Proceedings of the British Cardiac Society

THE AUTUMN MEETING of the British Cardiac Society was held in London on 13 and 14 November 1975. At the private business meeting the President, J. F. GOODWIN, was in the Chair. For the scientific meeting P. H. DAVISON was in the Chair. The three finalists in the competition for the Young Research Workers Prize were T. Pickering, P. A. Poole-Wilson, and K. M. Taylor. The Prize was won by T. Pickering. A Joint Symposium with the Cardiac Muscle Research Group was held with E. M. VAUGHAN-WILLIAMS in the Chair.

At the Dinner a presentation on behalf of the Society was made by the President to the retiring Executive Secretary, Miss E. G. North. Expressions of appreciation of Miss North's sterling work for the Society were made by Dr. J. P. Mounsey and Professor J. P. Shillingford.

Abstracts of Papers

Paroxysmal re-entry sinus tachycardia

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Paroxysmal re-entry tachycardia may affect the sinoatrial node (PRST): we have carried out intracardiac studies on 7 patients. The presentation was unrelated to PRST in 5 (that of sinoatrial disease, 4; WPW syndrome with paroxysmal tachycardia, 1); in 2 patients with palpitation, electrocardiograms suggested PRST. In 4, sustained PRST could be initiated and terminated by single critically-timed right atrial premature beats over a definite 're-entry zone'; in 3, PRST was poorly sustained, the re-entry zone small, and initiation or termination less reproducible. Rates during PRST ranged from 88 to 156. In all the P wave configuration and atrial activation sequence were virtually identical in PRST and sinus rhythm, high right atrial deflections differing slightly from those of sinus beats in 5. Verapamil prevented re-entry in 2 with sustained PRST and was ineffective in 2 who responded to ajmaline. Carotid sinus massage terminated PRST in 2, prolonged the cycle length without termination in 2, and had no effect in 1.

Sinus node re-entry deserves consideration as a cause of inapparent sinus tachycardia and can be confirmed electrophysiologically.

Torsade de pointes; an atypical ventricular tachycardia

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A typical ventricular tachycardia, often self-limiting, in which, characteristically, the QRS axis fluctuates, has been called 'torsade de pointes'. We have studied 15 patients with this disorder, and have found it as a complication of sinoatrial and atrioventricular block, hypokalaemia, and cardiac ischaemia, and as a toxic effect of various antiarrhythmic agents, phenothiazines, and tricyclic antidepressants. Similar appearances were also seen in one member of a family suffering from hereditary prolongation of the QT interval associated with sudden death. In a further 4 patients, it was induced during cardiac stimulation studies.

This arrhythmia appears to be caused by re-entry rather than enhanced ectopic activity. It can usually be recognized from the surface electrocardiograms, but in doubtful cases the diagnosis may be supported by recognition of possible underlying causes. While some cases are of long duration, it is not benign: we have observed the development of ventricular fibrillation in 5 of our cases. Antiarrhythmic agents usually effective in ventricular tachycardia are unhelpful and may be dangerous; cardioversion, rapid right atrial pacing, or intravenous isoprenaline may be needed, and correction of underlying factors (where possible) is of fundamental importance.

Clinical electrophysiological effects of mexiletine and its mechanism of antidyssrhythmic action

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Mexiletine 2 to 3 mg/kg body weight was given intravenously over 10 minutes. The blood levels at the beginning (0·98±0·1 μg/ml) and end (0·97±0·09 μg/ml) of the electrophysiological recordings were within the antidyssrhythmic therapeutic range (0·5 to 1·5 μg/ml).

No consistent effects were observed on the refractory period of the atrium or atrioventricular node. Significantly the relative refractory period of the His Purkinje system was shortened by mexiletine in all 9 patients in whom it could be measured, from a control value of 437±11 to 398±8 ms (P < 0·05). No significant changes occurred in sinus rate, atrioventricular or His Purkinje conduction time.

This study, therefore, shows that mexiletine has important clinical electrophysiological differences compared with other Class 1 agents, e.g. quinidine, procainamide, and the beta-adrenergic blocking agents. The observed effects on refractoriness in the His Purkinje system may explain the efficacy of mexiletine in re-entrant ventricular arrhythmias and indicates that its use should be relatively safe in patients with infra-His conduction disturbances.
Echocardiography in infective endocarditis
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Valvar vegetations of infective endocarditis have been shown by echocardiography. Six patients were investigated at the National Heart Hospital; 5 showed massive vegetations on the aortic valve and one showed a massive vegetation on the mitral valve. The aortic vegetations were so prominent as to fill the aortic root in diastole, and in 3 cases prolapsed into the left ventricular outflow tract; the mitral vegetations filled the mitral orifice in diastole. Echocardiography also showed thickening of the anterior sinus of Valsalva in one aortic case which proved to be an aneurysm.

The infecting organism was bacterial and different in each case. All 6 patients proceeded to valvar replacement; there were no deaths and the echocardiographic findings were confirmed in each case. The vegetations were florid and friable. The indications for surgery were failure to control infection despite adequate antimicrobial therapy with in vitro control in 3, and intractable heart failure in 3. We conclude that demonstration of massive vegetations by echocardiography indicates that the lesions are so severe as to preclude successful medical treatment and early valve replacement is indicated; furthermore cardiac catheterization before surgery may be hazardous.

Within-patient test of reliability of echocardiographic left ventricular dimensions and cavity and wall volume estimation
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This test is based on the incompressibility of myocardium which dictates that left ventricular wall volume remains constant throughout the cardiac cycle.

