Proceedings of the British Cardiac Society

THE AUTUMN MEETING of the British Cardiac Society was held at the Wembley Conference Centre, London, on Tuesday to Thursday, 22 to 24 November 1988. The President, E SOWTON, took the Chair during private business. D A CHAMBERLAIN was chairman of the scientific programme.

Abstracts of papers

ISIS-2: a large randomised trial of intravenous streptokinase and oral aspirin in acute myocardial infarction

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A total of 17 189 patients entering 417 hospitals up to 24 hours (median five) after the onset of suspected acute myocardial infarction were randomised, with placebo control, to receive either (a) a one-hour intravenous infusion of 1.5 million units of streptokinase; or (b) a one-month of 160 mg/day enteric-coated aspirin; or (c) both active treatments; or (d) neither. Each agent produced a highly significant reduction in five week vascular mortality: 774 (9.2%) vascular deaths among patients allocated aspirin v 954 (11.4%) among those allocated placebo tablets (odds reduction: 21% (5); 2p < 0.0001); 747 (8.9%) among patients allocated streptokinase v 981 (11.7%) among those allocated placebo infusion (odds reduction: 26% (4); 2p < 0.0001). The combination of streptokinase and aspirin was significantly better than either agent alone, and their separate effects on mortality seemed to be additive: 327 (7.8%) vascular deaths among patients allocated both active agents v 534 (12.8%) among these allocated neither (odds reduction: 42% (6); 95% confidence intervals 33 to 49%; 2p < 0.001). There was evidence of benefit from each agent even for patients treated late after pain onset (odds reduction at 0-4, 5-12, and 13-24 hours: 22% (7), 21% (7), and 17% (13) for aspirin alone; 37% (6), 15% (7), and 22% (12) for streptokinase alone; and 52% (6), 32% (9), and 37% (16) for the combination of streptokinase and aspirin). Aspirin significantly reduced non-fatal reinfarction (49% (9) reduction) and non-fatal stroke (37% (17) reduction) in hospital and was not associated with any increase in cerebral haemorrhage or bleeds requiring transfusion. Streptokinase was associated with more bleeds requiring transfusion (0.4% excess) and cerebral haemorrhage (0.1-0.2% excess), but with fewer other strokes.

There were significantly fewer reinfarctions, strokes and deaths among patients allocated the combination of streptokinase and aspirin than among those allocated neither. A median of 15 months follow-up indicated that the differences in early mortality also have a long term effect.

Anglo-Scandinavian study of early thrombolysis with recombinant tissue plasminogen activator (rt-PA): a six month mortality study

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A group of 13 323 patients with suspected acute myocardial infarction admitted to 53 coronary care units were considered for entry to a prospective double blind placebo controlled trial of recombinant tissue plasminogen activator (rt-PA) given within five hours of onset of major symptoms. 5016 (38%) of the patients were randomised to receive rt-PA or placebo. Of the remainder, 72% were excluded on the basis of time, and all the excluded patients were documented. Treatment began with an intravenous bolus of heparin 5000 units, followed by a bolus of rt-PA 10 mg (or placebo), then rt-PA 90 mg intravenously given as 50 mg in the first hour and 20 mg in each of the next two hours. All patients received heparin 1000 iu/hour for the next 21 hours. The trial infusion was prematurely terminated in 5% of the rt-PA group and 2.5% of the placebo group, the excess in the rt-PA group being mainly for bleeding complications. During the subsequent heparin infusion a further 6% were withdrawn in the rt-PA group and 3.5% in the placebo group. The one month overall mortality was 7.2% in the rt-PA group compared with 9.9% in the placebo group (relative reduction 26%, 95% CI 11 to 38%). About 70% of patients in both groups were diagnosed in...
hospital as having had an acute myocardial infarction, and their one month mortalities were rt-PA 9%, and placebo 13% (relative reduction 28%, 95% CI 14 to 41%). The six month follow up concludes in August 1988.

Interim analysis of the six month outcome in 2607 patients shows an overall mortality of 7-9% in the rt-PA group and 10-3% in the placebo group. Thus early treatment with rt-PA saves lives but carries a similar risk of bleeding complications as streptokinase.

Comparative one year mortality in patients treated with thrombolytic treatment at home and in the coronary care unit

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Eighty six patients with acute evolving myocardial infarction were seen by the Mobile Intensive Coronary Care Unit (MICCU) within six hours of the onset of symptoms. They were given 1-5 million units of streptokinase over 30 minutes. Thirty patients (mean age 60 years) were given streptokinase at home. The remainder (mean age 58-5 years) were treated in the coronary care unit (CCU). Previous history of ischaemic heart disease was 56-7% (MICCU group) and 51-8% (CCU group). Mean delay times for the two groups were 130-6 minutes (MICCU) and 178-4 minutes (CCU) (p = <0.01). Reperfusion times, judged clinically and electrocardiographically, were 83-8 minutes (MICCU) and 94-2 minutes (CCU). The inpatient vascular mortality for the MICCU group was 3-3% compared with 10-7% for the CCU group. The one year vascular mortality was 6-7% for those treated by the MICCU and 21-4% for those in the CCU (p = <0.05). There were no adverse events in either treatment group.

These data suggest that intravenous thrombolysis given outside hospital can provide a further reduction in mortality after acute myocardial infarction.

Comparison of recombinant tissue plasminogen activator and streptokinase for preservation of left ventricular function after myocardial infarction

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Streptokinase (SK) and recombinant tissue plasminogen activator (rt-PA) have been compared in terms of infarct artery patency after an acute coronary artery occlusion but there are few comparative data for effects on left ventricular function. In a double blind trial 244 patients presenting within three hours of a first infarction were randomised to streptokinase (1.5 × 10^6 u over 30 minutes) or to rt-PA (100 mg over three hours). Heparin was given for 48 hours. Aspirin and persantin were given on admission and continued until cineangiography was performed three weeks later. There were similar effects on left ventricular function: ejection fraction 58% (12%) SK, v 58% (13%) rt-PA; end systolic volume 63 (29) ml SK, v 66 (31) ml rt-PA. There was no difference in the frequency of bleeding (one patient had a cerebral haemorrhage after administration of rt-PA for reinfarction three weeks after receiving streptokinase.) Reinfarction rates were also similar (7% SK v 5% rt-PA). There was a higher 30 day mortality (7-8%, v 1-7%) and a higher incidence of hypotension (21%, v 8%) in streptokinase treated patients.

The effects of streptokinase and rt-PA on preservation of left ventricular function are similar.

Coronary lesion morphology in patients with acute myocardial infarction treated with streptokinase

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After thrombolysis for acute myocardial infarction (MI) there is a high incidence of recurrent angina and reinfarction. Morphological features of the coronary lesions may reflect their unstable nature, and predict the future clinical course. Quantitative angiography was performed in 50 patients, two to four days after streptokinase (SK) treatment for acute MI; a control group of 18 patients with stable angina was matched for age and sex. In the SK group, the infarct associated artery was patent in 36 (72%) and occluded in 14; in the control group there were three occlusions and 22 other important stenoses. There were no differences in stenosis length, severity, calcification or location of patent lesions at an acute bend or at a branch point. Lesions in the SK group were more often irregular (p < 0.001), eccentric (p < 0.001), had a shoulder (p < 0.001), and had globular filling defects (p < 0.025), and linear filling defects (p < 0.005). A previously described index of plaque ulceration was higher in the SK group (6-7 (9-7) v controls 3-2 (3-1), p < 0.01). Subsequently 13
of the 50 SK patients were maintained on heparin infusion until PTCA three to five days later. At repeat angiography immediately before PTCA, globular filling defects seen in four had all disappeared, whereas linear filling defects originally seen in six persisted in three. Fewer lesions were irregular (p < 0.005) and the ulceration index decreased from 9.3 (15-4) to 2.8 (1.6) (p < 0.01).

These data show that lesions in the infarct associated artery after PTK treatment are particularly irregular, with filling defects, perhaps corresponding to plaque fissuring and intraluminal thrombosis; these features partially resolve with maintenance heparin infusion.

Inhibition of coronary thrombosis with low dose tissue plasminogen activator (t-PA): the potential for modifications of endogenous t-PA activity

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t-PA is an endogenous protease with high affinity for fibrin bound plasminogen. Its concentration varies diurnally and in response to a variety of stimuli and it may be implicated in the homeostasis of thrombosis and thrombolysis in man. We and others have shown the effectiveness of therapeutic doses of t-PA in inducing coronary thrombolysis in experimental animals and in man. We hypothesise that doses of t-PA insufficient to induce effective thrombolysis might nevertheless inhibit clot formation in vivo because of its high affinity for nascent fibrin, but not fibrinogen. To test this hypothesis we studied the action of 10 000–200 000 IU/ml t-PA (6000–100 000 ng/ml) on thrombin clots using pooled citrated plasma, or euglobulin fractions from normal human subjects. The concentration of t-PA required to achieve the half maximum rate of lysis was reduced from 650 to 260 ng/ml for euglobulin fractions (n = 158), or from 6800 ng/ml to 2670 ng/ml for whole plasma containing endogenous plasmin inhibitors (n = 158). The studies were confirmed in vivo with 23 dogs. Coronary thrombosis (induced by the potent thrombogenic stimulus of a non-occlusive intracoronary copper coil) was inhibited by t-PA at concentrations of 140–430 ng/ml (n = 6) (but not at <140 ng/ml, n = 12), and totally prevented at >460 ng/ml (n = 5).

We conclude that low, sub-thrombolytic doses of t-PA can inhibit thrombus formation. This raises the therapeutic possibility of administering low-dose t-PA or augmenting endogenous t-PA activity in patients at high risk for occlusive thrombus formation.

Enterovirus RNA in myocardium of patients with dilated cardiomyopathy

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Two groups of patients with dilated cardiomyopathy (DCM) were studied. One was diagnosed clinically by haemodynamic function and angiography and proved by left ventricular biopsy. The second had end stage DCM and underwent orthotopic cardiac transplantation. Endomyocardial biopsy samples taken from group I at the time of diagnosis and tissue from the explanted hearts of group II were examined for virus RNA by molecular hybridisation with an Enterovirus group specific probe. In group I, biopsy specimens from 19 of 53 (36%) DCM patients were positive for virus RNA compared with none of 37 controls with other, specific heart muscle diseases (p = <0.002). In group II, tissue from six of 21 (29%) explanted hearts from patients with cardiomyopathy were positive for virus RNA compared with one of 19 controls with ischaemic heart disease (p = <0.05).

These data show that Enterovirus RNA is present in myocardium in a large proportion of patients with DCM and can persist until end stage disease. This association implicates Enterovirus infection in the pathogenesis of DCM.

Novel cardiac specific circulating autoantibodies in dilated cardiomyopathy

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Organ specific autoimmunity is characterised by circulating autoantibodies that are specific to the diseased target organ and do not cross-react with other tissues. Organ specific cardiac antibodies have not been reported. To determine whether organ specific cardiac antibodies are present in dilated cardiomyopathy, we tested 55 serum samples from patients with dilated cardiomyopathy, 225 with other cardiac disease, 166 from patients with polyendocrine autoimmunity without overt cardiac disease, and 200
normal subjects by indirect immunofluorescence on human atrium and skeletal muscle. Organ specific cardiac antibodies were more common (p < 0.01) in dilated cardiomyopathy (10/55, 18%) and in polyendocrine autoimmunity (31/166, 19%) than in patients with other cardiac disease (2/225, 1%) and normal subjects (6/200, 3%). Organ specific cardiac antibodies were IgG (titre range 1/10 to 1/80) and gave a cytoplasmic immunofluorescent staining only on heart tissue. Non-organ specific cardiac antibodies showed a sarcolemmal and striated immunofluorescent pattern both on the heart and on skeletal muscle, but in contrast to organ specific cardiac antibodies were detected with a similar frequency in dilated cardiomyopathy (6/55, 11%), polyendocrine autoimmunity (15/166, 9%), other cardiac disease (13/225, 6%), and in normal subjects (13/200, 6.5%).

These results support the existence of organ specific cardiac antibodies in dilated cardiomyopathy and suggest an autoimmune pathogenesis in some patients with this condition.

Is syndrome X a variable degree of myopathy?

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Syndrome X has been used to describe patients who complain of typical angina but have arteriographically normal coronary vessels. Between 1985 and 1988, 100 randomly selected patients with typical angina were studied, in whom musculoskeletal and gastrointestinal explanations for chest pain had been excluded. Of the patients, 58% were female and the age range was 29 to 64 years. None of the patients had evidence of obstructive coronary lesions at angiography, although in three patients ventricular function was somewhat reduced. Maximal symptom limited exercise tolerance tests were positive in 31% (ST shift >1 mm). Gated thallium scans were abnormal in 98%, with patchy defects affecting predominantly the septum and inferior surfaces. The distributions of defects were similar to those seen in patients with established myopathy, but could be clearly differentiated from scans obtained from normal volunteers (p < 0.01). The extent of left ventricular impairment was assessed quantitatively by two observers dividing the left ventricular region of interest in three projections (anterior, 40°, and 70° left anterior oblique) into a total of 45 segments. In those patients with positive exercise tests, mean left ventricular involvement was 33 (9%) compared with 28 (7%) (p < 0.05) when the exercise test was negative.

These results suggest that syndrome X may be a form of myopathy with a positive exercise test reflecting more substantial impairment of the myocardium.

Evidence for an abnormal vasodilator response in hypertrophic cardiomyopathy

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Sudden death is common in hypertrophic cardiomyopathy (HCM) but the mechanisms(s) have not yet been established. To identify those at risk of haemodynamic collapse, 103 patients with HCM, age 12–70 years (mean 40) underwent symptom limited treadmill exercise with standard cuff blood pressure recording (BP) at each minute of exercise and every 15 seconds during recovery for three minutes on at least two occasions. The test was repeated in 25 of the patients with direct arterial pressure measurement. Fifty one had normal, and 52 abnormal, BP responses with either hypotension during recovery or a fall during exercise of ≥ 20 mm Hg (range 20–100, mean 45 mm Hg) from the peak value to that recorded immediately before stopping. A family history of sudden death was more common in those with an abnormal exercise BP response (24/52 v 3/51, p < 0.001). To assess the mechanism of the hypotensive response, 10 with abnormal and seven with normal exercise BP responses underwent exercise haemodynamic studies, with continuous direct arterial pressure recording and measurement of cardiac output each minute. Systolic BP fell by 25–95 (mean 50) mm Hg in all 10. The rise in cardiac index on exercise (1/minute/m²) was similar in those with normal (2.3 (0.5) to 8.8 (2.4)) and abnormal BP response (2.1 (0.5) to 7.2 (1.1)). Systemic vascular resistance in the two groups at rest and after two minutes of exercise was similar, but at peak exercise fell significantly more in the hypotensive v normal BP responders (22 (6)% v 42 (4)% of resting values, p < 0.01). To assess the mechanism of this abnormal vasodilation, forearm plethysmography was performed during supine bicycle exercise in 11 hypertensive patients and 10 normal volunteers. Forearm vascular resistance fell by 18 (45)% in the hypertensive patients, but increased by 131 (45)% in the normal volunteers (p = 0.003).
Exercise hypotension is common in HCM, is strongly associated with a family history of sudden death, and is due to an inappropriate vasodilation of non exercising vascular beds.

**Objective assessment of exercise capacity in hypertrophic cardiomyopathy: association with clinical and prognostic features**

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Symptom limited treadmill exercise testing was performed in 91 consecutive patients with hypertrophic cardiomyopathy (age 12–70, mean 40 years) with objective assessment of exercise capacity by maximal oxygen consumption (VO$_2$ max) and ST segment and blood pressure monitoring. An abnormal fall in BP on exercise was seen in 47/91. ST segment changes were common (ST depression in 30 and pseudonormalisation of T waves in 11). Exercise capacity was abnormal even in NYHA Class I patients who had a VO$_2$ max of 28.5 (7.8) (normals >40), and was significantly lower in those with dyspnoea Class II and III (20.8 (5.6), p < 0.001). VO$_2$ max was lower in patients with chest pain (22.6 (5.4)) v those without (26.3 (8.6)) p < 0.05, and in those with non-sustained ventricular tachycardia on 24 hour ECG monitoring (21.5 (5.6)) v those without (26.2 (7.6)); p < 0.01), VO$_2$ max was related to age (r = −0.47, p < 0.001) and left atrial dimension (r = −0.43, p < 0.001). Reduced VO$_2$ max, abnormal BP response, and ST segment changes were not associated with maximum LV wall thickness, presence or magnitude of left ventricular gradient, conventional radionuclide indices of systolic and diastolic function, or to Doppler indices of diastolic function. A family history of sudden death was significantly more common in those exhibiting a hypotensive exercise response and those with pseudonormalisation of T waves. ST segment changes, however, were not associated with exertional chest pain.

In conclusion, exercise capacity was reduced even in symptom free patients with hypertrophic cardiomyopathy and was not associated with conventional indices of diastolic or systolic function, including the presence or magnitude of gradients. The association of a reduced VO$_2$ max, a hypotensive response, and pseudonormalisation of T waves with markers of adverse prognosis suggests a potentially important role of exercise testing in the assessment of high risk patients with hypertrophic cardiomyopathy.

**Hypertrophic cardiomyopathy without hypertrophy: a description of two families with premature cardiac death and myocardial disarray in the absence of increased muscle mass**

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The necropsy finding of widespread myocardial disarray typical of hypertrophic cardiomyopathy in three macroscopically normal hearts prompted clinical evaluation of the two families. In family A, the propositus, her daughter, sister, and nephew died suddenly, aged 37, 21, 42, and 17 years, while in family B only the propositus died a premature cardiac death at the age of 45. Eleven of the surviving first degree relatives were investigated. They were aged 12–49, median 18 years. Six had normal clinical cross sectional echocardiographic and radionuclide (Tc) findings. Five were symptom free but had electrocardiographic abnormalities including atrial fibrillation (n = 1), left axis deviation (n = 2) and widespread repolarisation changes (n = 5), with normal echocardiographic wall thickness and cavity dimensions, and radionuclide indices of left ventricular function. Three of the five patients with abnormal electrocardiograms had dilated left atriums associated with abnormal echocardiographic left ventricular relaxation and filling patterns.

The finding of myocardial disarray in the absence of left ventricular hypertrophy and in association with repolarisation changes in two families is consistent with a wider spectrum of hypertrophic cardiomyopathy than has been recognised, and suggests that hypertrophy may be a secondary rather than a primary abnormality in this condition.

**Magnetic resonance imaging of the great arteries in infants**

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Fifty two infants (age range 1–50 weeks) with congenital heart disease were studied with magnetic resonance (MR) imaging. A 1.5 T whole body MR imaging system was used with patients sedated and positioned supine inside a 32 cm diameter head coil. Multiple electrocardiographic gated sections 5 mm thick were acquired using a spin echo sequence with echo time of 30 milliseconds. A combination of standard and oblique imaging planes were used. Echo-cardiographic studies had previously been...
performed in all cases. Ventriculoarterial connections were defined in all 46 patients in whom full intracardiac imaging was performed. They were identified as concordant in 33 patients and discordant in five. Two patients had double outlet right ventricle (DORV), one a common arterial trunk, and five patients had pulmonary atresia. The aorta was identified posteriorly to the pulmonary artery in 38 patients, anteriorly in three, and to the left in the patient with DORV. In the patient with a common arterial trunk a single pulmonary trunk was identified arising anteriorly from the main trunk. Of the two patients with Fallot’s tetralogy the pulmonary trunk and branch pulmonary arteries were identified as hypoplastic in one, and normal in the other. In five patients with pulmonary atresia, confluent central pulmonary arteries were identified in three cases and were considered absent in two. Aorto-pulmonary collateral vessels were found in two cases. Aortic structure was clearly shown. An aberrant right subclavian artery, a large arterial duct, a double aortic arch and 14 cases of aortic coarctation have all been identified.

MR images provided consistently greater detail than echocardiography and appeared particularly suitable for imaging the great arteries, superseding the need for angiography in many instances.

Childhood blood pressure measurements in nine British towns

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Striking differences in the average blood pressure of middle aged men in 24 British towns have been reported. This study aimed to establish whether a similar pattern of differences could be detected in children 5–7 years of age in nine of the original 24 towns. Three towns with high, three with intermediate, and three with low average adult blood pressures were chosen. In each town stratified random sampling was used to select 500 children. Blood pressure was measured with a Dinamap 1846SX oscillometric blood pressure recorder. The overall response rate was 85.7% (n = 4114). Average blood pressures varied by 5.6 mm Hg (systolic) and 4.5 mm Hg (diastolic) between the highest town (Merthyr Tydfil) and the lowest (Guildford). The extent of variation was unlikely to be due to chance (p < 0.0001). Overall age standardised mean systolic pressures in the three towns with high adult blood pressure measurements combined were 2.35 mm Hg (95% CI 1.71 to 3.02: p < 0.001) higher than in those with low adult pressure measurements combined; the towns with intermediate adult pressures were in an intermediate position. The findings were not explained by observer bias nor by temperature differences between the towns.

The presence of geographical differences in blood pressure in 5–7 year olds resembling the adult pattern implies that the determinants of population blood pressure differences are present at an early age and that further aetiological studies should focus on the first decade of life.

Transoesophageal echocardiography, an improved diagnostic technique for subaortic membranes

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Fifteen adults with subaortic membranes underwent precordial and transoesophageal (TEE) studies to compare and contrast the information derived from cross sectional echocardiography and colour flow imaging (CFI). Precordial cross sectional echocardiography clearly showed the membrane septal origin in 11 patients but showed the lateral insertion in only eight. Precordial CFI studies clearly identified the site of left ventricular outflow tract turbulence in 14, but could not distinguish this abnormal flow pattern from that of hypertrophic obstructive cardiomyopathy or a fibromuscular tunnel. TEE in 15 patients clearly visualised the entire membrane, showing a wider range of both lateral and septal insertion patterns than that suspected by precordial echocardiography. Septal insertion varied from 0.1 to 4.3 cm below the aortic valve. Lateral insertion varied from (a) the aortic leaflets, (b) the left ventricular outflow tract lateral wall, (c) mitral chordae, or (d) reduplicated mitral tissue. CFI clearly defined both the obstruction site and minimal jet width. Associated mitral incompetence was accurately diagnosed and traded by CFI in 10/15 by TEE in contrast to 2/15 by precordial echocardiography.

There was a similar diagnostic improvement in evaluating the nature and severity of associated aortic valve lesions. In four patients the information derived from the transoesophageal study subsequently proved invaluable in intraoperative decision making.

We conclude that TEE provides important additional morphological and haemodynamic information over precordial echocardiography in this lesion.
We attempted the role of PBV in 102 normal human midtrimester fetuses, in 45 fetuses with gestational ages from 30–40 weeks, in 45 babies born at full term and 24 hours of age, and 80 neonates during the first week of life. Each fetus and neonate had a normal cross sectional echocardiogram. The Doppler sample volume was placed in the pulmonary artery and aorta, parallel to blood flow. The blood flow velocity wave form was displayed and recorded on videotape with the tracing speed set at 100 mm/second. The measurement of time to peak velocity (TTPV) was obtained subsequently from a frozen image from the videotape in 10 cardiac cycles, and the mean was obtained. The TTPV in the pulmonary arteries of fetuses of 30–40 weeks' gestation was no longer than in the midtrimester fetuses, although not significantly different from that in the aorta for the same gestational age. TTPV in the aorta did not show any significant alteration throughout pregnancy. In postnatal life TTPV in the pulmonary artery was much longer than in prenatal life.

These results suggest that the resistance faced by the right ventricle is higher than that faced by the left ventricle and diminishes progressively throughout pregnancy. The greatest fall in pulmonary artery pressure occurs after birth during the first day of life.

Percutaneous balloon dilatation for critical pulmonary stenosis in neonates and infants

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The role of balloon dilatation (PBV) in neonates and infants with critical pulmonary stenosis is unclear. We attempted PBV in 13 patients (median age 14 days, range 1 day to 9 months) with systemic or suprasystemic right ventricular systolic pressures, mean (SEM) ratio 1:3 (0:1), caused by pulmonary valve stenosis. Seven patients had a patent ductus arteriosus (PDA). In two neonates the pulmonary valve could not initially be crossed, but was crossed and successfully dilated at a later date after a systemic pulmonary shunt had been placed. One patient with a right ventricular pressure of 268 mm Hg and without a PDA developed hypotension and bradycardia when the pulmonary valve was crossed during the diagnostic study, and subsequently died after an emergency systemic pulmonary shunt. The right ventricular pulmonary systolic pressure gradients fell from 87 (8) mm Hg before PBV to 19 (3·2) mm Hg after PBV at 8·6 (2·2) months of follow up (p < 0·001). Only one patient required repeat PBV.

PBV is an effective treatment for critical pulmonary stenosis in neonates and infants: caution is advised, however, in patients without a PDA.

Presentation of ventricular tachycardia in children

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Twenty children were referred with ventricular tachycardia (VT) between 1978 and 1988; mean age was 4·5 years (range: birth–15·3 years), and the sex ratio was equal. Heart failure was the presenting feature in five (25%), mean age 7 months (range: birth–1·5 years). VT followed a viral illness in two and one child had a cardiomyopathy. Five (25%) presented with syncope, mean age 9·6 years (range: 6–15·3 years). One child had a long Q–T syndrome (LQTS), and one a cardiomyopathy. In two, syncopal attacks had been mistaken for epilepsy. Tachycardia during viral illness was the presenting feature in two (10%), aged 13 and 18 months. Two (10%) presented with palpitations aged 6·2 and 7 years. The finding of an irregular pulse first brought four symptom free children (20%) to medical attention, mean age 5·3 years (range: birth–13·8 years). VT was later shown on Holter monitoring. In one child this followed a viral illness and one child had a VSD. Torsade de pointe VT occurred in two children (10%) aged 3 months and 2·3 years, respectively, both with a LQTS. One occurred during routine Holter monitoring and one postoperatively (Fallot's tetralogy and pacemaker). VT was initially mistaken for a supraventricular tachycardia in seven (35%). Five of these had narrow complex His bundle VT, mean age 8 months (range: birth–1·5 years). Six received inappropriate initial antiarrhythmic drugs resulting in cardiac arrest in three.

