Proceedings of the British Cardiac Society

The 67th Annual General Meeting of the British Cardiac Society was held at the Belfast City Hospital on Wednesday and Thursday, 23 and 24 March 1988. The President, E Sowton, took the Chair during private business. R H Swanton succeeded D J Coltart as Secretary, P J Oldershaw was elected Assistant Secretary, and R W F Campbell was elected to Council on the retirement of D J Rowlands.

Deaths during the year: C W C Bain, C E Drew, R E B Hudson, A Hunter, O Kerr, C Symons, Helen Taussig.

New Ordinary Members 1988: N J Archer (Oxford); E J Baker (London); S G Ball (Leeds); J Beattie (Birmingham); S Campbell (Oxford); K S Channer (Bristol); R Collins (Oxford); J E Creamer (Manchester); A D Cunningham (London); A Davies (Middlesbrough); D W Davies (London); Heather M Dunn (Londonderry); A E Evans (Belfast); I N Findlay (Glasgow); E Jane Flint (W Midlands); M George (N Devon); A H Gershlick (London); B A Gould (Bristol); W J Grabau (Norfolk); L Guvendik (London); A M Heagerty (Leicester); R S Hornung (Glasgow); Patricia J Lowry (Birmingham); J M McLenachan (Glasgow); Jennie M Metcalfe (London); P Nihoyannopoulos (London); J E F Pohl (Leicester); Julia M Polak (London); P M Schofield (Liverpool); M Signy (London); W C S Smith (Dundee); L B Tan (Oxford); Ann C Tweddel (Glasgow); G I Verney (Cambridge); S C Webb (London); D A Wood (Southampton).

New Honorary Members: W Nelligan (USA); A Selzer (USA).

New Corresponding Members: S Furman (USA); M Kyriakidis (Greece).

New Overseas Member: Carole A Warnes (USA).

During the meeting the scientific sessions were held under the chairmanship of D Boyle and the following are abstracts of the papers that were presented.

Preliminary use of a dynamic rotating tip catheter for recanalisation of occluded vessels

M R Rees, A Gehani, P Thorley, D Richens
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Dynamic angioplasty (Kensey catheter) is a new treatment for vascular disease. The system consists of a French 8 catheter with a motor driven rotating cam at its tip that is capable of speeds of 90 000 rpm. The catheter has an inner lumen for the drive shaft and a fluid channel. The fluid is pumped through the catheter as a fine jet that is directed radially by the spinning of the cam. The rotating tip and the fluid jet abrade atheroma and calcified plaque but do not affect elastic tissue. We report the first use of this in the United Kingdom in five men with occlusive peripheral disease (four femoral/popliteal occlusions, one iliac occlusion), in whom conventional angioplasty had been unsuccessful. All five patients have showed significant clinical improvement after dynamic angioplasty. Two patients had complete occlusions recanalised with dynamic angioplasty (11 cm and 16 cm occlusions). One patient had recanalisation with a combination of dynamic and conventional angioplasty; the other two patients had partial recanalisation. In one patient a distal embolus developed eight h after the procedure; this was successfully treated with local streptokinase. Doppler pressures were measured before and after the procedure in four patients and showed improvement. In three of these patients clinical and Doppler improvements were confirmed by measurement of limb blood flow by radionuclide angiography.

Immediate and long term follow up after percutaneous transluminal coronary angioplasty: comparison of stable and unstable angina pectoris

K J Beatt, O Kamp, P W Serruys, P J de Feyter, H Suryapanata, G R Sutherland
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The clinical outcome of the first 896 consecutive patients (1000 lesions) who had percutaneous transluminal coronary balloon angioplasty (PTCA) between September 1980 and November 1985 was assessed. PTCA was performed in 530 patients who
presented with stable angina pectoris (group 1) and in 366 with unstable angina pectoris (group 2). Follow up data were assessed at three different times: within 24 hours of the procedure, within six months, and at the last available follow up (mean 2.1 years) after the procedure. Twenty six per cent of the procedures were done before the introduction of the steerable guiding systems in February 1983. There was no difference in the primary success rate (group 1 83%, group 2 87%) nor in the annual mortality rate, which was approximately 2% per year for both groups. In the group with unstable angina pectoris (group 2) the incidence of acute myocardial infarction within the first 24 hours was higher (9% versus 4%) and this difference persisted at the six month and long term follow up examinations. There was also an increased rate of coronary bypass surgery in group 2 at both six month and long term follow up (17% vs 13%, and 21% vs 15% respectively). There was no difference in frequency of repeat PTCA. At six month follow up 71% of group 1 and 72% of group 2 were event free in terms of death, myocardial infarction, coronary bypass surgery, and repeat PTCA. At long term follow up the percentages were 69% and 61% respectively.

