Proceedings of the British Cardiac Society

THE AUTUMN MEETING of the British Cardiac Society was held at the Royal College of Physicians, London, on Tuesday and Wednesday 19 and 20 November 1974. The President, JOHN GOODWIN took the Chair at 9.00 a.m. during Private Business. At the Scientific Session the Chair was taken by W. P. CLELAND.

Private Business

1 The President reported the deaths of Cornelio Papp and Douglas Robertson, and the members stood in tribute.

2 In the unavoidable absence of the Treasurer the President read his report, which was based on the unaudited figures to the end of September 1974. The Society’s finances were reasonably satisfactory. About two-thirds of the income from subscriptions was spent on secretarial and clerical services, stationery, the publication of the annual booklet, and miscellaneous general expenses; the remainder was split between subscriptions to other societies, a subsidy to the Thomas Lewis Lecture, and the cost of meetings.

Twenty-two people had received grants to attend the VII World Congress of Cardiology in Buenos Aires in September 1974 from funds made available by the British Heart Foundation, the British Council, and the Society.

There were no plans to increase members’ subscriptions next year, but if inflation continued at its present rate an increase soon after was unavoidable.

The Treasurer was considering whether the direct debit system should replace bankers’ orders for the payment of subscriptions.

The Treasurer’s report also stated that the Society of Cardiological Technicians was now very active, was recruiting new members, arranging scientific and social meetings, and taking its place in negotiations. They had asked whether it was possible for the British Cardiac Society to increase the annual grant of £50. Council recommended a grant of £150 this year, to help with their library project, and thereafter £100 a year, and this was approved. Council had also discussed the Technicians’ suggestion that the Society might help in some way with the Journal of Cardiovascular Technology and had agreed that Somerville, Linden, and Towers should look into the possibility. The Cardiological Technicians also asked if the British Cardiac Society would appoint a liaison officer to act with them, and Council suggested that the Honorary Assistant Secretary should take over this role. This was agreed.

3 Wade confirmed the invitation for the British Cardiac Society to hold the Annual General Meeting in 1975 in Manchester on 17 April, and reported that preparations were well in hand. The Honorary Secretary added that, because of the long delay involved in printing, the closing date for the submission of abstracts for the Manchester meeting was 17 January.

4 The Autumn Meeting in 1975 would revert to a Thursday and Friday, 13 and 14 November. This year’s meeting was on a Tuesday and Wednesday, and Council had agreed to send a referendum asking for members’ opinions as to which days were most convenient.

5 The Annual General Meeting in 1976 would be in Edinburgh on 1 April.

6 Edgar Sawton reported that the joint meeting with the Swedish Society would be held on 1 and 2 September and the morning of the 3rd, in Stockholm. It was planned to hold symposia on the mornings of the 1st and 2nd, with free communications on these two afternoons. The closing date for submission of abstracts of free communications was 18 April 1975. On the Wednesday morning the plan was to visit new hospitals and centres in Stockholm; a boat trip in the Stockholm archipelago was planned for the Wednesday afternoon. Travel agents were still reluctant to give firm quotations, but two package deals had been asked for—(1) for hotels and (2) for accommodation in University halls of residence, both to be for one week.

7 The Secretary reported that any inquiries regarding the European Travelling Fellowships should be addressed to Dr. Morris Butler at the British Heart Foundation.

8 The Specialist Advisory Committee on Cardiovascular Disease (one of several Advisory Committees to the Joint Committee on Higher Medical Training of the Royal Colleges of Physicians) was now engaged in visiting centres to look into the training programmes available for Senior Registrars, and where appropriate Registrars. Some nine centres had been visited by teams of 3 people (one from the SAC who acted as Chairman, one from the SAC on General Internal Medicine, and one other who might or might not be a cardiologist). The reports sent back to the SAC and the JCHMT were already proving of considerable value to the centres visited. The second edition of the training programmes was being revised and would be available shortly.

The turnover of members of the Specialist Advisory Committee had proved too rapid and in future two members would retire one year and one the next.

In reply to a question from Whitaker, the President explained that the Department of Health would do nothing about a request from a Region for a Senior
Registrar appointment until the relevant Specialist Advisory Committee had looked at the post, the training programme, and the centre, and reported to the Joint Committee on Higher Medical Training. If the report were favourable the request would go to the DHSS, to the Central Manpower Committee, and to the Senior Registrar Sub-Committee, and if the DHSS agreed the appointment would be allowed.

9 The Joint Liaison Committee (British Cardiac Society and Royal College of Physicians/Department of Health) had continued to meet and a short report on its activities would be sent out soon. Members were invited to submit problems of any sort connected with cardiology, including buildings. It was hoped to modify the Committee’s activities slightly, and instead of regular meetings it was decided to hold special meetings for major topics for which the DHSS had promised to mobilize the necessary people for the particular problem. Wade suggested that too little was known of this committee and that a circular should be sent to all members outlining its role and perhaps suggesting the type of problem which it could discuss. The President replied that he would prepare a short report and circulate it in the near future to all members.

10 Because some of the recommendations in the Report on Coronary Arteriography by the Cardiology Committee of the Royal College of Physicians had been questioned by members, Emanuel suggested that there might be a place for an editorial in the Journal outlining the problem. Goodwin thanked Emanuel for the considerable amount of work involved in preparing the report.

11 The President reported briefly on the VII World Congress of Cardiology held in Buenos Aires in September 1974; a short report was being prepared for publication in due course. The VIII World Congress would be held in Tokyo in 1978.

The President also reported that the question of the merger of the International Society of Cardiology (the scientific body dealing with Scientific Councils, World Congresses, etc.) and the International Cardiological Foundation (the fund-raising body) had been discussed at length by the Assembly meeting in Buenos Aires, and it was agreed by 37 to 5 that the merger should take place. This was subject to the agreement of the ICF who were meeting at the beginning of December. If the merger were rejected by the ICF then the American Heart Association would probably withdraw its support and the ISC would collapse. The British Cardiac Society was committed to nothing if the merger did not go through; if it did go through an increase in the Society’s present subscription was agreed in principle, but no figure had been recommended or agreed. Lyle Petersen had been elected President of the ISC, and John Goodwin President-Elect, to take office in 1976.

12 Brief reports were given on the Working Parties:

a) Lawson McDonald reported that the final edition of the report on Acute Coronary Care would be completed by the end of the year.

b) Semple reported that the Rehabilitation Working Party had held its last formal meeting. The report was still being independently checked and edited; this was primarily for general practitioners and family doctors, and publication and distribution were being discussed with the Department of Health. The report would probably be published in the Journal of the Royal College of Physicians, and the College would ensure that members of the British Cardiac Society would be sent copies. It would be some months before the report was ready.

c) Shaper reported that the Working Party on Primary Prevention of Cardiac Disease was attempting to provide general practitioners and physicians with the best possible advice which could be given at present on this subject, and was now working on the final recommendations for completion by the spring or early summer of 1975. It was hoped to produce a small cheap booklet which would reach every practitioner in the U.K.

d) Fleming reported that a good deal of information had been collected on the prophylaxis of infective endocarditis. He requested that basic case details of proven Strep. viridans endocarditis, especially in regard to previous dental work, should be sent to him and he thanked members for their co-operation so far. He also suggested that the topic might be discussed by the Society at the meeting in Manchester next April.