In conclusion, VT in children may be asymptomatic or present as a life threatening arrhythmia. Initial presentation and ECG may lead to difficulties in diagnosis and management.
Early coronary atherogenesis and myocardial infarction in Asians: a problem of glucose metabolism

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Ethnic differences in 128 consecutive male patients presenting with first myocardial infarction (MI) were examined. Infarction rates were derived using data from the 1981 census. Coronary arteriograms on 99 survivors were scored by two 'blinded' observers. Six months after the MI cholesterol (TC), triglycerides (TG), HDL cholesterol (HDL), and glucose (BS) were measured from a single oral glucose blood sample. Age and race matched controls were recruited for identical blood tests. Rates of MI were significantly (three times) higher in Asians in all age groups. Plaque numbers were greater in Asians (3.96 (2.27) ± 2.04 (1.65)) than in whites (p < 0.001). In the patient groups 34% of Asians v 13% whites (p < 0.005) had diabetes or impaired glucose tolerance (IGT). Cholesterol in whites was 6.18 (1.05) ± 5.56 (0.94) Asians (p < 0.002). TGs in Asians were 1.96 (1.06) ± 1.56 (0.76) (p < 0.05). HDL was lower in Asians (0.88 (0.24) ± 1.09 (0.24) (p < 0.001)). In controls 3% of Asians v 8% whites (p < 0.01) had diabetes or IGT. Cholesterol was higher in Asians (5.56 (0.99) ± 5.07 (0.86) in whites (p < 0.05)). TGs were 1.32 (0.70) in Asians v 1.25 (0.69) in whites p = NS. HDLs were 1.09 (0.34) in Asians v 1.19 (0.20) in whites (p = NS).

We conclude that rates of IGT and diabetes are higher in Asians than whites but not greater in Asians with MI than controls. Unfavourable lipid profiles with high TGs and low HDL were present in Asians with myocardial infarction. These data suggest that in Asians, adverse TGs and HDL concentrations are associated with MI but diabetes is not.

Comparative lack of free radical generation by the myocardium during cardiac surgery

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Diffuse impairment of ventricular function after cardiac surgery may be associated with the generation of oxygen derived free radicals during reperfusion of the myocardium. Ten patients undergoing elective coronary bypass surgery were studied using a previously described assay of peroxidised lipids as an index of free radical activity. Blood samples were obtained from systemic arterial, mixed venous, and coronary sinus catheters, and the ratio of octadeca-9, 11-dienoic acid to linoleic acid was measured in the plasma phospholipid and free fatty acid fractions by high performance liquid chromatography.

The 10 patients were men aged 49 to 67 years with good preoperative LV function (angiographic LV EF > 55%). All underwent coronary surgery on cardiopulmonary bypass with a membrane oxygenator, relative hypothermia 30°–34°C, and intermittent cross clamping of the aorta. During the ischaemic periods there were no changes; during the reperfusion phase there was a significant rise in peroxidised lipid ratio in the free fatty acid fraction in arterial and in mixed venous blood, but not in coronary sinus blood (arterial 3.60 (1.17) to 3.82 (1.13), p < 0.05; mixed venous 3.49 (1.17) to 3.97 (1.11), p < 0.05; coronary sinus 3.84 (0.96) to 3.87 (1.18), p = NS). There were no significant changes in the phospholipid fraction throughout the study period.

These data suggest that much of the observed rise in peroxidised lipids originates from tissues other than the myocardium. Caution will be needed in interpreting such measurements with regard to cardiac events unless samples are obtained specifically from the coronary sinus, as well as from systemic sites.

Influence of platelets on extent of myocardial infarction during coronary artery occlusion

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We have previously shown that platelet activation may contribute to the haemodynamic and arrhythmogenic effects after early coronary artery occlusion (CAO). To investigate the possible influence of platelets on the rate of myocardial necrosis, we measured extent of infarction following 30 minutes obtuse marginal CAO in the rabbit. Four sections of equal thickness from base to apex were obtained and after staining with nitroblue tetrazolium necrosed tissue was measured by planimetry. Results were calculated as the volume of myocardium infarcted and then expressed as % of the zone at risk. The zone at risk, obtained for each of the four sections from base to apex in five hearts after 90 minutes CAO, was 44%, 47%, 60%, and 80% (or 53% of the total myocardium at risk). After 30 minute CAO in 10...
controls, SAP decreased by 27% (p < 0.005), while heart rate remained unchanged and five developed VF. The extent of infarction (as % zone at risk) was 53%, 46%, 38%, and 31% from base to apex (or 43% of the total myocardium). Antiplatelet serum (APS, 1 ml/kg given intravenously before CAO) decreased platelet count by 98% (n = 9) and SAP decreased by 6% (NS); heart rate was unchanged and only two developed VF. The extent of infarction was 28%, 31%, 37%, and 21% of the zone at risk (29-5% of the total). Thus in the presence of platelet depletion there is a marked reduction in the rate of development of myocardial infarction especially in the basal region.

These findings indicate that platelet activation may contribute to the rate of necrosis in the early stages of acute myocardial infarction.

The role of echocardiography and Doppler and colour flow mapping before, during, and after dilatation of the mitral valve.

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Eighty seven cross sectional echocardiographic and Doppler and colour flow mapping studies in 24 patients undergoing dilatation of the mitral valve have been critically evaluated. Echocardiographic factors predictive of successful outcome included good main leaflet body mobility, lack of significant subvalvar calcification, and left atrial diameter < 6.5 cm. Contraindications were left heart thrombus or valvar regurgitation > grade 2. Assessment of the valve area by planimetry, leaflet mobility, calcification, Doppler maximum and mean pressure gradients, Doppler valve area (by pressure half time), and colour flow characterisation of left ventricular filling pattern were performed before, three days, six, and 12 months after the procedure. Mean echo/Doppler valve area increased from 1.11 cm² to 1.67 cm². Colour flow mapping showed increase in mitral regurgitation by one grade in three cases, transient small atrial septal defect in two, and complex dual left ventricular filling patterns in three where isolated medial or lateral commissure separation was seen on cross sectional echocardiography. Echocardiography and Doppler and colour flow mapping was performed during the procedure in nine patients and this monitored any increase in regurgitation, LV filling during inflation, and balloon position. In three cases pericardial effusion was detected and aspirated using echo guidance.

In conclusion, echocardiography and Doppler and colour flow mapping have an essential role in the selection of patients for dilatation of the mitral valve, intraprocedure monitoring, and long term follow up.

Clinical outcome and procedural risks of complete compared with incomplete revascularisation by percutaneous transluminal coronary angioplasty in patients with multivessel disease

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Coronary angioplasty (PTCA) is being increasingly performed in patients with multivessel disease (MVD) and emphasis is given to completeness of revascularisation to improve clinical outcome, although this is not always possible. We have retrospectively reviewed the clinical outcome in 86 patients with MVD who underwent PTCA in our centre between January 1985 and June 1988. Complete revascularisation was achieved in 29 patients (group I) and incomplete revascularisation in 50. Of the 50, complete revascularisation had been intended at the outset in 18, but only incomplete revascularisation could be achieved for technical reasons (group IIa). The remaining 32 patients had a planned incomplete revascularisation as it was felt that the non-dilated vessel supplied a smaller area of viable myocardium (group IIb). Over a mean follow up period of 16 (10) months, 21 (72%) in group I, 12 (67%) in group IIa, and 26 (81%) in Group IIb, remained symptom free or greatly improved. Three (10%) in group I, 2 (11%) in group IIa, and 2 (6%) in group IIb, had to undergo CABG. PTCA was repeated in four patients in group I, none in group IIa, and one in group IIb. One patient died after an MI in group I. There were no deaths in group IIa or group IIb. None of these differences was significant. Forty nine patients had PTCA to one vessel alone in the presence of MVD; a major procedure related complication (MI/CABG) occurred in two (4%) and there no deaths. The remaining 37 had PTCA to more than one vessel. Two patients (5.5%) suffered a major complication in this subgroup, and there were two (5.5%) procedural deaths (p < 0.05).

We conclude that in selected patients, incomplete revascularisation can be associated with a high incidence of clinical improvement and low subsequent cardiovascular event rate at follow up. The risk of the
A thrombotic tendency in systemic lupus erythematosus (SLE) has been associated with raised cardiolipin antibodies (CA). We studied 34 consecutive patients with SLE to find out whether these patients also have an increased incidence of cardiac abnormalities by complete echocardiographic and colour flow Doppler examination. The CA state of the patients was not known at the time of the ultrasound studies. The patients ages ranged from 16 to 64 years (mean 35.5). Fourteen (41%) of the patients had raised titres of CA. Thirteen (93%) of these had at least one cardiac abnormality. Eleven had abnormal valves (valvar thickening in two, regurgitation in five, and Libman–Sacks vegetations in four). Two patients had partial or global myocardial dysfunction, one of whom also had a small pericardial effusion. Only one patient with raised CA had an echocardiographically normal heart, and this patient had recurrent venous thromboses. In contrast none of the 20 patients with normal CA titres showed structural valvar disease although two had small pericardial effusions.

It is concluded that a raised CA is an indicator of probable cardiac impairment in SLE. This knowledge may be important in guiding decisions about treatment.

**Exercise renography in patients with heart failure**

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Renal blood flow is impaired in patients with heart failure, especially during exercise. This is difficult to evaluate using standard clearance methods. The radioisotope renogram has therefore been studied at rest and during incremental exercise to examine its role in the assessment of renal perfusion. Rest (n = 22) and exercise (n = 26) renograms were performed on 18 patients using 123-I orthohippurate. Renal counts were recorded for 30 minutes. Freewheel exercise using a bicycle ergometer was performed for the first three minutes. Thereafter the work was increased in 25 Watt steps every three minutes until the limit of work capacity. Radionuclide injection coincided with the start of exercise. The mean fractional shortening was 23 (11%). Most renograms done at rest were normal. Exercise renograms were of normal pattern in patients in NYHA class I (n = 5). There was a flattening of the washout phase in the
The effect of loading conditions on the right ventricular pressure-volume association

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In order to characterise the effect of loading conditions on the right ventricular pressure-volume association (RVPVR), calibrated biplane ventriculograms with simultaneous microtip pressure measurements were obtained from 16 patients with RV pressure load (RV systolic pressure 58 (9) mm Hg—group 1), five with RV volume load alone (RV systolic pressure 30 (6) mm Hg—group 2), and 10 after relief of RV pressure load (RV systolic pressure 28 (7) mm Hg—group 3). Left ventriculograms were obtained in a further three patients after the Mustard operation (where the left ventricle is the pulmonary ventricle). Pressure volume loops were constructed and compared with our previously published normal data. When compared to the more triangular normal RVPVR, loops obtained in group 1 were square or rectangular with little ejection occurring during pressure decline (p < 0.001). Indeed, they more closely resembled those described for the normal left ventricle. The shape of the loops obtained from patients in groups 2 and 3 were not significantly different from normal although end diastolic volume was increased in both. Interestingly, left ventricular pressure volume loops obtained after the Mustard operation were also indistinguishable from those of the normal RV, suggesting that the changes observed are independent of ventricular morphology.

In conclusion, the shape of the normal RVPVR reflects a series of load dependent phenomena. This may have important implications when attempting to measure right ventricular function.

Diagnosis of left ventricular pseudoaneurysm by colour Doppler flow mapping

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Left ventricular pseudoaneurysm (LVPA) is defined as a cardiac rupture contained by adherent pericardium. It is an unusual complication of myocardial infarction, cardiac surgery, and chest trauma. Due to the tendency of LVPA to rupture, early recognition is of paramount importance, as surgical repair may be curative. Eleven consecutive patients with cardiac

Neuroendocrine responses to captopril in acute heart failure after myocardial infarction

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Angiotensin converting enzyme inhibitors may have diverse neuroendocrine effects in heart failure. We measured atrial natriuretic peptide, angiotensin II, and noradrenaline in 20 patients with heart failure complicating myocardial infarction, and after incremental doses of captopril at hourly intervals to reduce pulmonary occluded pressure (POP) to < 16 mm Hg or to a total dose of 25 mg. Plasma angiotensin II (AII), atrial natriuretic peptide (ANP), arginine vasopressin (VAP), and noradrenaline (NA) were raised at 51 (21) pmol/l, 185 (25) pg/ml, 9-6 pg/ml, and 7-1 nmol/l, respectively. ANP correlated both with right atrial pressure (RAP) and pulmonary occluded pressure, both r = 0-5, p < 0-05, while AII correlated with POP, r = 0-4, but not with systemic vascular resistance (SVR). After captopril, AII fell to 6-8 (1-7) pmol/l, p < 0-001, with accompanying fall in POP from 25-7 (1-9) to 19-3 (1-4) mm Hg, and SVR from 1608 (83) to 1345 (52) dyn cm⁻². Plasma NA and AVP were unchanged at 7-1 (2-2) nmol/l and 7-9 (2-2) pg/ml, respectively. Despite RAP falling from 11-1 (0-8) to 8-7 (0-7) mm Hg, p < 0-001, ANP remained unchanged at 209 (22) pg/ml, being no longer correlated with either RAP or POP.

Thus the neuroendocrine and haemodynamic responses following captopril are complex. The persistent rise in ANP despite falls in RAP and POP suggest an additional beneficial mechanism of captopril in ameliorating heart failure by promoting natriuresis.
rupture forming left ventricular pseudoaneurysms (LPVA) have been studied using colour flow mapping (CFM), five with acute rupture following myocardial infarction, two following stab wounds, one late rupture of a calcified “true LV aneurysm”, one post surgical resection of an LV aneurysm and two resulting from LV venting. Recurrent rupture following repair of LPVA occurred in two patients. The study group therefore comprised 13 cases. The diagnosis was suspected clinically in only 6. Cross sectional echocardiography alone confirmed the diagnosis in 7/13. In all 13, CFM findings were compared with angiographic and surgical information. In all 13 CFM showed flow in and out of the pericardial cavity at the defect site as well as the abnormal flow pattern within the pseudoaneurysm. Pulsed Doppler at the defect site revealed a consistent mutiphasic “to and fro” flow pattern the peak velocity of which showed a characteristic respiratory variation. This intracardiac flow pattern is diagnostic of a pseudoaneurysm.

We conclude that in view of the above findings duplex scanning with integrated CFM is the diagnostic technique of choice of cardiac rupture.

Growth potential of the immature transplanted rat lung

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In heart lung transplantation in young children the potential for lung growth is uncertain. Thus unilateral left lung transplantation was carried out on immature rats (<6 weeks of age, when alveolar multiplication is incomplete). Isogeneic (Lewis) and non-isogeneic immunosuppressed (BN to Lewis) transplants were made, four in each group. All were killed six months postoperatively. Right and left lungs were analysed separately using light microscopic quantitative techniques, and the findings were compared with controls matched for age and strain. Body and heart weight was normal in all. The left lung was normal in volume, with alveoli of normal size and number in non-isogeneic rats. Lung volume was abnormally large (p<0.005) in isogeneic rats because of an increase in alveolar size (p<0.05), alveolar number being normal. The right lung of both non-isogeneic and isogeneic rats showed an increase in lung volume and alveolar number (p<0.01 for both). In addition, hilar stripping of the left lung (without transplantation) in Lewis rats of the same age (n = 4) showed that denervation alone did not affect left lung growth but led to a lesser but significant increase in right lung volume (p<0.005) due to a non-significant rise in alveolar size.

Thus after transplantation alveoli continued to form to achieve a normal number and normal lung volume, despite the non-isogeneic rats being immunosuppressed and showing evidence of low grade rejection activity. The hypertrophy of the contralateral lung seen in this model of unilateral transplantation was only partly attributable to denervation.

Coronary artery ectasia: prevalence, frequency of hyperlipidaemia, and influence on myocardial perfusion

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During a 12 month period 452 consecutive patients underwent coronary angiography for suspected coronary artery disease (CAD). Of these, 395 were abnormal and 18 (4.5%) showed either localised or diffuse coronary artery ectasia (CAE). All male patients below 60 years of age had their serum lipids and lipoproteins measured immediately before cardiac catheterisation. Their total number was 178 which included 10 patients with CAE. The mean value for total cholesterol (TC), triglyceride (TG), and low density lipoprotein cholesterol (LDL-C) in the CAE patients was no higher than in those with non-ectatic CAD, but the ratio of HDL-C:LDL-C was significantly lower (0.18:0.22, p = 0.05). Eight of the 10 patients with CAE were found to have TC, TG or LDL-C above age related 95th percentiles, or HDL-C below the 5th percentile of control subjects, compared with 80 of 168 (48%) of non-ectatic CAD patients (p = 0.05). In four of those with ectasia the only lipid abnormality was a low HDL-C (hypalophilipoproteinemia). Three patients were found to have non-obstructed ectatic vessels and underwent exercise stress testing with cross sectional echocardiography and thallium radionuclide scanning. Objective evidence of myocardial ischaemia in regions supplied by non-obstructed ectatic vessels was documented in two of these patients, both having a positive thallium scan, with one developing diagnostic ECG changes, and reversible regional wall motion abnormality on echocardiography.
In summary these findings show that serum lipid and lipoprotein abnormalities are common in CAE patients. This suggests that an imbalance between these lipoproteins may play a pathogenetic role in this uncommon form of coronary arteriopathy. Non-obstructed ectatic arteries may also be a substrate for exercise induced myocardial ischaemia.

Transcardiac changes in platelet function before and after angioplasty

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While not proved, restenosis after angioplasty is thought to be by proliferation of arterial wall smooth muscle cells, perhaps stimulated by the interaction between activated platelets and the balloon damaged vessel wall. To determine whether platelets are activated by coronary angioplasty we have assessed transcardiac changes in platelet function with aggregometry, and transcardiac platelet granule release by measuring β-thromboglobulin (BTG) before and after angioplasty. Samples of blood were taken from the aortic root and coronary sinus before dilatation (A1 and C1 respectively) and from the same sites (A2 and C2) within five minutes of the completed procedure, in a total of 21 patients. The platelet activation index, a sensitive measure of platelet hyperaggregability, was significantly higher in C2 (mean 9.4; 95% confidence interval 7.5 to 11.3, than in A1 (6.2, 4.8 to 7.7), (p = 0.014), A2(7.1, 6.4 to 7.8), (p = 0.039) or C1 (6.5, 5.7 to 7.3) (p = 0.013). Levels of BTG were higher in C2 (median 5.1 ng/ml; interquartile range 4.0-6.3) than A2(4.4 ng/ml, 3.3 to 5.7) and C1 (4.9 ng/ml, 3.5 to 5.8), but only reached significance when compared with A1 (4.3 ng/ml, 3.1 to 5.4) (p < 0.05).

Our data have shown that coronary angioplasty has a significant effect on platelet function. If angioplasty induced platelet aggregation could be pharmacologically prevented, subsequent restenosis might be reduced.

Collateral vessels after infarction: association with left ventricular damage and coronary disease, and influence on exercise testing and clinical outcome

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Associations between collateral vessels, left ventricular (LV) damage, and coronary artery (CA) disease, and their influence on submaximal exercise testing (SMET) and outcome after myocardial infarction (MI) are unclear. Of 302 consecutive survivors of MI, 262 underwent an early SMET. Of 139 patients with abnormal tests, 123 underwent cardiac angiography; extent of LV damage, CA disease, and collaterals to the infarct territory were scored. Extent of LV damage was not associated with the extent of CA disease or collateralisation but extent of collaterals was significantly related to extent of CA disease. Odds of dying in the first year following MI or suffering a further ischaemic event were doubled by an LV score ≥21 and a CA score ≥19, respectively. Collaterals did not influence outcome. Features of the SMET significantly associated with LV damage included lack of ST depression, but prolonged ECG recovery time if it occurred, an abnormal blood pressure response, and ST segment rises. Features associated with extent of CA disease included degree of ST segment depression, stage it first occurred, its presence in the infarct territory, ECG recovery time, and lack of ST segment elevation or ventricular arrhythmias. Multivariate analysis identified independent predictors of these variables: (a) LV score ≥21—lack of ST depression, ECG recovery time >2 mins, and ST elevation, and (b) CA score ≥19—ST depression ≥ 3 mV. Major collaterals were more likely with ST segment depression, a greater degree of ST depression, and absence of ventricular arrhythmias.

We conclude that SMET variables are useful predictors of the extent of LV damage and CA disease after infarction; the extent of collaterals, which can be predicted by SMET variables, is associated with the extent of CA disease but not with clinical outcome.

Comparative value of clinical and electrophysiological variables in the prediction of arrhythmic events after myocardial infarction

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The value of a number of different variables in the prediction of arrhythmic events was assessed in a series of 75 postinfarction patients. Late potentials (LP) were detected using Simson’s method; ventricular stimulation was performed using the Wellens protocol; exercise testing was performed using a
modified Bruce protocol, and ejection fraction was
determined using either gated blood pool scan or
cineventriculography. The mean age of the patients
was 56 years, median 55; 13% had had previous
infarctions. All tests were completed within six
weeks of infarction. During a mean of 18 (median 10)
months follow up, six patients developed symptom-
atic ventricular tachycardia (VT); there were no sud-
den deaths. Multivariate analysis showed that the
single most important predictor of arrhythmic
events was inducible, sustained, monomorphic VT
(sensitivity 5/6(83%), positive predictive accuracy
(PPA) 6/8(75%)). Induction of repetitive ventricu-
lar responses, non-sustained VT and polymorphic
VT or ventricular fibrillation, were poorly correlated
with the occurrence of arrhythmic events. Clinical
criteria were sensitive but inaccurate predictors of
arrhythmic events (occurrence of in hospital compli-
cations, sensitivity 5/6(83%), PPA 5/27(19%); eje-
tion fraction, sensitivity 4/6(67%), PPA 4/18(22%));
LP or an ectopic frequency of ≥10/hour were sensi-
tive (5/6(83%), and 4/6(67%)) but also inaccurate
predictors of arrhythmic events (PPA 5/20(25%),
and 4/13(31%).). The exercise test was of little value
in predicting arrhythmic events (PPA 2/42(5%),)
but was positive in all three patients who reinfarcted.
Combinations of the non-invasive tests did provide
increased accuracy (for example, LP + tape, PPA
4/6(67%)) but no combination was as sensitive or as
accurate as inducible, sustained monomorphic VT.

In conclusion, inducible sustained, monomorphic
VT is a sensitive and accurate predictor of arrhyth-
ic events after myocardial infarction; clinical data
and non-invasive electrophysiological tests are also
sensitive but comparatively non-specific prognostic
indicators.

Angioplasty of bifurcation lesions in major
coronary vessels

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Coronary stenoses affecting a bifurcation have pre-
viously been considered unsuitable for balloon
dilatation when occlusion of a major vessel may
occur in 30% of cases. Using techniques specifically
tailored to lesion morphology, successful angioplasty
was performed in 26 patients with lesions affecting
the origin of a large branch vessel. The lesions were
in the anterior descending/posterolateral in one. Simultaneous
dilatation with two balloons was performed in eight
patients. In four cases two guide catheters were used,
a single guide catheter being used in the remaining
four. In this group 2-5 mm balloons were used in
three cases and a balloon probe with a 3 mm mono-
rail in the remaining case. In 18 patients important
compromised branch vessels were protected by a
second guide wire. In these patients successful
angioplasty of the bifurcation was achieved but
unplanned dilatation was required in three patients
to correct iatrogenic stenosis in a previously
unstenosed vessel. Primary success was achieved in
all with no major complication. Serious disruption
of the vessel proximal to the bifurcation was seen in
three patients. In seven patients, however, lesions
recurred within six months in one or both limbs and
were successfully redilated in two.

Successful angioplasty of complex lesions can be
achieved if attention is paid to lesion morphology
and choice of technique.

Epicardial electrogram during successive
balloon occlusion during percutaneous
transluminal coronary angioplasty

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We have recorded an electrogram from the guide-
wire during percutaneous transluminal coronary
angioplasty (PTCA) monophasic action potentials
from the right ventricular septum, and an ECG, with
a view to a qualitative assessment of the procedure.
We have studied 20 patients undergoing PTCA for
lesions in the left anterior descending (n = 12)
circumflex (n = 3) and right coronary artery (n = 5).
Measurable changes in the electrogram were
obtained only from the left system. Of these 15
patients two showed no ST segment change during
the procedure. The remaining 11 patients registered
up to 5-4 mm of ST elevation in the electrogram with
the deflated balloon in position across the stenosis
(mean (SD) 1 8 (1-6) mm). During balloon occlusion
ST elevation was present up to 7 mm in 14 of the 15
patients (3-0 (1-9) mm). On deflation the electrogram
registered either reduction, no change, or an increase
in ST elevation compared with the previous control
value. After successive inflations and deflations a
final reduction of the initial control ST use of more
than 50% was associated with a good angiographic
result, whereas a fall of less than 50% or an increase
above the control value was associated with only a
moderate result. Although we were able to record
from the right coronary artery the signals were of low amplitude and registered only minimal ST and T changes, probably due to cancellation. There was no obvious correlation between angiographic evidence of collateral supply with either the changes in ST rise between inflations or the magnitude of ST elevation during inflation. In the 17 patients undergoing PTCA of the right and left anterior descending coronary artery, ischaemic changes in MAP duration were only registered in two (despite positioning the MAP electrode in the area normally supplied by the vessel) because of the extremely local nature of the 60 second ischaemic intervention. As would therefore be expected the electrocardiogram was less sensitive than the electrogram, ST changes often being minimal or absent and they did not provide useful sequential data.

Our results show that assessment of ST segment changes in the electrogram recorded from the PTCA guidewire during periods of balloon deflation between successive occlusions provide evidence of a good angiographic result when any initial ST elevation had subsequently been reduced by 50% at the end of the procedure. There was no correlation with angiographic evidence of collateralisation.

