Swedish Society of Cardiology and the British Cardiac Society

A Joint Meeting of the Swedish Society of Cardiology and the British Cardiac Society was held at the House of the Swedish Physicians in Stockholm from 31 August to 3 September 1975.

At the opening session a speech of welcome was made by the Chairman of the Swedish Society, H. Eliasch, to which the President of the British Cardiac Society, J. F. Goodwin, replied. An inaugural address on Carl Linnaeus was given by G. Strandell. The generous hospitality of the Swedish hosts included a dinner after the opening session, personal invitations to the homes of Swedish cardiologists, and dinner on the final evening. An official luncheon in Stockholm Town Hall on the closing day was preceded by visits to hospitals in the morning and followed by a boat trip in the Archipelago in the afternoon. A very enjoyable ladies’ programme was organized by Mrs. Harald Eliasch. The excellent organization of the meeting was in the hands of the Secretary of the Swedish Society, L. Mogensen.

The scientific sessions included two symposia. The Moderator for ‘The Prevention of Heart Disease’ was G. Björck and the speakers were June Lloyd, T. Lundman, M. Oliver, and L. Wilhelmson. The Moderator for ‘Physiological and Clinical Exercise Testing’ was E. Sowton and the speakers were G. Blonquist, R. Edwards, B. Jonsson, and P. Sleight.

The following communications were given.

Abstracts

Pre-hospital coronary care provided by ambulancemen

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The experimental use in Brighton of coronary ambulances manned only by ambulance personnel has been continued and developed during 1974. The special equipment is portable and compact, and can be carried on any vehicle. The ambulancemen monitor the heart rhythm of patients carried in the ambulance, use a defibrillator if indicated, and also administer intravenous lignocaine or atropine in carefully defined circumstances. Electrocardiograms are also recorded on magnetic tape for subsequent analysis in the coronary care unit. Over 1000 patients were carried during the year, and approximately half were suffering from acute myocardial infarction or coronary insufficiency. More than 90 per cent of rhythm diagnoses were identified and interpreted correctly by the ambulancemen. Atropine was given 68 times and lignocaine 41 times; in all but one instance the indications were entirely laid down. Fifteen patients were resuscitated successfully to be discharged from hospital alive. Fourteen of these had ventricular fibrillation, and in six cases the cardiac arrest had occurred before the ambulance arrived.

Effects of cardioselective beta-blockers on chest pain, ECG, and heart function in acute myocardial infarction

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Catecholamines were found to increase the experimental infarction size in dog hearts in situ and aggravate the ischaemic damage of the isolated rat heart. During the first two days of acute myocardial infarction there might be an inappropriate sympathetic activity, as judged from shorter systolic time intervals (PEP and ICT) and higher systolic blood pressure and heart rate compared to normal controls. A reduction in sympathetic activity occurs during the first week of infarction. Beta-blockage therefore seems reasonable early in infarction to reduce myocardial ischaemia. Therefore, a single intravenous injection of practolol (ev. 18-2 mg) was given to 10 patients on the first day of AMI. This reduced systolic blood pressure and heart rate and prolonged the systolic time intervals to normal control values. No changes were seen in the left ventricular ejection time corrected for heart rates (LVETI) or in the raised a/H-ratio. All patients given practolol got an immediate relief of the chest pain. A double-blind study of 30 patients confirmed these effects of practolol in AMI. In two double-blind studies including 40 patients with acute myocardial infarction similar effects were obtained on heart work and chest pain by the cardioselective beta-adrenergic blockers H 87/07 (similar to practolol) and H 93/26 (cardioselective with no intrinsic activity). The beta-blockade also reduced ST segments in these patients with acute myocardial infarction.

The injection of cardioselective beta-adrenergic blockers was found to reduce heart work, ischaemic chest pain, and ST elevation in patients with AMI with no major side-effects. Prolonged beta-blockade during the first days of AMI might decrease the severity of myo-
cardial ischaemia and limit the area of infarction as found in animal experiments.

Continuous recording of praecordial ST segment elevation during myocardial ischaemia

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Recent attempts to reduce the extent of necrosis after myocardial infarction have led to an interest in non-invasive methods of its assessment. The measurement of ST elevation from multiple praecordial sites has been used by many current methods of recording and analyses are cumbersome and not reproducible.