13 The President announced that Council had appointed Linden as the new Editor of Cardiovascular Research, and thanked Shillingford, on behalf of the Society, for his foresight and imagination in starting this Journal, and for setting and maintaining the high level of contribution, which had established the international standing of Cardiovascular Research.

14 The Cardiac Muscle Research Group had held a very successful meeting on Monday 18 December, and Council had agreed in principle to a joint half- or quarter-day with the Society next Autumn.

The Society dined together at the Zoological Gardens Restaurant with John Goodwin in the Chair. The guests included Mr. Harry Moore, Dr. Dennis Beddard, Dr. Stuart Reid, and Dr. E. Varnauskas.

Scientific Communications

Borderline hypertension – a specific entity?

J. B. Irving, H. Brash, F. Kerr (all introduced), and Brian J. Kirby
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The concept of borderline or labile hypertension as an early stage in the natural history of essential hypertension has attracted much interest, mainly on account of
its therapeutic implications. Several studies have demonstrated that this group of patients is characterized by pronounced lability of blood pressure and raised cardiac output at rest. We have investigated 32 individuals (8 normal subjects, 14 patients with borderline hypertension and 10 patients with established hypertension) by 24-hour monitoring of intra-arterial blood pressure using a radiotelemetry system and by haemodynamic studies during treadmill exercise.

Variability of blood pressure was measured by several methods including range of values, standard deviation, and an analysis of variance. No significant differences among the three groups were found.

The resting cardiac indices were: established group 2.54 l min⁻¹ m⁻², borderline group 3.22 l min⁻¹ m⁻² and normal 3.41 l min⁻¹ m⁻². The only significant difference in the various haemodynamic parameters was the resting heart rate (P < 0.01). The response to exercise was similar in all three groups except that patients with established hypertension had a steeper rise in blood pressure.

There is little evidence from these studies to suggest that patients with labile hypertension are a specific group.

Observations on the aetiology of unexplained or primary pulmonary hypertension

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Forty-four patients with ‘primary’ or ‘unexplained’ pulmonary hypertension have been analysed in relation to their aetiology.

In 3 cases the disease followed clinical pulmonary embolization and in 3 more there were congenital abnormalities of the pulmonary vessels with abnormal broncho-pulmonary communications which we thought were causal. These patients differed in age and sex preponderance and natural history from the remaining 38.

The 38 who are compared to the picture of primary pulmonary hypertension presented in published reports: 33 were female, with an average age of 33 years; 18 had borne children, and in 6 the onset of the disease related to childbirth. The cause was usually short with an average survival time from the onset of symptoms of approximately 4 years. Twelve patients presented evidence of a systemic disease of collagen type and were distinguished by the frequency of digital arterial disease, arthritis, and the presence of serological abnormalities. No such evidence could be adduced in the remainder but the clinical similarities suggest aetiological linkage. The pregnancy relation is discussed and it is suggested that the small pulmonary vessels respond abnormally to stress.

One familial case is reported in which the genetically determined defect may have been rheumatoid disease.

We suggest that a substantial proportion of cases of unexplained pulmonary hypertension may be examples of immune complex disease.

Observations on the origin of the impedance cardiogram

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Although they are quantitatively related to systemic stroke volume and pulse pressure, it has not yet been established how the variations in thoracic impedance originate during the cardiac cycle. Experimental work has suggested that pulmonary blood volume changes account for most of the systolic portion of the signal. To test this hypothesis, impedance cardiograms were recorded from 14 patients before and 6 months after surgical closure of atrial septal defects (mean Qp/Qs = 2.7 ± 0.3). Resting heart rate, and the amplitude (Dz) and maximum first derivative (dz/dt) of the impedance cardiogram were not significantly changed. Individual pre- and postoperative values correlated significantly. In contrast Dz and dz/dt were increased in patients with aortic incompetence. Comparison of impedance cardiograms in atrioventricular dissociation with sinus rhythm showed that the initial presystolic deflection was associated with atrial activity. Transient occlusion of the superior vena cava decreased Dz and dz/dt proportionally to reduced stroke volume but produced no waveform change. It is concluded that the impedance cardiogram is sensitive to changes in left ventricular stroke volume but insensitive to changes in right ventricular stroke volume, and that local venous pressure changes do not contribute significantly, even to the diastolic part of the impedance cardiogram signal.

Intermediate coronary care – a controlled trial

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A controlled trial of intermediate coronary care was carried out over a five year period at a district general hospital. One thousand consecutive male patients under 65 surviving to leave the Coronary Care Unit (CCU) were allocated at random into an intermediate care group kept in the same ward as the CCU and a control group discharged to a general medical ward. The intermediate care patients were nursed by the trained CCU staff, resuscitation equipment was immediately available, and there was an efficient emergency call system. The mortality was the same in both groups and no more patients survived cardiac arrest to leave hospital in the intermediate care group than among controls, though initial resuscitation was more often successful. The failure of intermediate coronary care was attributed to the rarity of primary ventricular fibrillation after discharge from the CCU.

Myocardial infarction in east London

Hugh Tunstall Pedoe (introduced by Wallace Brigden)
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The London Borough of Tower Hamlets was the
British centre in a European epidemiological study of coronary heart attacks, supported by the Department of Health and the World Health Organization. During 33 months 707 attacks in men under 65 were classified as Definite or Possible Acute Myocardial Infarction, with 271 deaths occurring within four weeks of onset (38%); and 171 in women with 79 deaths (46%).

The places of onset of the attacks and of death were recorded, as were details of the timing of these events and medical care. Despite the compactness of the area and a 97 per cent hospital admission rate for survivors, most fatalities occurred at the place of onset, and only 32 per cent in hospital. The survival curve showed a very high early fatality with an exponential fall with time. This fell was constant from 15 minutes to 4 weeks and, therefore, did not appear to change as the victims came under medical care. In order to reduce the proportion of medically unattended deaths emergency services would need to be speeded up geometrically. Halving the present time taken from onset to coming under care would anticipate only an additional 5 per cent of all deaths. The difficulties of interpreting these interesting data are discussed.

