actions within the cell.

CIRCULATORY EFFECTS OF DIGOXIN, ACID-BASE CORRECTION, AND VOLUME LOADING IN CARDIOGENIC SHOCK

By A. L. Muir, J. L. Anderton, D. M. Lawrie
(*all introduced*), and K. W. Donald

The mechanism of shock complicating acute myocardial infarction is obscure and therefore therapy is empirical. Many therapeutic regimens have been advocated but there are still few data on the effects of such therapy. In 5 patients with severe cardiogenic shock the circulatory response to therapy with digoxin, acid-base correction, and the use of a volume load was examined.

The patients were all in normal rhythm; auscultation did not suggest frank left ventricular failure. Circulatory studies showed a conspicuous reduction in cardiac output and stroke volume, and in all patients there was hypoxaemia and metabolic acidosis.

After the administration of digoxin there was no circulatory or clinical improvement. Similarly, no circulatory change was noted after the administration of bicarbonate. Where THAM was used to correct the acidosis, hypotension increased and arterial PO_2 fell to 25 mm. Hg in 2 patients despite oxygen therapy. In no patient was volume loading associated with an improvement in circulatory status, and in one patient there was a severe reduction in arterial pressure and cardiac output after the administration of 200 ml. 5 per cent laevulose. The lack of response to therapy suggests severe impairment of left ventricular function.

HAEMODYNAMIC EFFECTS OF OUABAIN IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION STUDIED WITH AN ELECTROMAGNETIC VELOMETER

By M. Thomas, C. Mills (*introduced*), D. Jewitt
and J. P. Shillingford

The use of cardiac glycosides in the treatment of advanced heart and circulatory failure in patients with acute myocardial infarction is widespread. Their haemodynamic and other effects in these circumstances are incompletely understood. One particular problem is the difficulty in measuring small changes in blood flow

in low cardiac output states using conventional indicator dilution techniques. With the development of the Mills electromagnetic velocity catheter, it is possible to measure accurately small changes in acceleration and peak velocity of blood, local flow, and vascular impedance in the aorta and other major arteries.

The communication gave results of investigations in patients with acute myocardial infarction, described some effects of intravenously administered ouabain, and discussed the significance of the information in the treatment of patients with circulatory failure after acute myocardial infarction.

CARDIAC PERFORMANCE AFTER DIAGNOSTIC CORONARY ARTERIOGRAPHY

By S. H. Rahimtoola, G. Gau, and M. J. Raphael
(the last two introduced by J. F. Goodwin)

Intracardiac and intravascular pressures, heart rate, cardiac output (determined by the indicator dilution technique), and blood haemoglobin, haematocrit, and mean corpuscular haemoglobin concentration were measured before and after diagnostic coronary arteriography in 19 patients. This involves rapid injection of small quantities (6–8 ml.) of contrast medium (hyperosmolar, sodium and iodine containing, radio-opaque agents) directly into the coronary arteries, repeatedly over 20–45 min., and is thus different from diagnostic angiocardiology where larger amounts of contrast medium are injected in 1–3 sec. into the cardiac chambers or great vessels.

Left ventricular end-diastolic and mean pulmonary artery wedge pressures consistently rose by 1–13 mm. Hg. The mean pulmonary artery, right ventricular end-diastolic, and mean right atrial pressures were unchanged in 3 patients and in the remainder rose by 1–10 mm. Hg. Mean systemic arterial pressure showed no consistent change.

The cardiac output and heart rate did not show any significant change, and the calculated left ventricular stroke work decreased in the majority of patients in whom it could be calculated.

All the patients in this study had heart disease and showed a depression of ventricular function after diagnostic coronary arteriography. To assess whether ventricular fibre length increases after diagnostic coronary arteriography, two dogs were subjected to coronary arteriography till the left ventricular end-diastolic pressure was raised. This was accompanied by an increase of left ventricular end-diastolic volume. Thus, alterations in ventricular function that we have observed can be considered on the basis of the Frank-Starling mechanism.