A system has been developed for the continuous recording on magnetic tape of the mean ST elevation from a maximum of 35 praecordial leads: on replay, a purpose-built computer provides further analysis and a running print-out of the sum of ST (ΣST). A simultaneous print-out of the heart rate has shown the relation between ΣST to rate change. This system has been compared to, and controlled by, a more orthodox measurement of a 35-lead ST praecordial map. The system has the advantage of preventing errors caused by repeated application of electrodes; of eliminating the subjective element in measurement; and providing relative convenience for the patients.

The natural history of ST segment variation has been studied over a 2-hour period in a group of patients admitted within 6 hours of symptoms of a heart attack. These results were described. In addition, the effect of antilipolytic treatment on the continuous recording of ΣST has been studied in patients within 6 hours of the onset of symptoms. The results from these studies indicate the value of this system in assessing any attempt to reduce the degree of acute myocardial ischaemia.

Prediction of mortality risk in acute myocardial infarction

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Serial serum estimations of total creatine kinase (CK) were obtained in 104 patients with acute myocardial infarction (AMI). Seventy of these had a history without a previous AMI. Relating the CK maximum to age, in this group, divided the patients into two subgroups, one with 49 per cent and the other with 3 per cent mortality during the hospital stay plus the next 90 days. This high or low mortality risk could be predicted in 68 of these 70 patients. Of 18 hospital and convalescence deaths, 17 could be correctly predicted, with an overprediction of 18 of the 50 survivors. This prediction could be done 3 to 8 hours after admission. The 34 patients with a previous AMI could also, relating CK maximum to age, be divided into two subgroups, one with 58 per cent and the other with 7 per cent mortality. These patients, however, were too few to permit prediction.

Myocardial scintigraphy with 99 mTc pyrophosphate and 201 pallidum

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Sixty-five patients admitted to the coronary care unit with suspicion of acute myocardial infarction (AMI) received 10 mCi 99 mTc pyrophosphate and were examined in the left anterior oblique position with a mobile gamma camera (Portacamera, General Electrics), 12 to 120 hours after onset of symptoms. Two scintiscans were obtained initially at injection and then every 15 minutes during an hour, the scintigrams were evaluated with regard to uptake in the myocardial area. The localization was decided after contrast enhancement.

In 5 of 6 patients with angina pectoris, uptake was found. In 51 patients with a myocardial infarction uptake was found in 48. In 39 of these there was a good correlation between electrocardiographic and scintigraphic localization. Eight patients were discharged with other diagnoses and only one showed uptake.

In all patients with coronary heart disease a time course was observed with high initial uptake, which diminished on the 15- and 30-minute scintigrams, and another uptake starting to increase on the 45- and 60-minute scintigrams.

Five patients were also investigated with 201 Tl. A good correlation between the area of increased uptake with 99 mTc pyrophosphate and the region of decreased uptake with 201 Tl existed.

Formation of coronary arterial thrombi in relation to onset of necrosis in acute myocardial infarction in man: a clinical and autoradiographic study

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The presence of radioactivity in coronary arterial thrombi was studied at necropsy in 12 patients with acute myocardial infarction who had been given radiolabelled fibrinogen for the detection of deep venous thrombi. Seven patients received 125I- and 5 patients 131I-labelled fibrinogen. The radioactivity in the coronary thrombi was detected with autoradiography. With a short interval, below 10 hours, between onset of symptoms and injection of fibrinogen the entire thrombus was radioactive in 4 of 5 patients, whereas with longer time intervals only parts or none of the thrombus contained detectable radioactivity. The findings give further evidence that thrombus formation in acute myocardial infarction probably is a slow process and that the major part of the thrombus may form after the onset of necrosis.
Ventricular arrhythmias three weeks and one year after acute myocardial infarction (AMI)

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A hundred consecutive patients under 66 years treated for an AMI have been investigated for ventricular ectopic beats (VEBs) with electrocardiograms obtained by telemetry to an ink-jet writer. Registrations were performed before discharge three weeks after the AMI and one year later. Recordings consisted of three hours of daily activities and exercise and three hours at night. No routine antiarrhythmic therapy was given.

Three weeks after an AMI, VEBs were seen in 70 patients—34 of them had had uniform only, 22 multiform only, a further 11 had paired VEBs, and another 2 showed the R/T phenomenon. One patient had ventricular tachycardia. VEBs were found in 59 per cent of night and 50 per cent of day recordings. The occurrence of VEBs was significantly associated with larger infarctions, reinfarction, ventricular tachycardia, or ventricular fibrillation during the acute phase.