Dynamic angioplasty in normal and abnormal coronary arteries

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The “Kensey” catheter is a new device for treatment of occlusive vascular disease. We have successfully used an 8 F device in peripheral arteries. We used a 1.8 mm resolution angioplasty to study the effects of an experimental 5 F Kensey catheter in normal pig and necropsy human coronary arteries. In 23 normal porcine arteries small intimal flaps were produced in 21 arteries. This occurred independent of tip rotation speed (20–80 000 rpm). These intimal flaps were usually single and small (<25% of the lumen). The catheter was seen to keep a central position in the arterial lumen when fluid was injected, resulting in only one perforation. In 25 normal porcine arteries the catheter was forced through single narrowings created by band ligation. The perforation rate increased with cam speed (0% at 20 000 rpm, 16% at 40 000 rpm, and 29% at 80 000 rpm, respectively). Perforations were rarely diagnosed angiographically, the common findings being of large multiple obstructive intimal flaps. In seven necropsy human coronary arteries angiography was more sensitive than angiography in detecting the aetiology of occlusion (thrombus v atheroma). It also detected dissections that were missed on angiography. Kensey atherectomy was performed on all seven arteries successfully reopening stenotic and occluded arteries with one perforation.

We conclude that (a) angiography is a sensitive method of detecting minimal vessel damage and (b) that the Kensey catheter has applications in the coronary artery, but that the approach should be cautious.

Comparison of visual estimation, measurement by hand held callipers, automated digital quantification, for the assessment of results of percutaneous transluminal coronary angioplasty

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Digital subtraction coronary angiograms of 27 patients who underwent coronary angioplasty to a total of 32 lesions were analysed using an automated border detecting computer program and hand held calliper measurement of diameter percent stenosis. The results were compared with visual interpretation of the 35 mm cineangiograms. Visual reports significantly overestimated the pre PTCA diameter percent stenosis (p<0.001) and underestimated the residual post PTCA narrowing when compared with the automatic computer program (p<0.001). Calliper measurements significantly overestimated the pre PTCA stenosis in comparison with the computer (p<0.01), but post PTCA the two methods did not differ significantly (p = 0.105). There was a positive but poor correlation between callipers and computer measurements (r = 0.43, p<0.005) performed on the pre PTCA digital angiograms. Post PTCA the two methods correlated better (r = 0.78, p<0.001) but further statistical analysis showed important discrepancies between them. The correlation between visual reports and computer measurements was poor before PTCA (Kendall’s τ = 0.32, p<0.05), and not statistically significant after PTCA (τ = 0.64, p = 0.5).

We conclude that there is observer bias in the visual reporting of angioplasty results such that pre PTCA lesions are overestimated whereas post PTCA are underestimated. Measurement by hand held callipers improves the assessment of coronary stenoses in comparison with the visual report, but still may not altogether eliminate observer bias, overestimating the pre PTCA stenoses compared with automated border detection.
Percutaneous angiography

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Percutaneous angiography has been limited in its applications because of poor optical resolution and difficulty in obtaining a bloodless field. We have developed a method of angiography that produces high optical quality imaging. Four arteries in three patients were viewed angioscopically using a high resolution Olympus 1-8 mm endoscope attached to a microchip video camera, with a xenon light source. The angioscope and video camera were sterilised in ethylene oxide, and the angioscope inserted percutaneously through an 8 F valved sheath into the artery. Sheaths of various length were used so that the angioscope could be positioned within the sheath close to the area to be inspected. The sheath was flushed with saline above arterial pressure. The angioscopic findings were compared with angiography performed before and after angioscopy. Angioscopy was able to distinguish between stenotic and occlusive disease, thrombus, and atheroma, and detected abnormalities not seen on angiography, including intimal flaps, fractured atheromatous plaque, and fresh thrombus in two of four introducer sheaths despite the patients having had large doses of heparin. In three of these arteries dynamic angioplasty was performed; angioscopy was again able to detect intimal damage and loose atheroma fragments not seen on angiography. The amount of flush solution used in each case was high (average 1-3 litres). Our patients were assessed carefully before and after angioscopy and none developed signs of fluid overload; further techniques to reduce the flush volume are needed, however, before this valuable technique can have a wider clinical application.

Laser thermal angioplasty: limitations in the coronary circulation

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To identify the factors responsible for the poor primary success rate of metal-capped optical fibres in the coronary circulation we performed the following experimental studies using an argon laser coupled to a 2-0 mm metal capped optical fibre. (a) The histo-

logical effects of laser were studied at 115 intracoronary sites in 12 cadaver hearts during aortic perfusion with either saline or blood. Endothelial thermoablation occurred at 76[23]J, and 47[22]J, respectively, (p<0.05) and perforation at 156[24]J, and 66[25]J, (p<0.001). Significant thermal damage to the contralateral normal arterial wall was a consistent feature in the presence of eccentric plaque, suggesting a less favourable outcome when this geometry exists. Furthermore, at energies required for thermoablation, adherence of the metal cap to the arterial wall resulted in trauma ranging from intraluminal flap formation to arterial intussusception. (b) The thromboembolic risk was assessed in an in vitro circuit during perfusion with 10 ml aliquots of blood at either 5 or 40 ml/minute during delivery of laser pulses of increasing power (4, 8, and 12 W) and time (3, 6 and 9 seconds). Nine aliquots were laser treated at each of the nine possible combinations of power and time; a tenth served as control. The effluent was filtered through five in-line filters to assess debris formation. Higher blood flow resulted in significant debris formation compared to controls at all combinations of power and time. At lower flow this difference was not significant at 4 W (6[14]μg) or 8 W for three seconds, but was significant for all remaining energies to a maximum of 198[35]μg at 12 W for three seconds (p<0.001). Scanning electron microscopy showed fragments >1 mm. (c) The risk of atheromatous embolism was assessed using a similar circuit incorporating coronary endarterectomy specimens treated with laser in saline. Sixty segments were treated: 10 at each of 4, 8, or 12 W for 10 or 20 seconds. Twenty other segments manipulated in a similar way but without being treated served as controls. Weight loss was greater in treated segments than controls (23[14] mg v 5[4:2:3] mg, p<0.001) although calcific foci were resistant. Total debris produced was greater than controls (275[118] μg v 155[65] μg, p<0.005). Most fragments were <50 μm although some of 200–300 μm were seen. In conclusion, although atheroma can be debulked, the application of these fibres is limited by arterial adherence, circumferential thermal damage in the presence of eccentric lesions, inability to ablate calcific plaque, and the potential for distal embolism.

Haemodynamic and renal effects of low dose infusions of atrial natriuretic peptide in chronic heart failure

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High dose infusions (>25 pmol/kg/minute) of atrial natriuretic peptide (ANP) cause a diuresis accompanied by a fall in systemic vascular resistance (SVR) and mean arterial pressure (MAP), but these infusions produce high plasma concentrations of the peptide. In order to elucidate the role of ANP at concentrations found in chronic heart failure we administered a low dose infusion (5 pmol/kg/minute) for four hours to five patients with chronic heart failure (NYHA class III) in a placebo controlled cross over study. Plasma immunoreactive ANP was raised at the start at 113 (24) pg/ml and rose during active infusions to 487 (37) pg/ml. This resulted in a significant rise in urine volume (placebo 2-7(0-6), ANP 4-5(0-9) ml/minute, p < 0.05) and fractional sodium excretion (placebo 0·6(0·2)%, ANP 2·9(1·0)%, p < 0·05). No significant differences were noted in renal blood flow, glomerular filtration rate, plasma renin activity, plasma angiotensin II, or plasma viscosity. Mean right atrial pressure fell from 9·03(2·3) mm Hg to 6·3(1·2) mm Hg, p < 0·05, mean pulmonary artery pressure from 28·2(2·9) mm Hg to 23·2(3·4) mm Hg, p < 0·05), and wedge pressure for 16·1(2·6) mm Hg to 13·4(1·8) mm Hg, p < 0·05, on placebo compared with ANP, respectively. No effects were noted on cardiac output, SVR, or MAP.

This suggests that low dose infusion of ANP that produces plasma concentrations seen in patients with chronic heart failure, acts primarily on the venous capacitance vessels with no significant effect on SVR. The marked diuretic and natriuretic properties are retained at this lower dose.

**Captopril does not potentiate the action of loop diuretics in patients with cardiac failure**

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Angiotensin converting enzyme (ACE) inhibitors reduce the plasma concentrations of the sodium retaining hormones angiotensin II and aldosterone. This should lead to an increase in natriuresis and diuresis; placebo controlled studies in heart failure have, however, shown an early weight gain and development of oedema after the introduction of ACE inhibitors. We studied the natriuretic and diuretic response to a 40 mg intravenous dose of furosemide in 12 supine patients with cardiac failure (NYHA III), and repeated these observations on another occasion one hour after a 25 mg oral dose of captopril. Urinary sodium, potassium, and creatinine concentrations were measured for four hours after the administration of furosemide. Plasma renin activity, angiotensin II, and aldosterone concentrations were measured at hourly intervals. Captopril caused a pronounced reduction in the furosemide induced natriuresis from 120 (9-6) to 67 (11·7) mmol, (p < 0·05), urine output also fell from 1166 (60) to 685 (77) ml, (p < 0·05). Captopril decreased creatinine clearance from 91 (7-2) to 57 (7·7) ml/minute. Systolic and diastolic blood pressures were 5 and 7% lower after captopril. Plasma renin activity rose from 3-53 (0-76) to 12·34 (4-1) ng/ml and plasma angiotensin II fell from 24·9 (5·05) to 10·72 (2·4) pg/ml with captopril. Plasma aldosterone concentrations were slightly lower with captopril.

ACE inhibitors produce an initial reduction in the supine natriuretic and diuretic responses to a loop diuretic. The dose of diuretics may therefore need to be increased in some patients.

**Comparison of the haemodynamic effects of dopexamine and dobutamine in patients with severe heart failure**

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Dopexamine is a novel compound with properties of DA_1 dopaminergic and β_2 adrenergic receptor agonism and noradrenaline reuptake inhibition. It has been shown to produce beneficial renal and haemodynamic effects in patients with severe heart failure (NYHA IV). Haemodynamic data were obtained using thermocoupling Swan-Ganz catheters and arterial cannulas before and during graded incremental doses of dopexamine (DP) from 0·5 to 6 μg/kg/minute and of dobutamine (DB) from 2·5 to 20 μg/kg/minute. The heart rates at basal and at peak infusion rates were similar (DP 87 (17) to 100 (14), DB 91 (18) to 103 (17) per minute), and so were the pulmonary artery occluded pressures (DP 24 (6) to 22 (6), DB 25 (9) to 24 (10) mm Hg), the cardiac indices (DP 1·7 (0·5) to 2·8 (1·1), DB 1·8 (0·5) to 3·0 (1·1) 1/minute/m^2 and the systemic vascular resistances (DP 1553 (221) to 1117 (432), 1721 (347) to 1280 (433) dyn.s.cm^-5). The peak left ventricular stroke work index (DP 20 (9), DB 27 (15) g.m/m^2, p < 0·05) and peak cardiac power output (DP 0·71 (0·36), DB 0·93 (0·46) W, p < 0·05) were different.
These data and calculations using an analytical technique of differentiating inotropic from vasodilatory effects showed that dopexamine produced milder positive inotropic effects than dobutamine. Because of the beneficial central haemodynamic effects and its known direct renal vasodilatory property, dopexamine can be considered to be the drug of choice in the short term treatment of the subgroup of patients with heart failure and compromised renal perfusion.

**Thrombolytic treatment in cardiogenic shock**

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Twenty-five patients aged 40-76 (mean 60.8) with acute myocardial infarction complicated by cardiogenic shock were admitted over a two year period. Systolic blood pressure was less than 80 mm Hg despite aggressive inotropic treatment. Onset of symptoms to treatment was less than six hours. Sixteen patients (mean age 56.2) were given 1.5 million units of streptokinase intravenously as well as heparin. Nine patients (mean age 68.8) had conventional treatment without thrombolytic treatment. Delay times to treatment were 132 minutes in the thrombolytic group and 70.5 minutes in the conventional treatment group. All patients had a previous history of ischaemic heart disease. In the streptokinase group, 12 patients (75%) are alive (mean 19.3 months), and four died in hospital. In the conventional treatment group, eight patients died in hospital and one survived for five months after discharge. The observed difference in hospital mortality, despite the small number of cases, is highly significant (p = 0.003). Mean time to reperfusion in the streptokinase group was 70.4 minutes. Angiography was performed in only nine patients in the streptokinase group and evidence of reperfusion in the infarct related artery was seen in all.

Cardiogenic shock has a high mortality. Survival after thrombolysis at one year is significantly improved in this condition, thus preserving myocardium and improving morbidity and mortality.

**Alcohol and cardiovascular mortality**

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A number of studies have shown that both non-drinkers and heavy drinkers have higher total mortality rates than light to moderate drinkers, and some studies show an inverse relation between alcohol intake and cardiovascular mortality. In the British Regional Heart Study of 7735 middle aged British men there have been 504 deaths during 7-5 years follow up. There is a U shaped relationship between alcohol intake and total mortality and an inverse relationship with cardiovascular mortality even after adjustment for established cardiovascular risk factors. The relation between alcohol intake and mortality was examined separately in men with and without recall of cardiovascular or cardiovascular-related doctor diagnoses at screening. The U shaped curve for total mortality and the inverse relationship with cardiovascular mortality were seen only in men with cardiovascular related illness present at initial examination. The data suggest that the U shaped total mortality pattern and the inverse cardiovascular mortality pattern are produced by pre-existing disease, and the movement of men with disease into non-drinking and occasional drinking categories.

The apparent "protection" effect of moderate alcohol intake may be a consequence of this phenomenon and not due to any benefits from alcohol.

**The national history of idiopathic dilated cardiomyopathy: a comparison of population based and referred cohorts**

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Studies documenting the natural history of idiopathic dilated cardiomyopathy (DCM) report a high annual mortality rate. Since these studies are based on data from tertiary referral centres, however, the observed high mortality may be due to referral bias. We studied survival in a population based cohort of DCM (n = 41), identified through the unique Mayo Clinic records linkage system and representing all incident cases of DCM in the population of Olmsted County, Minnesota, for the 10 years 1975-1984. The results were compared with a previously defined cohort of patients referred to the Mayo Clinic with DCM. The median ages at diagnosis in the population based and referred cohorts were 52 and 49 years; sex ratios (M:F) were 3:6 and 1:4, respectively. There was no significant difference between the proportions of the population based and referred cohorts that had cardiomegaly (cardiothoracic ratio on chest x-ray >0.50), cardiac failure, or electrocardiographic abnormalities at diagnosis or...
entry into the study (73% v 87%, 93% v 80%, and 66% v 73%). Survival rates, relative to the United States West North Central population in 1980, were 95% v 69% at one year (p < 0.001), and 94% v 40% at five years, (p < 0.001) in the population based and referred cohorts, respectively.

These results suggest that the natural history of unselected DCM is more benign than previously reported and that high mortality in DCM may be a reflection of referral bias.

In vitro flow studies to improve hydrodynamic design of Fontan reconstruction

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After Fontan reconstruction, fluid energy losses in the reshaped pathways of flow may critically raise the already raised systemic venous pressure. We performed in vitro flow studies with a view to improving the hydrodynamic design of atrio-pulmonary connections. Silicone rubber casts of reconstructed heart cavities provided a basis for making models in which a suspension of aluminium dust was used to look at areas of turbulent flow. Smoothing of contours at cavity outflow tracts and 90° junctions was found to reduce turbulence; associated pressure head losses fell from levels equivalent to those of a 50%, to those of a 20%, area stenosis. Pulsation of a valveless cavity in a continuous flow circuit was found to hinder rather than assist net forward flow, turbulence being increased by pulsation. These findings recorded on videotape illustrate the importance of streamlined shaping of anastomotic sites, and show that inclusion of a valveless, contractile chamber, as in a conventional Fontan circulation, may be detrimental in terms of local flow dynamics.

The studies supported our decision to opt for total cavopulmonary connection as an alternative to atrio-pulmonary connection in 22 children who required Fontan reconstruction. Postoperative angiographic studies shown favourable cavopulmonary flow in these patients.

venous compared with anatomical repair for double outlet right ventricle with sub-pulmonary ventricular septal defect

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Thirty patients with double outlet right ventricle and subpulmonary ventricular septal defect, Taussig-Bing disease, who underwent repair over a 14 year period (1973–1987) have been reviewed for their early, intermediate, and long term status. At operation their ages ranged from two months to 8 years (median 12 months) and weight from 3 to 16.5 kg (median 7.5 kg). All 14 patients who had undergone previous palliative operations had pulmonary artery banding and in addition, five of these required repair of a coexisting aortic coarctation, one of an interrupted aortic arch, and one had a Blalock-Hanlon septectomy. Fifteen patients underwent inflow correction of the venous return at atrial level with a Mustard procedure in conjunction with patch repair of the associated ventricular septal defect; in this group there were nine hospital deaths (60%).

Fifteen patients had anatomical correction, in one patient by an intracardiac repair which was not successful, and in the others by an “arterial switch” Jatene’s operation, with two hospital deaths (13%). By multivariate analysis, the change over point from the Mustard to the arterial switch procedure was the only event that significantly affected the hospital mortality (p 0.025). The 18 survivors have been followed for a period of two months to 148 months (median 23 months). There were two late deaths, both in patients who had repairs by atrial redirection (33%). One patient who had the arterial switch operation required closure of a residual ventricular septal defect four months post-operatively. All long term survivors are in functional class I. Postoperative catheterisation in five patients (mean interval 5-8 months) shows normal ventricular function, no valvar regurgitation, and no coronary stenosis.

The improved survival rate, which we expect to continue, together with the satisfactory intermediate functional results have shown that Jatene’s operation is the operation of choice for double outlet right ventricle with subpulmonary ventricular defect.

Sinus venosus atrial septal defect: investigation and repair

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Most sinus venosus atrial septal defects (SVASD) are not diagnosed preoperatively, and postoperative atrial arrhythmias are common due to the proximity of the SA node to the site of repair. A review of the
217 ASD repairs performed here in the 10 year period up to March 1988 showed that 57 (26\%) were for SVASD. Of the latter, 34 were male and 23 female patients whose ages ranged from 1 to 58 years (mean 14 years). Sixty per cent were symptom free. Echocardiography was performed in 54 patients (95\%) but correctly diagnosed the defect in only 4\%. Cardiac catheterisation was performed in 35 patients (61\%) and was correct in 54\%. Overall 38\% were correctly diagnosed preoperatively. Operative correction, on cardiopulmonary bypass with hypothermia and cardioplegia, was by pericardial xenograft patch (n = 31), Dacron patch (n = 21), Goretx patch (n = 1), or direct suture (n = 4). Interrupted sutures to hold the patch were placed from outside the heart to avoid the SA node. There was one operative death (1.8\% mortality). Follow up ranges from 1 to 96 months (mean 34 months). Two cases were lost to follow up, two patients remain dyspnoeic, and one has a nodal rhythm. The remainder are symptom free and in sinus rhythm.

In conclusion, most SVASDs are still not diagnosed before operation. Because patients are now usually accepted for surgery on echocardiographic evidence alone, the need for careful inspection of cardiac anatomy in theatre is emphasised. The technique of external suture placement may avoid long term rhythm problems.

**Total correction of the tetralogy of Fallot: assessment of survival**

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During the period from January 1970 to December 1985, 277 patients underwent total correction of the tetralogy of Fallot at two cardiac centres. The mean (SD) age at correction was 7.5 (15.5) years. In 158 patients total repair was performed as a primary procedure whilst 119 previously had a palliative operation (Blalock-Taussig shunt in 72\%, Brock procedure in 21\%, and Waterston shunt in 7\%). A transannular patch was required in 119 cases and 36\% of patients received a monocusp homograft patch. The overall hospital mortality was 11.5\%. There were only two deaths outside the immediate postoperative period (0–28 days). There was no significant difference in actuarial survival between centres ($\chi^2 = 2.25, p > 0.1$), or between primary corrective procedures and staged repairs ($\chi^2 = 0.68, p > 0.1$). Significant differences were found between those patients less than two years at total repair (mortality 18.6\%) compared with those older than two years at operation (mortality 8.4\%, $p < 0.01$) and between those corrected before 1977 (mortality 15.2\%) compared with those repaired during the latter half of the study period (mortality 7.5\%, $p < 0.05$). Multivariate analysis of risk of hospital death showed that the independent risk factors were high postoperative RV:LV pressure ratios ($p < 0.001$) and low body surface area ($p < 0.02$). No incremental risk was shown for a transannular patch when a monocusp homograft was incorporated.

In conclusion though hospital mortality from total repair of Fallot's tetralogy has fallen throughout the period of our study, small size at repair and diminutive pulmonary arteries remain important risk factors.

**Mustard's operation: long term haemodynamics in symptom free children and balloon dilatation for systemic venous obstruction**

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Twenty symptom free children were electively catheterised 10 years after Mustard's operation for simple transposition of the great arteries. Another two patients with signs of superior vena cava (SVC) obstruction were added and underwent balloon dilatation. All patients were anaesthetised and the study was carried out in air and in 100% oxygen. Oxygen consumption was measured with respiratory mass spectrometry and end tidal gases were monitored throughout the study. Mean right ventricular end diastolic pressure was 87 (4.6) mm Hg (range: 5 to 20 mm Hg). Two patients had important left ventricular outflow tract obstruction (> 50 mm Hg), and two had minor intracavity obstruction (20 and 25 mm Hg). Cardiac output was normal in 15 and below normal in 5 patients (mean = 3.45 (0.8) l/m²/minute). Pulmonary vascular resistance (PVR) in air of < 3 units was found in 16/19 patients. Gross increase in PVR in air and in 100% oxygen (> 20 units) was present in one patient. Baffle dysfunction was present in 11 patients: 10 with gradients > 5 mm Hg between the SVC or inferior vena cava (IVC) and the systemic venous atrium, and five had a leak identified either by a left to right shunt or by the catheter course. Balloon dilatation was attempted in
eight patients with gradients of > 5 mm Hg in the IVC channel, and SVC dilatation was an additional procedure in two of them. Mean gradient was 6.4 (2.3) mm Hg before the dilatation and fell to 4.1 (2.2) mm Hg after the procedure. No pulmonary venous obstruction was identified.

In this selected group of symptom free children, about half had a detectable haemodynamic abnormality. Transvenous balloon dilatation is effective in some patients with systemic venous obstruction.

Heart and heart-lung transplantation in children

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Since August 1984, 76 children aged under 15 years have undergone transplantation. Thirty six patients aged 9 days to 14 (mean 7.5) years underwent heart transplantation (7% of the total population). The main indications were dilated cardiomyopathy in 15 and congenital heart disease (CHD) in five. Forty patients aged 10 weeks to 14 (mean 7.8) years underwent heart-lung transplantation (HLT) (23% of the total) for pulmonary vascular disease, either primary in five or secondary to CHD in 16. Four patients had parenchymal lung disease. There were eight (23%) and 13 (30%) early deaths: moderate pulmonary hypertension in the recipient and bleeding from previous thoracotomy were important risk factors in each group, respectively. With a mean follow-up of 21.6 and 15.4 months, there were one and two late deaths in each group, respectively. To avoid the first risk factor we are now using “primed” hearts from “live” donors. Postoperatively patients were maintained on cyclosporin and azathioprine. Routine steroids are not given. All patients are well, growing normally, and attending normal schools. One patient developed lymphoproliferative disease, which has regressed completely. Two patients after HLT developed obliterative bronchiolitis at 12 and 24 months, and one patient required retransplantation. Routine annual investigations have shown no evidence of chronic rejection in either group.

It is concluded that transplantation offers a chance of remarkable improvement in children with end stage heart and lung disease but long term function, particularly of the lungs, continues to need evaluation.

Decreased pulmonary microvascular permeability in patients with severe mitral stenosis

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In mitral stenosis (MS) a considerable rise in the pulmonary capillary pressure may occur without the development of pulmonary oedema. The mechanism protecting against pulmonary oedema is not known, but might be the result of a reduction in pulmonary microvascular permeability (PMVP). PMVP was measured non-invasively using a previously validated double isotope scintigraphic method that permits calculation of a plasma protein accumulation index (PPA index) for the lung fields. Studies were performed in nine patients with severe MS (Gorlin valve area 1 cm² or less, mean pulmonary capillary wedge pressure 27 (4) mm Hg) and in 10 control patients. The PPA index was significantly lower in the MS patients, being 0.17 (0.53) × 10⁻³/minute, and 0.46 (0.54) × 10⁻³/minute in the control subjects (p < 0.05). Furthermore, within the group of patients with MS, there was a correlation between the PPA index and the severity of MS, as reflected by the Gorlin valve area (G): PPA index = 1.35 (G) – 0.67, r = 0.7, p < 0.05.

These data indicate that in severe MS, PMVP is significantly reduced, providing a mechanism that protects against pulmonary oedema despite a chronic rise of the pulmonary capillary pressure.

Balloon dilatation of the mitral valve by double and single balloon techniques

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Balloon dilatation of the mitral valve was attempted in 35 patients with symptomatic rheumatic mitral stenosis (mean age 48 years, range 20–80). The valve was successfully dilated in 30 patients. In 20 patients two guide wires were passed through the left heart and stabilised in the descending aorta. This allowed two balloons (15 mm × 2 or 25 mm + 10 mm in size) to be inflated simultaneously in the mitral valve. In six patients a bifoil balloon (19 mm × 2) was positioned on a single wire and in four patients a single balloon (25 mm) was used. The end diastolic mitral
valve gradient was reduced from 14·2 (6·1) to 4·9 (4·2) mm Hg, and mean left atrial pressure fell from 22·6 (6·3) to 13·7 (7·5) mm Hg. The calculated mitral valve area increased from 1·17 (2·9) to 2·28 (3) cm$^2$. At cross sectional echocardiography and Doppler assessment within one week of dilatation, the mitral valve area had increased from 1·1 to 1·7 cm$^2$. Mitral regurgitation of grade 3/4 developed in one patient who required early mitral valve replacement. Three patients developed tamponade, which was successfully relieved. At follow up (mean 9 months) all patients had improved symptomatically and on repeat echocardiography and Doppler examination the mean mitral valve area was 1·6 cm$^2$.