OBSERVATIONS ON MITRAL STENOSIS

By Robert Walmsley and Hamish Watson

Hearts with mitral stenosis that have not been modified by surgeons or pathologists have become rare. One found recently in a female cadaver at the University of

St. Andrews has been examined in an attempt to elucidate further the clinical anatomy of rheumatic lesions affecting the heart. Thin sections of the entire left heart were prepared and were presented as stained transparencies to illustrate the nature of the cusps in calcific stenosis of the mitral valve. The contribution was further illustrated by the presentation of small portions of the heart which had undergone pathological changes. It was shown that there was a considerable proliferation of the collagenous tissues of the heart, and this was particularly apparent in the gross enlargement of the cusps of the aortic valve.

REOPERATION FOR MITRAL STENOSIS

By A. Gray (introduced), A. H. Kitchin,
A. Logan, and R. W. D. Turner

In a consecutive series of 720 patients submitted to mitral valvotomy for mitral stenosis and followed annually for 1 to 18 years, 200 required further operation in the form of valvotomy (165) or valve replacement (35). Restenosis was the commonest cause (170); other causes were briefly discussed.

The annual incidence of restenosis has been analysed in relation to factors present before the first operation, including age, sex, cardiac rhythm, heart size, associated mitral regurgitation, aortic valve disease, and occurrence of systemic embolisms, to the presence of calcification or sclerosis of the valve and the extent of valvotomy; and to the incidence of post-operative embolism. Comparison was made with patients who did not develop restenosis.

The cumulative incidence of restenosis increased from 5 per cent at 5 years to nearly 50 per cent at 10 years. In patients developing restenosis the average interval between operations was 7 years.

Changes in the state of the valve at operation were analysed. Operative mortality for repeat valvotomy was low and operative complications rare, but the long-term results were less good than after the first operation.

The indications for valve replacement in mitral restenosis were discussed.

RUPTURE OF CHORDAE TENDINEAE OF MITRAL VALVE

By James Fleming and John Hamer

The clinical features, chest x-ray, cardiac catheterization data, and angiocardigraphic appearances of 8 cases of chordal rupture have been reviewed. Open heart operation for severe mitral regurgitation was required in 7.

Two groups can be distinguished on clinical and anatomical grounds. In the first group (3 patients) there was a history of rheumatic fever, atrial fibrillation was the rule, and a conspicuous deterioration in symptoms was the clue to the diagnosis. At operation, in addition to chordal rupture, the valve cusps were noted to be fibrosed and thickened.

The second group of patients had no history of heart disease until rupture of the chordae resulted in the sudden onset of left ventricular failure. The clinical diagnosis of severe mitral regurgitation secondary to chordal rupture is easily made in this group if the patient is seen at an early stage, the chief distinguishing features being the persistence of sinus rhythm, a small left atrium on chest x-ray, and a tall v wave (over 40 mm. Hg) in the left atrial pressure record. The apex cardiogram shows a large presystolic wave and the ultrasound cardiogram shows an abnormally mobile anterior leaflet of the mitral valve.

When the patient is first seen several years after the event, the diagnosis is more difficult. Severe pulmonary hypertension was found in one patient who presented in congestive cardiac failure, and in 2 further patients the left atrium was large and there was atrial fibrillation. One patient, after an attack of pulmonary oedema at the onset, did well with medical treatment.

SURGERY OF SUBVALVULAR MITRAL REGURGITATION

By Stuart Lennox and George Sutton (*introduced*)

In a series of over 300 open operations on the mitral valve at the Brompton Hospital between 1964–1968, the lesion was subvalvar in 24. Nineteen patients had ruptured mitral chordae, while 5 had papillary muscle dysfunction. In 14 patients (58%), it was possible to repair the valve, though 2 of these subsequently had the valve replaced. Valve replacement was performed as a primary procedure in the remaining 10 (42%), using a Starr-Edwards prosthesis or mounted aortic homograft.

Aetiology of chordal rupture included rheumatism, bacterial endocarditis, degeneration of the valve, and trauma. All patients with papillary muscle dysfunction had severe ischaemic heart disease.

In the group with chordal rupture, there was one (5%) hospital death (due to air embolism), and 2 (11%) late deaths (due to bleeding while on anticoagulants, and bacterial endocarditis, respectively). In the group with papillary muscle dysfunction, there were 3 (60%) hospital deaths and one (20%) late death (from progression of ischaemic heart disease).