Positive correlation of coronary angiographic index with maximal exercise level in angina pectoris

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There has been no reliable method to date of predicting the severity of coronary artery disease before angiography. An angiographic index has therefore been designed to estimate the proportion of ischaemic myocardium at induction of angina pectoris. This has been shown to give a better correlation with exercise performance than the standard methods of angiographic scoring. The number of major coronary vessels distal to an 80 per cent or more stenosis is expressed as a percentage of the total number of such vessels. Seventy-five patients with angina pectoris and angiographically demonstrated coronary artery disease were studied. A low ($r=0.27$) but significant ($P<0.05$) correlation was found between the index and the maximum exercise load, which was not found when the angiogram was classified according to the number of the three major vessels diseased, whether or not the degree of stenosis was taken into account. There was also a positive correlation ($r=0.29; P<0.05$) between the maximum increase in heart rate during the test and index. It is suggested that patients with poor exercise performance should be more urgently considered for angiography. The index which gives a more quantitative assessment of the severity of disease may also help to define more clearly those patients with poor prognosis who might be considered for prophylactic surgery.

Experience with left main stem coronary stenosis

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Left main coronary lesions are reported to have a poor prognosis, and a high investigative and operative mortality. Eighteen (3%) of 600 patients studied had left main stem stenoses of 75 per cent or more. All had angina (average grade 2.7/4), which in 11 started or increased in the previous 6 months. Eleven (61%) had electrocardiographic or clinical evidence of previous infarction. The average maximum exercise load achieved on the bicycle ergometer was 490 kpm min$^{-1}$ (4802 Nm min$^{-1}$). Eleven were paced to angina at rates from 110 to 170/min. Mean resting left ventricular end-diastolic pressure was $16\pm9$ mmHg ($2.1\pm1.2$ kPa). 75 per cent lesions were present in at least one other coronary artery in 17 patients. Segmental left ventricular dyskinesia was present in 10 and diffuse in 7. One refused surgery, 1 died awaiting operation, and 1 had diffuse ventricular dysfunction. Fifteen underwent surgery. One died during induction of anaesthesia. The follow-up on the 14 operated averaged 10.9 months (1 to 36 months). Twelve (88%) are asymptomatic. Two (14%) have grade 1 angina. It is concluded that patients with left main stem stenosis present a similar clinical picture to those with severe coronary disease. Catheterization with the Sones technique was safe and there was only one operative death. Results of surgery are similar to other patients, the majority are relieved of symptoms, and all are improved.

Value of left atrial vectorcardiography in assessment of left ventricular function after acute myocardial infarction

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The pulmonary arterial diastolic pressure, a timed vectorcardiogram, and a standard electrocardiogram were recorded simultaneously in 32 previously well patients after acute myocardial infarction. The recordings were made immediately after the patients were free of pain (2 to 18 hours). If the pulmonary arterial diastolic pressure exceeded $15$ mmHg ($2.0$ kPa), the recordings were repeated twice daily until the pressure fell below this or became stable. Timed vectorcardiograms were also obtained from 60 ‘normal’ hospitalized subjects.

The correlation coefficient between the initial left atrial electrical activity calculated from the timed vectorcardiogram and the pulmonary arterial diastolic pressure was $0.85$ ($P<0.001$). The correlation between $P$ terminal force calculated from $V_1$ of the standard electrocardiogram and the pulmonary arterial diastolic pressure did not reach significance. The vector results
at the initial investigation enabled the patients to be separated into three groups corresponding to the pulmonary diastolic pressure: (a) Normal (<15 mmHg (2.0 kPa)); (b) mildly raised (16 to 20 mmHg (2.1 to 2.7 kPa)); (c) severely raised (21 to 30 mmHg (2.8 to 4.0 kPa)). The values for the left atrial electrical activity for the 60 'normal' subjects all fell within the range for normal pressure. However, the correlation between the vectorcardiogram and the pressures was poor when those patients with initially high pressures were re-investigated more than 36 hours after infarction.

These findings indicate that the timed vectorcardiogram is a valuable indicator of left ventricular filling pressure within the first 36 hours after infarction.

An angiographic investigation of aortocoronary vein grafts

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Repeat angiography was performed in 90 patients after aortocoronary bypass grafts at periods varying from 2 weeks to 4 years (mean 13 months). This included patients with residual symptoms. Out of 186 grafts 168 were entered and shown to be patent, representing an overall patency rate of 91 per cent. The early patency rate (less than 3 months after operation) was 96 per cent (61 grafts); between 6 months and 1 year the patency rate was 86 per cent (46 grafts) and between 1 and 4 years it was 90 per cent (79 grafts). All patients had 1 or more functioning grafts. Patency of grafts to right coronary was 92 per cent (69 grafts), to anterior descending 90 per cent (67 grafts) and to circumflex 95 per cent (30 grafts). The patency rate was not affected by age of patient, presence of hypertension or diabetes, previous infarcts, or additional endarterectomy. There was no evidence of progressive narrowing of the grafts with time. Progression in native vessels was observed in 15 per cent of the patients, mainly in non-grafted and in proximal segment of grafted vessels.

It is concluded that coronary vein grafts continue to function satisfactorily up to 4 years and that accelerated progress in native vessels after grafting is uncommon.

Post-infarction ventricular septal rupture: Clinicopathological study

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Between 1969 and 1974, 19 patients with acute ventricular septal rupture were seen at Harefield Hospital, 17 were treated surgically. Their ages varied from 44 to 76 years. Rupture occurred 4 to 8 days after infarction. The lesions could be classified into 4 clinicopathological types according to the site of rupture in relation to the ventricular septum (anteroapical, posteroapical, central, and posterobasal). Accurate preoperative diagnosis of the type could be made depending on the location of the systolic thrill, the presence or absence of atroventricular conduction defects, and the distribution of the QRS abnormalities. Coronary angiography was performed in 4 patients during circulatory support using intra-aortic balloon counterpulsation. The timing and type of surgical repair depended on the clinicopathological type. In the antero and posteroapical types excision of the infarcted septum with the rupture was performed. In the central type, a double patch of 'dacron' was used, and in the posterobasal type, through a right ventriculotomy, the septum was reattached to the posterior ventricular wall after intra-aortic balloon counterpulsation for 2 weeks.

It is concluded that preoperative diagnosis of the clinicopathological type of ventricular septal rupture is possible and helps in management of these patients.

Morphological observations in specimens of tricuspid atresia with reference to bulbo-ventricular morphogenesis

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There is controversy regarding the mode of formation of the normal right ventricle. Some contend that it is formed entirely from the primitive bulbus; others that its infundibulum is of bulbar origin, while its sinus is developed from the primitive ventricle. Since it can be argued that the chambers in specimens of tricuspid atresia represent the bulbar and ventricular chambers, respectively, it seemed that study of such specimens may elucidate the problem of right ventricular development. We have studied 26 examples of tricuspid atresia or hypoplasia. Our findings indicate that the so-called right ventricle is indeed the primitive bulbus, and that the so-called left ventricle is composed of the sinus portions of both right and left ventricles, and represents the primitive ventricle. The anomaly results from failure of rightward expansion of the primitive atrioventricular canal. The observation that the endocardial cushions were fused eccentrically to form a 'central' fibrous body on the right margin of the canal and a single mitral orifice supports this hypothesis. Subsequent fusion of the atrial septum with their eccentrically positioned cushions, therefore, produces the blind right atrium. It will be demonstrated that evidence of the right ventricular sinus is observed in the 'left ventricles' of most specimens, and that the 'dimple', frequently seen in the right atrial floor, communicates with this particular part of the 'left ventricle' rather than with the 'right ventricle'. In one specimen a partly formed valve ring was observed in this position. The tissue considered to represent the ventricular septum is, in fact, the bulboventricular septum, and evidence of the posterior interventricular septum is
usually present in the 'left ventricle'. The significance of these findings to 'single ventricle with outlet chamber' was presented.