The volumes occupied by the left ventricular cavity, by ventricular cavity plus wall, and hence by ventricular wall alone, can be estimated from echocardiographic measurements of wall thickness (W) and cavity dimension (D). A discrepancy between wall volume estimates at end-systole and end-diastole indicates that either the dimensions are unreliable or that the assumptions of ventricular geometry on which the volume calculations are based are incorrect.

Forty patients were studied. Wall volumes were determined by assuming an ellipsoid shape \((4/3\pi(W+D/2)(W+L)-(D/2)L)\); the semimajor axis (L) being predicted from a regression equation relating angio-graphic and ultrasound dimensions, and also by the cube method \((2W+D)^3-D^4\).

Using the ellipsoid formula, values for wall volume ranged from 92 to 719 ml; end-systolic and end-diastolic wall volumes correlated closely \((r=0.95, \text{mean difference}=6.7 \pm 1.0 \text{(SEM)}\%\), supporting the reliability of the echocardiographic measurements. In the 7 patients with very large end-diastolic cavity dimensions \((6.5-8.6 \text{cm})\), however, correlation was poor \((\text{mean difference}=18.5 \pm 2.7 \text{(SEM)}\%\). Using the cube method, wall volumes were greater \((102-986 \text{ml})\) but correlation was similar \((r \times 0.91, \text{mean difference}=7.2 \pm 1.1 \text{(SEM)}\%\).

This test can be useful for assessing the reliability of echocardiographic ventricular measurements in individual subjects.

Description of segmental wall motion abnormalities in patients with coronary artery disease
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Forty patients being investigated for chest pain were studied at routine cardiac catheterization by performing 3 sequential left ventriculograms at rest, after pacing and after nitroglycerine.

Using single plane RAO angiograms and dividing the ventricle into 8 segments based on a conventional long axis measurement, normal values for systolic segment motion have been defined in 10 patients without coronary disease. The change in segment areas between end-diastole and peak systole varied from 45 per cent \((SD=11\%)\) in the posterior wall of the left ventricle to 65 per cent \((SD \times 10\%)\) on the high anterior wall and were reproducible between sequential studies.

Patients with coronary artery disease who developed angina during pacing showed changes in systolic wall motion in territories of poor coronary supply which were reversed by nitroglycerine. Assessment of changes in wall motion by long axis measurement depends on the position of the apex which may alter during ischaemia, and a comparison between conventional segmental description and a technique depending upon radial measurements from the centre of gravity of the single plane view was presented.

Heart scanning with \(^{99m}\text{Tc}\) stannous pyrophosphate
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\(^{99m}\text{Tc}\) stannous pyrophosphate is a radioactive marker which is cheap, safe, and generally available. It labels freshly damaged but not normal or scarred myocardium, thus giving a positive scan in recent infarction.

Using simple mobile apparatus, heart scans have been obtained at the bedside in 52 patients. The anterior scan was the most useful, an abnormal result being indicated by asymmetry about the sternum.

In acute myocardial infarction, positive scans were found between 17 hours and 18 days after the onset of symptoms. When the infarction was definite on other grounds, the scan was positive in all of 15 patients, when likely in 12 of 16, and when doubtful in 9 of 21 patients.
Heart scanning with "Tc" stannous pyrophosphate is useful in the assessment of suspected myocardial infarction. Preliminary studies suggest that it is reliable, sensitive, and of particular value when previous damage makes electrocardiographic interpretation difficult, and when raised enzyme levels cannot be attributed with confidence to the heart.

Prognosis of early myocardial ischaemia

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A simple screening examination was made of 16 016 male civil servants aged 40 to 59 and included a standardized chest pain questionnaire, electrocardiography, and assessment of the main coronary risk factors. In the ensuing 5 years, 181 of these men died of coronary heart disease. 'Angina', with a prevalence of 4.3 per cent, carried a mortality ratio of 5.4 and predicted 19 per cent of these deaths. For a history of 'pain of possible infarction' the prevalence was 6.5 per cent and mortality ratio 5.8; 29 per cent of deaths from coronary heart disease were predicted. 5.6 per cent showed electrocardiographic abnormalities suggesting ischaemia, with a mortality ratio of 7.0 and prediction of 29 per cent of deaths. Altogether 14.3 per cent of men were positive to one or other of these indices of suspect ischaemia; this combined category carried a mortality ratio of 6.4 and predicted more than half of all the 5-year deaths from coronary heart disease. Among the men with suspect ischaemia the predictive significance of blood pressure, plasma cholesterol, glucose tolerance, overweight, and cigarette smoking seemed to be as great as in their contemporaries without ischaemia.

These findings were discussed in relation to policy on the detection and management of early myocardial ischaemia.

Fibrinolytic activity and risk factors for coronary heart disease

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The response of the fibrinolytic system to stress (venous occlusion) has been examined in healthy middle-aged men who had consistently shown either high or low resting fibrinolytic activity (FA) on repeated studies over several years. There was a close relation between resting FA and the response to venous occlusion, though subjects with low resting FA showed considerable variation in response. The group with high resting FA and a uniformly good response to stress had significantly lower levels of plasma cholesterol and triglycerides, lower Quetelet's index, lesser skinfold thickness, and lower blood pressure. This group also included more non-smokers and more physically active subjects. Subjects who had never smoked clustered in the area of high response to stress. When the low resting activity group was divided into those with low or high response to stress, differences in several risk factors were present and showed an interesting trend.

Hypotheses relating the risk factors to atherosclerosis and to coronary heart disease are presented and it is suggested that the response of the fibrinolytic system to stress may provide an index of atherosclerosis.