What factors predict recurrent restenosis after coronary balloon angioplasty?

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Restenosis after percutaneous transluminal coronary angioplasty (PTCA) has been much investigated, but scant attention has been paid to those patients who have recurrent (≥2) restenoses of their initial lesions. To determine the frequency of recurrent restenosis after PTCA and the factors that may predict this occurrence we examined the records of 1011 patients undergoing PTCA in our centre from 1 March 1980 to 1 June 1987. Of these patients, 262 (26%) had a single restenosis of one or more of their initial lesions and 38 (3.8%) had recurrent restenosis. We selected a random sample of 125 patients (group A) from among those patients with a single restenosis and compared their characteristics with those of the 38 patients with recurrent restenosis after PTCA (group B). There was no significant difference between the two groups for age and sex distribution, angiina state, clinical presentation at the time of angioplasty, frequency of coronary risk factors, and coronary angiographic fea-

ures. Univariate analysis of procedural factors showed that important predictors of recurrent restenosis were a greater number of inflations (6.3 (3.9) (mean (SD)) in group B patients and 4.9 (2.7) in group A; p < 0.01) and a shorter balloon inflation time (50 seconds (mean) in group B and 64 seconds (mean) in group A; p < 0.06). Neither the maximum balloon inflation pressure nor the balloon size predicted recurrent restenosis.

These data indicate that the recurrent restenosis rate after angioplasty is low and may be reduced by careful attention to procedural details, in particular, by use of fewer but longer inflations.

Assessment of regional wall motion abnormalities during coronary angioplasty by an algorithm of coronary artery shortening: correlation with changes in regional blood flow and myocardial metabolism

R A Perry, P F Wankling, A Seth, A Hunt, S C H Smith, Faith Westwood, J A Newell, M F Shiu
University Department of Cardiology, Queen Elizabeth Hospital, Edgbaston, Birmingham

Balloon occlusion during coronary angioplasty causes ischaemia of the myocardium distal to the balloon and induces abnormalities of left ventricular wall motion. Assessment of this regional contractile dysfunction during balloon occlusion is difficult. We have previously used shortening of the coronary artery branch junction as a measure of myocardial contraction and have now studied changes during angioplasty using a computer assisted algorithm for sequential frame tracking of branching points. We studied 12 men undergoing elective angioplasty to the left anterior descending artery. In addition to measurements of great cardiac vein flow (GCVF), myocardial oxygen consumption (MVO₂), and lactate production (LER) the distal arterial lumen was imaged during the last 10 s of a 60 s balloon inflation by injecting dilute contrast through the distal balloon lumen. The relative maximal percentage shortening of septal branching points through a cardiac cycle during the second balloon inflation was compared with control films. Arteriograms were digitised on an Olivetti M24 PC and a semiautomatic tracking program was used to enter branching points on sequential frames. The mean percentage end diastolic to end systolic branch shortening during control films was 17 (9%). This was significantly lower during balloon occlusion 5 (10%) (p < 0.0001). There was also a significant fall in GCVF...
Angioplasty of total coronary occlusions: improved success rates with the use of a wire splinting technique