Surgical repair of conotruncal abnormalities

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It has recently been shown that transposition and mal-position are morphogenetically closely linked. It follows, therefore, that an understanding of the developmental processes and positional variations of the underlying right ventricular anatomy can make for easier understanding of these complex anomalies, and help in their corrective surgery.

The development and position of the septal insertion of the conus septum influences the intraventricular repair of conotruncal abnormalities.

Nine patients, 4–20 kg, have been studied and corrected.

1) Four had 'complete' 1-transposition of the great arteries with a subaortic ventricular septal defect.
2) One had double outlet right ventricle with a subaortic ventricular septal defect.
3) Two had double outlet right ventricle with a sub-pulmonary ventricular septal defect.
4) Two had double outlet right ventricle with l-malposition of the aorta and a subaortic ventricular septal defect.

In (1) the conus septum is hypoplastic or absent. Therefore, the ventricular septal defect is immediately subaortic. In (2) the septal insertion of the conus septum blocks off the left anterior half of the ventricular septal defect. In (3) and (4) the septal insertion of the conus septum is to the posterior part of the interventricular septum, the primary bulboventricular foramen persists as the ventricular septal defect and is beneath the left-sided conus, which in (1) and (4) gives rise to the aorta, but in (3) the pulmonary artery.

In corrective surgery the intraventricular repair must either re-route blood from the left ventricle into the aorta (2, 4) or the pulmonary artery (3). After this, in (1) and (3) venous inflow must be re-routed at atrial level by an interatrial baffle. Eight children are well, and one died 3 months after operation.

Current surgical management of transposition of great arteries in infancy

J. Stark, M. de Leval, D. J. Waterston (last two introduced), and R. E. Bonham Carter
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The natural history of transposition of the great arteries has been considerably modified by palliative procedures. However, despite an initial improvement, continuing morbidity and mortality still make an early repair desirable.

In a series of 325 Mustard operations, 73 were performed in infants. Sixty patients had simple transposition of great arteries; there were 5 early and 3 late deaths. Twelve patients had transposition of the great arteries and ventricular septal defect; there were 6 early and 3 late deaths. One patient had transposition of the great arteries and pulmonary stenosis, and he survived. A high incidence of systemic venous obstruction, mainly in the group of infants where a 'dacron' patch was used, led us to go back to using pericardium and to change the size and shape of the intra-atrial baffle. The results of this new technique are encouraging.

Our present policy can be summarized as follows: Balloon septostomy is performed at initial catheterization in all infants. Simple transpositions of the great arteries are reinvestigated at 4 to 5 months of age. Mustard operation is then performed between 6 and 12 months of age, or earlier if deterioration occurs. Because of the high incidence of heart failure and pulmonary vascular disease, patients with transposition of the great arteries and ventricular septal defect are operated in infancy. In patients with transposition of great arteries, ventricular septal defect, and pulmonary stenosis, a systemic to pulmonary artery shunt is performed in infancy if necessary.

Long-term cardiorespiratory assessment after surgical closure of ventricular septal defect in childhood

Katherine A. Halidie-Smith, R. E. Edwards, R. Wilson, and E. Zeidifard (last three introduced)
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This study was designed to find out whether surgical correction of a large ventricular septal defect in childhood can result in normal cardiorespiratory function in adult life.

Twenty-six patients aged 3 to 12 years at operation now 9 to 16 years after operation, had evidence of pulmonary vascular disease preoperatively, with one exception.

All are now leading normal lives and only one admits to fatigue on effort. Respiratory function tests and exercise studies on 18 patients showed normal values for FEV1,0.2, vital capacity, and transfer factor for CO. Exercise tolerance was assessed with an increasing work load test and was essentially normal for the group. Heart rate, ventilation, and pulmonary gas exchange increased normally with exercise. In a constant work-rate exercise test, cardiac output, dead space, tidal volume ratio, and blood lactate concentration were within the normal range.

Cardiac catheterization in 11 patients showed progressive pulmonary hypertension in comparison with earlier study 8 years previously (1 to 8 years post-operatively) (Hallidie-Smith et al., 1969) and there was a striking increase in pulmonary artery pressure during supine and erect bicycle exercise.

The severity of the pulmonary hypertension is in contrast with the functional status and may imply an unusually benign course. Nevertheless, these results
strengthen the case for early closure of ventricular septal defect.

Reference

Late results of homograft reconstruction of right ventricular outflow tract in infants and children

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Between March 1970 and July 1974, 21 children underwent successful reconstruction of their right ventricular outflow tract with adult-sized fresh aortic homografts. Their ages were between 4 months and 13 years. Twelve patients were below 4 years of age. The lesions corrected were truncus arteriosus, pulmonary atresia with ventricular septal defect, pulmonary atresia with intact septum, absent pulmonary valve, double outlet right ventricle, transposition of the great arteries with ventricular septal defect and pulmonary stenosis, and severe Fallot's tetralogy.

Ten patients have been reinvestigated 2 weeks to 50 months after operation. In no patient is the gradient across the pulmonary valve more than 12 mmHg (1.6 kPa) and the homograft is functioning well. The pulmonary artery pressure is now at systemic level in 2 patients who underwent operation at 7 and 12 years of age, respectively. In the remaining patients the pulmonary artery pressure is between 20 and 40 mmHg (2.7 to 5.3 kPa). There is no evidence of reopening the ventricular septal defects.

It is concluded that homograft reconstruction of the right ventricular outflow tract in infants and children gives satisfactory results.

Critical outflow valve obstruction under the age of 1 year

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This report represents the Brompton Hospital experience of infants and neonates with critical pulmonary valve stenosis and aortic valve stenosis. There were 19 patients with critical pulmonary valve stenosis and 8 with critical aortic valve stenosis.

Critical pulmonary stenosis: Among the 19 patients up to 1 year of age, 8 were girls, and the commonest presenting symptom was cyanosis seen in 15, the commonest physical sign being a systolic ejection murmur with a single 2nd sound. A more detailed analysis of the symptoms, signs, and findings at special investigation will be presented. The evolution of a satisfactory technique of operation will be indicated in these critically ill patients, and the late follow-up will be presented.

Critical aortic stenosis: There were 8 patients in this group all of whom were male. Dyspnoea was the commonest presenting symptom with a systolic ejection murmur and hepatomegaly as the commonest signs. All patients in this group underwent open aortic valvotomy and/or valvuloplasty, with 3 deaths. A late follow-up of the survivors will be presented.