The best results were obtained in the younger patients with mobile valves, but useful improvement was achieved in older patients with calcified valves. The double balloon or bifoil balloon was superior to the single balloon technique. Balloon dilatation of the mitral valve is an effective procedure for the treatment of patients with mitral stenosis and an important alternative to surgery.

**Doppler echocardiographic characteristics of prosthetic valves in the tricuspid position**

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There is little information available concerning prosthetic tricuspid valve (TV) function. Thirty two patients with prosthetic TV and prosthetic mitral valves (MV) were studied using simultaneous imaging and pulsed wave Doppler echocardiography to measure peak velocity (PVk) m/s and pressure half time (Pt 1/2) m seconds. There were 26 Björk-Shiley and six Carpentier-Edwards xenograft valves in each position. The time from implantation to study ranged from 6 months to 18 years (median 10·5). Twelve native tricuspid and mitral valves were studied as a control group. Doppler data for prosthetic MV and TV flow were obtained in 27 patients with clinically normal valves. Pt 1/2 was significantly longer in the TV 38–197 (median 105·3) mseconds than in the MV, 46–104 (73) msec (p < 0·02), but no significant difference in PKv was found, TV 0·6–1·50 (1·02) m/s, and MV 0·40–1·20 (0·91) m/s. There was no correlation between PKv or Pt 1/2 and time since surgery, or valve size. Five patients had clinically abnormal prosthetic TV with: PKv 1·53–1·94 (1·73) m/s, Pt 1/2 237–560 (308) mseconds. Values for the normal TV control group were: PKv 0·34–0·84 (0·54) m/s, Pt 1/2 31–71 (57) mseconds, and for the normal MV were: PKv 0·40–1·20 (0·91) m/s, Pt 1/2 30–65 (51) mseconds.

Hence data derived from the prosthetic TV cannot be extrapolated to the prosthetic TV. A Pt 1/2 >200 mseconds seems to distinguish important prosthetic TV obstruction from clinically normal prosthetic TV.

**Balloon dilatation of the aortic valve: an in vitro assessment of the mechanism, magnitude, and duration of its effects**

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Balloon dilatation of the aortic valve (BAV) in adult calcific disease remains a controversial method of treatment with uncertainty as to the magnitude and duration of its effects. We analysed the changes in aortic valve area (AVA) and transvalvular mean systolic gradient (MSG) seen after valvuloplasty in calcified valves removed at operation (n = 17) or necropsy (n = 1). The intact valves were mounted in a pulsatile flow rig that delivered a constant 4 l/minute saline at 37°C with an ejection period of 45% and pulse rate of 70 per minute. Aortic valve motion was videotaped with a magnifying lens and the ABA then planimetered from the screen. A 20 mm balloon inflated to 3 atm for 30–60 seconds was used for dilatation. The AVA and MSG were measured before, immediately after, and at 5, 10, 20, 30, 60, 120 minutes, 3 days, and 1, 2, and four weeks after dilatation. AVA increased from (mean (SD)) 0·52 (0·16) cm$^2$ to 0·78 (0·17) and the MSG decreased from 54 (27) to 33 (8) mm Hg (both p < 0·001) immediately after BAV. In only three valves was the peak AVA after BAV 1·0 cm$^2$. At five minutes the AVA decreased to 0·73 (0·16) and at 4 weeks to 0·70 (0·15) (NS). The MSG increased at five minutes to 35 (10) and at four weeks to 40 (11) (NS). Radiographs showed fractures of calcific deposits almost exclusively at the site of commisural splitting. In six valves stretching alone, without commisural splitting occurred and these had a smaller increase in AVA (32% v 60%) and a lesser fall in MSG (21% v 48%).

Significant though small haemodynamic improvements were obtained by BAV using a 20 mm balloon. These were more pronounced with commisural splitting that with simple valve stretching alone. At five minutes after BAV there was already a partial reversal of these peak effects which then persisted up to four weeks in the absence of body repair mechanisms.
Aortic root abscess complicating aortic valve endocarditis

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Development of aortic root abscess (ARA) in the course of infective endocarditis is a major determinant of the outcome of the disease. In a retrospective study of 49 patients (40 men and 9 women; median age 53, range 21–80) referred to a cardiothoracic centre with aortic valve endocarditis between 1982 and 1987, 42 patients underwent aortic valve surgery; 23 (46%) of the 49 patients had ARA (13 with prosthetic valve and 10 patients with native valve endocarditis). Intracardiac conduction disturbance (prolonged PR interval in 13, complete heart block in three, and bifascicular block in two patients) occurred in 15 (65%) of the 23 patients with ARA and in only three (11%) of 26 patients without ARA. Echocardiography correctly identified the presence of ARA in six (23%) patients. In four (17%), presence of ARA was unsuspected preoperatively. Streptococcus viridans was the most common infecting organism in the whole group (42%) but no particular organism was identified as predisposing to the formation of ARA. Early postoperative paraprosthetic leak of mild to moderate severity was noted in eight (40%) of 20 patients with ARA as opposed to only two (9%) of 22 patients with ARA. Surgical mortality was significantly higher (16.6%) in patients with ARA compared with 2.4% in patients with noninfected aortic roots (p < 0.05).

Aortic valve endocarditis complicated by aortic root abscess formation carries a high operative and postoperative mortality and is commonly associated with postoperative paraprosthetic leak. In this series, conduction disturbance on the ECG was the most common indicator of the presence of an aortic root abscess.

Comparative value of precordial and transoesophageal echocardiography in the spectrum of infective endocarditis

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We report 26 consecutive patients with clinically suspected endocarditis (all bacteriologically culture positive) in whom the findings from standard precordial cross sectional echocardiography (PE) were compared and contrasted with those from transoesophageal echo (TEE). Seventeen patients had suspected native valve endocarditis and nine had suspected prosthetic valve endocarditis (one triple valve replacement, seven aortic prostheses, and two mitral prostheses). PE suggested intracardiac vegetations in only five of 26 studies and identified no infective sequelae. TEE identified imaging evidence of vegetations in 18/26 with vegetations alone visualised in 10 patients. In eight patients vegetations plus evidence of infective sequelae were seen while in the remaining eight patients no vegetations were seen but the diagnosis was inferred by imaging infective sequelae. The infective sequelae visualised by TEE in the 16 patients included seven mycotic aneurysms, one aortic cuspid perforation, five mitral chordae ruptures, four prosthetic valve dehiscences, and one left ventricular free wall intramural abscesses. In no case was the infective sequelae visualised by PE. In every case TEE images were diagnostic and allowed correct clinical management decisions to be made without recourse to further investigation.

We conclude that TEE is the ultrasound diagnostic approach with the highest yield in the investigation and management of endocarditis and its complications, especially in the setting of suspected mitral prosthetic valve endocarditis and aortic root or septal infection. It would seem to be mandatory where appropriate clinical suspicions are present and precordial imaging is either equivocal or negative.

Emergency operation after failed percutaneous angioplasty: how late?

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Immediate surgical revascularisation is the ideal in reversing myocardial ischaemia after failed percutaneous transluminal angioplasty (PTCA) but this may not be always achievable. Between January 1983 and December 1987 we provided surgical cover for 667 patients of whom 25 (3.7%) came to emergency coronary bypass surgery. The revascularisation time—that is the time between the onset of the ischaemia at PTCA and the opening of the coronary bypass grafts at surgery—ranged from 160 minutes to 390 minutes, with a mean of 285 minutes. Cardiac-selective enzymes (creatine kinase MB) were recorded preoperatively, and at the same time as the
ECG for three days postoperatively. Seventeen patients required a single graft, five had double grafts, two had triple grafts, and one had a quadruple graft. There were no early or late deaths, and at follow up 6–48 months postoperatively (mean 27 months), 20 of the patients were free from angina, three had mild angina, and two had moderate angina. Ten patients had myocardial infarction (MI) as shown by CKMB and ECG readings post operatively although three of these had enzymatic evidence of MI preoperatively. The mean revascularisation time of the patients who developed a MI was 250 minutes, while those who did not develop a MI had a mean revascularisation time of 280 minutes. There was no correlation between the development of a MI after surgical revascularisation and the number of grafts inserted.

Though earlier revascularisation after failed PTCA should be aimed for, it seems reasonable to carry it out up to 6½ hours after the onset of ischaemia.

Long term follow up after percutaneous transluminal coronary angioplasty in patients with chronic total occlusions

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Percutaneous transluminal coronary angioplasty (PTCA) was performed on 90 total occlusions by a single operator between December 1982 and June 1988, with a 52% (47/90) primary success rate. Acute occlusions (<2 months duration) were present in 27 (30%) with a primary success rate of 63% (17/27). Chronic total occlusion with no antegrade flow (TIMI grade 0, 1) was present in 63 (70%). The mean estimated duration of occlusion was 19 months (>2–120). All patients had severe angina pectoris with strongly positive exercise tests. Successful dilatation was achieved in 40% (25/63). The success rate was 51% in the second half of the group and 35% in the first half. The successfully dilated vessel was infarct related in 64% (16 of 25). Residual stenosis after PTCA of 15 (10%) was present with good antegrade flow (TIMI grade 3). A limited dissection was present at the site of PTCA in six (24%). Clinical and exercise assessment with cardiac catheterisation were undertaken at 18 months (>2–57) after PTCA. There was a 40 (15%) stenosis at the site of successful PTCA in 19 of 25 patients. Five patients (20%) had reoccluded; of these two had limited dissection at PTCA. One other patient had a coronary artery vein graft. Four of five patients who reoccluded had a recurrence of angina. The remaining 19 patients with patent vessels were free of angina and had negative exercise tests.

Despite the comparatively low primary success rate of PTCA in chronically occluded vessels there is symptomatic improvement with good long term patency rates.

Importance of procedural factors in the development of recurrent restenosis after coronary balloon angioplasty

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Previously we have presented preliminary data regarding factors responsible for the development of recurrent restenosis (RRS) after coronary balloon angioplasty (PTCA). We now report the final results of our investigation conducted in all 1011 patients undergoing successful first PTCA at our centre between March 1980 and June 1987. Clinical restenosis (recurrence of angina and >50% stenosis at a site with primary success) developed in 204 patients. A second PTCA was performed in 198 of these 204 patients, with primary success being achieved in 183 (87%). Of these 183 patients, 47 (26%) developed a second restenosis of their initial lesion(s). Comparing clinical and arteriographic variables including age, degree of angina, time between PTCAs, severity of disease, and lesion morphology there were no significant differences between the 47 patients with RRS (group A) and the 136 with single restenosis (group B). Maximum balloon size and maximum inflation pressure used at the second PTCA were similar for the two groups. Univariate analysis showed that important predictors of RRS were the greater number of inflations, mean (SEM) 6·4 (0·6), in group A, 4·4 (0·2) in group B; p < 0·0001 and the shorter maximum inflation time (48 (4) seconds in group A, and 69 (3) s in group B; p < 0·005). When the longest balloon inflation time was >60 s, the incidence of RRS was 20%, as opposed to 40% when it was ≤60 s (p < 0·05). These data suggest that the incidence of recurrent restenosis may be reduced by the use of fewer, more prolonged inflations when dilating initial restenosis.
Management and outcome of patients with recurrent restenosis after coronary balloon angioplasty

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Patients who develop restenosis (R) after first coronary balloon angioplasty (PTCA) are generally treated with a second PTCA. Less, however, is known about the management of patients who develop a second restenosis after repeat PTCA. To determine the outcome of such patients we reviewed the records of all 1162 patients undergoing PTCA at our centre between March 1980 and June 1987. Initial angioplasty was successful in 1011 patients (87%). Two hundred and four patients (20%) developed clinical R (recurrence of angina and >50% stenosis at a site with primary success). Of these 198 were treated with redilatation, which was successful in 183 (92%). After the second dilatation, R developed in 47 patients (26%). Of these, 41 (87%) were treated with a third PTCA, with primary success in 38 (92%). A further restenosis developed in 13 of these 38 patients (34%). Eight patients were treated with a fourth angioplasty, with restenosis occurring in four (50%). Two of these four patients underwent a fifth PTCA (with continuing success at long term follow up in both). Overall, 14 of the 47 (30%) patients who developed recurrent R were eventually treated with coronary bypass surgery. Most of these patients (33), however, were treated only with repeated angioplasties. Of these 33 patients, 27 were treated with a third angioplasty, four with a fourth procedure, and two with a fifth. Twenty nine (88%) remain free at a mean follow up of 28 months (range 8–86 months). The combined success rate for a third, fourth, and fifth angioplasty was 94%.

These data suggest that most patients with recurrent restenosis after angioplasty may be managed, both successfully and safely, with repeated redilatations.

We investigated the ability of serial exercise testing to predict restenosis after coronary angioplasty (PTCA). Thirty one consecutive patients with single vessel disease who had successful PTCA (≥30% reduction in stenosis diameter measured by computerised quantitative angiography) underwent treadmill exercise testing treatment before PTCA and at three days, and one, three, and six months afterwards. All 31 patients had positive exercise tests with ST-segment depression ≥0·1 mV before PTCA. At angiography six months after PTCA, restenosis (≥70% loss of the initial gain in luminal diameter) had occurred in 14 patients (group 1) but not in 17 patients (group 2). At three days after PTCA, 11 patients in group 1 had a positive exercise test compared with three patients in group 2 (p = 0·09). At one, three, and six months, all 14 patients in group 1 had positive exercise tests, compared with only one in group 2 (p < 0·01). The ischaemic threshold was assessed by measuring the heart rate systolic blood pressure product (RPP) at 0·1 mV ST-segment depression or peak exercise if the test was negative. In group 1 the mean (SD) RPP increased from 16270 (3100) before PTCA to 20400 (3680), (p < 0·01), at three days, but fell to 16090 (5150) at six months. In group 2 the RPP increased progressively from 14840 (4400) before PTCA to 27372 (4177), (p < 0·001) at six months. A significant difference in RPP between the two groups was first seen at one month (20870 (3110) in group 1, 22177 (2913) in group 2, (p = 0·05).

Patients with restenosis after PTCA show a deterioration in ischaemic threshold after the first month, whereas it progressively improves in those without restenosis. In these patients the predictive value of a positive exercise test for restenosis is 79% at three days, and 100% at one and six months, but the predictive value of a negative exercise test is 82% at three days, and 94% at one and six months.

Long term clinical follow up after coronary angioplasty for single vessel disease

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The efficacy of coronary angioplasty in selected patients with symptomatic coronary artery disease has been established, but information on the long term benefit of the procedure remains incomplete. We have analysed the acute and long term results of coronary angioplasty in 336 patients with single vessel disease (mean age 53 years, range 27–76). Before

Can exercise testing predict restenosis after coronary angioplasty?

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Angioplasty 74% complained of severe (grade 3–4) angina and 29% had a history of myocardial infarction. Initial angiographic success was achieved in 286 patients (85.1%), but complications occurred in 13 of these, resulting in an overall primary success rate of 81.3%. At one year 4.5% of successfully treated patients had undergone coronary surgery, and 15% had required repeat angioplasty. During follow up ranging up to five years, over 75% of all patients remained free from angina, over 80% were improved by at least two grades, and 59% were able to work. Cumulative cardiac event rates were calculated for patients with primary success. The five year cumulative freedom from coronary artery surgery was 88.0%, and from repeat angioplasty was 81.4%. The cumulative five year non-fatal myocardial infarction rate was 4.9%. Cumulative five year cardiac survival was 94.8%, both for successful cases and for all patients. Intention to treat analysis showed a five year survival from all causes of 89.3%.

These data indicate that after coronary angioplasty for single vessel disease three quarters of all patients remain free of angina in the long term, with a low risk of late cardiac death.

Hazards associated with the use of temporary pacemakers before permanent pacing

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The records of 145 consecutive patients who were treated with a temporary pacemaker before insertion of a permanent pacemaker for chronic atrioventricular block (110) or sinoatrial disease (35) were reviewed. The temporary pacing lead was inserted in a districted general hospital in 116 (80%) and after arrival at the cardiac centre in 29 (20%). Problems were noted in 74 (51%), with at least one major problem in 52 (36%). These included failure to pace (46 episodes in 40 patients), systemic infection (n = 9), pericarditis (n = 6), haemothorax (n = 2), fatal pulmonary embolism (n = 1), and inadvertent placement in the left ventricle (n = 1). The incidence of problems was inversely related to local experience with the procedure. Thus temporary pacing was associated with a major problem in 7% of patients treated at the cardiac centre and 43% of patients treated in one of the 10 district general hospitals who contributed patients to this series. Furthermore, there was an inverse correlation between the incidence of problems and the number of patients referred by each district general hospital (p < 0.05, Spearman's rank correlation test).

These findings emphasise that temporary pacing may be ineffective and may result in serious complications, particularly when undertaken by inexperienced operators, and suggest that in many cases the risks may exceed the benefits.

New insights with computerised intraoperative epicardial mapping of QRS alternans during orthodromic tachycardia

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Electrical alternans of the QRS is reported to occur during orthodromic tachycardia. To investigate further the mechanism of QRS electrical alternans, we analysed beat to beat epicardial isochronal and isoaera maps during orthodromic tachycardia in 10 patients. Isochronal maps were generated using a 63 shock array electrode, and isoaera maps were obtained by computing the time integral of the local electrograms with reference to a baseline between the onset of R wave and the T wave offset. Isoarea difference maps were also calculated by subtracting the two successive isoaera maps. Of 13 episodes of sustained orthodromic tachycardia, 11 (85%) showed QRS alternans and the remaining two (15%) did not. Analysis of isochronal maps during orthodromic tachycardia showed that alternans in electrograms occurred in local regions and corresponded to areas with conduction delay. Beat to beat changes in total activation time were independent of alternans. There was no difference in isoaera maps or isoaera difference maps suggesting the phenomenon of alternans is associated with conduction delay rather than changes in action potential duration. Following cryoaablation of the accessory pathway, atrial pacing did not induce electrical alternans. Similarly, at postoperative electrophysiological studies, atrial pacing at cycle lengths identical to those of orthodromic tachycardia did not produce electrical alternans.

These observations, not previously reported, suggest that QRS alternans in orthodromic tachycardia might be caused by local conduction delay within the ventricular myocardium.

Transoesophageal DC cardioversion

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Transchest cardioversion is a procedure of low efficiency as only a small fraction of the energy delivered passes through the heart. We have developed a stable oesophageal electrode system and describe our initial experience for elective DC countershock of atrial dysrhythmias. The oesophageal system consists of four electrodes, each of coiled gold plated copper wire mounted on PVC tubing, the external diameter of which ranges from 5–9 mm. Using this oesophageal electrode system placed 35–40 cm from the mouth to the tip of the system, we have carried out six successful cardioversions in five patients; four were in atrial fibrillation and one had recurrent atrial flutter necessitating two cardioversions within four weeks. Transchest impedances were initially recorded using R2 pads, one positioned in the right infraclavicular position, the other over the apex. Impedances were also recorded between the oesophageal system and an R2 pad on the anterior chest wall. Impedances measured with the oesophageal system (range 28–56.5, mean 42.3Ω) compare favourably with those recorded for the same patients using chest electrodes (range 37–70, mean 55.7Ω). Peak current at defibrillation measured in four patients ranged from 18–26, mean 22.7 A. Delivered energies for successful cardioversion ranged from 30–70 J, mean 48.5 J. Three patients had previously undergone elective countershock using the standard transchest approach, and two of these had required delivered energies of 200–360 J for successful cardioversion. All patients tolerated the procedure well and no adverse effects occurred.

Thus transoesophageal defibrillation allows successful DC conversion using much lower energies than would be required using the conventional transchest approach.

Incidence of malignant vasovagal syndrome in 332 syncope patients: results of cardiac pacing

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Three hundred and thirty two patients with recurrent syncope underwent electrophysiological study (EPS) between January 1984 and June 1988. Complete atrioventricular block (AVB) was found in 68 (21%), intermittent block in 44 (13%), sick sinus syndrome (SSS) in 69 (21%), carotid sinus syndrome (CSS) in 32 (10%), tachyarrhythmia in 19 (6%), and no abnormality in 100 (30%). Head up tilt testing at 60° was performed in 67 patients with normal EPS and it reproduced syncope in 50 (75%). Twelve with atrioventricular block were tilted without syncope. Two of 11 with SSS were syncopal during tilt (18%). Six of 12 with CSS were syncopal on tilt. Of 50 with normal EPS and positive tilt, 40 received DDI pacing, and 10 were unpaced. At 22 (4-3) and 20 (15-4) months follow up, respectively, 14 (35%) had continuing syncope with 1 (1.3) episodes, and in the unpaced group eight (80%) were still syncopal, with 2.75 (1.1) episodes (p < 0.01).

We conclude that a 60° head up tilt identifies a subgroup of patients with recurrent syncope and normal EPS who may benefit from cardiac pacing.

QT prolongation in diabetic autonomic neuropathy

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Congenital QT prolongation, which is associated with cardiac arrhythmias, may be caused by abnormal sympathetic cardiac innervation, but the effect of acquired autonomic neuropathy is uncertain. We have compared the maximum QT interval on 12 lead electrocardiograms recorded at 100 mm/second at rest in 34 diabetics and 20 normal controls. Five tests for autonomic neuropathy (heart rate response to deep breathing, Valsalva manoeuvre, and standing; blood pressure response to standing and hand grip) were scored 0 for normal, 1 for borderline, and 2 for abnormal. Diabetics were grouped as no neuropathy (score 1–3; n = 7), borderline (score 4–7; n = 12), and definite neuropathy (score 8–10; n = 15). The uncorrected QT interval was similar in controls (381 (28 mseconds) and diabetics (376 (31 mseconds), but the mean RR interval was significantly shorter in the diabetics (736 (106) compared with 809 (136) mseconds; p < 0.05). The QT plotted against RR interval was above the 95% limit for the controls in five diabetics with definite neuropathy (33%), but in none of the others. Multivariate analysis showed that RR interval alone explained 48% of the QT variability in the total sample, and RR interval and autonomic group together explained 59% (p < 0.025). Age, sex, or the presence of symptoms of autonomic neuropathy did not contribute significantly to QT variability.

We conclude that QT prolongation is common in diabetics with autonomic dysfunction and is a possible cause for the high incidence of sudden death in these patients.
Accuracy of a semiautomatic defibrillator during cardiac arrests

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Semiautomatic defibrillators incorporating microprocessors for the detection of ventricular fibrillation are now used in the training of paramedical and lay personnel. Using these systems clinically few have reported the results of continuous analysis of the electrocardiogram (ECG) during the cardiac arrest. The Marquette 1200 defibrillator analyses cardiac arrest rhythms in continuous 8-12 second episodes and advises whether DC shock is indicated. It was used at 57 arrests in 55 patients and in one patient who had ventricular tachycardia (VT). Initial rhythm was ventricular fibrillation (VF) in 40 arrests and was correctly identified in 37 (92.5%). Of non-VF initial rhythms 17/18 (94%) had no shock advised. Of 438 episodes VF/VF flutter a shock was correctly advised in 358 (sensitivity 82%); of 80 incorrectly identified 52 had cardiopulmonary resuscitation, 19 were low amplitude (<0.25 mV), in eight VF started late in the episode, and one had jagged wide complexes resembling wide QRS complexes throughout the VF. Of 4157 non-VF episodes no shock was advised in 3901 (specificity 94%). False positive identification occurred during cardiopulmonary resuscitation (n = 112), VT (n = 98), slow sinusoidal rhythms (n = 4), agonal rhythms (n = 21), generalised fitting (n = 6), frequent ventricular extrasystoles (n = 9), and high ST segment rise causing QRS distortion (n = 3). During analysis, in three episodes a late change to non-VF occurred.

Excluding episodes with cardiopulmonary resuscitation, a high sensitivity of 91% (304/334) and specificity of 96% (3543/3687) for VF detection were recorded.

Stress testing: a district audit

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The safety and consequences for patient management of 777 consecutive exercise electrocardiograms (ECG) undertaken over a two year period were reviewed. The usual indications for testing were for the diagnosis and assessment of angina, but included post myocardial infarction patients. All tests were maximal and patients were on no treatment. There were no deaths, episodes of ventricular fibrillation, or myocardial infarctions during or within 24 hours of the test. Twelve patients developed exercise induced arrhythmias, all of which were self limiting. Two hundred and eighty three (36%) patients had positive exercise ECG, and 153 (19%) had angina. Ninety one patients (13% women; 87% men) were referred for coronary angiography, either because of strongly positive exercise tests or because they had less strongly positive tests but an inadequate response to medical treatment. Twelve patients were referred for angiography without exercise ECGs, mainly for unstable angina. Sixty one per cent of patients undergoing angiography (8% women; 92% men) were referred for coronary artery surgery or angioplasty. During this period of the review 42 exercise isotope ventriculograms, 4 exercise thallium scans, and 20 dipyridamole thallium scans were also undertaken within this district.

Our annual rate per million population was 2158 exercise tests, 286 coronary angiograms, and 175 bypass operations or angioplasties; the regional average is 188 operations or angioplasties. Exercise testing within a district general hospital is a safe and cost efficient means of selecting patients for coronary angiography and surgery.

Is left ventricular dysfunction in mitral stenosis caused by isolated posterobasal wall abnormality?