Comparison of antihypertensive effectiveness of beta-adrenoceptor antagonists with different pharmacological properties

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There is little information on the comparative antihypertensive activity of beta-adrenoceptor antagonists with different spectra of ancillary pharmacological properties. Both the immediate and the long-term blood pressure lowering activity of propranolol, oxprenolol, practolol, tolamolol, and metoprolol were, therefore, compared at rest and during treadmill walking in patients with stable, uncomplicated essential hypertension (diastolic pressure 110 to 120 mmHg (14.6 to 16.0 kPa)). The trial was designed as a randomized double-blind cross-over study with particular respect to dose-response relations.

With all drugs there was a similar significant reduction in blood pressure within an hour, which was maintained for over 8 hours, greater for systolic than diastolic pressure and greatest during exertion. There was a significant relation between the reductions in systolic pressure and the logarithm of dose of each drug. These effects were enhanced during long-term treatment where again there was a logarithmic relation between dose and blood pressure lowering activity.

It is concluded that the blood pressure lowering activity of beta-receptor blocking drugs is greatest on systolic pressure, greatest during exertion, enhanced by continuous treatment, and independent of their ancillary pharmacological properties.

Effect of selective and non-selective beta-blockade on coronary circulation in man

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Eight patients undergoing cardiac catheterization for suspected coronary artery disease were studied. Myocardial blood flow was measured using a Ganz thermodilution catheter placed in the coronary sinus. Systemic arterial pressure and arterial blood samples were obtained from a brachial artery cannula.

Myocardial arterial and coronary sinus blood samples, systemic blood pressure, and heart rate were obtained before and during pacing to angina threshold, ST-T changes or 150 beats/minute. After the administration of 0.1 mg/kg propranolol (4 patients) or atenolol (4 patients) measurements were repeated at the achieved heart rate and at the heart rate immediately before drug administra-
tion. The heart was then rapidly paced to the same rate as previously.

Neither the non-selective beta-blocker propranolol, nor the relatively selective atenolol produced significant coronary vasoconstriction. Changes in coronary resistance were related to changes in myocardial oxygen consumption, myocardial oxygen extraction remaining unchanged.

Thyroid stimulating hormone levels in atrial dysrhythmias

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A study of 80 clinically euthyroid patients who had cardiac disease and/or atrial dysrhythmias has been made, in which the level of thyroid stimulating hormone (TSH) and the response to thyrotrophin (TRH) has been estimated in conjunction with clinical assessment and conventional thyroid function tests, to detect occult thyroid disease. Forty of the patients had established atrial fibrillation, 20 others had paroxysmal atrial dysrhythmia, and 20 more had heart disease without dysrhythmia.

Although 3 patients were found to have true hyper-thyroidism, the overall results were contrary to what had been expected. Twelve cases were disclosed (10 with chronic atrial fibrillation, 2 with paroxysmal dysrhythmia), where the basal TSH level and its response to TRH was exaggerated, despite the absence of thyroid hypofunction as judged clinically or from the standard tests of thyroid function. No patient from the group with heart disease alone had raised TSH levels. It is apparent that any patient with established atrial fibrillation from any cause is likely to have abnormally high TSH levels, both basally and after TRH stimulation, compared with those with paroxysmal dysrhythmia or those with heart disease but without rhythm changes. Two possible explanations were discussed: i) that thyroid autoimmune disorder may be closely linked to atrial muscle damage; ii) that circulating metabolites of thyroid hormones partially inhibit thyroid function but affect cardiac muscle excitability.

New observations on systolic anterior motion of mitral valve leaflets in hypertrophic cardiomyopathy

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Eighteen patients with hypertrophic cardiomyopathy were scanned with the Siemens 'Vidoson', a real-time dynamic B-scanner, only recently introduced for routine two-dimensional cine-echocardiography. The diagnosis was proven by angiography and high fidelity pressure measurements in 12 patients, and in the remainder was made by both clinical and M-scan echocardiographic examinations. This study shows that the two-dimensional cross-sectional display of the Siemens 'Vidoson' is superior to the M-scan technique for the diagnosis of hypertrophic cardiomyopathy, since the latter is dependent on beam direction.

Furthermore, real-time scanning, being extremely rapid, allows true anatomical relation within the heart to be seen, and quantitative assessments of the exact location and extent of the septal bulge to be made.

In particular, the motion of the prominent hypertrophied papillary muscles is easily seen. Evidence is presented to support the contention that the characteristic systolic anterior motion is caused by an exaggerated upward motion of the chordae tendineae, brought about by synchronous displacement of the rigid left ventricular posterior wall and attached papillary muscles in systole.

The results suggest that the systolic anterior motion is produced by reflection of the chordae tendineae, which traverse the sound beam at certain phases of the cardiac cycle, rather than by the tips of the mitral leaflets themselves. A typical example was illustrated by a cine film.

Arterial oxygen tension and response to oxygen breathing in differential diagnosis of congenital heart disease in infancy

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Arterial oxygen tension was measured from radial artery samples in 285 infants referred for cardiological investigation. Values obtained during air breathing in infants with congenital heart disease showed considerable overlap between 'cyanotic' and 'acyanotic' groups and are of limited diagnostic use. In contrast, values obtained while breathing oxygen in concentrations of over 80 per cent, measured in 182 infants, allowed clear differentiation between these groups.