In summary, a series of 27 patients up to the age of 1 year with critical obstruction of either the pulmonary or aortic valves will be presented. This is one of the larger series to be reported in this age group.

YOUNG RESEARCH WORKERS PRIZE

Role of pulmonary veins in regulation of lung capillary blood flow

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Lung capillary blood flow is pulsatile. The patterns of capillary blood flow in normal man and in patients with diseases affecting the right and left heart have previously been documented from Oxford and elsewhere. These studies suggested that both pulmonary arterial and venous mechanisms played a part in maintaining lung capillary blood flow pulsatility in arterialized form.

Previous work from this laboratory, both in animal model studies and in man, demonstrated that lung capillary blood flow remained normally pulsatile over a wide range of pulmonary artery pressures. This was shown to be because of a hyperbolic relation between pulmonary artery resistance and compliance.

Some of the human capillary flow studies had also shown that left atrial pressure transients, such as cannon waves in patients with complete AV dissociation, did not impede capillary blood flow until the mean left atrial pressure was grossly raised. This findings led me to the investigation of pulmonary venous pressure-flow relations and their role in regulating capillary blood flow.

The pulmonary veins are collapsible structures and so special methods were developed for measuring blood flow in them, using electromagnetic flow meters. It was found that the pattern of pulmonary venous blood flow was a mirror image of the left atrial pressure fluctuations, and that there was considerable variation in venous flow waveform.

Pulmonary venous and capillary flows were measured simultaneously in order to investigate whether the changes in venous waveform were reflected in capillary flow. This was found not to be the case.

A hypothesis based on the collapsible nature of the pulmonary veins was formulated to explain the decoupling of the capillary bed and left atrium described above. The consequences of this hypothesis were then tested by appropriate experiments. The results confirmed the existence of both the decoupling phenomenon and the collapsible behaviour of the main pulmonary veins.

Pulmonary venous velocities were then studied in man and the results confirmed that the results of animal studies were broadly applicable to man.
The role of this reservoir function of the pulmonary vein in protecting the lung capillaries from left heart pressure transients in the normal state and in left heart disease is discussed.

**Biological and pharmacological myocardial elimination rate of propranolol**

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Recent reports have suggested that propranolol should be withdrawn several weeks before open heart surgery. However, early withdrawal has resulted in rebound crescendo angina and infarction. The purpose of this study was to define a safe period for withdrawal of propranolol therapy. $^{14}$C propranolol was administered orally, 41-43 mg (25-75 μCi) or intravenously, 1 mg (25 μCi) to patients undergoing mitral valve replacement. Plasma, skeletal muscle, adipose tissue, and myocardial tissue were assayed. After intravenous propranolol plasma $^{14}$C varied between 1.5 to 5.0 hours with little change in total plasma radioactivity within ten hours. No propranolol was detected in the myocardial tissue 8.25 to 10.5 hours from dose, though the total tissue radioactivity indicated the presence of small amounts of metabolites. These are biologically inactive, since isoprenaline’s chronotropic and inotropic challenge had returned to control levels by 6-5 hours. After oral propranolol, most of the plasma radioactivity was caused by metabolites of the drug, indicating first passage hepatic extraction of propranolol. In one patient, low levels of radioactivity were detected in the myocardium at 28 hours; tissue $^{14}$C propranolol level in this patient was below the threshold of pharmacological activity. No myocardial radioactivity was detected in other patients undergoing surgery 24 to 97 hours after the oral dose. No radioactivity was detected in skeletal muscle tissue, but adipose tissue showed some depot unchanged propranolol.

*In vitro* myocardial dose-response curves to isoprenaline showed no difference in tissue obtained from patients who had previously received propranolol and those who had not.

In a consecutive series of patients, the operative mortality of patients who had never received propranolol was 5 per cent and 3 per cent in those in whom propranolol was discontinued between 1 and 11 days before surgery. This study defines 24 hours as an adequate safe withdrawal period for complete myocardial elimination of propranolol and pharmacologically active metabolites in patients undergoing operation or other interventions.

**Myocardial preservation during elective cardiac arrest**

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Elective cardiac arrest may be induced by a variety of procedures used either singly or in combination and with or without coronary perfusion. Each procedure has a different biochemical basis and thus has different metabolic and functional consequences. In studies with a rat heart model we have shown that methods which induce cardiac arrest without depleting cellular energy reserves (e.g. coronary perfusion with hypothermic solutions or solutions containing high concentrations of potassium) permit extended periods of arrest followed by good functional recovery. In contrast, methods causing a depletion of energy reserves (e.g. ischaemic arrest or electrically induced ventricular fibrillation) render the heart susceptible to damage and lead to a poor functional recovery. The results stress the importance of ensuring continuous coronary perfusion during arrest in order to maintain adequate cellular energy production for myocardial preservation.

**Myocardial biopsy: description of technique, comparison of right and left ventricular biopsy**

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Using a long sheath technique via the venous transseptal, arterial left ventricular, and venous right ventricular route, over 250 myocardial biopsies have been performed from the right/or left ventricle in over 100 patients. Biopsies were performed in patients with aortic valve disease, cardiomyopathy, or rheumatic heart disease but were not undertaken if there was any evidence of ischaemic heart disease.

Complications, all associated with right ventricular biopsy, occurred in 1.2 per cent of biopsies, haemopericardium of no haemodynamic significance in 2 patients, and ventricular tachycardia requiring electroversion in another patient. The biopsies were examined by both light and electron microscopy, and by histochemical staining.

In 20 patients, both left and right ventricular biopsies were obtained; the right ventricular morphology differed conspicuously from left ventricular morphology in 59 per cent of cases. The right ventricular biopsy consisted of fibrous tissue or adipose tissue in 24 per cent, and in 2 cases it included pericardium. The histological appearance was similar in biopsies from both right and left ventricles in 17 per cent.

Analysis of myocardial cell size suggests that biopsies taken from the right ventricular surface of the interventricular septum consist of a mixed population of right and left ventricular muscle cells, and may thus afford some indication of disease processes affecting the left ventricle. However, accurate placement of the biopsy on the septum cannot be ensured. Our experience would suggest that left ventricular biopsy is: a) safer than right ventricular biopsy; and b) more likely to obtain a biopsy sample that is representative of pathological changes affecting the myocardium which are more often pronounced in left than right ventricle.
Percutaneous technique of left ventricular biopsy, and comparison between right and left ventricular myocardial samples

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Endomyocardial biopsy has been performed from the left ventricular cavity previously, but it could only be achieved by means of an arteriectomy of the carotid or brachial arteries. The improved design of the King’s endomyocardial biopsyte has facilitated the development of percutaneous transfemoral left ventricular biopsy with a sheath technique. Repeated left ventricular sampling has been made possible without recatheterization of the aortic valve and the technique has been uncomplicated.

The clinical value of ventricular biopsy, particularly of the left ventricle, is discussed. Sixty-five patients have been biopsied, and in 15 patients biventricular biopsy has been performed. All samples have been evaluated by histological, histochemical, and ultrastructural methods, and have shown no major differences between right and left ventricular samples, though minor differences do exist between the ventricles. Diffuse disease processes such as congestive cardiomyopathy have been found to show identical features in the right and left ventricles.