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In order to determine the mechanism and prevalence of left ventricular (LV) dysfunction in mitral stenosis (MS), the left ventriculograms of 192 patients mean (SD) age 49 (11) years with mitral valve mean gradient of 10-22 mm Hg were analysed. Three cycles of non-post extrasystolic beats were analysed per patient. Left ventriculograms from 25 patients aged 47 (7) years without valvar or coronary artery disease were used as control. Regional wall motion (RWM) was derived from the fractional shortening, from diastole to systole of six hemiaxes along the long axis. Compared with normal control, mean LV ejection fraction (EF) was significantly reduced in MS (65 (14%)%) compared with 73 (76%), p = 0.003). One hundred and sixteen patients had reduced EF (<64%). The mean left ventricular end diastolic volume index (LVEDVI) and left ventricular end systolic volume index (LVESVI) of the study and control of patients were 95 (49) ml/m², 112 (26)

Proceedings of the British Cardiac Society
From peripheral to coronary arteries: experience with percutaneous laser thermal angioplasty

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The femoral artery—a testing ground for angioplasty advances applicable to coronary artery disease—is a larger calibre, straighter, motionless vessel with less spasm potential and a shorter percutaneous route to the lesion than the coronary vessels. Peripheral artery laser probe (LP) angioplasty is performed with an inflexible 2.0 mm oval metal tipped fibre enabling comparatively forceful coaxial advancement. We used the LP to cross eight of 10 peripheral occlusions (range 1-11 cm) but in none was the LP channel alone of adequate calibre before balloon dilatation. More flexible 1.3-1.7 mm tip "over the wire" LPs were developed to gain access to the coronary tree. In two patients with circumflex artery stenoses we found the loss in axial force was critical: in neither could the lesion be crossed during energy delivery, with a perforation in one (which sealed spontaneously); in the other exchange to a balloon led to a successful dilatation. Our cadaver studies showed that a vessel:probe diameter ratio of 2:0:1 was optimal to minimise lateral wall damage. In a patient with a 95% LAD stenosis a probe with vessel:probe ratio of 1:7:1 was able to cross the lesion "cold" but intractable spasm occurred after energy was delivered to debulk the lesion.

Coronary LP angioplasty is likely to find its most useful role in total occlusions by creating the initial "break through" channel while simultaneously avoiding contact with normal endothelium and lateral heat transmission to the medial coat. New LPs maintaining axial strength with optimal tip size and combined with a balloon are to be tested next in short segment coronary occlusions both peroperatively and during percutaneous angioplasty.

Prospective study of the frequency of phrenic nerve paralysis after cardiac surgery in children

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In retrospective studies damage to the phrenic nerve during cardiac surgery has been reported in 1.3-1.6% of cases. Diaphragmatic paralysis can be an important cause of postoperative morbidity, especially in infants, because of its essential role in respiration. This prospective study was undertaken to determine the frequency of phrenic nerve paralysis after cardiac surgery in children. Sixty patients undergoing palliative or corrective surgery for congenital heart disease have been studied pre- and postoperatively. Their ages ranged from one day to 14.5 years (median 17 months). The phrenic nerve was electrically stimulated percutaneously at the posterior border of the sternomastoid muscle at the upper edge of the thyroid cartilage. The diaphragmatic muscle action potential was detected by surface electrodes at the seventh intercostal space at the anterolateral aspect of the chest wall. Thirty five patients had median sternotomies, 19 left lateral thoracotomies, four right lateral thoracotomies, and two bilateral thoracotomies. Four patients (6.6%) had phrenic nerve palsies, all on the left side. Two of these had their initial operations at 2 days of age and subsequently required surgical plication of the diaphragm for episodes of respiratory distress.

Early detection of phrenic nerve injury helps plan postoperative management of the respiratory system.

Transvenous His bundle ablation: short term and long term (6-36 months) results in 15 patients. Can success be predicted?

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Fifteen patients with drug resistant supraventricular tachycardia (SVT) exhibiting a satisfactory unipolar His potential recording underwent transvenous electrical ablation of the bundle of His to achieve com-
plete heart block (CHB). Patients received either two consecutive unipolar shocks (group 1) or a single unipolar shock (group 2) from a defibrillator (350 J), or from a "National Heart Hospital" ablator (35 J) through the distal pole of a bipolar 6F USCI electrode. In group 1 (seven patients) acute CHB was achieved in all patients, with a wide QRS escape in five and a narrow QRS escape in two. One of the narrow QRS escape resumed atrioventricular (AV) conduction in right bundle branch block (RBBB) 24 hours later with the return of the tachycardia, but this was controlled by drugs. The other together with six in wide QRS escape, continued in CHB during the long term follow up. In group 2 (n = 8) acute CHB was achieved in five patients, with a wide QRS escape in two, and a narrow QRS escape in three. The remaining three resulted in RBBB with patent AV conduction. The two in wide QRS escape have remained in CHB during long term follow up. Those with narrow QRS escape resumed AV conduction in RBBB within 24 hours of the procedure. In all five unsuccessful patients tachycardia returned that was poorly controlled on drugs. Repeat attempt by single shock in three resulted in acute CHB with a wide QRS escape in one and a narrow QRS escape in two, both resuming AV conduction in RBBB. One with wide QRS escape remains in CHB 20 months later. In the two repeat unsuccessful cases, one required a third attempt by double shock technique to achieve CHB, and the other developed CHB spontaneously three months later.

We believe that by the conventional technique, successful electrical ablation of the His bundle generally requires high cumulative energy. Relief from tachycardia is usually not obtained by partial conduction damage. Recording of a satisfactory His bundle potential does not always guarantee success. A wide QRS escape rhythm seems to be a positive predictor of a successful ablation.

Are Doppler diastolic indices associated with functional capacity in patients with hypertrophic cardiomyopathy?

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Diastolic flow velocity waveforms obtained with pulsed wave Doppler have been used to assess left ventricular diastolic performance in patients with hypertrophic cardiomyopathy (HCM). The association between Doppler derived indices with the patient's symptomatic state has not previously been ascertained. Complete echo Doppler studies and exercise testing with measurement of oxygen consumption (VO₂) and anaerobic threshold were performed in 72 consecutive patients with HCM and sinus rhythm. Thirty six normal individuals also had echo Doppler studies for comparison. Early (E) and late (A) mitral peak velocities (cm/second), E wave duration (DF) (msec), and E wave deceleration (EF) (cm/second²) were measured. Patients with HCM differed from normals in every Doppler index: E = 55 (16) v 62 (13), (p < 0.03), A = 46 (21) v 39 (10), (p < 0.04), DF = 300 (9) v 231 (38), (p < 0.001), and EF = 283 (178) v 457 (127) (p < 0.001).

Doppler echocardiographic indices identified and defined the abnormalities in left ventricular filling in patients with HCM. Although these indices measured at rest did not correspond to the patients' functional state, they were a strong indicator of functional capacity assessed by VO₂ and anaerobic threshold.

Evidence for increased tissue type plasminogen activator inhibitor complexes in atherosclerotic patients

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Impaired endogenous fibrinolysis in blood may contribute to the development of, or be a consequence of, atherosclerosis (AS). Because of the known circadian fluctuation of fibrinolytic components in blood, we took three hourly venous samples over 24 hours in six patients (mean age 65 years) with angiographically documented diffuse AS and in six normal subjects (N, mean age 28 years) and measured plasma concentrations of tissue type plasminogen activator (t-PA) antigen (Ag) by ELISA (ng/ml) and of t-PA activity by parabolic rate chromogenic assay (mIU/ml). Specific t-PA activity was calculated as the activity:Ag ratio (mIU/ng).
In both AS patients and normal subjects t-PA Ag showed a sinusoidal circadian variation (peak v nadir: p < 0.01) with similar phase (peak: nine hours, nadir: 21 hours) and similar amplitude (3 ng/ml). In AS patients, however, mean (SD) t-PA Ag concentration over 24 hours was raised: 14 (1) v 5.5 (1) ng/ml (AS v N, p < 0.001). Specific t-PA activity also showed sinusoidal circadian variation (p < 0.05) in both AS patients and normals, with similar phase (peak: 18–21 hours, nadir: 3–6 hours); mean 24 hour concentration was, however, lower in AS v N (11 (14) v 72 (70) mIU/ng, p < 0.05) and the amplitude of change smaller (39 (35) v 190 (129) mIU/ng, p < 0.05).

In conclusion, compared with normals, patients with AS have a similar circadian pattern of t-PA Ag and specific t-PA activity in plasma; in AS patients, however, t-PA Ag concentration is increased by 150% and specific activity decreased by 85%, suggesting increased formation of circulating t-PA inhibitor complexes. The comparative role of age compared with AS merits further investigation.

**Indium-111 labelled monoclonal antimonyosin antibody for the detection of myocardial necrosis in subendocardial infarction and unstable angina**

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The distinction between subendocardial infarction and unstable angina is a common clinical problem. We describe the use of indium-111 labelled monoclonal antimonyosin antibody (AMA) antigen binding fragment to detect and localise acute myocardial necrosis in patients with subendocardial infarction (n = 7) and unstable angina (n = 7). Subendocardial infarction was defined as chest pain with no Q waves, persistent ST and T wave changes, and a rise in creatine kinase to more than twice the upper limit of normal. The criteria for unstable angina were transient ST/T wave changes in the presence of chest pain, with normal cardiac enzymes. AMA was injected intravenously within 48 hours of the onset of chest pain. Images were acquired at 24 and 48 hours in three projections and graded in intensity (0–4) by two independent observers. All patients with subendocardial infarction had positive AMA images (grade 2, n = 3; grade 4, n = 4) that were diffuse and poorly localised. Five of the patients with unstable angina, however, also had positive images (grade 2, n = 3; grade 3, n = 2). The remaining two were negative (grades 0 and 1). The positive images in unstable angina tended to be diffuse and corresponded poorly with the site of transient ST and T wave changes. In all but three patients (subendocardial infarction, n = 2; unstable angina, n = 1) there was no difference between the 24 and 48 hour AMA images.

This study indicates that diffuse myocardial necrosis may occur in patients with “unstable angina” as well as those with “subendocardial infarction” despite an absence of an enzyme rise in unstable angina. The image patterns are similar to those previously reported with technetium labelled pyrophosphate.

**Improvement of subclinical left ventricular dysfunction in obese subjects after weight loss**

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Radionuclide ventriculography was performed at rest (R) and during exercise (Ex) in 34 obese subjects weighing +30% their ideal body weight (Wt) before and after weight loss (four week supervised diet of 330 kcal in hospital). Fourteen were hypertensive (Ht) with blood pressure (BP) >160/95 mm Hg, nine had coronary artery disease (CAD), and 11 were asymptomatic normotensive (Nt) subjects. After dieting the mean (SD) body weight in the whole group fell by 9.6 (3.5) kg (p < 0.0001), and the BP fell from 151 (21)/92 (14) mm Hg to 134 (18)/82 (11) mm Hg, p < 0.0001, respectively. In the subgroups, there were similar falls in both systolic and diastolic BP. Weight loss produced an overall reduction in mean resting left ventricular ejection fraction (LVEF) from 61 (10)% to 56 (7)% (p < 0.005). Response to Ex showed a fall in LVEF by 2 (7)% (NS), which reversed after weight loss increasing by 5 (4)% (p < 0.0001). The Ht group showed the most pronounced changes, where weight loss produced a fall in mean (R) LVEF from 66 (8)% to 57 (6)% (p < 0.0005), and a fall in Ex LVEF of 6 (9)% (NS) was reversed to become an increase of 7 (4)% (p < 0.002). The CAD showed similar but insignificant changes. The Nt group also showed a fall in (R) LVEF after dieting, but the Ex LVEF increased by less than 5% from its resting value which after weight loss showed a significant improvement of 6 (2)% (p < 0.001).

These data suggest the presence of subclinical left ventricular dysfunction in obesity which may be corrected by weight loss and a fall in BP.
Cardioscint: a new system for the continuous evaluation of left ventricular function

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A new portable bedside monitoring system (Cardioscint) has been developed for continuous monitoring of left ventricular (LV) function using a miniature CsI/photodiode scintillation detector (45 × 40 mm) and an Olivetti M24 computer (CSD). Special purpose modules inside the computer perform signal processing, and gated or beat to beat LV activity curves are continuously displayed together with an averaged ECG from which ST segment values are obtained. At the end of each acquisition the ejection fraction (EF), relative volumes, diastolic filling parameters, and ST value are displayed, and can be trended or stored on disc, or both. Validation was performed on 15 patients undergoing gamma camera (G) radionuclide ventriculography within 20 minutes and 6 hours later. The mean (SD) difference between CSID and G was 5.6 (7.9)% (early) and 4.5 (7.3)% (late), (range of EF: 20%-78%).

Thus the portable probe system can accurately measure LV ejection fraction, and the accuracy was maintained after 6 hours. The small size and low cost of this system make it suitable for long term monitoring and the rapid response time allows accurate LV function assessment following acute interventions.

Role of signal averaged electrocardiograms compared with myocardial biopsy specimens in ventricular tachycardia associated with no obvious cardiac abnormality

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Primary myocardial abnormalities in patients with ventricular tachycardia (VT) suggest adverse prognosis. To assess the value of signal averaged electrocardiograms (ECG) in predicting abnormal endomyocardial biopsies, 38 patients (mean age 42, range 15-70 yrs), with VT were investigated. All had normal QT interval, and coronary angiography, and no regional wall motion abnormalities. High gain signal averaged ECGs were performed using Simon's technique. Late potentials were defined as present if the duration of the filtered QRS was greater than 110 ms and the root mean square of voltages in the last 40 ms was less than 25 µV. Right and left ventricular biopsy specimens (average 5-7/patient) were reviewed on light microscopy by an independent pathologist. Fibrosis in the specimens was quantified by a point counting method and compared with a control group of myocardial biopsy specimens (n = 24) from subjects who had died accidentally and who had normal hearts at necropsy. In these the proportion of collagen to muscle was <10%; 15 (40%) patients had abnormal biopsy specimens, six of these had late potentials that were seen in only one of the patients with normal biopsy specimens (p < 0.001). In all patients with late potentials and abnormal biopsy specimens the relative amount of collagen was >15% and the biopsy specimens showed moderate interstitial fibrosis. In patients with abnormal biopsy specimens and no late potentials, collagen was <15% and the biopsy specimens showed mild sub-endocardial fibrosis or fatty infiltration. Other findings in the abnormal biopsy specimens were cellular hypertrophy and atrophy. No patients had changes to suggest myocarditis. Late potentials were 96% specific and 40% sensitive for abnormal biopsy specimens with positive predictive accuracy of 87%.

Thus in patients with VT and clinically "normal hearts", the pretnce of late potentials was suggestive of abnormal endomyocardial biopsy specimens with a moderate increase in fibrosis.

Normal and stenotic aortic valve area measurement: comparison of an in vitro physiological method with Doppler echocardiography


Aortic valve (AV) area (A) estimation in patients with aortic stenosis using Doppler echocardiography (DOP-ECHO) has been found to correlate well with invasive Gorlin AVA estimations. Neither method, however, has been well validated with operative or necropsy AVA measurements which in themselves are not physiological. The Gorlin formula is also known to over or under estimate AVA at high and low cardiac outputs, respectively. In an attempt to measure accurately the physiological AVA in intact valves we mounted such AVs in a pulsatile flow rig. Saline at 37°C was delivered at flow rates from 1-5.4 l/minute with ejection periods of 25-45% and a heart rate of 70/minute. AV motion was videotaped with a magnifying lens to permit derivation of leaflet infor-
Demonstration of the fatigue and slow recovery in the human atrioventricular node

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Atrioventricular nodal response to an abrupt increase in the atrial rate has a complex pattern and varies according to the stimulation protocols. The "fatigue phenomenon" already described in the rabbit atrioventricular node is defined as a time dependent prolongation in atrioventricular nodal conduction time during fast pacing rate over several minutes. The characteristics of atrioventricular nodal fatigue and recovery in man were less defined. In 12 patients, a beat to beat analysis of atrioventricular nodal conduction (AH interval) was done during atrial pacing over periods of 30 seconds to two minutes. Computer assisted analysis of digitised signals (sampling rate: 2 KHz) from tape recordings was used. The basic cycle lengths were 800 or 700 ms. Fatigue was determined during fast atrial pacing cycle lengths of 500 or 400 ms and the recovery was determined during return to basic cycle length. In all cases, an instantaneous prolongation in the atrioventricular nodal conduction was observed at the onset of the fast rate which stabilised with some degree of beat to beat variation in atrioventricular node conduction (5–20 ms). Upon return to the basic drive (recovery), a beat to beat decrease in atrioventricular nodal conduction was observed for several beats depending on the cycle length and duration of the fast rate.

These observations suggest that fatigue and slow recovery do occur in intact human atrioventricular node and can be manifested only with fast pacing rate for long periods. These findings provide insights on the mechanisms of cycle length alteration at the onset of supraventricular tachycardia.

Magnetic resonance imaging of cardiac sarcoidosis

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Cardiac impairment by sarcoidosis is often occult but its detection is important because it most often presents as sudden death. Diagnosis is usually based upon cardiac abnormality in the presence of known sarcoidosis, but even myocardial biopsy can be unreliable. We have studied 13 patients with cardiac sarcoidosis by magnetic resonance imaging, and shown myocardial, epicardial, and pericardial abnormalities in 10. In one patient the diagnosis was established by cardiac biopsy, and in the others the diagnosis was made from proved impairment of another organ with evidence of rhythm disturbance, conduction disturbance, or heart failure. The Kveim test was positive in nine. Spin echo (TE 26 ms, 40 ms, and 120 ms), field echo (cine imaging), and inversion recovery (TI 100 ms for fat suppression) sequences were used, although not every sequence was used in each patient. The images were scored by two observers independently for areas of high signal within the myocardium, and differences were resolved by consensus. Areas of high signal were seen in seven patients, most commonly in the proximal septum and in the posterolateral wall. Epicardial thickening with high signal was seen in two, and the fat suppression sequence was used to show that the high signal was not due to epicardial fat. Using the longer spin echo sequence, the abnormalities were more obvious. Pericardial thickening was seen in three with underlying effusion in two. The cine field echo sequence provided a distinction between pericardium and effusion, which was otherwise difficult. Abnormal left ventricular wall thickness and wall motion were seen in four. No abnormality was detected in three, all of whom had rhythm or conduction disturbance without evidence of active disease.
Magnetic resonance imaging shows myocardial, epicardial, and pericardial abnormalities in cardiac sarcoidosis. Further studies are warranted to compare its sensitivity with other techniques (such as echocardiography and thallium scintigraphy) and to investigate the specificity of abnormalities.

Non-invasive measurement of phosphorus metabolism in the human heart during exercise by phosphorus-31 magnetic resonance spectroscopy

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The ratio of phosphocreatine (PCr) to adenosine triphosphate (ATP) is an important indicator of myocardial energy status and can be determined noninvasively by phosphorus-31 magnetic resonance spectroscopy (31P MRS). Because many indices of cardiac function display abnormalities only during exercise, we developed an exercise protocol for stressing the heart in the confined space of the whole body magnet while collecting 31P MR spectra. We compared the physiological responses of this prone exercise to those during the Bruce treadmill test and then documented the myocardial PCr:ATP ratio at rest and during steady state exercise. Six healthy normal subjects (age 25 (3) years) lay prone with the heart positioned over a double surface coil. 31P MR spectra were acquired at rest over 30 minutes. They then exercised by lifting 5 kg weights attached to both feet by cables at a rate of 50/minute/leg for 30 minutes. Spectra were collected during exercise. At rest the mean (SD) heart rate/minute was 59 (10), SBP 114 (6) mm Hg, DBP 73 (3) mm Hg, VO2 3.6 (0.4) (ml/kg/minute). The responses during the prone exercise compared with Bruce stage I were: HR 86 (19) v 91 (13); SBP 146 (16) v 133 (17), DBP 100 (6) v 73 (6), rate pressure product 12670 (3580) v 12290 (3028), VO2 9.2 (2) v 14.5 (2.9). Technically satisfactory spectra were obtained in five subjects. The PCr:ATP was 1.56 (0.2) at rest v 1.58 (0.14) during exercise.

There is no difference in myocardial PCr:ATP between rest and exercise in normal young men using this exercise protocol. Thus in cardiac muscle increased metabolic demand at this degree of exercise is met by increased oxygen consumption without the need to break down PCr.

Association between blood pressure, left ventricular mass and free cytosolic calcium in primary hyperparathyroidism and essential hypertension

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Primary hyperparathyroidism is associated with left ventricular hypertrophy which is said to be out of proportion to mild hypertension. It has been suggested that parathyroid hormone could act as a growth factor but more recently there has been an interest in intracellular free calcium, which is known to affect vascular tone and cell growth. We therefore measured blood pressure, left ventricular mass, and platelet cytosolic free calcium using Quin 2 in 23 patients with primary hyperparathyroidism, 20 normal controls, and 22 patients with essential hypertension. Blood pressure (mean (SEM)) in the hyperparathyroid group was higher than in controls (141 (5)/85 (2) mm Hg v 123 (2)/79 (2) mm Hg, p < 0.001), but lower than in hypertensive patients (175 (5)/108 (2) mm Hg, p < 0.001). Median left ventricular mass index was 113 g/m2 in the hyperparathyroid group v 87 g/m2 in controls (CI -40-9 to -7, p = 0.006) and 143 g/m2 in hypertensive subjects (CI -38 to 12, p = 0.31). Median [Ca2+]i in the hyperparathyroid group was slightly lower than in controls (82 nM v 88 nM, CI -3.2 to 18.6, p = 0.15) and significantly lower than in hypertensive subjects (101 nM, CI -27.3 to -4.8, p = 0.005). There was no correlation between [Ca2+]i and LV mass but there was a negative correlation between [Ca2+]i and diastolic blood pressure in hyperparathyroidism (r = 0.46, p < 0.05).

In primary hyperparathyroidism left ventricular mass is consistent with mildly raised blood pressure. In contrast to essential hypertension, patients with high blood pressure and hyperparathyroidism show low concentrations of cytosolic free calcium.

Electrophysiological changes in an animal model of experimental chronic cardiac failure

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Many patients with cardiac failure die suddenly, presumably from ventricular arrhythmias. We have studied the electrophysiological changes in New Zealand white rabbits with experimental cardiac failure.
cardiac failure induced by the administration of adriamycin 1mg/kg intravenously twice weekly for eight weeks, and saline injected controls. Recordings of the Stimulus-R interval (Stim-T) of the paced evoked response, an index of ventricular repolarisation time, and effective refractory period (ERP) were made in conscious animals using previously implanted bipolar permanent pacing electrodes. Progressive shortening of Stim-T and ERP occurred in the adriamycin treated animals with a reduction of 12% and 14% in repolarisation and refractoriness, respectively, by week 10. No significant changes in Stim-T or ERP were found in controls. Papillary muscles from adriamycin treated animals in vitro also showed significant shortening of intracellular action potential duration and ERP compared with controls. The changes noted in the adriamycin treated animals were not attributable to a direct, acute electrophysiological effect of adriamycin nor to differences in plasma electrolytes, noradrenaline, or renin.

Shortening of ventricular repolarisation and refractory period in heart failure may represent an intrinsic mechanism predisposing to arrhythmias and sudden death.

**Role of wall stress in the pathogenesis of ventricular arrhythmia**

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This experimental study in 84 isolated working hearts investigated the role played by wall stress in the pathogenesis of ventricular arrhythmia and was followed by a clinical study of 58 untreated hypertensive patients. The results showed that raised amounts of wall stress were associated with an increased prevalence of ventricular arrhythmia. Acute changes in wall stress (increase or decrease) were arrhythmogenic; acute increases, however, produced the most serious arrhythmias. Cation depletion (potassium and magnesium) increased the frequency and severity of this arrhythmia. Hypertrophy protected hearts from the development of arrhythmia but also sensitised the myocardium to cation depletion. In hypertensive patients there were some interesting correlations with the experimental findings. They had a higher prevalence of arrhythmia than the normal controls, showed the protective effect of left ventricular hypertrophy, and also had a significant adverse interaction between left ventricular hypertrophy and potassium depletion.

There are several important implications from these findings. Firstly, raised wall stress, a common occurrence in cardiac disease, may be arrhythmogenic. Secondly, left ventricular hypertrophy exerts a protective effect against this arrhythmia. Lastly, the deleterious effect of left ventricular hypertrophy is probably due to sensitisation of the myocardium by diuretic induced, cation depletion. These results may explain some mechanisms underlying sudden death in hypertension.

**Cardiac sympathetic neural activity in ischaemic heart disease**

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Sympathetic neural activity has a profound effect on the susceptibility of ischaemic myocardium to serious ventricular arrhythmias yet has not been systematically studied in man. Radiotracer noradrenaline (NA) kinetics were used to evaluate the effect of myocardial ischaemia on cardiac and overall sympathetic activity in man. Atrial pacing in 15 patients with ischaemic heart disease (IHD) caused angina in 13, but neither cardiac (C) nor whole body (WB) NA spillover were increased. (C: 16 (5) ng/minute before, and 20 (7) ng/minute during pacing. WB: 306 (35) ng/minute before, and 359 (45) ng/minute during pacing (p = NS)). Subjects with recent unstable ischaemic symptoms (n = 10), had, however, greatly increased basal cardiac NA spillover (24 (7) ng/minute) compared with subjects (n = 10) with stable angina (6 (1) ng/minute, p < 0.001). Low level supine bicycle exercise in 29 patients with IHD and in six controls without serious cardiac disease resulted in an increase in cardiac and whole body NA spillover in all. Patients with IHD who did not develop myocardial ischaemia on exercise and normal controls had similar increases in cardiac NA spillover (6 (1) ng/minute to 19 (2) ng/minute and 5 (1) ng/minute to 18 (2) ng/minute, respectively). Patients developing myocardial ischaemia (n = 20) had similar basal cardiac NA spillover (8 (1) ng/minute) but exercise values were higher (30 (5) ng/minute, p < 0.05). In two patients developing severe and prolonged ischaemia cardiac, NA spillover was further increased 10 minutes after exercise, while in all other subjects it had declined towards the pre-exercise value at this time.