All infants with 'acyanotic' but only 2 of 109 with 'cyanotic' lesions achieved an arterial oxygen tension of more than 150 mmHg (20.0 kPa). In the 'cyanotic' group, the response to oxygen breathing was significantly greater with common mixing situations and the hypoplastic left heart syndrome than with either pulmonary outflow tract obstruction or transposition of the great arteries. Of 23 infants with primary lung disease, suspected initially to have cyanotic congenital heart disease, 7 achieved arterial oxygen tensions of more than 150 mmHg (20.0 kPa) during oxygen breathing and, on this basis, cardiac catheterization was not performed.

We, therefore, submit that measurements of the arterial oxygen tension while breathing high concentrations of oxygen should be routinely performed in the initial assessment of sick infants with suspected congenital heart disease.

Thromboembolic complications after aortic valve replacement: comparative study

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The long-term results after aortic valve replacement with either a Starr Edwards silastic ball prosthesis (model 2320, cloth covered cage), or Björk Shiley tilting disc
prosthesis, have been analysed, with particular attention to complications or death as a result of thrombosis of the valve or embolism from it. The incidence of thromboembolic complications in these two groups was compared with that of a similar sized group of consecutive patients who had undergone homograft replacement of the aortic valve. Most patients undergoing Starr valve replacement were treated for about three months with phenindione which was subsequently discontinued. On the other hand, patients in the Björk Shiley group received no postoperative anticoagulation, and no anticoagulation was used with the homograft group.

The incidence of thromboembolism in the homograft group was negligible. A small number of patients with Starr valves had serious thromboembolic complications. However, non-anticoagulated patients with the Björk prosthesis in the aortic position had an unacceptably high incidence of emboli and/or valve thrombosis. This incidence was so high that we have discontinued this study group and anticoagulated all patients with Björk Shiley prosthesis.

Myocardial infarction in patients undergoing coronary artery bypass grafting

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The incidence of perioperative myocardial infarction in 250 consecutive patients undergoing coronary bypass grafting was studied. Patients who had additional procedures (excision of aneurysms or infarcts or valve replacement) were excluded from the study. Myocardial infarction was diagnosed by set electrocardiographic criteria. Sixteen patients (8%) developed evidence of new myocardial infarction. Of these, 1 died early and 1 late. The site of the infarct was in the territory of a grafted vessel in all patients.

Cardiac enzymes, including the cardiospecific isoenzyme CK-MB, were evaluated in 50 patients. These were abnormally high in 8 patients (16%).

Sixteen patients with perioperative infarction were followed up for periods of up to 5 years. All patients showed symptomatic improvement.

Repeat coronary and left ventricular angiography showed patent grafts to the infarcted area in 15 and occluded in 3 (a patency rate of 83%). The infarct did not influence left ventricular ejection fraction in 14 and produced reduction in the other 4. This was not related to graft patency.

It is concluded that perioperative infarction is not related to graft patency and influences left ventricular function in a proportion of cases.

Currently we are investigating the use of beta-blockers and hypotensive agents during and immediately after operation to reduce the incidence and severity of this complication.

The watershed: a factor in coronary vein graft occlusion

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Of 215 patients with one or more aortocoronary saphenous vein grafts, all 16 survivors with a poor clinical result and 34 others with a good result were investigated by angiography to determine graft patency, half within one month of operation and half after a longer interval. Seventy per cent of the grafts were patent but the patency rate of grafts inserted into arteries with a proximal occlusion was found to be significantly higher (P < 0.05) compared with those inserted into arteries with a proximal stenosis; this difference was greater (P < 0.025) in those investigated after more than one month. A watershed was shown at the anastomosis in one patient, with a graft inserted into a non-occluded artery: the distal run-off appeared good, but reflux of contrast up the coronary artery into the aorta occurred when injecting into the graft, and vice versa. It is suggested that this watershed is operative to a minor degree in all grafts inserted into non-occluded arteries and, by causing stasis at the anastomosis, is the explanation for the higher incidence of graft occlusion in this group. Ligation of the coronary artery proximal to the anastomosis may, therefore, be necessary to achieve the highest patency rate.

Total correction of tetralogy of Fallot postoperative haemodynamic assessment

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Postoperative haemodynamic evaluation was performed on 15 patients approximately 11 years after total intracardiac repair of Fallot's tetralogy. At the time of the postoperative study all the subjects were asymptomatic.

The cardiac output and right heart pressures were measured at rest and after 5 minutes' sub-maximal exercise on a bicycle ergometer. It was shown that the degree of residual obstruction of the right ventricular outflow tract was slight; the RV-PA systolic gradient exceeded 20 mmHg (2.7 kPa) at rest in only 3 patients. The mean cardiac index at rest was 5-4 l/min per m² which increased to 8-3 l/min per m² during exercise. The response of the cardiac output to exercise was subnormal in one patient but either normal or supranormal in the others. Pulmonary regurgitation was present in 5 of the 15 patients but these patients showed no significant difference in their cardiac function compared to those with competent pulmonary valves.

Six of the patients in the present study had cardiac catheterizations performed 1 to 4 years after operation. The later study shows remarkably little change in their haemodynamic status. These results are encouraging but the long-term effects of the right ventricular pressure and volume overload are unknown.
Homograft reconstruction of right ventricular outflow tract in pulmonary atresia—late results

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The 24 consecutive survivors of right ventricular outflow reconstruction with an aortic homograft for pulmonary atresia and extreme Fallot and failed correction of Fallot have been studied 3 to 10 years after operation. There have been no late deaths, no haemolysis of thromboembolism, and no patients have been lost to follow-up. One patient, the first patient operated on aged 9 years, had reoperation after 9½ years for right ventricular obstruction which had occurred because the homograft was the smallest used (Size 1) and had not grown with the boy; right ventricular failure had occurred in the eighth postoperative year.