In patients with primary left ventricular pathology such as hypertrophic cardiomyopathy or left-sided valvar lesions, biopsy of the left ventricle is preferable.

Oxyfedrine-induced changes in the structure and function of cardiac muscle

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Oxyfedrine (L-3-methoxy-(1-hydroxy-1-phenylisopropylamino)-propiophenone hydrochloride) is an effective antianginal drug. When given acutely it acts as a partial agonist at beta-adrenoceptor sites, but prolonged administration results in the establishment of beta-adrenoceptor blockade.

When injected intraperitoneally into rabbits oxyfedrine causes dose-dependent changes in the fine morphology of cardiac muscle cells. These changes are characterized by a proliferation of the cell membrane, lysis of the myofibrils, and vacuolation of the mitochondria. The Z band substance remains intact and attached to the cell membrane, despite evidence of tissue swelling. These changes are not caused by a release of endogenous catecholamines, because the noradrenaline content of rabbit heart muscle remained unchanged despite the occurrence of these other indices of cell damage. Other oxyfedrine-induced changes in the fine morphology of heart muscle cells included thickening of the basement coat substance, and evidence of collagen deposition. Heart weight to body weight ratio remained relatively constant, but high concentrations of oxyfedrine caused the rabbits to lose weight.

Relatively high doses of oxyfedrine interfered with the ability of the microsomal fractions to accumulate Ca²⁺ by the binding process. Under in vivo conditions it reduced the activity of the Ca²⁺-activated myofibrillar ATPase enzyme.

Primary restrictive cardiomyopathy

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During investigation of patients with cardiomyopathy a new group has been recognized. These patients show a unique fault in left ventricular diastolic function. This fault is manifested by restriction of left ventricular volume, without reduction of the rate of ventricular filling, and has been attributed to endocardial disease.

Seven patients aged 32 to 60 years were seen over a 2-year period, and were studied by echocardiography and angiography. All except one were Europeans. Of the 7, 6 had chest pain. In 4, the fault was confined to the left ventricle. There was involvement of the right ventricle also in 3, and these 3 patients showed some clinical resemblance to constrictive pericarditis.

The left ventricular diastolic pressure showed a steep early diastolic rise to a plateau without a prominent a wave in 4 out of the 7 (the early diastolic pressure ranged between 2 and 7 mmHg (0.3 and 0.9 kPa) and the end-diastolic pressure was 18 and 40 mmHg (2.4 and 5.3 kPa)). Cineangiography showed a small thick-walled left ventricle with a smooth rather featureless outline (end-diastolic volume 46 to 86 ml m⁻²). Systolic contraction was normal (ejection fraction 73 to 87%) and the end-systolic volume ranged between 10 and 17 ml m⁻². The distinctive nature of the haemodynamic fault was further revealed by study of the instantaneous pressure-volume relation in 3 out of the 7 patients. At the beginning of diastole both the pressure and volume were within normal range, but towards the end of diastole, the pressure climbed steeply to higher levels while the left ventricular volume failed to increase correspondingly. The coronary arteries were normal. Cardiac biopsy was carried out in 2 patients. One patient died and necropsy was performed. The pathological findings were those of moderate left ventricular hypertrophy and fibrosis with thick endocardium, but there was no eosinophilic infiltration.

The terms restrictive and constrictive cardiomyopathy have been used before but the left ventricular fault has not been examined in detail. A restrictive form of cardiomyopathy has now been defined in which the haemodynamic fault differs fundamentally from that in the hypertrophic or diluted cardiomyopathies, from amyloid heart disease or endocardial fibroelastosis. The dysfunction is believed to be secondary to endocardial disease in most, if not all cases.

Continuous assessment of left ventricular shape in man

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Frame-by-frame analysis of left ventricular angiograms has been performed in 10 normal subjects and 90
patients with heart disease. As well as calculating standard parameters, left ventricular shape index was derived as \((\text{cavity area}/(\text{perimeter}))^2\), which has a maximum value when the outline is circular. In normal subjects, systole was always associated with progressive reduction in shape index. This change was less pronounced in patients with low ejection fraction, and also when anterior or inferior ischaemia was present, even though ejection fraction was normal. During early diastole, shape index rose rapidly due to an increase in minor diameter occurring throughout the period of rapid filling. This preceded any changes in long axis, which were associated with upward movement of the aortic root as well as outward movement of the apex. The pattern of wall movement during atrial systole differed from that in early diastole in being mainly caused by an increase in long axis. These results have clear functional implications, and suggest that wall movement during filling, in particular, may be strikingly non-uniform, and that tacit assumptions about cavity shape used in the derivation of wall properties from estimates of ventricular volume may require modification.

**Observations on relation between heart sounds and valve movements by simultaneous echocardiography**

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By recording echoes from all four valves with high-frequency phonocardiograms, temporal relations between valve motions and heart sound components can be identified.

This technique has been used clinically for several months. Preliminary results indicate that:

1. Each valve closure is precisely coincident with the onset of a high frequency sound.
2. In normal subjects with a single first sound, mitral and tricuspid valves close almost simultaneously.
3. In normal subjects with two major first sound components, the first coincides with mitral closure and the second with tricuspid closure.
4. Where mitral and tricuspid closures are grossly asynchronous, as in right bundle-branch block or paced patients, each valve closure has an associated sound.
5. Ejection sounds occur at the moment of maximal semilunar valve opening. Pulmonary ejection sounds occur at this point, whether associated with pulmonary stenosis or hypertension.
6. Midsystolic clicks associated with the mitral valve prolapse occur when displacement of the valve leaflets towards the atrium is halted.

It thus seems reasonable to postulate that high frequency cardiac sounds are associated with the halting of valve leaflets after movement initiated by pressure changes.

**Echocardiographic estimation of the systolic pressure gradient in aortic stenosis**

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Values for systolic wall stress have been shown to be similar for normal and hypertrophied left ventricles in the absence of heart failure.

A constant representing systolic wall stress was derived from echocardiographic measurements of left ventricular systolic wall thickness and cavity minor axis, together with systolic blood pressure measured by sphygmomanometer, in 12 normal subjects. This constant (mean value \(110, SD \pm 7\)) was validated in 12 patients with left ventricular hypertrophy not due to aortic stenosis.

Using the simple relation: Left ventricular systolic pressure = \(110 \times \text{systolic wall thickness} - \frac{\text{minor axis}}{2}\), and sphygmomanometric measurements of systemic pressure, systolic aortic pressure gradients were calculated in 20 patients with varying degrees of aortic stenosis. These estimates compared favourably \((r=0.84, P<0.001)\), with the gradients measured at left heart catheterization, which were in 5 cases simultaneous.