The presentation and investigations of these patients were reviewed, and the series discussed in relation to the operations of other patients with mitral valve disease.

MITRAL VALVE REPLACEMENT WITH THE INVERTED AORTIC VALVE HOMOGRAFT

By J. Keith Ross

In view of the satisfactory long-term performance of homograft aortic valves when used to replace the aortic valve and the need for a suitable alternative to the prosthetic valve, it was decided to develop a technique for mitral valve replacement using inverted homograft aortic or pulmonary valves.

The use of a semilunar valve for mitral valve replacement is not a new concept and was first described by Gordon Murray of Toronto in 1954.

Since April 1968, 18 patients have had mitral valve replacements using inverted aortic or pulmonary valve homografts in my service at the National Heart Hospital. On 7 occasions there were multiple valve problems and in 6 patients homograft aortic valve replacement was carried out as well as replacement of the mitral valve.

Patients selected for this procedure have suffered from a spectrum of mitral valve disease extending from pure stenosis to pure regurgitation, and detail of the clinical presentation, haemodynamic data, and radiological investigations were given.

The technique of the operation and its development were described.

There has been one hospital and one late death in the series to date. The results of follow-up clinical assessment and of special investigations were presented.

EXPERIENCE WITH A DISC VALVE PROSTHESIS

By Ary Blesovsky

Series of 55 patients with Beall valves used to replace the atrioventricular valve or valves.

	No. of patients	Deaths
Mitral replacement	34	5
Mitral and tricuspid replacement	10	8
Mitral + Starr aortic	2	2
Mitral + tricuspid + Starr aortic	2	1
Tricuspid	7	0

In 2 patients death could be related to the valve.

Haemodynamically the valve was satisfactory though some valves had a small gradient across them. Embolic incidents presented in 3 patients: one mitral + tricuspid had a transient monoplegia, one mitral + Starr aortic died from cerebral embolism after electric conversion of atrial fibrillation to sinus rhythm, and one mitral had a left hemiplegia after replacing the Beall by a Starr valve.

Haemolytic anaemia was the major problem: one patient after triple valve and one after mitral valve replacement had gross haemolysis associated with a trivial leak through a loosened suture; the double replacement who died from cerebral embolism and a second mitral replacement had severe anaemia without any incompetence.

One triple valve and 3 mitral replacements have low haemoglobin levels (75%–80%) but are otherwise satisfactory.

There has been no haemolytic problem with the valve in the tricuspid area.

Because of excessive haemolysis with and without leaks around the prosthesis the Beall valve is unsuitable for routine replacement of the mitral valve but suitable for the tricuspid valve.

SEQUENTIAL ATRIOVENTRICULAR PACING IN MYOCARDIAL INFARCTION COMPLICATED BY HEART BLOCK

By D. A. Chamberlain, R. C. Leinbach, J. A. Kastor, C. Vassaux, and C. A. Sanders (*all introduced by John Hamer*)

Sequential atrioventricular (AV) pacing was compared with ventricular pacing alone on 10 occasions in 9 patients with acute myocardial infarction, complicated by heart block. Bipolar pacing electrodes were positioned in the right atrium and in the right ventricle and the electrodes were connected to a pulse generator which could be set to deliver paired stimuli at an AV interval of approximately 150 m.sec. Arterial pressure was measured by a catheter placed in the radial artery, central venous pressure by a catheter in the superior vena cava, and cardiac output by a dye dilution technique. Cardiac output was augmented consistently by AV pacing, the mean increment being 25 per cent ($p < 0.01$). Systemic arterial pressure was also increased significantly and mean central venous pressure was lowered. All patients showed clinical improvement when AV pacing was initiated, and 5 survived to leave hospital. No complications resulted from the pacing technique. The restoration of a normal AV relation increases cardiac output and arterial pressure when heart block complicates myocardial infarction, and the augmentation can be of prime importance when the output during ventricular pacing is critically low.

METABOLIC ACIDOSIS IN CYANOTIC CONGENITAL HEART DISEASE IN INFANCY

By S. C. Jordan

The acid base state of 52 infants with cyanotic congenital heart disease was investigated. "Arterialized" capillary blood obtained by heel puncture or arterial blood was used to estimate pH, standard bicarbonate, total buffer base, base excess, and PCO_2 by the method of Astrup. Serial studies were carried out on 39 of the patients.