Thus myocardial ischaemia induced by atrial pacing does not increase cardiac sympathetic tone, but myocardial ischaemia enhances the cardiac sympathetic response to exercise and a period of unstable
ischaemic symptoms causes a sustained and specific increase in cardiac sympathetic tone. These findings may in part explain the adverse prognosis and increased likelihood of arrhythmias in patients with low level exercise induced ischaemia and unstable ischaemic syndromes.

Value of colour Doppler flow mapping and digital computer analysis in the measurement of mitral regurgitation in vitro

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Mitral regurgitation (REG) was simulated in a physiological pulsatile flow model with REG orifices of 0.2, 0.6, or 2.0 mm², varying haemodynamic conditions of REG volumes, and REG pressure drop. Colour Doppler flow mapping (CDFM) was performed in velocity (VEL) variance and power modes at constant gain and 4 kHz pulse repetition frequency. For each valve, colour jet area correlated linearly with REG pressure \( r = 0.98, 0.2 \text{ mm}^2 \); \( r = 0.97, 0.6 \text{ mm}^2 \); \( r = 0.98, 2.0 \text{ mm}^2 \) but a tenfold increase in REG volume (7.2 ml/minute at 0.2 mm² v 78.4 ml/minute at 2.0 mm²) produced only a twofold increase in colour area (1.8 cm² v 3.6 cm²). Digital computer analysis with multiple alias unwrapping also provided actual VEL assignments for the blue, red, and green components of each colour pixel in the REG jet. Total jet energy estimated by the summation of all pixel VELs \((A \times V)\) and the summation of the square of each pixel VEL \((A \times V^2)\) correlated better with REG pressure \( r = 0.72, A \times V; r = 0.84, A \times V^2)\) than with REG volume \( r = 0.56\) and \( r = 0.50\) respectively for all REG valves combined. Summation of the digital computer colour intensities of the power mode correlated well with REG volume \( r = 0.80\) and not with REG pressure \( r = 0.42\).

Spatial distribution of REG jets imaged by CDFM is associated with their kinetic energy and therefore with REG pressure, and results should be interpreted with knowledge of the patient’s haemodynamic function. Summation of power mode information may improve quantification of valve REG.

Proceedings of the British Cardiac Society

Is there any indication to perform ambulatory ST segment monitoring in patients with coronary disease and a negative exercise test?

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We investigated 264 patients (222 men) aged 30–77 years with proved coronary artery disease (CAD) (67 single, 81 two, 116 three vessel disease) to establish the association between the frequency and duration of silent ischaemia and a negative exercise test (Ex test). Patients underwent treadmill exercise testing (Ex test) \((n = 255)\) and 48 hours of ambulatory ST segment monitoring \((\text{total} = 11389 \text{ hours}\); 114 patients were receiving standard antianginal drugs, and 150 were off all treatment. Of the 255 exercise tests performed, 85 (33%) were negative for ischaemia. During the 11389 hours of ST segment monitoring a total of 600 episodes of silent ischaemia \(> 1 \text{ mm ST depression for} > 1 \text{ minute}\) were recorded. Those with a negative Ex test had a mean of 0·41 episodes of silent ischaemia, of mean duration 6·1 minutes/24 hours \((\text{total} 51 \text{ episodes,} 8.5\%)\). Those with a positive Ex test had a mean of 1·93 episodes of mean duration 42 minutes/24 hours \((\text{total} 549 \text{ episodes,} 91.5\%)\) \( (p < 0.001 \text{ in both cases}) \). The association between silent ischaemia and a positive Ex test was highly significant in those patients off and on therapy. Only one patient \((1.1\%)\) with a negative Ex test had > 4 episodes of silent ischaemia/24 hours of monitoring. This patient had documented coronary spasm.

In conclusion, silent ischaemia is rare in patients with CAD and a negative Ex test, whether on or off drugs. There is little indication to perform ambulatory ST segment monitoring in these patients, except when the history is suggestive of coronary spasm, or when patients cannot undergo a meaningful Ex test.

High density lipoprotein cholesterol, triglycerides, and ischaemic heart disease: a reappraisal

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Previous British Regional Heart Study (BRHS) findings suggested that high density lipoprotein (HDL) cholesterol was not a major risk factor for IHD events. A recent overview of four American prospective studies and the BRHS concluded that all
showed a consistent inverse relationship between HDL cholesterol and IHD events. Current analysis of BRHS data concerns 417 major IHD events (fatal and non-fatal) in 7415 middle aged men followed up for 7-5 years. The relative odds of an event occurring in the highest fifth of the total cholesterol and HDL cholesterol distributions compared with the lowest fifth were 3·6 and 2·4, respectively. Adjusted for each other, and for age, smoking, systolic blood pressure, and body mass index, the relative odds were reduced to 3·5 and 2·0, respectively. Non-fasting triglycerides were measured in 5675 men with 324 IHD events in 7·5 years follow up. The relative odds of an event for the highest fifth of the triglyceride distribution was the lowest fifth was 2·3, and after adjustment for smoking, systolic blood pressure, body mass index, and total cholesterol was 1·3. With HDL cholesterol included in the adjustment, the relative odds fell to 0·98.

Total cholesterol remains the most important blood lipid risk factor. HDL cholesterol is relevant but of less importance. Triglycerides do not appear to have prognostic importance once other risk factors have been taken into account.

Is the site of myocardial ischaemia reproducible?

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Angina may be produced by ischaemia occurring at variable sites within the myocardium, which may be influenced by the type of stress used to induce ischaemia. The extent of this variability has been studied in 17 male patients with documented coronary artery disease and positive exercise tolerance tests in whom drug treatment had been withdrawn. In 12 patients gated thallium imaging was performed after symptom limited maximal exercise testing on both an erect and a supine bicycle ergometer. In five patients, scans were performed following two symptom limited maximal erect exercise tests four weeks apart, and a third test performed to the same workload as the initial test. Scans were reported visually semiquantitatively by two observers from the gated image for three left ventricular segments in the anterior and 45° left anterior oblique projection (LAO), and four segments in the 75° LAO projection. Perfusion defects are thought to reflect the region of maximal ischaemia. The mean difference between observers was 2(2)%.

Effect of exercise training on myocardial perfusion in angina pectoris

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Forty male patients with stable angina pectoris and no previous myocardial infarction were randomised to exercise training and control groups. The training group carried out daily exercise using the Royal Canadian Airforce Schedule for Physical Fitness. Myocardial perfusion was assessed initially and at one year by thallium 201 perfusion scintigraphy after treadmill exercise. Planar thallium images were recorded in the anterior 45° LAO and 65° LAO positions and analysed by circumferential profile analysis. The profiles obtained were compared with a set of profiles from patients with normal coronary arteries. The number of abnormal segments in each view was recorded as a percentage of total circumference and the area of abnormality was also recorded as a percentage of normal curve area. The abnormalities obtained in the three views were in addition summed to produce total scores of percentage circumference and percentage area of abnormality. The training group achieved an 11·4% increase in double product on the treadmill (p < 0·05). In spite of this increase in cardiac work the total percentage circumference abnormality decreased from 26 (15)% to 18 (12)%, (p < 0·01). The total area abnormality decreased from 4·3 (4)% to 2·6 (2·6)%, (p < 0·05).

Analysis of individual views showed the greatest improvements in this anterior view and the 65° LAO view with lesser degrees of improvement in the 45° LAO views. This may reflect a tendency for collaterals to develop most readily between the LAD and right coronary arteries. No significant changes were found in the control group.
Clinical experience of coronary angiography by the femoral route using a multipurpose catheter (Sones technique from the leg)

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Manipulation of a multipurpose Schoonmaker catheter permits left ventricular, right, and left coronary angiography by the femoral route. We report our experience in 1137 cases over four years with five operators. Catheterisation was completed with a Schoonmaker catheter alone in 62% (710 of 1137) of cases. A second catheter was used in 28% (n = 313) and a third in 10% (n = 115) giving a mean of 1.5 catheters per case. The Schoonmaker was successful for left ventricular angiography in 97%, right coronary angiography in 83%, and left coronary angiography in 73%. The screening time was 7:34 (5:2) minutes per case, which was twice that for studies with preshaped catheters (3:75 (1:3) minutes). A learning curve was evident for the four operators without previous experience. Comparison of their first and last 50 cases showed an increase in cases completed with a single catheter from 48% to 67%. Total catheters used for the first and last 200 cases fell from 351 to 279 compared to 600 needed if Judkins catheters had been used. The mean screening time also fell from 8:3 (4:8) to 6:1 (4:0) minutes.

The change from preshaped catheters to a single catheter system has been achieved with a saving of £17,300 in this series. More substantial gains are achieved after completing the learning curve with a 54% reduction in catheters used, saving approximately £16 per case. The increase in screening time fell with experience and represents a small increase in radiation exposure.

Intracoronary injections of salbutamol show the presence of β2 adrenoceptor mediated activity in the human heart

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In the human heart β2 adrenoceptors (B2ADR) constitute 20–60% of total β adrenoceptor number, a greater proportion than any other mammal. In vitro, human cardiac muscle strips respond to stimulation of B2ADR by increasing contractile force. The demonstration of a functional role for these receptors in intact man is confounded by the peripheral vasodilation produced by B2ADR stimulation that produces secondary cardiac effects by reflex vagal withdrawal and sympathetic activation. We gave salbutamol, a selective β2 agonist, to three groups of six patients. The drug was injected into the right coronary artery in order to stimulate the sinoatrial node directly while avoiding peripheral vasodilator actions. Incremental doses of salbutamol were given until they caused a sinus tachycardia. As a control the same doses were injected into the aortic root where they caused no change in heart rate. In the pilot group of six patients the mean dose required to increase heart rate by 30 beats per minute (IHR30) was 2.6 μg. To confirm that salbutamol was acting via B2ADR a further group of 12 patients were studied. They were randomised to receive either practolol 8 mg intravenously (β1 selective blockade) or propranolol 4 mg intravenously (β1, and β2 blockade), 15 minutes before administration of salbutamol. These doses have equal β1 blocking activity as defined by equal inhibition of exercise tachycardia. After practolol had been given the mean IHR30 dose was 2.1 μg whereas after propranolol the mean IHR30 dose was 64 μg (practolol v propranolol p < 0.001).

This study shows that direct cardiac β2 adrenoceptor stimulation in intact man has a positive chronotropic effect.

Intimal proliferation in an organ culture of human saphenous vein

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Intimal proliferation is a principal cause of late vein graft occlusion. Its aetiology is uncertain, however, partly because its progression is difficult to study in humans. To overcome this we attempted to produce intimal proliferation in an organ culture of human saphenous vein. Vein (1 cm length) was pinned out intimal surface uppermost and maintained at 37°C in 6 ml of RPMI 1640 medium containing 30% donor calf serum, 0.8 mM glutamine, 2.5 μg/ml of gentamicin, 100 μg/ml of penicillin, and 100 μg/ml of streptomycin. Medial viability was assessed by ATP concentration (nmol/g wet weight), endothelial integrity by scanning electron microscopy, and cell
proliferation by preincubation for 24 h with 6 μCi of [6-3H]thymidine followed by fixation, paraffin embedding, transverse sectioning, and autoradiography. Freshly isolated vein had a mean (SEM) ATP concentration of 300 (30) (n = 16), had about 80% of endothelial coverage, and had few dividing cells in any cell layer. Vein surgically prepared for coronary bypass grafting had an ATP concentration of 130 (20) (n = 17, p < 0.001), < 20% of endothelial coverage, and few dividing cells in any cell layer. Freshly isolated vein cultured for two weeks also had an unchanged ATP concentration of 250 (30) (n = 8), it was covered by enlarged endothelial cells, and had many proliferating cells that were confined to the endothelial and immediately subendothelial layers. Surgically prepared vein cultured for two weeks also had an unchanged ATP concentration of 180 (30) (n = 9), which was still significantly lower than freshly-isolated vein (p < 0.01), it was < 20% covered by endothelial cells, and had a similar number and pattern of proliferating cells as cultured freshly isolated vein.

We have succeeded in showing intimal proliferation in human saphenous vein in organ culture. This was not influenced by surgical preparative damage despite the observation that neither the media nor the endotherium recovered during culture. This system provides, for the first time, an experimental model in which the progression of intimal proliferation can be studied in human tissue.

Do tight coronary stenoses impede access of cold cardioplegia: protection or not?

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The two major elements in the use of a cardioplegic technique are the high potassium concentration that is used to arrest the heart, and cooling that protects the myocardium during the period of arrest. The cardioplegic solution infused into the coronary arteries through the aortic root may not reach myocardium beyond tight stenoses. The question arises as to whether these areas are protected by hypokalaemic arrest. We have recorded a monophasic action potential (MAP) from the epicardium in an area supplied by either the left anterior descending or circumflex coronary arteries during infusion of cold cardioplegic solution during routine coronary artery surgery as an index of the arrival of cardioplegic solution to the area of interest. The net effect of cardioplegia on action potential duration is variable due to the opposite influences of potassium (shortening) and cooling (lengthening) on repolarisation time. The effect on diastolic potential is a rapid and substantial reduction, however, due primarily to the influence of raised potassium concentration; cooling had no effect. Although measurements of diastolic potential in long term recordings may be problematical, short term recordings over a few beats are acceptable with our electrode design. Fourteen patients were studied during infusion of the cardioplegic solution using either one or two MAP electrodes. Satisfactory recordings (16) were obtained from the left anterior descending territory in 10 and circumflex area in six (both together in three). Recording from an area distal to a tight stenosis with angiographic evidence of antegrade diastolic potential fell by a mean (SD) of 3.9 (2.5) mV, n = 10. Recording from an area distal to a tight stenosis but without evidence of antegrade filling the diastolic potential fell by a mean of 0.2 (0.4) mV, n = 5. In one patient where the recording area was supplied by a non-stenosed circumflex coronary artery infusion of the cardioplegic solution reduced the diastolic potential by 4.5 mV. In three patients in whom simultaneous records were obtained from areas supplied by non-occluded and totally occluded arteries the contrasting changes in diastolic potential were striking. When a change in MAP duration (rate corrected) occurred this was always associated with a pronounced fall in diastolic potential.

Our results show that myocardium distal to tight stenoses may be electrophysiologically uninfluenced by the high potassium cold cardioplegic solution, thereby raising the question as to whether parts of the heart are inadequately protected during the procedure.

Improved patency rates to small vessels with the internal mammary graft

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Ninety five of 330 patients undergoing isolated coronary artery surgery between January 1985 and June 1987 agreed to recatheterisation and exercise electrocardiogram (ECG) one year postoperatively. These 95 patients had received 103 pedicled internal mammary artery (IMA) grafts and 272 saphenous vein grafts (SVG). Of the 330 patients, there were four hospital deaths (1%) and no late deaths. No significant difference was found between the study group (95) and the total group (330) for age (54 ± 57
years, sex (85% v 82% men), severity of disease (14% v 8% left main stem; 86% v 82% 3VD; 12% v 15%, 2VD; or 2% v 1% 1VD) or number of grafts performed (6% v 6%: two grafts; 38% v 35%: three grafts; 41% v 46%; four grafts; 15% v 12%; five grafts). At one year, overall NYHA status was improved with 86% of patients in NYHA Class 0 (preoperatively 82% were in NYHA III or IV), and 92% of patients had negative exercise ECGs: 25% at stage 5, 35% at stage 4, and 32% at stage 3. Overall graft patency was 90% for IMA and 79% for SVG (p = 0.025, \( \chi^2 \) test). The size of vessel grafted had a significant effect on graft patency when SVG was used: 82% for 2-0 mm vessel v 70%, for 1-25 mm (p = 0.05), but no such difference for IMA (100%, for 2-0 mm v 100% for 1-25 mm). Overall graft patency in women was pronouncedly worse than the total group at 54% (57% for IMA and 41% for SVG; p = 0.01) and this was independent of the size of the vessel grafted.

The internal mammary artery provides improved one year patency rates, which unlike saphenous vein are not related to the size of the vessel being grafted.

Late left ventricular function after anatomical correction of anomalous left coronary artery

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Seven children aged 10 weeks–10 years who underwent anatomical correction of anomalous origin of the left coronary artery (LCA) from the pulmonary artery, between 1979 and 1985, have been followed up for a minimum of 3-5 years. Five infants aged 10 weeks–6-5 (mean 4) months presented in severe cardiac failure; two of these patients (aged 4 and 6-5 months) had ECG evidence of extensive anterior myocardial infarction, and one died at operation. Two children aged 5 and 10 years presented with dyspnoea on exertion. At operation the LCA was reimplemented directly into the left coronary sinus of the aorta in all. Mitral annuloplasty was performed in one. With a follow up of 3-5–10 (mean 6-6) years, there have been no late deaths. No child is on drugs and exercise tolerance is normal. Ischaemic changes on the ECG have improved in all except one patient who had preoperative myocardial infarction. Left ventricular function has been monitored by M mode and cross sectional echocardiography, radionuclide and left ventricular angiography in all patients. Abnormalities of anterior wall motion have improved and the left coronary artery is patent on coronary angiography with normal perfusion patterns in all patients. It is concluded that anatomical correction of anomalous left coronary artery can be carried out safely at a young age and offers the best hope for near normal left ventricular function.

Intraoperative cerebral monitoring with a cerebral function analysing monitor predicts neuropsychological outcome after coronary artery surgery

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We have recorded cerebral electrical activity using a cerebral function analysing monitor (CFAM) in 58 patients undergoing elective coronary artery bypass surgery. All patients underwent detailed neuropsychological testing with a series of 10 tests before surgery, and at eight days and eight weeks after operation. Focal neurological deficit, unresolved at the time of hospital discharge, occurred in three patients (70% confidence interval (CI) 2-5% to 10-5%), which resolved in all but one patient by eight weeks (70% CI 0-3% to 6-2%). A deterioration in neuropsychological performance compared with preoperative scores was seen in 57% (70% CI 47% to 63%) at eight days, and 33% (70% CI 26% to 40%) at eight weeks. Abnormalities in CFAM traces were seen in 13 patients (70% CI 17% to 30%). Abnormalities were defined as abrupt changes in the mean CFAM voltage or enlargement in the voltage envelope of greater than 10% accompanied by an increase in low frequency electroencephalographic waveform activity not recovering within a three minute period. Those patients with neuropsychological impairment at eight weeks had an increased incidence of CFAM abnormality (\( \chi^2 = 4-265, p = 0-04 \)), whereas there was no significant association at eight days (\( \chi^2 = 0-002, p = 0-96 \)). The magnitude of neuropsychological impairment at both eight days and eight weeks was significantly greater in those patients with abnormal CFAM records (\( t = 2-29, p = 0-02 \) at 8 days; \( t = 2-09, p = 0-013 \) at eight weeks: unpaired t test). There was no association between CFAM abnormality and focal deficit at the time of hospital discharge (\( \chi^2 = 0-445, p = 0-50 \)).

Abnormalities of CFAM activity are a significant predictor of diffuse neuropsychological impairment following coronary artery bypass surgery but do not predict focal neurological deficit.

Proceedings of the British Cardiac Society
Effect of aprotinin on blood conservation during cardiac surgery

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Because bleeding after cardiopulmonary bypass (CPB) remains a source of morbidity usually requiring blood transfusion, treatment with epsilon-aminocaproic acid and desmopressin to reduce this need has been investigated. The serine proteinase inhibitor aprotinin was studied in a randomised, placebo-controlled, double blind study in 80 patients having primary aortocoronary bypass grafting. Volume replacement in the postbypass period was with nonhaemorrhagic colloids. Bank blood was only given if the haematocrit was below 30%. Blood concentrations of 400 kU/l of aprotinin were obtained at the beginning of CPB. Mean (SD) chest drainage in the 40 patients given aprotinin was significantly less than the control group: 309 (133) ml v 573 (166) ml, p < 0.01. Total haemoglobin loss into the chest drains was also significantly reduced from 37.7 (18.3) g to 12.0 (12.6) g. Bank blood requirements were less in the aprotinin group of patients who received a total of 13 units. The control group received a total of 78 units. Thirty two of the treated patients were not transfused at any stage whereas 35 of 37 controls required blood. Venous haemoglobin concentrations preoperatively, and on the first and the seventh postoperative days did not differ. Template bleeding times were similar preoperatively, 90 minutes postoperatively, the increase seen in the control group was not seen in the aprotinin patients. This drug has also been used in two other groups of patients at much higher risk of bleeding—those having reoperations and those with acute sepsis at the time of surgery. In a randomised trial, 11 of 22 patients having reoperations were given aprotinin. Blood loss postoperatively was reduced from 1509 (388) ml to 286 (48) ml in the treated patients (p < 0.001). Only four treated patients were transfused with a total of five units of bank blood compared with all the untreated patients who were given 41 units. Fifteen patients, all requiring operation for acute infective endocarditis received a slightly higher dose of aprotinin. Five had evidence of intravascular coagulation and two were receiving dialysis. Mean blood loss in this group was 388 ml. Six patients were transfused with a total of 11 units of bank blood. No adverse effects attributable to the drug were seen.

These data show that aprotinin reduces the need for bank blood after cardiac surgery with safety. Patients who are likely to benefit most are those at high risk of postoperative bleeding, those having reoperations, those with infective endocarditis, and those who for religious or other reasons cannot or will not have blood transfusions. Its use in cardiac surgery offers a major contribution to blood conservation.

Contraction excitation feedback: a new approach

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The Valsalva manoeuvre provides a convenient means of manipulating left ventricular volume. These volume changes result in pressure changes associated with the three phases of the Valsalva manoeuvre: (1) an initial increase in left ventricular volume and pressure as blood is squeezed into the heart; (2) a subsequent decrease caused by impairment of venous return, and (3) an increase in pressure and volume when venous return is restored at the end of the manoeuvre. It is well known that the electrical signal before any contraction influences that contraction. The reverse phenomenon whereby ventricular loading conditions influence electrical activation and hence the subsequent beat has recently been shown in man (contraction excitation feedback). Acute elevation of ventricular pressure has been shown to shorten repolarisation and hence refractoriness. We have conducted a study intended primarily to examine the interrelation of acute volume changes. Monophasic action potentials (MAPs) were recorded from the left ventricular endocardium together with femoral artery pressure during the performance of a controlled Valsalva manoeuvre (maintenance of a steady 40 mm Hg for 15 seconds).

Monophasic action potential duration was measured at 90%, repolarisation in two ways: comparing beats with similar preceding cycle lengths, and using a rate correction. Nine patients were studied; two had poor left ventricular function (ejection fraction of less than 20%,); of the other seven patients who were receiving calcium antagonists. The effects of the Valsalva manoeuvre on MAP duration was highly reproducible. In the five patients not receiving calcium blockers and with good left ventricular function during the first few beats (phase 1) mean (SD) systolic pressure increased from 127 (12) to 184 (22) mm Hg and MAP duration increased from 353 (34) to 365 (33) ms; during phase 2 left ventricular pressure fell to 74 (18) mm Hg and MAP duration decreased to 336 (31) ms; during phase 3 systolic pressure increased to 189 (20) mm Hg and MAP duration increased to 440 (43) ms. In the two patients with poor left ventricular function a similar
Development of peptide containing neural and endocrine elements in the human heart

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Several peptides have been identified in mammalian cardiac nerves and myocardial cells and are thought to act as neuromodulators or transmitters, or circulating hormones, respectively. There is a lack of information regarding the expression of these peptides during human development.

We have used indirect immunofluorescence staining techniques and antisera to general neural markers, neuropeptides, and atrial natriuretic peptide (ANP) to examine neural and endocrine elements in fetal hearts (n = 25, 7–24 weeks' gestation) obtained after spontaneous or legal abortion. Ganglion cells and nerves were present in the atrial subepicardium from seven weeks' gestation, concentrated around the sinus node and atrioventricular junction. Ventricular innervation became apparent at about 12–14 weeks forming a prominent epicardial perivascularplexus. The density of innervation increased up to 20–24 weeks, but was always greater in the sinus and atrioventricular nodes than in other regions. Many nerve fibres contained neuropeptide Y and are probably of sympathetic origin. Other nerves displayed somatostatin or vasoactive intestinal polypeptide immunoreactivity. ANP was found from 7 weeks onwards, localised in both atrial and ventricular myocardial cells. Cells of the ventricular conduction system also contained ANP whereas the sinus and atrioventricular nodes were unreactive.

The findings show that putative endocrine elements express ANP at an early stage in the fetal heart and there is a progressive, regional development of human cardiac innervation by nerves which may also contain peptides.

Human left ventricular twist: measurement by magnetic resonance tagging

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It has been postulated that rotation of the left ventricular (LV) apex with respect to the base (twist) is a component of normal systolic function in man but, until now, it has not been possible to measure twist non-invasively. Tagging is a magnetic resonance imaging technique which labels specific areas of myocardium by narrow selective radio frequency excitation. If applied at end diastole, the tags will move with the myocardium through systole. We have used tagging to measure non-invasively LV twist in man. Eight normal volunteers (aged 24–38) were studied in a 0.38T resistive magnet. Five short axis LV planes were obtained separately at end systole, four radial tags having been inserted at end diastole, intersecting the myocardium at eight locations. Twist was defined as the angular displacement of each epicardial and endocardial tag point from its diastolic position with the base as zero and measured using an online coordinate system and a systolic centroid. Twist of the apex relative to the base was clockwise (20°) in 11 patients; the same in all radial positions, Epicardial twist (clockwise, 11 (3°), p < 0.001) was 9° less than endocardial twist (p < 0.01), but greater (6°) in the anterolateral than posterior positions (p < 0.01). Endocardial twist was not site specific.

Thus twist is a component of LV ejection in man, endocardial twist is homogeneous whereas epicardial twist is site dependent, and greater endocardial than epicardial twist suggests shearing or sliding of myocardial layers upon one another.