**Early diastolic events in cardiac disorders**

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Left ventricular events during early diastole have been studied using non-invasive techniques in 52 patients. Simultaneous echocardiograms, showing mitral valve cusps and transverse dimension, apex cardiograms, and phonocardiograms were recorded and digitized. These were correlated with micromonometer pressure traces and frame-by-frame analysis of cineangiograms. Events were studied during:

1. Isovolumic relaxation, from aortic valve closure (A2) to mitral valve opening.
2. Early filling, from mitral valve opening to the o point on the apex cardiogram.
3. Period of ‘rapid filling’ on the apex cardiogram, from the o point to the third heart sound.

Increased transverse diameter before mitral valve opening was demonstrated in all patients, except those with severe mitral regurgitation or left ventricular disease. Significant left ventricular filling (dimensional changes after mitral valve opening) occurred in all patients while left ventricular pressure was still falling. ‘Rapid filling’ on the apex cardiogram and the third heart sound bore no consistent relation to wall movement, suggesting that factors other than filling contribute to their genesis. On the basis of these results, a model of left ventricular relaxation and early diastolic filling was presented.
Echocardiographic assessment of severity of aortic regurgitation

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Echocardiographic measurement of left ventricular dimensions may offer a non-invasive technique for documenting the severity of aortic regurgitation and providing criteria on which to base the long-term assessment of left ventricular function in this lesion.

Left ventricular diameter was measured by an ultrasound technique using a Smith Kline Ekoline 20 ultrasonicoscope in 20 patients with aortic regurgitation, and in 20 normal subjects. Estimates were made of the stroke volume, left ventricular output, and ejection fraction by the method of cubing the left ventricular diameter measured at end-diastole and end-systole.

In patients with aortic regurgitation of more than mild severity, the left ventricular end-diastolic diameter, stroke volume, and left ventricular output were increased compared with normals. The mean left ventricular end-diastolic diameter was 8.1 cm (range 5.4–9.1) in the patients, compared with 4.9 cm (range 4.2–5.8) in the normals, and the mean stroke volume 230 ml (range 125–465) compared with 83 ml (range 46–158). The mean left ventricular output was 16.7 l min⁻¹ (range 10–31) in the patients, compared with 5.3 l min⁻¹ (range 3.0–8.5) in the normals. The ejection fraction was similar in both groups.

The severity of aortic regurgitation was also graded by clinical and radiological methods. The echocardiographic data were compared with this grading to assess the value of the method.

Pulsus alternans: force-velocity and angiographic volume analysis in man

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Pulsus alternans was studied haemodynamically in 8 patients: 6 had aortic valve disease, 1 mild aortic and mitral valve disease with severe coronary artery disease, and 1 a cardiomyopathy.

Cardiac output was measured by indocyanine green dye dilution. Pressures for force-velocity analysis were taken using Telco (MM52) or Millar catheter tip manometers, and Kᵥmax derived. Angiographic volume analysis was performed using a light-pen computer system, with films taken in the right anterior oblique projection.

The only features shared by all 8 cases were: a low cardiac index; alternating LV systolic pressures; alternating diastolic filling period; alternating max and min dP/dt, and alternating Kᵥmax.

Inconsistent factors in this study of pulsus alternans were: Alternating left ventricular end-diastolic pressure, alternating left ventricular end-diastolic volume, alternating left ventricular end-systolic volume, absolute left ventricular end-diastolic volume, ejection fraction, end-diastolic wall stress, wall stiffness, ventricular mass, and ratio of mass to left ventricular end-diastolic volume.

Thus the change in contractility seems to be a primary phenomenon in the human heart, and is not necessarily linked to a change in left ventricular end-diastolic pressure, left ventricular end-diastolic volume, or left ventricular end-systolic volume. We do not think alternans is caused by changes in end-diastolic fibre length, decreased diastolic filling period, or decreased diastolic compliance before the small beat.

Pulsus alternans is primarily caused by an alteration of contractility.

Preoperative assessment of patients with Wolff-Parkinson-White syndrome

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Successful surgical correction of the WPW syndrome depends upon accurate preoperative localization of the accessory pathway and confirmation that the accessory pathway participates in circus tachycardia. The accessory pathway was localized at surgery by epicardial mapping in 13 consecutive patients (6 in the lateral mitral ring, 2 in the lateral tricuspid ring, and 5 in the septum). With lateral accessory pathway, preoperative pacing of the atrium adjacent to the accessory pathway caused more pre-excitation than pacing the contralateral atrium distant from the accessory pathway. Pacing from both sides in 3 of 5 patients with septal accessory pathway caused equal pre-excitation in 1, and more pronounced pre-excitation from the right atrium or left atrium in 2. During circus tachycardia the QRS complex showed no delta wave and simultaneous recordings from the low lateral right atrium, low atrial septum, and left atrium (via coronary sinus) revealed that retrograde atrial activation occurred early in all cases and in an abnormal sequence in those with lateral accessory pathway. The earliest atrial activity always occurred adjacent to the accessory pathway during circus tachycardia and during ventricular pacing with retrograde conduction over the accessory pathway. The accessory pathway was successfully divided in 9 of these 13 patients and arrhythmias were abolished.

Surgical treatment of ventricular tachycardia following epicardial mapping studies

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Four patients with intractable ventricular tachycardia were studied using programmed electrical stimulation of the heart, and shown to have a re-entry mechanism as the basis for the tachycardia. All 4 patients underwent epicardial mapping studies at open heart surgery. In each patient the tachycardia was initiated and the epi-
cardial activation times from 40 to 50 points on each ventricle were obtained using a bipolar electrode. After this the epicardial activation sequence during tachycardia was plotted in the form of an isochrone map. In Case 1 the re-entry activation front was found to begin in the posterobasal region of the left ventricle; in Case 2 the re-entry front was found in the anterobasal region of the right ventricle; in Case 3 the re-entry front was found to be in the outflow tract of the right ventricle involving the adjacent interventricular septum, and in Case 4 the re-entry front was found on the anterior surface of the left ventricle adjacent to the interventricular septum. In each patient an appropriate transmyocardial incision was made across the anterograde part of the re-entry front in order to interrupt the re-entry pathways.

Cases 1, 3, and 4 had short-lived attacks of tachycardia in the immediate postoperative period, but all 4 patients are free from tachycardia.

Repetitive and established paroxysmal tachycardias

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In repetitive paroxysmal tachycardia (Parkinson Papp syndrome) episodes are usually separated by no more than one or two sinus beats, and are initiated by extrasystoles that activate a re-entry circuit; the location may be atrial, AV nodal, or ventricular. We have observed 7 patients who met these criteria and confirm the usual absence of underlying disease, relative freedom from symptoms, and benign prognosis. A different form of AV nodal reciprocating tachycardia has recently been characterized, usually in children or young adults, and we describe 9 cases in most of whom programmed electrical stimulation of the heart was included in the diagnostic assessment. The oldest subject was 27. Attacks tend to be of long duration and also to be separated by relatively brief episodes of sinus rhythm or AV nodal dissociation. During attacks, the electrocardiogram usually suggests 'left atrial rhythm'. We have also recognized 4 cases in whom this mechanism has initiated reciprocating tachycardia in the Wolff-Parkinson-White syndrome. In these tachycardias reciprocation is induced by critical increases in heart rate; therapy and prophylaxis require control of sinus atrial and nodal discharge as well as modification of AV nodal and/or 'bypass' conduction.