Approximately two-thirds of the patients showed some degree of metabolic acidosis at some stage. There was no close correlation with arterial oxygen desaturation. Moderate or severe acidosis was generally accompanied by clinical evidence of cardiovascular and central nervous system depression. In unoperated patients metabolic acidosis adversely affected the prognosis.

Oral or intravenous administration of sodium bicarbonate produced an improvement in clinical status in about half of the patients treated, and side-effects were uncommon.

It is suggested that monitoring the acid base state in infants with cyanotic congenital heart disease may have a prognostic value, and that correction of metabolic acidosis is of therapeutic value.

MITRAL VALVE IN AORTIC REGURGITATION

By Ronald B. Pridie (*introduced by Celia Oakley*)

The movements of the mitral valve in aortic regurgitation have been examined in 60 patients.

Severe aortic regurgitation results in a raised left ventricular diastolic pressure, which in the absence of mitral stenosis can reverse the end-diastolic atrioventricular gradient and bring about premature closure of the mitral valve. This can be recognized readily in the mitral ultrasound record because closure of the valve occurs before the start of left ventricular systole. This premature closure was particularly conspicuous in patients with acute aortic regurgitation after infective endocarditis, in whom the duration of effective diastole was seen to be drastically curtailed.

The mitral diastolic murmur described by Austin Flint is not uncommon in patients with severe aortic regurgitation in whom the question of associated mitral stenosis may then be raised. When complicating mitral stenosis was present, the ultrasound record showed that the mitral valve remained fully open throughout diastole and just as in patients without aortic valve disease the rate of closure of the valve could be correlated with the severity of the mitral valve obstruction. In patients with aortic valve reflux but no mitral stenosis the ultrasound record showed either normal movements of the valve or premature diastolic closure. In patients with an Austin Flint murmur premature closure of the mitral valve was always found and could be distinguished readily from the prolonged diastolic opening found in patients with mitral stenosis. Subsequent operation or haemodynamic study revealed no false results.

It was concluded that ultrasound readily enabled mitral stenosis to be confirmed or excluded in patients with aortic regurgitation and that the Austin Flint murmur was caused by presystolic closure of the mitral valve and functional mitral stenosis.

PULMONARY AUTOGRAFT REPLACEMENT FOR SEVERE AORTIC VALVE DISEASE

By D. N. Ross, R. Radley-Smith (*introduced*), and Jane Somerville

Since July 1967, 35 patients at the National Heart Hospital, aged 13 to 56 years, with severe aortic valve disease, had aortic valve replacement by pulmonary valve autograft and an aortic homograft valve placed in the pulmonary position (Ross, 1967). Two patients had additional tricuspid and mitral valve operation. Seventeen patients had left ventricular or congestive failure before operation, and the rest had angina or dyspnoea with electrocardiographic signs of severe left ventricular hypertrophy.

Eight patients died at or soon after operation. There were no late deaths. Of the 27 surviving patients, 26 have returned to work and are symptom-free. One patient has dyspnoea due to important aortic regurgita-

tion, but is working 15 months after operation. A further 8 patients have an immediate diastolic murmur but only 3 have alterations in the pulse pressure suggesting aortic regurgitation. In 2 patients investigation has shown pulmonary regurgitation and trivial aortic regurgitation. Patients have been followed up for up to 18 months and no calcification has yet appeared in the grafts. One patient, early in the series, developed an aneurysm of the right ventricular outflow tract which ruptured 6 weeks after operation and was satisfactorily repaired.

In the first 15 patients there were 5 deaths, a further

4 with post-operative myocardial infarction, and high incidence of distressing dysrhythmias. Evidence is available that these serious complications were related to long periods of ischaemic arrest and coronary artery damage in removing the pulmonary valve. With a change of technique, there have been no deaths, no evidence of myocardial infarction, and less dysrhythmias in the last 15 patients.

REFERENCE

Ross, D. N. (1967). Replacement of aortic and mitral valve with a pulmonary autograft. *Lancet*, **2**, 956.