All patients lead normal asymptomatic lives and two had uncomplicated pregnancies, delivering normal children. Pulmonary regurgitation was present in 70 per cent and did not progress. Pulmonary valve closure (P2), from the aortic homograft, remained clear, despite radiological appearances of calcium in the graft aortic wall in 22 patients.

Cardiac catheterization was performed in 21 and repeated in 10. Resting gradients across the graft were 5 to 35 mmHg (0.7 to 4.7 kPa) and did not increase except in the first patient.

The difference in these results and some transatlantic reports may be explained by different methods of sterilization and selection of patients. From this study we see no reason to abandon the use of homografts for reconstruction of the right ventricular outflow tract in cyanotic congenital heart disease.

Intra-aortic balloon assistance in cardiogenic shock after myocardial infarction or cardiac surgery

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Sixty-five patients with cardiogenic shock were referred for consideration for intra-aortic balloon assist (IABA). Fifty-two were accepted, 30 patients with myocardial infarction and 22 patients who could not be weaned off cardiopulmonary bypass. Twenty-one of the accepted patients were referred from the region.

Criteria for acceptance after myocardial infarction were a systolic arterial pressure of <90 mmHg (12.0 kPa) persisting for >2 hours, with a urine output <20 ml/hour, and cool peripheries or mental clouding. In 8 of the 30 patients death occurred within one hour and before IABA could be established. In the 22 patients established on IABA, the initial mean arterial pressures were 80 ± 2.5 mmHg (10.6 ± 0.4 kPa) systolic, 66 ± 2 mmHg (8.8 ± 0.3 kPa) mean, and 55 ± 2 mmHg (7.3 ± 0.3 kPa) diastolic. Sixteen patients showed clear haemodynamic improvement during IABA, of whom 5 survived to leave hospital; a hospital survival of 23 per cent.

Appropriate early angiography and coronary artery surgery on IABA could probably further improve this survival rate. The study clearly showed in 15 patients that IABA could be satisfactorily established in a peripheral hospital, continuing during transport and subsequent coronary angiography. The gain in survival from cardiogenic shock complicating myocardial infarction should be compared with the hospital survival of 6 from 16 (38%) cardiac surgical patients who could not initially be weaned from cardiopulmonary bypass.

‘Isolated’ pulmonary valve stenosis: a possible misnomer

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Histological studies have been made on the myocardium of both ventricles, the coronary arteries, and the ascending aorta in 23 specimens with ‘isolated’ pulmonary valve stenosis from children aged 6 days to 9 years. Serious abnormalities in these areas have been found in 23. Myocardial infarction and dysplasia in both ventricles was common and widespread obstructive changes in the coronary arteries were found in 17. The media of the ascending aorta were abnormal in 12.

Clinical scrutiny of a different series of 10 children aged 8 months to 3 years after open pulmonary valvotomy for simple pulmonary valve stenosis showed examples of unexplained pulmonary oedema, persistent low cardiac output, subsequent left ventricular enlargement, and systolic hypertension.

In an older group of 43 patients aged 4 to 20 years who had pulmonary valvotomy, there were 7 who developed obvious left-sided problems for no apparent reason. Two developed the features of classical obstructive cardiomyopathy 6 and 8 years later.

It is suggested that some of the unexpected clinical features in pulmonary valve stenosis may be explained by the pathological abnormalities. This also suggests that pulmonary valve stenosis may be on occasion part of a more widespread cardiovascular disease. This should be suspected in infants with severe pulmonary valve stenosis, those who present with thickened tricuspid valves, and in children with stigmata of congenital abnormalities outside the cardiovascular system.

Experience with an extractable aortic flow probe after open heart surgery

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Precise, sensitive haemodynamic monitoring is essential in the intensive care period after cardiac surgery. With the innovation of the Williams-Barefoot extractable electromagnetic aortic flow probe, a system has been developed for post-cardiac surgical monitoring which provides continuous on-line information concerning
Tricuspid atresia—morphology and classification

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Eighty-six hearts have been examined in which the morphological tricuspid valve was either absent or imperforate. Five specimens possessed an imperforate valve situated between the right atrium and a poorly formed right ventricle. The right ventricle contained tension apparatus of the valve. The remaining 81 hearts all had the ventricular morphology of 'primitive ventricle', 78 possessing an outlet chamber and 3 being examples of tricuspid atresia in primitive ventricle without outlet chamber. All the primitive ventricular hearts were from solitus individuals and 75 had atrioventricular concordance. In the 6 examples with atrioventricular discordance, the left atrium was blind-ending and a mitral valve was placed between the right atrium and the primitive ventricle. The outlet chamber was left sided. Striking variation was noted in ventriculo-arterial relations. Fifty-four hearts had normally related great arteries and 18 had transposed arteries. Three examples of anatomically corrected malposition, one example of double outlet malposition from the outlet chamber, and 5 examples of single arterial trunk were observed. The single trunk was a common truncus in one instance and the aorta in the remainder. Pulmonary stenosis was encountered in 40 hearts. It was most frequently the result of a constrictive bulboventricular foramen in the presence of normally related great arteries. A constrictive foramen in association with transposed arteries was seen 7 times with aorto isthal hypoplasia. The findings emphasize the necessity of adopting a segmental approach in order to describe adequately hearts with tricuspid atresia.