Myocardial hypertrophy and mechanical restitution in isolated human myocardium

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Small strips of human myocardium were obtained at open heart surgery from 58 patients; 24 patients had
Evidence for active filling of the left ventricle: a study of the association of ventricular torsion and ventricular volume

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Three dimensional motion of coronary artery bifurcation points was studied. The three dimensional data were derived from computer analysis of calibrated blipane coronary arteriograms of the left coronary artery in five apparently normal patients. The arteriograms were performed at 50 frames per second and gated to the electrocardiogram. Corrections were made for pincushion distortion and magnification effects. The resulting data were corrected for the translation component of cardiac motion and the final 25 to 30 three dimensional coordinates for each frame were analysed for the torsional component. A time dependent long axis formed by the frame was analysed for the torsional component. A time dependent long axis formed by the frame was analysed for the torsional component. A time dependent long axis formed by the frame was analysed for the torsional component. A time dependent long axis formed by the frame was analysed for the torsional component. A time dependent long axis formed by the frame was analysed for the torsional component. A time dependent long axis formed by the frame was analysed for the torsional component. A time dependent long axis formed by the frame was analysed for the torsional component.
details. Fourteen patients were heavy drinkers (> 30 u/week), eight were moderate (15–30 u/week), nine were light (1–15 u/week) and 19 drank occasionally (< 1 u/week). Twelve patients had evidence of IgA deposition on myocardial cell membranes. The diagnoses in these patients were IDC in five and coronary heart disease in seven. Of these, nine admitted to heavy, and three to moderate, alcohol consumption.

IgA deposition on myocardial cell membranes is a highly specific and moderately sensitive marker of alcohol related myocardial dysfunction and may also have a role in the pathogenesis of this condition.

Range of coronary artery lesions and the requirement for coronary arteriography in post-infarct ventricular septal defects

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In our experience a combination of ultrasound imaging methods (cross sectional echo, continuous wave Doppler, and colour flow imaging) have effectively supplanted left ventricular angiography in the diagnosis and haemodynamic assessment of post infarction ventricular septal defects (PIVSD). The requirement for preoperative coronary arteriography in such lesions remains a subject of much debate. It therefore seemed appropriate to review the coronary lesions present in large series of PIVSDs to attempt to define any correlation between PIVSD type and coronary morphology. CFM defines three distinct PIVSD types: (1) apical, (2) central trabecular, and (3) posterior. In order to determine the incidence of critical coronary stenoses in patients with PIVSD, we reviewed 112 consecutive coronary arteriograms collected from three cardiac centres. PIVSD morphology was assessed at surgery or necropsy (apical 26, central trabecular 29, and posterior 57). A critical coronary stenosis was defined as greater than 50% luminal narrowing in a first order or major second order vessel. Single vessel disease (infarct vessel only) occurred in 23/26 apical PIVSDs (88%), in contrast to 22/29 central trabecular (74%), and 26/57 posterior (46%) PIVSDs. In addition the spectrum of associated multivessel disease differed significantly between central trabecular and posterior PIVSDs with much more severe disease in the latter group.

We conclude that the requirement for preoperative coronary arteriography seems to differ significantly between the three PIVSD types. In our experience coronary arteriography may be superfluous in the apical and central PIVSD groups but may be mandatory in the posterior PIVSD group.

Using the Rose angina questionnaire

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Since Rose introduced his questionnaire on chest pain brought on by exertion as an epidemiological method of determining angina in 1962, it has been extensively used in many countries. The self administered version introduced in 1977 has proved particularly useful in postal surveys. We report on two methodological issues based on our experience in the British Regional Heart Study (BRHS): (1) Most reports have restricted themselves to reporting on "definite" angina (that is, chest pain brought on by exertion, situated in the sternum or left anterior chest, which forces a person to slow down or stop and which disappears within 10 minutes). We argue that there is a loss of sensitivity and no useful gain in specificity by imposing the criteria additional to exertion. In the BRHS, men with "possible" angina (that is, chest pain on exertion, but not satisfying all the other criteria) had similar rates of ECG abnormalities to those with "definite" angina. Of men with definite angina, 45% could recall a doctor telling them they had ischaemic heart disease, compared with 30% of men with "possible" angina, and 2-5% of other men. Most importantly, individuals with "possible" angina were at the same risk of heart attack during 7-5 years follow up as men with "definite" angina. (2) In the BRHS the age standardised prevalence of angina was the same whether assessed by administered questionnaire, as at the initial screening, or by a postal questionnaire filled in by 99% of survivors five years later. This is at variance with reports that claim a doubling of prevalence estimates when a self administered questionnaire is used.

Prevalence of heart failure in two general practices

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There is little information on the epidemiology of heart failure. The Framingham study, 20 years ago, suggested a prevalence of 1%. The current study assessed the prevalence in two large (20,000 patients) general practices in north west London. It was assumed that patients who had heart failure would be on diuretics and in a three month period would have to receive prescriptions from the general practitioner. Consequently, all patients in the practices receiving diuretic prescriptions over a three month period were noted, and a subsequent analysis was made of their practice records, which included hospital correspondence. In each of the two practices, the number of patients with heart failure was the same. The total number was 66 patients, a prevalence of 0.3%. There were 32 women and 25 men with a mean age of 72.5 (10.3) years. Only 11 patients were under 65 years old. The aetiology was thought to be coronary artery disease in 36, valve disease in 13, hypertension in four, and unknown in 13. The initial diagnosis of heart failure was made in hospitals in 51, and solely by the general practitioner in 15. Irrespective of whether the diagnosis had been made in hospital or by the general practitioner, only 60 patients had an ECG, only 58 a chest radiograph, and only 13 echocardiography. In one practice, of 90 patients taking diuretics, 41 were for hypertension (46%), 34 were for heart failure (38%), and the remainder were for leg oedema, "needing to pass more urine", and so on.

In the population studied, heart failure was less common than is generally believed and is predominantly a problem in the elderly. A cardiac abnormality is only proved in a minority of patients. Contrary to popular teaching, hypertension is an uncommon cause of heart failure.

Effects of digoxin in infants with ventricular septal defects and cardiac failure

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The clinical and haemodynamic effects of digoxin in infants with ventricular septal defect (VSD) and cardiac failure have been studied prospectively in 13 infants (mean age = 3.5 months, range 1-6 months). After stabilisation of other antifailure treatment, infants were given digoxin or placebo in a randomised double blind cross over trial. Clinical, echocardiographic, and Doppler measurements together with serum digoxin concentrations were assessed at baseline, 4-8 hours after a loading dose, and after one week of long term treatment. In all 13 infants data were obtained on the acute effects of digoxin and in 10 on maintenance treatment. Serum concentrations of digoxin were in the therapeutic range in both the short (mean = 1.3, range = 0.6-2.3 µg/l) and long term (mean = 1.2, range = 0.6-2.3 µg/l) phases. During the short term there was an increase in fractional shortening (FS) (p < 0.01) but this was not associated with a change in heart rate (HR), respiratory rate (RR), left ventricular ejection time (LVET), stroke volume (SV), or pulmonary to systemic blood flow ratio (Qp/Qs). During long term treatment the HR fell (p < 0.001) but so did LVET (p = 0.05), and there was no change in RR, FS, SV or Qp/Qs. These results show that after the short term administration of digoxin there was an isolated increase in FS. During long term treatment the fall in heart rate was not associated with improvement in other measurements and might simply reflect the action of digoxin on the sinus node.

The study suggests that digoxin is of no benefit in treating cardiac failure in infants with VSD.

Densitometric angiographic assessment of normal and diseased coronary arteries: validation of technique

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Computer assisted quantitative analysis (QA) systems for coronary arteriograms are now commercially available, but their ability to measure dimensions of diseased coronary arteries (CA's) accurately has not been fully evaluated. A QA facility with some original features has been developed. To validate the algorithms used, segments of normal and diseased CAs were excised at necropsy, perfuse fixed, filled with 5% barium sulphate gel, decalcified, and set in wax blocks. x ray films were taken from two approximately orthogonal views using both screen film and cine fluorography, both without scatter material and with a plastic bone phantom to provide realistic scatter flux. The films were digitised, mask subtracted, and analysed with the QA facility. Photographs of the exposed sections of the blocks were taken as they were serially cut with a 5 µm microtome. The true cross sectional area of the CAs were measured. This technique ensured accurate registration of measurements and ensured that the CAs did not change dimensions between the x ray picture and cutting. For normal CAs (< 1 mm to > 4 mm in diameter) both edge detection and
Densitometric analyses were more accurate than those calculated from edge detection data only. Densitometric units were converted to true dimensions. For diseased CAs with screen film, linear regression yielded a slope of 1.13, correlation coefficient 0.97 for 84 samples, standard error of the estimate (SEE) 0.47 mm². Correlation of 29 measurements of estimated area from the cinefilms with scatter material gave a correlation coefficient of 0.97, slope 1.05, SEE 0.47 mm².

We conclude that this system provides valid estimates of lumen dimensions in normal and diseased segments of CAs.

Haemodynamic assessment of mitral stenosis in the presence of atrial fibrillation

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In patients with atrial fibrillation (AF) the haemodynamic assessment of mitral stenosis (MS) is complex, requiring measurement of the mean valve gradient over 10 cycles, and the cardiac output. We describe a new method for assessing the severity of MS which is reliable and simpler to perform. In 20 patients with MS and AF, PAW and LV pressures were recorded simultaneously. The end diastolic pressure gradient was related linearly to RR interval in each patient and was unchanged by exercise. The slope (S) and intercept (I) varied with the degree of MS, and correlated with the Gorlin valve area (G): S = 10.4(G)–1.7, r = 0.8, p < 0.005; I = 1460–662 (G), r = 0.9, p < 0.005. The area (A) between PAW and LV diastolic pressure signals was measured and rose with increasing RR interval to a plateau (A-max). A-max correlated closely with the Gorlin valve area: A-max = 21.1–9.9 (G), r = 0.9, p < 0.001. Thus in MS complicated by AF, the relation between RR interval and end diastolic gradient permits accurate assessment of severity of stenosis by calculation of simple parameters (S, I, A-max) for which we have derived regression equations; they enable the construction of nomograms relating end diastolic pressure gradient to Gorlin valve area at different heart rates.

The method correlates closely with the Gorlin valve area but is simpler to perform, being independent of cardiac output measurement and heart rate.

Demonstration of myocardium at further risk after thrombolysis in acute myocardial infarction

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Although thrombolysis reduces mortality in myocardial infarction (MI), the extent to which it may salvage myocardium in individual patients has not been shown. Accordingly, 15 patients treated with streptokinase in acute MI were studied during early elective percutaneous coronary angioplasty (PTCA) to a patent infarct related artery. Left ventricular (LV) performance before and during PTCA was examined with intravenous DSA and analysed using both regional wall motion and phase/amplitude methods. During balloon inflation maximum ST elevation (mean 3.1 mm) was seen in 13 patients while mean LV ejection fraction (EF) fell in all cases (from 60% to 47%, p < 0.001) despite pre-existing Q waves in nine patients. In the eight patients undergoing LAD PTCA the fall in LVEF (54% to 40%, p < 0.001) was associated with a reduction in apical regional shortening (19% to 1%, p < 0.01). LVEF fell similarly in seven patients during right coronary PTCA (68% to 55%, p < 0.001) associated with a reduction in inferior shortening (41% to 16%, p < 0.01). Phase amplitude analysis showed similar regional abnormalities in 13 patients.

A deterioration in LV performance during balloon coronary occlusion in these patients suggests that thrombolysis had salvaged myocardium at risk during thrombotic occlusion. In patients with pre-existing Q waves, LV ischaemia during balloon inflation implies that this ECG abnormality does not necessarily indicate complete infarction.

Use of Doppler echocardiography in determination of left ventricular dP/dt max

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Doppler echocardiography is emerging as a powerful tool in measuring indices of left ventricular contractility. In human subjects peak acceleration has been shown to correlate closely with ejection fraction, and in dogs peak aortic flow velocity has been shown to correlate closely with maximum dP/dt. Our study verifies the correlation of peak velocity with...
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dP/dt max in human subjects and validates our theoretically derived formula that showed a better correlation with dP/dt max. Using the concept for conservation of energy, by assuming sinusoidal aortic flow and pressure patterns and a constant of proportionality between the area of LV outflow and ascending aorta, the following is derived:

\[ \frac{dP}{dt} \text{ max} = \frac{V_p^2}{T} + \frac{\text{pulse pressure}}{T_C} \text{ (pulse P)}; \]

where \( K = \text{constant}; T = \text{time to peak velocity}; V_p = \text{peak velocity}; \) and \( T_C = \text{corrected ejection time}. \) The validity of this equation was tested in 11 patients undergoing diagnostic catheterisation using a catheter tip micromanometer in the LV for dP/dt max and a 4 MHz continuous wave Doppler transducer for aortic flow signals. While \( V_p \) and mean acceleration (MA) correlated with dP/dt max (\( r = 0.837 \) and 0.811, respectively) a better correlation (\( r = 0.866 \) p < 0.001) was found for \( \frac{K V_p^2}{T} + \frac{\text{pulse P}}{T_C}. \)

This method may prove useful in the non-invasive estimation of dP/dt max under differing clinical conditions, particularly if there are rapidly changing (beat to beat) variations in contractility.

Magnetic resonance imaging of atrioventricular septal defects in children correlates with echocardiographic, angiographic, and surgical findings

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Magnetic resonance (MR) imaging was performed in seven patients (age range 11 days—14 months) with atrioventricular septal defects (AVSD). Echocardiographic studies had previously been performed in all patients, and five subsequently had angiography. Three patients have since been surgically corrected. Associated defects present were Fallot’s tetralogy in one case, and right atrial isomerism with pulmonary atresia in another. A 1·5 T whole body MR imaging system was used with patients sedated and positioned supine inside a 32 cm diameter head coil. Multiple ECG gated sections 5 mm thick were acquired using a spin echo sequence with echo time of 30 ms. A combination of standard and oblique imaging planes were used. The MR images were compared with the other investigations in order to assess the size of the ventricular component of the defect, the attachments of the atrioventricular valve, and the left and right ventricular dimensions. MR predicted the size of the ventricular component of the defect in every case with full agreement with angiographic and surgical findings. In all cases MR demonstrated the defect in more detail than echocardiography. In four patients the attachments of the superior and inferior bridging leaflets to the ventricular septum were clearly shown. In this respect there was full agreement with those patients who had surgery. MR was an accurate method of determining ventricular dimensions. In one patient MR correctly predicted severe ventricular disproportion which was confirmed at operation.

MR imaging is capable of providing detailed morphology data in infants with AVSDs. This information is likely to be of value in the preoperative assessment of these patients.

Thrombin inhibition by heparin or hirudin decrease platelet thrombus deposition and vasospasm after arterial angioplasty

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To test the hypothesis that localised vasoconstruction after arterial injury is related to platelet deposition, the effects of heparin and recombinant hirudin (a more specific thrombin inhibitor than heparin without intrinsic vasodilating properties) were tested on platelet deposition and spasm following experimental angioplasty. After heparin (35 u/kg bolus + 35 u/kg/hour infusion, n = 10, or 250 u/kg + 250 u/kg/hour, n = 9) or hirudin (1 mg/kg + 1 mg/kg/hour, n = 5) pigs underwent bilateral carotid angioplasty (five inflations for 30 seconds to 6 atmospheres, 60 seconds apart). Vasoconstruction (1% luminal diameter narrowing immediately distal to the dilated segment) was assessed by computer assisted analysis of angiograms taken before and 1 minute after injury. The time course of spasm after injury was assessed by serial angiograms (1, 5, 15, 30, and 60 minutes after injury) in 11 animals. Platelet deposition \( \times 10^6/cm^2 \) of deep arterial injury) was measured using indium III labelling. Platelet deposition in the low dose heparin group was 305 (77) and was less with high dose heparin (59 (18), p < 0·002) or hirudin (28 (8), p < 0·001). Mural thrombus occurred in 72% of deeply injured segments in the low dose heparin group, and occurred less often in the high dose heparin group (10%, p = 0·001) or with hirudin (0%, p = 0·001). Vasoconstruction at 1 minute was positively correlated with platelet depo-
position (r = 0.32, p < 0.01). Vasocostrction at 1 minute was 49 (3)% in the low dose heparin group and was less after high dose heparin (26 (5)% , p < 0.01) or after hirudin (33 (5)% , p < 0.02). Vasocostrction was most intense one minute after injury (37 (4)% ) and almost absent at 60 minutes (15 (2)% , p < 0.01).

The results show that inhibition of thrombin by heparin or hirudin reduces thrombosis and vasocostrction associated with arterial injury. These effects are related to their ability to inhibit thrombin induced platelet activation. The results underline the importance of platelets as mediators of vasospasm after arterial angioplasty.

Reduction in major morbidity and mortality by heparin in acute myocardial infarction

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About 5700 patients with suspected acute myocardial infarction (AMI) have been randomised in 20 trials of subcutaneous (sc) or intravenous (iv) heparin. Individually, these trials were too small to provide reliable estimates of treatment effects on major morbidity events and death. An overview of the trials, however, in which results from individual studies were combined, provides strong evidence of beneficial effects. Overall, among all patients allocated heparin, the "typical" odds of death was 17% (7) lower (2 p = 0.005) than among those allocated control. Furthermore, treatment with heparin was associated with significant reductions in deep vein thrombosis (66% (7); 2 p < 0.001), pulmonary embolus (54% (8); 2 p < 0.001), reinfarction (22% (10); 2 p < 0.05) and stroke (50% (16); 2 p < 0.005).

The effect of heparin on total stroke suggests that in these trials any increase in cerebral haemorrhage resulting from anticoagulation was outweighed by a reduction in cerebral infarction. The heparin regimen tested in about 90% of patients entailed doses of 20 000 IU or more daily, given subcutaneously in about half the patients.

In the UK and USA, such doses of sc or iv heparin are used only in a minority of patients with suspected AMI. Some physicians may wish to reconsider this policy in the light of the present results. Others may feel that further randomised evidence is needed about the effects of heparin in the current era of treatment with thrombolytic and antiplatelet agents.

Electrophysiological study of survivors outside hospital from ventricular arrhythmias unassociated with acute myocardial infarction

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We studied 32 survivors (25 men) of life threatening ventricular arrhythmias not due to acute myocardial infarction (MI) that occurred outside hospital. Mean age was 57 years (range 9-79). Electrophysiological study (EPS) included three programmed extra stimuli in sinus rhythm and ventricular pacing drive rate of 100, 120, and 140 per minute (S4 protocol) from at least two right ventricular sites. Twenty patients had a previous history of MI. In 10 patients the initial documented rhythm was ventricular fibrillation (VF), in 12 ventricular tachycardia (VT), and in 10 VT deteriorated into VF. During EPS non-sustained VT was induced in nine patients, sustained VT in 15, and VF in both the survivors. Of patients with induced VT, 19 had cycle length < 300 msecs. VT or VF was induced in 16 of 20 patients with left ventricular ejection fraction (EF) > 30%, and in 10 of 12 patients with EF < 30% (no significant difference). During follow up (four weeks to 35 months) ventricular arrhythmias were suppressed by treatment for underlying heart disease and EPS directed treatment in 16 patients. In six patients serious VT recurred, and in two was due to poor drug compliance. Serious VT recurred in one of six patients with initial negative EPS. One death occurred during follow up, in one of six patients whose EPS induced VT could not be completely suppressed by drug treatment. Inducibility of VT/VF during EPS was not related to previous MI or whether documented prehospital rhythm was VF or VT.

We suggest that EPS in survivors of ventricular arrhythmias occurring outside hospital is of value in determining prognosis and treatment.

Rise in left atrial pressure does not limit exercise capacity in patients with hypertrophic cardiomyopathy

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The hypothesis that exercise capacity in hyper-
trophy cardiomyopathy (HCM) is limited by a rapid rise in left atrial pressure as a result of a systolic gradient or impaired diastolic function has not been tested. Twenty three patients, age 15-70 (mean 36) years, in whom exercise capacity was limited only by dyspnoea were selected. Patients with severe mitral regurgitation, pulmonary disease, or an inability to exercise for any reason other than dyspnoea or fatigue, were excluded. Pulmonary capillary wedge pressure (PCWP), maximal oxygen consumption (VO₂ max) and anaerobic threshold were measured during symptom limited treadmill exercise testing. VO₂ max was reduced (28.1 (7.5) ml/kg/minute; normals > 40) as was anaerobic threshold (21.5 (6.1) ml/kg/minute; normals > 30), even in 12 of 13 NYHA Class I patients. All patients stopped because of dyspnoea. Supine resting PCWP (mm Hg) was 15 (5). Erect PCWP rose from 5 (5) at rest to 24 (11) at peak exercise, and cardiac index (l/minute/m²) from 2.4 (0.6) to 8.1 (2.1). VO₂ max and anaerobic threshold were related to peak cardiac index (r = 0.65, p < 0.001, and r = 0.46, p = 0.03, respectively) as well as the rise in cardiac index on exercise (r = 0.68, p < 0.001, and r = 0.51, p = 0.016, respectively) but not to resting cardiac index, resting and peak PCWP, the rise in PCWP on exercise, or PCWP at a submaximal oxygen consumption value of 15 ml/kg/minute.

These findings confirm previous reports of an abnormal rise in left atrial pressure but are not consistent with the hypothesis that raised left atrial pressure is the major determinant of exercise capacity in patients with HCM, and suggest that as in patients with chronic heart failure, other mechanisms should be considered.

**Myocardial perfusion SPET scintigraphy with technetium 99 methoxy-isobutyl-isonitrile in coronary artery disease: comparison with thallium 201**

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Technetium-99m MIBI (methoxy-isobutylisonitrile) has recently been introduced as a new radiopharmaceutical for myocardial perfusion scintigraphy. Labelled with 99Tcm, it may be superior to the standard thallium²⁰¹ in the detection and localisation of coronary disease. Twenty nine patients (aged 41-72 years, mean 55 years) with established coronary artery disease (CAD) (50% or greater diameter stenosis on coronary angiography in at least one vessel) were investigated. Stress and rest delayed studies were carried out with ⁹⁹Tcm⁹⁹MIBI (370 MBq at peak stress and rest 24 hours or more later) and ¹³¹I (74 MBq at peak stress) within seven days of first test. Each patient achieved identical rates of exercise on graded supine bicycle ergometer. Post stress (5-10 minutes of ²⁰¹TI and one hour of ⁹⁹Tcm MIBI injection) and delayed/rest SPET studies were performed in each case using IGE 400 AC/STARCAM and 400A STAR gamma camera computer system. Myocardial ischaemia (as expressed by reduced tracer uptake) was seen in 66 of 145 (46%) myocardial segments with ²⁰¹TI and 70 of 145 (48%) segments with ⁹⁹Tcm MIBI. Four segments showed normal uptake of ²⁰¹TI but reduced uptake of ⁹⁹Tcm MIBI while opposite was seen in two segments. Reversible or irreversible myocardial ischaemia as predicted by ²⁰¹TI was present in 26 out of 29 patients (90%) whilst ⁹⁹Tcm MIBI predicted disease in 28 out of 29 patients (95%). Both radiopharmaceuticals could accurately localise coronary lesions and their functional significance. The quality of the ⁹⁹Tcm MIBI tomogram was similar or better than that using ²⁰¹TI.

This study demonstrates the high sensitivity obtained with ⁹⁹Tcm MIBI in the detection of coronary disease. It has the advantages of easier availability than ²⁰¹TI, and exercise testing may be performed at a site remote from the nuclear imaging system. This will reduce the number of exercise tests needed in the investigation of coronary disease.

**Magnetic resonance chemical shift imaging of human atheroma**

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Magnetic resonance can show protruding atheromatous plaques either using conventional imaging or angiographic techniques, but chemical shift imaging also allows assessment of the lipid content of a lesion. This is of considerable clinical importance because the ability to modify atheroma in a patient either pharmacologically or mechanically depends upon its composition.

Twelve necropsy specimens of human descending aorta and the aortas of two patients with documented atheroma were studied using a spin echo sequence to localise protruding lesions. The specimens were
maintained at 37°C and were distended at 80 mm Hg pressure. Chemical shift imaging was performed using the technique described by Dixon that produces maximum signal for a 50% mixture of lipid and water and no signal for either pure lipid or pure water. Since the specimens contained less than 50% lipid, the signal strength was proportional to lipid content, and the signal from atheroma was expressed as a proportion of that from extravascular fat. Calibration was performed using fat and water mixtures. After imaging the specimens were examined histologically using haematoxylin and eosin, elastic van Gieson, and oil red O fat stains. The lipid content of the plaques was scored as either absent, low, medium, or high, and the distribution of lipid within the plaque and between intima and media was noted. Lipid content assessed by magnetic resonance ranged from 8% to 50% compared with extravascular fat. There was good agreement with the histological findings and all but one plaque with signal intensity over 25% had high lipid content histologically. The distribution of lipid within the plaques also agreed with histology and intimal lipid was particularly prominent in the majority. In one patient, atheroma in the descending aorta was shown to have a high lipid content and in the other, a low lipid content. Histological confirmation was not possible.

Magnetic resonance chemical shift imaging provides a method of assessing the lipid content of atheromatous plaques. Validation in vivo is more difficult than in vitro but results can be obtained. The usefulness of the technique in routine clinical practice remains to be established.

Impact of colour Doppler flow mapping on the assessment of congenital heart disease

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The role of cardiac catheterisation in the investigation of congenital heart disease is reassessed in the light of the increased ability of non invasive techniques with the development of colour Doppler flow mapping (CDFM) which enhances the ability of echocardiography to demonstrate intracardiac lesions.

During a seven month period 142 patients underwent surgery (59 having had previous catheterisation) and 111 catheterisation (26 for interventional procedures). In those undergoing catheterisation full echocardiographic and spectral and CDFM studies were performed, and a form stating the exact non-invasive diagnosis with assessment of the haemodynamic measurements (particularly shunt size and pulmonary pressure) was completed before the procedure, and the contribution of catheterisation to the final diagnosis assessed. Of those catheterised there was no major discrepancy in the assessment of the diagnosis, shunt size, or the severity of obstructive lesions. Ultrasound was unable to provide sufficient detail to permit surgical intervention in those with complex venous abnormalities, coronary artery fistula, or distorted pulmonary arteries. In three cases venous abnormalities not commented on before catheterisation were recognised afterwards. Little additional information was obtained from catheterisation in 50% of patients but the decision on the need for surgery would not necessarily have been the same had it not been undertaken.