To date 79 patients have been followed for one year. Ten have died, 4 instantaneously. Another patient had circulatory standstill but was resuscitated. A further 7 were not examined because of reinfarction. Sixty-one patients were reevaluated for arrhythmia. The incidence of VEBs increased from 58 per cent to 77 per cent in patients who had a primary infarction whereas those with initial reinfarction had an incidence of 83 per cent on both occasions.

Procainamide and phenytoin: a comparison of two antiarrhythmic drugs

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In order further to evaluate procainamide and phenytoin, considering both efficacy and side-effects, some comparative studies on the two drugs have been performed. The antiarrhythmic effects were studied in 81 patients with suspected or proven myocardial infarction. Those patients who developed ventricular arrhythmia requiring treatment during the first 8 hours in hospital were randomised to a procainamide or phenytoin group. *Therapeutic plasma levels were rapidly achieved* by intravenous and oral loading doses. An electrocardiogram was continuously recorded during the 24-hour trial and analysed minute by minute. During the first 2 hours after initiation of therapy, a significantly higher frequency of therapeutic failure was found in the phenytoin group (23 of 35 patients) compared with the procainamide group (13 of 39 patients). Also side-effects were more frequent in the phenytoin group. The haemodynamic effects of the two drugs, given intravenously during conventional cardiac catheterization on 19 patients with congenital heart defects, have also been studied. With equipotent plasma concentrations of the drugs phenytoin had the most pronounced negative inotropic effect.

Thus, the results of these investigations show the overall advantage of procainamide over phenytoin during short-term therapy after acute myocardial infarction.

Analysis of preoperative and operative factors influencing outcome of aortocoronary bypass surgery in 146 patients

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Thirty-five (24%) of 146 consecutive patients followed for at least 6 months (mean 13.4 months) had recurrent angina after coronary artery bypass. They were compared with the asymptomatic group to determine which pre- and intra-operative factors are important in the recurrence of angina. There was no difference in the pre-operative age, sex, duration or grade of angina, mean serum cholesterol, incidence of myocardial infarction, or graft flow. The maximum level in the preoperative exercise and the maximum pacing rate were higher in the asymptomatic group (P < 0.01 and P < 0.05, respectively). Left ventricular end-diastolic pressure, left ventricular angiographic, and total coronary scores were similar in the two groups, but there were fewer coronary scores of 10 and 11 (of 12) in the angina group (P < 0.025).

The angina-free group had more grafts (P < 0.025), and more left ventricles were judged normal at operation (P < 0.01). Scoring grafted vessels as zero, they had a lower coronary score after operation (P < 0.01), and more had zero scores (P < 0.01). All postoperative myocardial infarcts occurred in the patients with angina.

We conclude that the degree of revascularization is the most important factor in determining the outcome. Patients with demonstrably good stress tolerance are more likely to be relieved of angina. The occurrence of clinical postoperative myocardial infarction has an adverse effect.

Evaluation of early and long-term function of aortocoronary saphenous vein grafts

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During the past 3 years, 180 patients (with 310 grafts) underwent repeat angiography after aortocoronary saphenous vein bypass graft operations. This constitutes 40 per cent of the total number of patients undergoing this operation at Harefield Hospital and included routine restudies as well as patients with residual or recurrent symptoms.

The overall graft patency rate was 91 per cent while 96 per cent of the patients had 1 or more patent grafts. The patency rate in patients studied during the first 3 months after operation was 96 per cent while in those studied 4 months to 4½ years the patency rate was 82 per cent. There was no difference in the patency rate of grafts to different coronary vessels. Additional gas endarterectomy (performed in 25% of the cases), diabetes, hypertension, previous infarcts, or venosclerosis...
affecting the saphenous vein did not seem to affect the patency rate. There was no evidence of progressive dilatation or narrowing of the grafts. Left ventricular function, assessed by ventriculography did not change in the majority of patients, though in some cases there was improvement in ejection fraction and pattern of left ventricular contraction. There was no evidence of significant accelerated progress in the native vessel disease.

It is concluded that aortocoronary saphenous vein grafts continue to function satisfactorily for periods of up to 4½ years.

Haemodynamic long-term follow-up of patients with aortic valvular disease
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In an attempt to find measurements of prognostic significance 128 patients with aortic valvular disease were studied by repeated graded exercise tests including determinations of cardiac output. The patients were forced to the limit of tolerance with exhaustion, excessive dyspnoea, or intense angina pectoris as restricting factors. More than 400 exercise tests of this kind have been performed without serious complications. Normal physical working capacity (PWC) was defined as 600/900 kpm/min (women and men, respectively) during at least 4-5 minutes.