Overdrive suppression and other tests of sinoatrial function

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The features of chronic sinoatrial disease are now a well-recognized entity and the diagnosis is relatively straightforward. These patients usually have sinus bradycardia in association with sinus arrest, junctional rhythm, and bouts of atrial flutter/fibrillation. However, in many instances, though a history of syncopal attacks and palpitations is obtained in association with bradycardia, it is often hard to prove that sinoatrial dysfunction is responsible since serial electrocardiograms may fail to capture the offending rhythms. Special investigations are needed in order to distinguish between physiological and pathological bradycardia. The paper reports the results of atrial overdrive suppression, premature atrial stimulation, and His bundle electrocardiography as means of attempting this distinction. Forty-eight cases of suspected sinoatrial disorder were investigated and compared with 63 patients with heart block but with no evidence of sinoatrial disease.

Extending the life of a pacemaker

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For the past 6 years all pacemakers removed electively from patients at St. George’s Hospital have been placed in an incubator to continue simulated pacing conditions. The units have been checked at regular intervals to determine how they have been functioning and in particular to establish the modes of failure.

Using these results, it has been possible to extend reliably the period of patient-use to 3 years for a fixed rate Devices unit. However, the major cause of premature failure is associated with batteries. In order to safeguard against this a unit has been developed which enables the patient to check the functioning of his pacemaker daily (or more often if necessary) at home without the need for frequent visits to the pacing clinic. This in turn has led to a total decrease in attendances at clinics without a diminishing service to pacemaker patients.

24-hour electrocardiogram monitoring of ambulatory outpatients suspected of dysrhythmias

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Continuous 24-hour electrocardiogram monitoring using the Oxford tape recorder system was performed on 130 ambulant outpatients who presented to the General Medical Clinic with symptoms of blackouts, dizzy turns, faints, or palpitations. Twelve-lead electrocardiogram at rest and on exercise was normal in 56 per cent and abnormal but not diagnostic in 36 per cent. In these patients 24-hour monitoring of the electrocardiogram revealed dysrhythmias accounting for the symptoms in 60 per cent, and in a further 24 per cent dysrythmias were seen but not sufficient to account for the
symptoms. The remaining 16 per cent were in sinus rhythm throughout.
In those 8 per cent with 12-lead electrocardiograms considered diagnostic, additional unsuspected dysrhythmias were revealed in 63 per cent.
Supraventricular dysrhythmias alone were seen in 46 per cent, ventricular dysrhythmias in 6 per cent, and both supraventricular and ventricular dysrhythmias in 32 per cent. Episodes of ventricular tachycardia were seen in 5 per cent of patients and atrial arrest (2 to 30 s) with no ventricular escape in 8 per cent.
We conclude that in patients suspected of dysrhythmias 24-hour monitoring of the electrocardiogram gave positive diagnosis even in the presence of a normal or non-diagnostic 12-lead electrocardiogram.

Computer and human interpretation of electrocardiogram correlated with cardiac pathology

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To assess the performance of the Cardionics 12-lead electrocardiogram computer programme in patients with heart disease, comparisons have been made with both the patients’ clinicopathological data and the electrocardiographic interpretations of two human readers.
In the study were 12 patients with aortic valve disease, 22 with mitral valve disease, 15 with coronary artery disease, 10 with cardiomyopathy, and a miscellaneous group with congenital heart disease and pulmonary hypertension. The clinico-pathological data were obtained from clinical examination, biochemical studies, lung function tests, radiology, echocardiography, cardiac catheterization, cardiac surgery, and necropsy material. The electrocardiograms were read initially double-blind by the human readers. They then compared and combined their interpretations to arrive at the interpretation used for the study.
From the clinicopathological data it was estimated that 45 patients either had left ventricular hypertrophy or conditions causing it. The computer diagnosed 28 correctly, with 2 false positives; the human readers diagnosed 22 correctly with 2 false positives. 35 patients similarly had right ventricular hypertrophy. The computer diagnosed 6 with 1 false positive; the human readers diagnosed 11 with 2 false positives. 14 patients had anterior myocardial infarction. The computer diagnosed 5 with 12 false positives; the human readers 4 with 8 false positives. 12 patients had lateral infarcts. The computer diagnosed 2 with 4 false positives; the human readers 3 with 0 false positives. 13 patients had infero-posterior infarcts. The computer diagnosed 4 with 2 false positives; the human readers 4 with 1 false positive.
It is concluded that present techniques of 12-lead electrocardiographic diagnosis of patients with heart disease, either by computer or by human reader, give a correct diagnosis in only 38 per cent of cases.

Effects of clonidine on baroreflex arc in conscious rabbits and man

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The action of clonidine (Catapres) on single aortic nerve baroreceptor fibre discharge was examined in anaesthetized normotensive and renal hypertensive rabbits. There is an increase in discharge at a given arterial pressure which is dose dependent.
We also compared the effects of graded doses of intravenous clonidine (0.25–3.0 μg kg⁻¹ min⁻¹) and clonidine injected into the lateral cerebral ventricle 0.5–1.5 μg kg⁻¹ on the mean arterial pressure-pulse interval response curves of unanaesthetized normotensive and renal hypertensive rabbits in order to assess in each the excitability of the cardiac autonomic effectors. In both normal and hypertensive rabbits there was a striking increase in the gain of the baroreflex arc controlling heart rate. This was largely because of a sensitization of vagal motoneurones.
It is concluded that at least part of the hypotensive effect of clonidine is mediated through increased sensitization of the baroreflex arc.
Preliminary results of the action of clonidine on the baroreflex arc in man are presented.

Problems associated with development of radioimmunoassay for urinary digoxin and its use in clinical assessment of biological availability

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Determination of urinary digoxin excretion seems to offer advantages since single plasma measurements cannot account for fluctuating levels, and serial measurements are inconvenient. Urinary excretion depends on plasma levels over a period of time, and reflects dose administered and biological availability. Studies using single doses of tritiated digoxin suggest a high recovery of digoxin in the urine, with little catabolism.
This study employs a simple specific radioimmunoassay for urinary digoxin, developed from one previously described for plasma, applied to the measurement of digoxin excreted by patients in a steady state, on maintenance therapy with several oral digoxin preparations.
Twenty-four urinary levels were found to be proportional to dosage in each patient, and varied between individuals, though consecutive 24-hour collections from an individual were similar. Total urinary recovery was low. This is possibly because of analytical errors (unlikely because of the high precision and accuracy of the assay and reproducibility using another assay), high biliary excretion, or, more likely, the excretion of significant concentrations of a digoxin metabolite not detected by the assay.