Congenital aortic valve disease with rupture of mitral chordae tendineae

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A new clinical entity is described in which free aortic regurgitation from congenital aortic valve disease causes rupture of the chordae to the anterior leaflet of the mitral valve. Comparing these patients with those who rupture mitral chordae in association with rheumatic heart disease and patients with spontaneous chordal rupture, differences were evident. The cause of chordal rupture in these cases was in part progressive left ventricular dilatation, direct trauma to the anterior cusp of the mitral valve, and possibly a genetic factor.

Seven cases are described, all men between the ages of 45 and 63 years (average 52). None had a history of rheumatic fever and none had active infection. The typical picture was of acute mitral regurgitation into a small left atrium with severe pulmonary oedema which was often resistant to medical treatment. All patients were treated surgically. Anatomical and histological features of the affected valves are described. Early
operation with replacement of the aortic and mitral valves is recommended if the present high mortality (5 out of 7, 70%) is to be reduced.

**Angiocardiographic/pathological correlations in congenital mitral anomalies**

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The angiocardiographic findings were reviewed in 21 patients with congenital mitral valve disease in whom the exact anatomical diagnosis was known from surgery or necropsy. Patients with corrected transposition and simple atroventricular canal defects were excluded. Contrast medium had been injected immediately downstream to the mitral valve in each patient.

No precise anatomical diagnosis was made in five patients (24%), all of whom had a cleft anterior leaflet or congenital short chordae. In the remainder, the specific diagnosis could have been made by downstream injection alone in 15 (71%). Upstream injection was performed in 13 of these but would have made the precise diagnosis in only 3 (23%). In one patient both upstream and downstream injections were necessary.

The anomalies giving rise to characteristic angiocardiographic appearances were parachute valve, atresia, annular hypoplasia, dysplasia, anomalous arcade, and anomalies associated with subaortic stenosis.

Thus injection downstream to the valve is more valuable than upstream injection, because information can be obtained not only about tensor apparatus, but also about annular dimensions and the patency and competence of the valve. The ability to show the relation of the mitral annulus to the primitive ventricle had important practical and theoretical consequences in two patients with mitral atresia and primitive ventricle.

**Isotope electrocardiograph-gated angiocardiology**

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A technique has been devised for assessing cardiac structure and function by studying one cardiac cycle after an injection of 

\[^{99mTc}\text{HSA} (0.15 \text{ mCi/kg})\]

into an antecubital vein, using a gamma camera and low energy collimator. Synchronized triggering of the R wave of the electrocardiogram is used in conjunction with a small computer for data storage and processing. The anatomy of the heart is delineated by sequential passage of the bolus of isotope through the various chambers.

Thirty one patients have been studied: 8 with hypertrophic obstructive cardiomyopathy, 9 with valvular disease, 6 with left-to-right shunts, 4 with ischaemic heart disease and dyskinesia of the left ventricle, and 4 with normal hearts.

The computer has been programmed to generate time-activity curves to obtain volume and flow values by isotope dilution curves. The radiocardiograms thus made have permitted measurement of cardiac output, pulmonary transit time, shunt ratios, ejection fractions, and ventricular volumes.

The radiation dose involved is 1 per cent of that during angiocardiology using contrast medium.

**Importance of patient-selection in evaluation of cardiac ambulance service**

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This paper describes an evaluation of a cardiac ambulance service manned by trained ambulance personnel.

The overall mortality of patients with heart attacks, including those dead on arrival at hospital and those dying in hospital, was 40 per cent in the group carried by the cardiac ambulance, compared with 51 per cent in the group carried by the routine ambulance service when the cardiac vehicle was not available. However, the apparent reduction in mortality associated with the cardiac ambulance was balanced by a high mortality (68%) among patients brought to hospital by a routine ambulance at times when the cardiac vehicle was available but not used. This high mortality was associated with a short duration of symptoms, a heart attack occurring away from home, and a call for an ambulance by a member of the public rather than a general practitioner.

It is suggested that there was inadvertent selection of low risk cases for transport by the cardiac ambulance, and the difficulties of assessing the value of such services will be discussed.

**Atrial receptors and a possible naturally occurring diuretic agent**

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Atrial receptors are believed to be one link in the regulation of blood volume. These receptors are known to discharge less frequently in heart failure in animals (Greenberg et al., 1973). In normal animals stimulation of atrial receptors produces a diuresis which is thought to be mediated by a diuretic agent (Kappagoda et al., 1975). The present report is concerned with experiments in which a bioassay was used as a possible means of detecting this agent.

Distension of balloons to stimulate atrial receptors in anaesthetized dogs resulted in a diuresis; plasma samples were obtained. Malpighian tubules of *Rhodnius prolixus* (blood sucking bug) were suspended in test and control plasma; the mean difference in secretion of the tubules (test control) was \(27 \pm 7.1\) per cent (SEM; \(P < 0.01\)). A similar result was obtained after extracting the plasma with butanol. These differences were abolished by cutting or cooling the vagi.

It is concluded that the bioassay using the Malpighian tubules of *Rhodnius prolixus* is capable of detecting a difference between control and test plasma. This difference, like the increase in urine flow, is dependent
on the presence of intact vagi. It is suggested that the substance detected by the tubules may be the hypothetical diuretic agent.

References

Autonomic features of supraventricular tachycardia
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Forty patients with electrocardiographic evidence of paroxysmal supraventricular tachycardia were investigated, using previously introduced autonomic methods of evaluating atrial pacemaker function (Dighton, 1974). The atrial rate responses to a Valsalva manoeuvre and an intravenous bolus dose of isoprenaline (5 μg/70 kg B.W.) were obtained with patients in sinus rhythm and compared with the responses of 22 normal control subjects.