The courses were poorly correlated to aortic valvular area, degree of regurgitation, left intraventricular pressures, heart enlargement, and electrocardiographic abnormalities. Patients with a normal PWC attaining effective stroke volume index (SVI) above 50 ml/m² and cardiac index (CI) more than 71/min per m² on maximal load, had an unchanged course during 3 to 5 years, while those with SVI and CI below these limits deteriorated clinically and haemodynamically during the subsequent 1 to 3 years. Patients with subnormal PWC and effective SVI around or below 40 ml/m² on maximal load had a bad prognosis with further deterioration and even sudden death (11 cases) within less than one year. Some younger patients with SVI exceeding 40 ml/m² were improved, while others presented an unchanged course or deteriorated in parallel to a reduction of SVI below 40 ml/m² on maximal load.

Influence of preoperative left ventricular function on results of homograft replacement of aortic valve for aortic stenosis
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The effect of preoperative left ventricular function on early and late prognosis was assessed in 50 consecutive patients with aortic stenosis, who underwent left ventricular angiography before homograft replacement of the aortic valve. Patients were divided into 2 groups, those with a left ventricular ejection fraction of 0·46 or more (group A), and those with an ejection fraction of 0·45 or less (group B). Other indices of poor left ventricular function in group B included a low cardiac index with raised left ventricular end-diastolic pressure. Group A consisted of 29 patients, with a mean age of 52 years, and group B of 21 patients, with a mean age of 55 years.

There were no operative deaths in either group and no late deaths in group A. One patient in group B (4%) died late. The length of follow-up varied from 3 months to 5½ years. Symptomatic improvement was observed in all patients in both groups. Ninety per cent of patients in group A moved to Class I of New York Heart Association Classification compared with 70 per cent of group B. Regression of electrocardiographic and radiological signs of left ventricular hypertrophy were more consistent in group A. Repeat angiography in 10 patients of group B showed improvement of ejection fraction in 7.

It is concluded that poor left ventricular function in patients with aortic stenosis does not increase the risk of homograft valve replacement. Though many patients with poor left ventricular function derive considerable benefit from the operation, the overall improvement and probably the long-term survival is less than in patients with good left ventricular function.

Aortic valvular surgery using hypothermia without coronary perfusion
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At the University Hospital of Lund aortic valvular surgery has been performed since 1970 using Björk-Shiley disc prosthesis inserted under local hypothermia with the aid of 'Bretschneider's solution', making coronary perfusion unnecessary. This paper deals with the first 50 patients operated in this way. The preoperative mortality was 8 per cent (4 patients). A further 8 patients died from a variety of causes during the next 6 months.

The paper comprises: (1) a brief description of the method used, and (2) a comparison between preoperative findings and results obtained one year postoperatively in 38 patients, including physical working capacity (PWC), haemodynamics during maximal work load (cardiac index, CI; stroke volume index, SVI; and intra-arterial blood pressures), electrocardiographic abnormalities, and radiological heart size.

In summary there was a significant increase of PWC, a striking improvement of CI and SVI on maximal work load, a significant regression of electrocardiographic signs of left ventricular hypertrophy and strain, and a reduction of heart size in most patients. Angina pectoris which was a common symptom preoperatively was seen in only 3 patients one year after operation.

Pulmonary autograft for aortic valve disease: 3 to 8 years later
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Between 1967 and 1971, 100 patients, aged 12 to 54 years, whose diseased aortic valve was replaced by their
own pulmonary valve (autograft) and a cadaveric aortic homograft placed in the right outflow, have been followed for 3 to 8 years with haemodynamic studies. Of these, 65 had operation for lone or dominant aortic regurgitation and 35 had severe stenosis. After operation important aortic regurgitation occurred in 16, dating from early postoperative weeks in the majority and in patients operated on for serious aortic regurgitation. Re-operation (6%) constantly confirmed malpositioning of autograft. Acute valve failure occurred in one patient—the only patient in whom a frame-mount was used. Late mortality was low (3) and so was the incidence of bacterial endocarditis (3). There was no haemolysis or gradients above 10 mmHg (1.3 kPa) across the autograft; neither was there progressive obstruction on either side of the heart nor need for anticoagulants. There was a slight increase in aortic regurgitation in those in whom it was present after operation after 5 years. Trivial diastolic murmurs occurred in 56 patients without evidence of important or increased pulmonary or aortic regurgitation. Histology in the few autografts examined suggests the presence of living cells in the valve up to five years after operation. In spite of the higher early mortality in comparison with other forms of aortic valve replacement and the added morbidity from the right-sided homograft, the low incidence of late complications suggests this may be the best aortic valve replacement, particularly in the young patient.