CDFM adds to the diagnostic ability of imaging and spectral Doppler in the assessment of congenital heart disease and will further reduce the need for catheterisation before surgery.

Prediction of outcome after acute uncomplicated myocardial infarction: a study of 394 patients with five years follow up

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Three hundred and ninety four consecutive patients were studied prospectively after an acute uncomplicated myocardial infarction by predischarge maximal treadmill exercise testing and coronary angiography at six weeks. Logistic regression analysis was used to produce a simple predictive index that could be applied at the end of the exercise test to predict the coronary anatomy and the therapeutic outcome in these patients. Four models were constructed out of 11 variables, with age as a continuous variable. The best model for predicting coronary anatomy was found to consist of angina, exercise time, maximal ST segment depression, and myocardial infarction site. Coronary anatomy then added most to the combined predictive power of exercise time and ST segment depression, which together dictated the outcome. The sensitivity and specificity of the exercise tests were 94 and 92%, respectively. Based on this model 214 patients required coronary bypass surgery during the follow up period. Of these 141 patients had inferior, 63 had anterior, and 10 had
Right ventricular dysfunction enhances mortality after myocardial infarction

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After myocardial infarction, left ventricular ejection fraction is known to predict mortality, but the predictive value of right ventricular dysfunction is less clear. Right and left ventricular ejection fraction (RVEF and LVEF) were measured in 81 consecutive patients with myocardial infarction at one day (EF 1), three days (EF 2), before discharge (EF 3), and at a mean of 16 months later (EF 4) using gated blood pool ventriculography. In 36 patients with anterior myocardial infarction mean LVEF did not alter significantly throughout the study period but RVEF deteriorated from day 1—RVEF 36-8 (9-9)% to 16 months 32-4 (7)%—(p < 0.01). For the 45 patients with inferior myocardial infarction, mean left ventricular function deteriorated slightly in hospital and subsequently improved by follow up (LVEF 1 31-3 (8)% and LVEF 4 34-1 (9-9)% p < 0.02) but right ventricular function did not change. Mortality was 31% (11/36) for anterior infarcts and 16% (7/45) for inferior infarction. If a LVEF < 30% was considered abnormal, 33% (16/45) were dead at follow up compared with 6% (2/33) of those with preserved left ventricular function. With right ventricular dysfunction (RVED < 35%) 25% were dead at follow up, and 19% of those with preserved right ventricular function had died. Twenty nine patients had both right and left ventricular dysfunction, however, and the mortality was 41% (12/29) compared with 22% (4/18) in those with only left ventricular dysfunction.

In conclusion, this suggests that after myocardial infarction right ventricular dysfunction enhances the risk of mortality in patients with left ventricular dysfunction.

Diagnostic importance of reciprocal ST segment depression in acute myocardial infarction

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The interpretation of reciprocal ST segment depression (RSTD) in acute myocardial infarction (AMI) remains controversial. It might represent acute ischaemia of the underlying myocardial segment. One hundred and forty two patients with a classical AMI underwent early submaximal exercise tests (SM-ET). All patients with positive SM-ET (n = 76) and one third of those with negative SM-ET (n = 30), chosen at random, had coronary angiograms shortly afterwards. RSTD was observed in 65% of patients with inferior MI and 57% of those with anterior MI. Of the 52 patients with RSTD who had coronary angiography, 33 had significant lesions of the coronary artery that subserves the RSTD territory (63%), while of the 46 patients with positive SM-ET, 31 had comparable lesions of the coronary artery that supplies the ischaemic territory (67%). The incidence of multivessel coronary artery disease in the two groups was 79% and 76%, respectively.

This study has shown that RSTD is a common electrocardiographic finding in acute myocardial infarction. It is associated with stenosis of the RSTD subserving coronary artery in nearly two thirds of the patients, and with multivessel coronary artery disease in almost four fifths. RSTD is as good as the SM-ET at predicting the anatomy of the underlying coronary artery disease.

Prognostic value of heart rate variability after myocardial infarction

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The prognostic value of heart rate variability was studied in 54 patients within two weeks of infarction. Ten patients either died or developed ventricular tachycardia (VT), and five had non-fatal rein-
Characteristics and prognosis of non-Q wave myocardial infarction

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Survivors of non-Q wave myocardial infarction are reputed to have a comparatively high risk of subsequent cardiac events including sudden death, reinfarction and angina. The frequency of non-Q wave infarction and subsequent prognosis were therefore investigated in 337 survivors of definite myocardial infarction. The results of predischarge submaximal exercise testing were available in all patients, coronary angiography in 109 patients, and all were followed for 12 months. Non-Q wave infarction occurred in 80 patients (24%) of whom two died (2.5%), seven reinfarcted (9%), and 32 developed angina (40%) during the 12 month follow up. Of those with Q wave infarction, 12 (5%) died, 21 (8%) reinfarcted, and 99 (39%) developed angina. Positive predischarge exercise tests were recorded in 47 (59%) patients with non-Q wave infarction and 138 (54%) patients with Q wave infarction. A positive test predicted cardiac events with similar frequency in non-Q wave infarction (73% subsequent events) and Q wave infarction (83% subsequent events). The ratio of silent to painful predischarge ischaemia during submaximal exercise testing was identical in both groups. Coronary angiography was available in 109 patients, 84 with Q wave and 25 patients with non-Q wave infarction. Single vessel disease was found in 12 of 25 (48%) with non Q wave infarction and 25 of 84 (30%) with Q wave infarction. Double and triple vessel disease were equally distributed in the remaining patients.

Thus non-Q wave infarction was seen in 24% of infarct survivors and had a similar clinical outcome in the ensuing 12 months. This is supported by identical predischarge exercise test characteristics and a similar distribution of coronary artery disease.

Breathlessness, lung function, and risk of heart attack

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Diminished pulmonary vital capacity and dyspnoea are recognised as consequences of advanced heart disease. Their relevance in earlier stages of heart disease is less well documented. In a prospective study of 7735 British men aged 40–59 years and followed up for 7.5 years, men with moderate or severe breathlessness had a greater than two fold risk of suffering major ischaemic heart disease (IHD) compared with men with no evidence of breathlessness. After adjustment for other risk factors, including cigarette smoking, the relative risk remained two fold. Men in the lowest fifth of the FEV₁ distribution also had a two fold risk of IHD compared with men in the highest fifth, after similar adjustment. Reduced lung volume rather than obstruction seemed to be the determinant of risk as FVC exhibited a similar association with FEV₁, while FEV₁ to FVC% was less strongly associated with risk. The role of breathlessness as a risk factor for new IHD was partly explained by its strong association with pre-existing but usually undiagnosed IHD. Both a reduced FEV₁ and breathlessness,
however, were associated with raised risk of major IHD events even in men without any evidence of IHD at screening.

The incidence of angina during the five years after screening was strongly associated with breathlessness grade at screening, suggesting that breathlessness is an early indicator of IHD in the absence of other symptoms.

One hundred and thirty three cases of spontaneous atrial flutter: electrophysiological characteristics and factors affecting successful conversion

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One hundred and thirty three unselected consecutive cases of atrial flutter were studied in 114 patients (86 men, 28 women, mean age 54, range 18–79). In 118 cases the flutter was of the common type. Duration of flutter ranged from 10 minutes to 16 years (median four days). The mean (SD) atrial cycle length during flutter was 223 (35) ms. Drug treatment included digoxin (n = 40) and amiodarone (n = 13). Atrial mapping was performed in 75 cases (424 sites) and showed predominantly downward activation of the lateral right atrium and upward activation of the atrial septum in common flutter. Sites exhibiting a reset response to atrial programmed extrastimuli (APE) were most commonly found in the low lateral, septal, and posterior right atrium, indicating preferential inclusion of these sites in a reentry circuit. Conversion was not attempted in 17 cases (12 proceeded to catheter ablation, five had areas of coexisting atrial fibrillation (AF)). In the remaining 116, 1–4 APEs resulted in conversion to sinus rhythm (SR) in 23 cases (20%), and to AF in five (4%). In 82 cases, conversion was then attempted using a single long constant current pulse (atrioversion). SR was achieved in 36 cases (44%), and AF in 30 (37%). In 29 cases who had failed either one or both of the above attempts, rapid atrial overdrive pacing (RAP) achieved SR in 12 cases (41%), and AF in 13 (48%). Overall, 72 cases (62%) were converted to SR and 39 (34%) to AF (12 reverted spontaneously to SR in less than 24 hours). Five cases (4%) remained in flutter.

Factors significantly improving successful conversion included coronary artery bypass grafting in the previous month (SR in 86%), congenital heart disease (SR in 89%), and stimulation at a site exhibiting reset. Thus aetiology and detailed atrial mapping may significantly affect successful conversion.

Verapamil or adenosine for supraventricular tachycardia

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The place of intravenous verapamil as the treatment of choice for supraventricular tachycardia (SVT) has recently been challenged by the use of intravenous adenosine, a rapidly metabolised naturally occurring compound. We have reviewed 105 spontaneous episodes of paroxysmal SVT in 38 patients (aged 16 to 79 years; mean 44 years) treated acutely with either agent. Verapamil was given to 22 patients during 65 episodes of SVT, and adenosine to 21 patients on 40 occasions. Five patients received both compounds. Verapamil, 3 to 30 mg (mean 7.7 mg), restored sinus rhythm in all 22 patients, in 57 of 65 episodes (88%), within 40 minutes of administration. In four patients verapamil (5 to 25 mg) failed to terminate SVT in 8 of 22 episodes. Hypotension (systolic BP < 90 mm Hg) occurred after verapamil in seven episodes (10%) in four patients, requiring emergency DC cardioversion on three occasions. Arrhythmia recurred following verapamil within one hour in only one patient and within 12 hours in two. Adenosine was administered by incremental doses, from 2.5 to 25 mg (mean effective dose 10.4 mg). Sinus rhythm was restored in all 21 patients in 38 of 40 episodes (95%) within one minute of administration. In one patient SVT persisted despite adenosine (15 and 20 mg) in two of 14 episodes. Early recurrence of SVT (within 10 minutes) occurred in eight patients (36%). No haemodynamic deterioration was observed after adenosine, but minor transient side effects were common (77%).

In conclusion, both verapamil and adenosine are highly effective in the treatment of SVT. Adenosine acts more rapidly and is safer, but its effect is short lived and arrhythmias may recur.

Comparative efficacy and safety of adenosine, lignocaine, disopyramide, flecainide, and sotalol in the termination of ventricular tachycardia

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Drug termination is frequently used in the acute management of ventricular tachycardia. The comparative merits of the various drugs available are not known. In this study drugs were given intravenously as bolus doses (adenosine and sotalol) or infusions (disopyramide, flecainide, and sotalol) during induced, sustained, haemodynamically stable ventricular tachycardia in serial trials. Tachycardia was terminated by programmed stimulation if it continued 10 minutes after the end of drug administration. Twenty four patients underwent 105 trials with termination of ventricular tachycardia in 35 trials by the drug. In six patients no drug terminated ventricular tachycardia. Adenosine did not terminate ventricular tachycardia in any patient (so acted as a control), lignocaine in 7/23 patients, disopyramide in 12/24 patients, flecainide in 11/20 patients, and sotalol in 5/15 patients. If the ventricular tachycardia was not terminated by flecainide, then in only 3/9 patients was it terminated by another drug. This was much lower than seen with disopyramide (6/12), lignocaine (10/17) and sotalol (6/10). Serious adverse effects during ventricular tachycardia were seen in 5/20 patients with flecainide (4 proarrhythmic, 1 haemodynamic), in 2/15 patients with sotalol (both haemodynamic), and in 1/24 patients with disopyramide (proarrhythmic), but were not seen with lignocaine or adenosine.

In conclusion, flecainide is the most effective drug at terminating ventricular tachycardia but has the most adverse effects. Lignocaine was both significantly safer and less effective (p < 0.05). Disopyramide and sotalol were more effective but had more side effects than lignocaine and were safer but less effective than flecainide.

**Efficacy of flecainide for atrial arrhythmias after open heart surgery**

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The antiarrhythmic effects of intravenous flecainide and intravenous digoxin were assessed in 29 patients (26 men aged 43 to 73 years, mean 60) who developed atrial arrhythmias in the first 96 hours after open heart surgery. Twenty seven had AF and two had atrial flutter. Patients were entered into the study only if the arrhythmia was observed for at least 15 minutes with a ventricular rate of more than 120/minute. Patients were randomised to flecainide or digoxin. Flecainide was given as a bolus of 1 mg/kg over 10 minutes followed by infusion of 0.25 mg/kg/hour for the rest of the 24 hour study period. Digoxin was given as three bolus doses of 0.5 mg followed after six hours by 0.25 mg and after another six hours by 0.25 mg. In both groups, verapamil was subsequently given for 45 minutes if no reversion to sinus rhythm or adequate ventricular rate control (<100/minute) was achieved. Fifteen patients were given flecainide, five of whom required verapamil. Fourteen patients were given digoxin, 13 of whom required verapamil. The antiarrhythmic efficacy was assessed by 24 hour Holter monitoring and 15 second rhythm strips taken at 1, 2, 3, 6, 12, and 24 hours. After flecainide arrhythmia terminated in 9/15 within one hour and did not recur during the 24 hour study period. In 1/15 arrhythmia persisted with a controlled ventricular rate that was maintained throughout the study period. Flecainide with verapamil terminated arrhythmia in 1/15, controlled the ventricular rate in 1/15, and failed to control arrhythmia in 3/15. Digoxin alone failed to terminate arrhythmia in all 14 and controlled the ventricular rate in only 1/14 (p = 0.001). Digoxin with verapamil terminated arrhythmia in 1/14, controlled the ventricular rate in 1/14 and failed to control arrhythmia in 11/14. None of the patients in the two groups had major side effects.

We conclude that intravenous flecainide is more effective than conventional drug treatment for postoperative atrial arrhythmias.

**Prediction of life threatening arrhythmias in hypertrophic cardiomyopathy using high gain, signal averaged, electrocardiography**

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The prediction and prevention of sudden death remains a major objective in the management of hypertrophic cardiomyopathy (HCM). We have investigated the value of late potentials (LP) detected using high gain, signal averaged electrocardiography in predicting arrhythmias in 31 HCM patients. Maximum echocardiographic wall thickness was 15–29 mm, median 22 mm; 15 patients were taking amiodarone (median dose, 200 mg/day); 12 of the patients were young (<25 years), and of these three had been resuscitated having gone into ventricular fibrillation (VF) when not in hospital. All 31 patients had 48 hour electrocardiographic monitoring; eight had nonsustained ventricular tachycardia (VT), and one had sustained VT. None of the three patients with a history of VF had arrhythmias on electrocardiographic monitoring. LP were detected using Sinnow’s method; patients with bundle branch block were
excluded, and a prolonged filtered QRS was not considered positive for LP if the surface QRS was >120 ms. LP were present in 7/31 (23%). There was no difference between the incidence of LP in those who were and were not taking amiodarone, and there was no correlation with the degree of echocardiographic left ventricular hypertrophy. Of the four patients with a history of life threatening arrhythmias, three (the one with sustained VT and two of the three with VF) had late potentials. The specificity for the prediction of life threatening arrhythmias by LP was thus 75%, the specificity 85%, and the positive predictive accuracy 43%. Of the nine patients with non-sustained VT four had LP compared with 3/22 without VT (p < 0.05). All eight patients with non-sustained VT have had a benign course.

In conclusion, the presence of LP in patients with HCM correlates both with the occurrence of life threatening arrhythmias and non-sustained VT, a known prognostic marker in HCM. Signal averaged electrocardiography may therefore be of value in the assessment of the risk of sudden death in HCM, especially in the younger age group, where currently no prognostic indicator exists.

**Hospital mortality of ventricular tachycardia and fibrillation**

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Factors influencing hospital mortality of ventricular tachycardia (VT) and/or ventricular fibrillation (VF) were prospectively investigated in 124 consecutive patients between January and June 1988. Seventy seven (62%) were men, and 102 (82%) had ischaemic heart disease. Fifty five had had acute myocardial infarctions (MI). Age, sex, previous history of VT/VF, aetiology, site of MI, number of previous MIs, drug history, and NYHA functional state were recorded. Seventy three patients (59%) survived to leave hospital. The following were statistically significantly associated with hospital mortality by univariate analysis: age >60 (p < 0.05), female sex (p < 0.01), first episode of VT/VF (p < 0.01), anterior MI (p < 0.01), use of inotropes (p < 0.01), NYHA 3 or 4 (p < 0.001). Other features showed no significant correlation with outcome. Hospital mortality rose in association with increasing numbers of "risk" factors: hospital mortality was 4·7% when no factor or one alone was present, 30·6% when two or three factors were present, 63·6% when four factors were present, and 94·5% when five or six factors were present.

This study shows that VT or VF, or both, carry an appreciable hospital mortality (41%); several clinical features correlate with the risk of death. Previous history of VT/VF was not associated with an increased hospital mortality. The natural history of and risk factors for VT and Vf whether or not in the context of acute ischaemia are poorly documented.

**Role of transoesophageal echocardiography in the diagnosis of aortic dissection: a cooperative multicentre trial**

Erasmus University, Rotterdam, The Netherlands, University of Mainz, Westfälische Universität, Münster and Medizinische Hochschule Hannover, West Germany, and Academisch Ziekenhuis, and St Anthonius Ziekenhuis, Amsterdam, The Netherlands

Transoesophageal echocardiography (TEE) can view the entire thoracic aorta and thus could provide all the required diagnostic information about the type and complications of aortic dissection. In a multicentre cooperative study both the sensitivity and specificity of this method were tested in 181 patients with proved (99) or excluded (82) aortic dissection. The echocardiographic findings were compared with computed tomographic (CT), angiographic, and surgical or necropsy findings, or both, in 75 patients; with CT and angiography in 89 patients; and with surgical findings alone in 17 patients. In the 164 patients with correlative angiographic, CT and TEE findings, TEE correctly defined an aortic dissection in 81/82 patients. One false negative result occurred in a type II dissection. In addition these were two false positive diagnoses. In comparison, CT gave false negative results in seven patients and no false positive results. Aortography gave false positive results in three patients and false negatives in nine. The sensitivity and specificity of TEE were 99% and 98%, respectively, with a positive and negative predictive accuracy of 98% and 99%. For CT the sensitivity measured 83% and specificity 100% with (+) and (-) predictive accuracies of 100% and 86%, respectively. For aortography the comparable sensitivity and specificities were 80% and 94%, respectively, and the (+) and (-) predictive accuracy 96% and 84%. Aortic dissection was reliably differentiated from aortic aneurysm and aortic dilatation. Colour flow imaging clearly defined the
abnormal flow patterns in the true and false lumens and the site of the proximal entry point(s).

We conclude that TEE is the diagnostic technique of choice in aortic dissection.

An evaluation of homovital aortic valve homografts

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Between January 1980 and April 1988, 99 homovital aortic valves, obtained under sterile conditions at the time of cardiac transplantation, were used for aortic valve replacement (61 patients) or aortic valve and root replacement (38 patients). The valves were kept at 4°C until used 1-5 to 72 hours later. ABO compatibility was achieved in 63 (63%) patients. Twenty four patients were immunosuppressed with azathioprine and prednisolone for three months. Additional procedures were performed in 21 patients (21%). There were two (2%) early deaths, both in patients with additional procedures, and four (4%) late deaths during a follow up period ranging from 1 to 99 months (mean 22-5 months). The five year actuarial survival for patients with isolated aortic valve replacement was 95%. Valves failed in three patients and this was related to endocarditis in two patients (30 and 48 months after operation), and degeneration eight years postoperatively in one patient. All three patients were reoperated on successfully. The cumulative probability of freedom from endocarditis and valve failure was 94% at 6 years.

It is concluded that the medium term results of homovital homografts are good, with no evidence of early rejection.

Is there a limit to operability in aortic stenosis?

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It has been argued that elderly patients with aortic stenosis, and those with poor left ventricular function, should be considered for valve dilatation as they are at high risk from surgery. To investigate this assumption, a retrospective study was made of all 74 patients who underwent valve replacement for pure aortic stenosis, under the care of a single surgeon, between January 1982 and December 1987. This included patients having simultaneous coronary artery surgery. There were 46 men and 32 women. Median age was 66 years (range 29-86). The median aortic gradient was 80 mm Hg (range 20-140). Poor left ventricular function was assessed by (a) presence of clinical left ventricular failure, (b) poor left ventricular function at cardiac catheter, (c) left ventricular end diastolic pressure over 25 mm Hg, and (d) left ventricular ejection fraction under 30% (MUGA).

All four signs were not available for all patients. Ten patients were classed as having poor left ventricular function based on the presence of two or more criteria. There was no significant difference in age or aortic gradients between the good and poor function groups. Inotropic support was required by 2/64 of the good LV group compared with 2/10 of the poor group. Median hospital stay was 10 days (range 6-32) for the good LV group and 8-5 days (range 6-10) for the poor group, (p = 0-5, Mann-Whitney U test). Median follow up was three years. No perioperative deaths occurred in either group.

We conclude that neither age nor poor left ventricular function need be a deterrent to surgery in aortic stenosis.

What is normal function of an artificial valve? A colour and continuous wave Doppler assessment

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To evaluate Doppler characteristics of normal artificial valves we studied 335 patients with 368 prostheses; 179 were in the aortic position (AVR), 98 Starr-Edwards (SE), 50 Björk-Shiley (BS), 7 Duomedics (DM), 12 Mitralflow (MF), 14 Hancock-Pericardial (HP) and 8 Carpenter-Edwards (CE) and 189 in the mitral position (MVR) (116 SE, 43, BS, 3 DM, 12 MF, 8 HP and 7 CE). Peak flow velocities (m/s) and the derived transvalvular pressure gradients (mm Hg) were measured; for MVR pressure half time (m/sec) was also calculated. Prosthetic valve regurgitation was assessed using colour and continuous wave Doppler. The average pressure gradients were significantly higher (p < 0-01) in mechanical AVR (SE = 29 (15), BS = 31 (13), and DM = 18 (13)) than in bioprostheses (MF = 14 (9), HP = 17 (8), and CE = 19 (8)). Pressure gradients in mechanical MVR (SE = 10 (4), BS = 9 (4), and DM = 13 (2)) were not significantly higher than in bioprostheses (MF = 8
Sympathetic neurohormonal response to dynamic exercise early after orthotopic cardiac transplantation

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We investigated the sympathetic neurohormonal response to dynamic exercise of seven healthy male orthotopic transplant recipients, mean age 35.4 (range 18–61) years, six (range 3–11) months after transplantation. They were compared with seven normal subjects mean age 35.1 (range 23–58) years. The subjects performed progressive dynamic exercise on a cycle ergometer. Blood samples were obtained from an indwelling venous cannula in a peripheral vein. Noradrenaline and adrenaline concentrations were measured by high performance liquid chromatography using electrochemical detection. All results are quoted as mean values (SD). Before exercise noradrenaline concentrations were 3.95 (1.6) nM in the transplant recipients and 2.9 (0.6) nM in the normal subjects. Noradrenaline concentrations were 4.8 (1.7) and 2.8 (0.9) nM, respectively, at 30 watts and 6.2 (1.7) and 3.8 (1.9) nM, respectively at 70 watts. Analysis of variance for repeated measures showed a significant difference between the noradrenaline concentrations in the two groups (p < 0.05), a significant increase in noradrenaline concentrations with exercise (p < 0.001), and a trend for a difference in the way noradrenaline increased with exercise (p = 0.06). The adrenaline concentrations at rest were 0.27 (0.12) nM in the transplant recipients and 0.36 (0.15) nM in the normal subjects. At 30 watts the concentrations were 0.33 (0.2) and 0.36 (0.17) nM, respectively, and at 70 watts 0.38 (0.3) and 0.45 (0.13) nM, respectively. Analysis of variance showed that adrenaline concentrations increased with exercise (p < 0.05) but there was no significant difference between the 2 groups. At maximum exercise noradrenaline concentrations were 15.3 (9) nM in transplant recipients and 12 (4) nM in the normals (p = NS), and adrenaline concentrations were 1.1 (0.9) and 1.9 (0.7) nM, respectively (p = NS).

This study has shown increased sympathetic activity in the transplant recipients before and during exercise.

Colour flow mapping as intraoperative angiography in ventricular septal defects: advantages and pitfalls?

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Fifty one patients undergoing surgical repair of one or more ventricular septal defects (VSD) were studied before and after cardiopulmonary bypass using an epicardial intraoperative technique to determine whether a combination of cross sectional imaging, contrast echocardiography, and colour flow imaging (CFI) could reliably predict (a) successful VSD closure, (b) the presence of further unsuspected VSDs, or (c) any resultant complications. Cross sectional imaging alone detected a minor degree of patch dehiscence in one patient. CFI after bypass showed immediate, well localised, trans patch or peripatch VSD jets in 26/51 patients (multiple jets 16/26). In none of these patients did contrast echocardiography suggest an important interventricular shunt. Early and late postoperative studies showed the disappearance of the narrow jets in 24/26 patients with "VSD closure" almost invariably occurring >6 hours after bypass. In a further four patients broad extensive CFI VSD transpatch or peripatch jets were immediately identified in all of whom contrast echocardiography suggested a small but important interventricular shunt. In none of these cases was bypass recommended as the defect appeared small. After bypass, further important distal VSDs (undetected by preoperative angiography) were noted in two patients, and bypass was recommended to effect closure. Late patch dehiscence (that is, >6 hours after bypass) was clearly defined in a further two patients.

We conclude that a combination of cross sectional imaging, contrast echocardiography, and colour flow imaging can provide the surgeon with a remarkably accurate intraoperative monitoring technique that can both confirm defective closure and exclude the presence of important residual VSDs.