On the basis of routine electrocardiographic analysis and autonomic responses, three groups emerge.

1) 14 cases: Atrial flutter or fibrillation associated with sinus rhythm; 3 of the 14 cases had reduced autonomic responses, indicating sinoatrial disease, one thyrotoxic had supersensitive responses and the remaining 10 cases were normal to testing.
2) 10 cases: Atrial flutter or fibrillation associated with sinus bradycardia (rate < 55/min at rest). Five of the 10 cases had sinoatrial disease while the remaining 5 were normal on investigation.
3) 16 cases: Rapidly conducted atrial tachycardia of undefined mechanism, associated with normal sinus rhythm at times. No electrocardiographic evidence of pre-excitation. One case had responses indicative of sinoatrial disease; 6 were entirely normal; and 9 had responses greater than any control subject.

Results of this investigation suggest 3 possible mechanisms for supraventricular tachycardia: sinoatrial disease, physiological (or undetectable abnormality), supersensitivity to catecholamines. Those with sinoatrial disease may need to be followed with a view to pacing since digoxin and cardiac suppressants may be contraindicated. Those with normal atrial pacemaker function should be able to take these drugs without complication. In those with supersensitivity to catecholamines causes for anxiety and aggravation should be sought and thyrotoxicosis carefully excluded, or treated as necessary. Anxiolytic drugs and beta-blockers may prove the most useful drugs in this group of patients.

Reference

JOINT SYMPOSIUM WITH THE CARDIAC MUSCLE RESEARCH GROUP

Clinical prospects for reducing infarct size
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The prognosis after acute myocardial infarction depends to an important degree upon the extent of muscle necrosis. Ultimate infarct size is determined in part by events that occur some hours after the onset of symptoms. The balance between delivery and use of oxygen within an ischaemic area influences the degree of hypoxia and the secondary pathological changes which lead to irreversible cell death. Therapy should, therefore, be directed to reducing myocardial oxygen requirement and preserving the integrity of cells within the damaged myocardium. Experimental evidence suggests that infarct size can be limited in this way, and numerous varieties of treatment have been proposed for clinical use. None has yet gained wide acceptance partly because of the difficulty in assessing their value. The prospect of translating promising experimental techniques into routine clinical procedures awaits the development of practical methods for monitoring the extent of myocardial injury and necrosis.

What is the evidence that therapeutic intervention in acute myocardial ischaemia can preserve useful functioning myocardium?
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Therapeutic intervention in the natural history of disease can be dramatically obvious in its effect, but more often new treatment requires detailed analysis before acceptance or rejection as 'orthodox'. Proof of efficacy varies widely in quality, from direct knowledge of lives saved, improvement in tissue function, or symptomatic aid in man, to indirect suggestion from experimental data and theory. In myocardial ischaemia, benefit may be had from a primary increase in blood supply, a reduction in metabolic demand, or factors that attenuate intracellular metabolic disease at a given level of ischaemia. Metabolic approaches, purporting to ameliorate intracellular disease and preserve the myocardium for future function are difficult to assess. Prevention of catecholamine drive, modifying potassium and glucose metabolism, blocking free fatty acid utilization are subject to controversial debate. A particular instance is the use of β-blocking agents in unstable angina and acute myocardial infarction in man. Measurement of their influence in patients is limited and extrapolation from indirect indices of benefit uncertain. We need to examine the proof available and to apportion weight to the case for changing clinical practice.
Does ST segment mapping accurately reflect myocardial damage?

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Over the past several years there has been increasing interest in the development of tools which might give the clinician the ability to quantify the ontology of myocardial ischaemia in patients experiencing acute myocardial infarction.

Such tools, by necessity, would need to predict the future history of electrical and mechanical function of acutely ischaemic areas. If it were possible to predict the hysteresis of an infarcted area, then one could make evaluations of various interventions which might change the predicted history of an area of ischaemic myocardium.

Naturally the quantification of unipolar or bipolar electrocardiograms, whether epicardial, transmymocardial, or chest wall would seem a reasonable way to evaluate ischaemia.

In the early 1960's Sayen and colleagues showed that unipolar epicardial electrogram ST segments were poor indicators of transmymocardial ischaemia when compared with intramyocardial oxygen electrodes and myocardial dyskinesis, especially in larger infarctions in dogs.

This technique was revived in the early 1970's by Maroko and Braunwald who suggested that epicardial unipolar ST segment elevations reflected myocardial cellular injury, and that the magnitude of ST segment elevation correlated well with altered myocardial metabolism and subsequent histological evidence of myocardial infarction. This group and several others have subsequently extrapolated these techniques in clinical precardial ST segment mapping of anterior myocardial infarctions.

Recently, however, several studies have shown poor correlation of the unipolar epicardial ST segment mapping techniques with the microcirculatory perturbations of acute ischaemia in dogs. These studies have again questioned the reliability of unipolar ST mapping.

We would like to clarify these conflicting data by examining the biophysics of unipolar ST elevation.

Serum enzymes: what can they really tell us about myocardial damage?