Plasma aldosterone levels in patients with severe, treated heart failure

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The availability of patent aldosterone antagonists has raised the question of the incidence and importance of hyperaldosteronism in congestive heart failure. The development of a sensitive method for aldosterone, based on radio-immunoassay, has allowed it to study the plasma levels of this hormone in patients with severe treated heart failure. The findings have been correlated with plasma levels and total exchangeable sodium and potassium by radioisotope dilution methods: in some patients extracellular fluid volume was also measured as the sulphate space.

Twenty-five patients were studied in a stable state on unmodified optimal treatment, mostly while awaiting valve replacement.

The plasma aldosterone was ten times the normal level in some patients. The changes were related to the severity of the heart failure, as judged by the right atrial pressure at cardiac catheterization (r=0.63), the fall in total exchangeable potassium (r=0.60), and the intensity of diuretic treatment measured as a daily dose of frusemide (r=0.77). There was little relation to the plasma electrolyte changes.

Although aldosterone antagonists are useful to maintain plasma potassium levels during intensive diuretic therapy, the effect on tissue electrolytes is complex, and may not be entirely beneficial.

Prolonged hyperaldosteronism may be a factor in the production of cardiac cachexia and the loss of cellular potassium cannot necessarily be replaced by supplements in this situation.

Consideration should be given to possible surgical treatment before heart failure reaches this degree of severity.

Echocardiographic diagnosis of primitive ventricle

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Recent morphological studies in our institute have shown that the pathognomonic feature of ‘primitive ventricle’ (considered by us to be a better term than any of the synonyms ‘single’, ‘common’, or ‘double inlet left’ ventricle) is absence of the posterior segment of the interventricular septum. The anterior part of the septum may or may not be present. When present, it, together with a bulboventricular ridge, forms an anterior or bulboventricular septum. This feature enables the condition to be classified into primitive ventricle with or without an outlet chamber. While presence of this outlet chamber is of significance, and can be established angiographically, knowledge of the presence or absence of a posterior septum separating the atrioventricular orifices is of paramount importance to subsequent surgical management. In our experience this diagnostic feature cannot be unequivocally proven using angiographic techniques. However, use of strip chart, single beam M mode echocardiography enables presence or absence of this part of the septum to be elucidated. When the posterior septum is absent, the septal cusps of the atrioventricular valves approximate each other during diastole. In contrast, when the septum is present the typical bilaminar septal echoes are seen separating the echoes from the valve septal cusps.

We have found this technique of considerable value in distinguishing between the diagnoses of transposition with ventricular septal defect and primitive ventricle or double outlet right ventricle and primitive ventricle. In addition, the technique has enabled deformities of one or other atrioventricular valve to be identified in primitive ventricle, again a matter of considerable importance in determining future surgical management of the anomaly.

Value of echocardiography in diagnosis of congenital mitral stenosis

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Ten patients with congenital mitral stenosis have been studied by conventional single element echocardiography. The diagnosis was verified at operation or necropsy in 9 patients. One of the patients had an isolated
congenital mitral stenosis. In the other patients this lesion was combined with different congenital heart malformations (coarctation of the aorta 6 patients, ventricular septal defect, and/or persistent ductus arteriosus 3 patients). Three patients were operated upon with a mitral valvotomy together with a correction of the associated heart malformation.

The first echocardiographic examination was in 5 patients performed during the first year of life. The deviations from normal in the echocardiogram consisted of a reduced closing velocity of the anterior mitral leaflet during the rapid filling period in diastole and a more or less reduced amplitude of opening movement of the anterior mitral leaflet in the beginning of diastole.

In all patients it was possible to diagnose the mitral stenosis by echocardiography and to separate those with a severe degree of stenosis from those where the degree of stenosis was slight. A clearly reduced amplitude of movement seems to indicate that it is not possible to perform only a mitral valvotomy.

**Repair of mitral regurgitation in paediatric age-group**

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Between May 1971 and December 1974, 25 infants and children aged between 7 months and 14 years (8 were below the age of 4 years) presented with severe mitral regurgitation. A reconstructive valve-conserving operation was performed in all patients except 1, who was treated by homograft valve replacement. The aetiology of the valve disease was congenital in 13 and rheumatic in 5 patients. Associated lesions included atroventricular defects in 5, 1 or whom had a complete atrioventricular canal, Marfan’s syndrome in 1, ventricular septal defect in 2, and corrected transposition with Ebstein’s anomaly in 1 patient. Severe pulmonary hypertension was present in 10 patients.