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Associated with acute myocardial ischaemia are numerous metabolic and morphological changes. The time of onset and magnitude of these tissue changes are dependent upon the duration and severity of ischaemia. Of the many hundreds of biochemical changes that occur in the myocardium, a few, for example potassium loss, pyruvate and lactate changes, and the leakage of intracellular cardiac enzymes to the plasma, have been singled out as valuable markers of ischaemic damage. The observed leakage of cardiac enzymes has formed the basis of modern diagnostic enzymology. The state of the art has evolved such that the appearance of one or two cardiac enzymes in the plasma is not only used for the diagnosis of the presence, approximate magnitude, and time of onset of myocardial infarction but it has been suggested that mathematical analysis of enzyme leakage allows us to predict the progression and exact size of the infarct. What is the basis of these suggestions? Why and by what mechanism do enzymes leak from the cell? What are the cellular implications of enzyme leakage and does it signal the onset of irreversible damage? Why do we choose CK, LDH or AST? Are they in some way unique? Can we really equate enzyme leakage with cell death? Is there a precise and defined relation between the rate and extent of enzyme release and the amount of cell damage? How specific are these tests and what benefits are there in studying isoenzyme as opposed to enzyme leakage?

YOUNG RESEARCH WORKERS' PRIZE

Learned voluntary control of autonomic functions: investigations of mechanisms and possible clinical applications

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The autonomic nervous system is not usually thought to be susceptible to direct voluntary control, and autonomic changes during voluntary acts are assumed to depend on changes produced by skeletal muscles. The ability to learn more direct voluntary autonomic control was investigated with biofeedback techniques in patients in whom skeletal muscle control was severely impaired.

In 8 patients with poliomyelitis or muscular dystrophy small (±3 mmHg (0.4 kPa)) but significant changes of diastolic pressure could be produced in either direction during 90-second trials, without changes in breathing or muscle tension.

Twenty-five per cent of tetraplegic patients with complete cervical cord transections learned to increase systolic pressure (+7 to +24 mmHg (+0.9 to +3.2 kPa)) during 5-minute trials. Heart rate also increased, but decreased during spontaneous pressure rises. Voluntary pressure control could be blocked by atropine (which itself raised pressure), suggesting a vagal mechanism. Respiratory manoeuvres and attempted muscle contraction could not reproduce the voluntary pressure rises.

It is concluded that voluntary control of autonomic functions may be learned, and is not necessarily secondary to changes in skeletal muscle. Possible therapeutic applications are described.

Glycoside inotropy and K+ efflux in presence of an acidosis

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Although the cardiac glycosides are widely used in clinical practice, their mode of action is controversial. The augmentation of Ca++ influx associated with their
inotropic effect has been attributed to an increase of intracellular sodium or to an increase of K⁺ efflux. This study was designed to examine these two hypotheses.

The inotropic effect of 1·25 × 10⁻⁶ M acetylstrophanthidin (ACS) and the influx and efflux of ⁴⁰K⁺ were studied in the arterially perfused rabbit interventricular septum under control conditions and during a respiratory acidosis. An increase of the CO₂, with which the modified Ringer’s solution was equilibrated, from 5 to 30 per cent reduced the perfusate pH from 7·37 to 6·66. The increment of developed tension in the presence of ACS was 3·0 ± 0·2 g (n = 10) under control conditions but was greater, 7·1 ± 0·9 g (n = 9) in an acidosis (P < 0·001). The net K⁺ loss resulting from an increase of K⁺ efflux was 1·9 ± 0·2 mmol/kg wet wt in control experiments but only 0·1 ± 0·1 mmol/kg wet wt under acidic conditions (P < 0·001); in 7 out of 9 experiments in a respiratory acidosis no increase of K⁺ efflux occurred despite a striking positive inotropy. In three septa K⁺ influx was reduced by ACS during a respiratory acidosis.

These results indicate that though ACS in an acidosis inhibits Na⁺–K⁺ ATPase and causes an inotropic effect, it does not increase K⁺ efflux. K⁺ efflux cannot be coupled with Ca⁺⁺ influx or be closely linked to the mechanism of the inotropy induced by glycosides. The results give further support to the proposal that the inotropic effect of glycosides is secondary to enhancement of a Na⁺–Cr⁺ exchange system. The findings are of relevance to the use of glycosides in those clinical situations in which there exists an intracellular acidosis.

Pituitary-adrenal axis during cardiopulmonary bypass

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The response patterns of plasma cortisol, corticotrophin (ACTH), and growth hormone were investigated in patients undergoing elective cardiac surgical procedures using normothermic cardiopulmonary bypass. Patients undergoing closed mitral valvotomy were used as controls.

Total plasma cortisol levels fell at the onset of extracorporeal circulation (ECC) and did not rise significantly throughout the period of ECC. The peak cortisol level was delayed, occurring at 24 hours postoperatively. This pattern was highly significantly different from the control patients (P < 0·001) who showed the typical cortisol stress response pattern.

Stimulation of the adrenal cortex during ECC with synthetic ACTH produced a positive response (i.e. a rise in plasma cortisol > 200 nmol/l) in all patients studied. This group was significantly different from 6 mitral valvotomy patients who had no response to synthetic ACTH administered during operation (P < 0·01).

Plasma ACTH levels showed a progressive fall during the period of ECC from a pre-ECC primary peak such that levels in the latter half of ECC were in the non-stress range (i.e. < 100 ng/l). One hour after the end of ECC a striking secondary ACTH response was seen. Plasma growth hormone levels showed a similar response pattern to that of ACTH.

It is concluded that the cortisol response during cardiopulmonary bypass differs significantly from the typical cortisol response to stress, and that the differences appear to be related to the period of extracorporeal circulation. The secretion of cortisol during ECC is submaximal but the adrenal will still respond to exogenous ACTH. In addition, the response patterns of ACTH and growth hormone suggest that the reduced cortisol secretion is a direct result of generalized anterior pituitary hypofunction during bypass.