Surface-induced profound hypothermia was used in 5 patients. There were no operative or late deaths. One child had residual cerebral damage.

Clinical evaluation with a follow-up period of 3 months to 4 years (mean 23 months) showed evidence of good correction of the lesion in all but 1 patient who has the signs of residual moderate mitral regurgitation.

It is concluded that conservation of the valve is possible in the majority of infants and children with severe mitral regurgitation. The early and late results of this procedure have been encouraging.

**Repair of ventricular septal defects in infancy**

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Between May 1971 and March 1975, 31 infants under 1 year of age underwent closure of a ventricular septal defect at the Hospital for Sick Children, Great Ormond Street, London. Their weight ranged from 2.5 kg to 6.5 kg (mean of 4.6 kg). The indication for surgery was intractable heart failure in spite of maximal medical support. An inadequate banding of the pulmonary artery had been done in 7 of the infants before closure of the ventricular septal defect. All the patients had a left-to-right shunt greater than 2:1, pulmonary hypertension (Pp/Ps greater than 0.6), and normal pulmonary vascular resistance. There were associated cardiac anomalies in 12 cases. Both the techniques of circulatory arrest, with deep hypothermia and conventional cardiopulmonary bypass were used. The defects were closed through a right atriotomy or a right ventriculotomy. There were no operative deaths and one late death which was not related to the cardiac disease. The ventricular septal defect re-opened in 2 cases, one of which was reoperated successfully.

Our current policy is to close the ventricular septal defects with large left-to-right shunts rather than to band the pulmonary artery when the infants fail to improve with medical treatment.

**Congenitally corrected transposition of the great arteries: a clinical study of 101 cases**

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The anatomy, natural history, clinical features, and symptomatology of 101 cases of congenitally corrected transposition of the great arteries (C-TGA) diagnosed over a 20-year period at The Hospital for Sick Children, Toronto, Ontario, is reported. Only 1 patient had no associated lesion and only 14 an intact ventricular septum. Pulmonary stenosis was present in a little less than half of the patients. Left AV valve involvement existed in 21 patients.

In spite of the high incidence of associated lesions the prognosis was better than expected. Seventy-six patients were alive at the end of the study, no one being critically ill. No death occurred among the patients with intact ventricular septum. Patients with pulmonary stenosis as a group developed symptoms later, were less handicapped, and had a better prognosis than those without a pulmonary stenosis. One-third of the patients had dysrhythmias. Eight patients had a 3rd degree AV block.

Auscultatory findings were not very helpful when diagnosing C-TGA. Forty-seven patients had a single second sound; of these, 37 had pulmonary stenosis. In 60 per cent of the patients, where information was available, the second sound was reported to be of normal intensity.

Our surgical experience is documented and discussed. Palliative procedures were done with successful results and low mortality. Totally corrective procedures were performed in 9 patients with 4 deaths.
Association of secundum atrial septal defect with abnormalities of atrioventricular conduction or left axis deviation: a new syndrome

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A genetic analysis was made of 10 families in which the propositi had a secundum atrial septal defect associated with abnormal atrioventricular conduction (first, second, or third degree heart block) or unexplained left axis deviation, or a combination of these conduction disturbances. Diagnostic information was available on 51 (81%) of the 63 first degree relatives. Three of the families appeared to be examples of a new syndrome which, with variable expression, was inherited as a non-sex-linked autosomal dominant. The main features were a secundum atrial septal defect, disease of the conducting tissue, which in some cases was progressive, unexplained left axis deviation, and unexpected death. These families did not appear to be examples of the Holt Oram syndrome for the upper limbs were clinically and radiologically normal in the 19 members examined.

The importance of recognizing this syndrome is the occurrence of progressive disease of the conducting tissue and the risk of sudden death.

In the remaining 7 families there was only one affected first degree relative out of 39 examined. He was the son of one of the propositi and had paroxysmal coronary sinus rhythm with an intact atrial septum.

Effect of therapeutic doses of lignocaine hydrochloride on effective refractory period of right ventricle in humans

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The effect of lignocaine on the effective refractory period of the right ventricle (EVRP) was measured in 10 patients using the extra-stimulus technique of progressive introduction of premature stimuli until a response could not be elicited. In 4 patients lignocaine produced a significant decrease in the EVRP, in a further 4 patients there was no change in EVRP, and in the remaining 2 patients lignocaine produced a significant increase in the EVRP. The effect of the drug on the EVRP could not be correlated with the serum potassium levels.

The reasons for the variable effect of lignocaine are attributed to the different action of lignocaine on different components of conduction within the ventricle.

Electrophysiological properties of right atrium in relation to physical training and heart rate

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The monophasic action potential (MAP) and the effective refractory period (ERP) of the right atrium were determined in eight healthy volunteers before and after hard physical training. The duration and amplitude of the MAP increased in seven individuals after training. ERP showed no consistent changes. One possible explanation for the findings is an accumulation of intracellular potassium. The duration of the MAP was well correlated to maximal heart rate.

Management of paroxysmal supraventricular tachycardia using a scanning pacing system

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Programmed stimulation of the heart has shown that a re-entry process is commonly the mechanism of supraventricular tachycardias. Under these circumstances it is usually possible to terminate the tachycardia by suitably timed premature beats. However, it has been found in any one patient that the point at which premature beats can terminate a tachycardia varies over 20 to 30 ms. This means that premature beats with preset coupling times cannot be relied on consistently to terminate an attack. A pacing system has, therefore, been developed which is automatically activated when tachycardia occurs and induces single or double atrial or ventricular premature stimuli with a preset coupling time. The first stimulus occurs within the refractory period of the stimulated chamber, then one second later a second stimulus is induced but is delayed by 5 ms. Five millisecond increments in delay occur every second until 100 ms has been scanned. When the tachycardia is terminated stimulation ceases. This scan can occur using either single or double stimuli. Two patients with intractable supraventricular tachycardia have been fitted with a Devices 4273 miniscan. In one patient the cardiac cycle is scanned with single ventricular premature beats and in the other double ventricular premature beats.

In both patients their tachycardias are now consistently terminated within 5 to 20 seconds of the spontaneous onset of the tachycardia.

Ultrastructural investigation of cardiac biopsies

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Twenty-five patients who underwent complete cardiac investigation including endomyocardial biopsy from the right ventricle are presented. The clinical and haemodynamic findings are presented together with comments on the biopsy method used. Special attention is given to the ultrastructural changes, with particular reference to the occurrence of artefacts. Different fixation methods have been used in order to obtain an optimal result from a technical point of view. Careful and uniform handling of the tissue samples seems to increase the theoretical as well as the practical usefulness of cardiac biopsy. On the basis of our experience in more than 150 cases the method used is considered simple, reliable, and safe.
Adaptation of left ventricle to sudden changes in heart rate in patients with artificial pacemakers

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In five patients treated with external pacemaker for complete heart block, the adaptation of left ventricular volume to sudden changes in ventricular rate has been analysed on a beat-to-beat basis, by one-plane cineangiography and simultaneous aortic pressure recordings. The results are unambiguous and show: (1) When heart rate is changed above a certain lower limit left ventricular stroke volume varies inversely with heart rate. (2) The changes in stroke volume are brought about by changes in left ventricular end-diastolic volume proportionate to the duration of mechanical diastole and filling energy. The end-diastolic volume is unchanged. (3) Left ventricular end-diastolic volume and stroke volume adapt, on a beat-to-beat basis, to changes in heart rate. (4) There is a lower border frequency at which the ventricular end-diastolic volume is maximal. Below this frequency, a further increase in length of diastole does not increase the stroke volume.

Functional defect in amyloid heart disease
‘the stiff heart syndrome’

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Left ventricular performance was studied in three patients with heart failure as a result of amyloid. The diagnosis was proven by cardiac biopsy in 2 patients and by rectal biopsy in the third. The left ventricular diastolic pressure was high throughout diastole and exceeded the pressure in the right ventricle by at least 10 mmHg (1.3 kPa) in each case. Pressures were recorded through a catheter tip manometer during injection of contrast medium into the left ventricle allowing simultaneous volume measurement. The left ventricular volumes were not increased at end-diastole (62–118/m^3) despite very low ejection fractions (between 40 and 43%). The left ventricle was shown to be stiff since for each diastolic volume the pressure was higher than normal and for each volume change (AP) was greater than normal.

The unyielding poorly contracting and slowly filling left ventricle explains the slow stroke output and hypertension. The left ventricular angiogram showed normal contours but coarsened trabeculation and prominent papillary muscles.

Both the functional defect and the angiographic appearance are unique to amyloid and differ from the dysfunction caused by other known disorders of the myocardium, the generalized systolic impairment of dilated cardiomyopathy, the focal faults caused by coronary artery disease and the diastolic abnormalities of hypertrophic cardiomyopathy, restrictive cardiomyopathy, and constrictive pericarditis.

Left ventricular pressure-volume relations in hypertrophic cardiomyopathy, with effect of beta blockade

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Left ventricular diastolic compliance was studied in 8 patients with hypertrophic cardiomyopathy (HOCM) of the left ventricle, before and after the intravenous administration of 0.3 mg practolol per kg body weight. Simultaneous measurements of left ventricular pressure and volume were made throughout ventricular diastole. The pressures were recorded as contrast medium was injected into the left ventricle through a catheter sheath surrounding a catheter-tip manometer. Left ventricular volumes, calculated by planimetry from the right anterior oblique cineangiogram were directly related to the simultaneously recorded pressures by a linking system. Peak positive and peak negative dP/dt were also determined.

Analysis of the pressure volume data showed that the relation in this condition could be expressed as a straight line (P = aV + b) with a high degree of accuracy both in the control state and after practolol administration (mean R value 0.95). Practolol produced no consistent alteration in the slope (a), but uniformly depressed the pressure intercept (b) mean change 6.7 mmHg (0.9 kPa).

Peak negative dP/dt increased (mean 274) after practolol. Peak positive dP/dt did not alter.

It is concluded that the diastolic pressure volume relation in HOCM is linear within the limits of pressure and volume encountered and that it is unnecessary to use an exponential or other complex function to describe it, as well as being theoretically unsound.

We have shown that practolol accelerates relaxation of the series elastic element of the myocardial muscle fibre but that it does not alter compliance (\(\frac{\Delta P}{\Delta V}\)). Contractile function as indicated by dP/dt and ejection fraction appears to be unaffected by practolol.

Chronic beta-adrenergic receptor blockade in congestive cardiomyopathy

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Conventional therapy in congestive cardiomyopathy has failed to change the final outcome of the disease. The treatment has been so unsatisfactory that cardiac transplantation might be considered in desperate cases. In a recent study immediate relief of ischaemic chest pain was reported when beta-blocking agents were given to patients with acute myocardial infarction. In some patients with high heart rate the injection of beta-blockade was followed by disappearance of the signs of left-sided backward heart failure.

In the present study adrenergic beta-blocking agents were, therefore, given to seven patients with advanced
congestive cardiomyopathy who had tachycardia at rest (98 ± 13 beats/min). The patients were on beta-adrenergic receptor blockade for 2 to 12 months (av. 5.4 months). One patient was given alprenolol, 50 mg twice daily, and the other patients were given practolol, 50–400 mg twice daily. Virus infection was seen in six of the patients before the onset of symptoms of cardiac disease. All patients were in steady state or were progressively deteriorating at the start of beta-adrenergic receptor blockade. Conventional treatment with digitalis and diuretics was unaltered or reduced during treatment with beta-blocking agents. A striking improvement was seen in their clinical condition shortly after administration of the drugs. Chronic treatment resulted in an increase in physical working capacity and a reduction of heart size.

Noninvasive investigations including phonocardiogram, carotid pulse curve, apex cardiogram, and echo-cardiogram showed improved ventricular function in all cases. The present study indicates that adrenergic beta-blocking agents can improve heart function in at least some patients with congestive cardiomyopathy. Furthermore, it is suggested that the presence of catecholamines may be an important factor for the development of this disease, as has been shown in animal experiments.

**Viscous properties of cat myocardium**

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The measurement of ventricular diastolic compliance is complicated by the presence of viscous properties. However, quantitative assessment of these properties has not been made. In order to approach this problem, cat papillary muscles contracting isometrically at 15/min at 26°C were stretched at different velocities during diastole. At a given length there was no excess of force above that defined by the static force-length curve until velocity of stretch exceeded one muscle length/second. At greater velocities of stretch there was an increase of force with increasing velocity of stretch denoting viscous resistance to stretch. This viscous resistance to stretch increased with muscle length. At a given muscle length potentiation of contractile state by paired pulse stimulation caused a change in the diastolic force length curve but no apparent change in viscous resistance to stretch, ie there was no apparent relation between viscous resistance to stretch and contractile state or the length of the 'series viscous element'. These results would lead one to expect viscous resistance to stretch during rapid filling of the intact left ventricle.