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For chronic total coronary artery occlusion recanalisation by percutaneous transluminal coronary angioplasty (PTCA) is increasingly considered. The conventional technique is to explore and cross the occluded segment with the soft steerable wire and to attempt balloon passage across the lesion only if it can be crossed with a wire. The success rate of PTCA of total coronary occlusions is significantly lower than that of non-occluded vessels and the failures are the result of the inability of the soft flexible tip of steerable wire to cross the occlusion. We have developed a new technique of splinting the floppy tip of the steerable wire with the balloon catheter which permits crossing and successful dilatation of a higher proportion of chronic total occlusions. PTCA was performed on 46 totally occluded coronary arteries in a series of 354 elective PTCA procedures. Procedures for acute myocardial infarction are excluded from this series. Successful dilatation leading to revascularisation was achieved in 68% (31/46). There were no major complications. Duration of occlusion was less than two months in 20 patients (group 1), 2-6 months in 11 (group 2), and more than six months in 15 (group 3). The success rate decreased with duration of occlusion: 80% in group 1, 73% in group 2, and 46% in group 3. Until June 1986 only the conventional technique was used (period A) and after June 1986 the “wire splinting technique” was applied if the conventional technique failed. Twenty total occlusions were attempted in period A and 26 in period B. Though the success rates for occlusions of less than two months’ duration in the two periods were not significantly different, the success rates for longer standing occlusions (more than two months) were 3/10 in period A vs 12/16 in period B (p < 0.05).

The wire splinting technique is safe and led to a higher rate of successful revascularisation of long-standing total coronary occlusions than the currently published series. These results also have implications for the design of balloon catheters.

Laser coronary angioplasty under direct vision

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We have developed a system of angioscopy that uses a 2.5 mm flexible angioscope with a working channel for laser angioplasty. The angioscope has an integral coherent optical bundle for visualisation and two working channels. A 400 μm glass fibre is inserted into one channel and is used for illumination. The other channel is for saline flushing. The 400 μm fibre can be attached to an Nd-YAG laser for illumination by the helium-neon aiming beam. The fibre can be manoeuvred into position by extending it from the end of the angioscope once the target has been visualised. It therefore has a dual role—illumination and transmission of laser energy. A video will be shown of the angioscope being used to create a laser channel through a stenosis in the right coronary artery of a postmortem heart.

Coronary artery surgery in the elderly is effective in the relief of angina

J M Morgan, H H Gray, J C Clague, D G Gibson
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Financial constraints may prevent older patients from being considered for coronary artery bypass grafting (CABG). CABG was performed on 315 elderly patients (244 men, age 65-79, mean 68 years) between 1981 and 1986. All patients had limiting angina; 38% had rest pain and 80% had triple vessel disease. There was no impairment of left ventricular function in 46%, mild impairment in 20%, moderate impairment in 23%, and severe impairment in 10% of patients. Grafts (saphenous vein or internal mammary artery) were inserted into three vessels (52%), four vessels (42%), five vessels (6%), and six vessels
Minor changes in QRS configuration during unstable angina: evidence for recurrent myocardial “micro-infarctions”?  

D C Russell, D P de Bono  
Cardiovascular Research Unit, Department of Medicine, University of Edinburgh  

Myocardial “micro-infarctions” can follow peripheral microthromboembolism during unstable angina; their frequency is, however, unknown. Similar size lesions in animals cause minor distortion of QRS configuration on high-fidelity recordings. To determine whether such changes occur in man we compared QRS configurations in high-fidelity vectorcardiograms recorded over three successive days from 24 patients with unstable angina of recent onset (without evidence of infarction on 12 lead electrocardiograms) and 20 patients with stable angina on effort. High resolution QRS complexes were derived by signal averaging of unfiltered Frank lead vectorcardiograms in duplicate over 100 beats by use of a trolley mounted microcomputer system with 1 KHz sampling frequency and 12 bit resolution. Minor distortions of QRS configuration were quantified from “counts” of inflections of the derived instantaneous spatial velocity of the QRS vector loop (that is $\sqrt{(dx/dt)^2 + (dy/dt)^2 + (dz/dt)^2}$). Inflection “counts” ranged from 5 to 27 in individual patients and were highly reproducible in duplicate recordings ($r = 0.99$). The initial distribution of inflection “counts” was similar in the groups with stable and unstable angina (16.7 (3.8) vs 15.6 (4.8) “counts”). Over the three successive days, however, 8/24 (33%) of patients with unstable angina showed either loss of spatial velocity inflections or appearance of new inflections outside the ranges seen in stable angina (mean $\Delta$ “counts” 2.9 (2.9) vs 0.7 (0.9) $p < 0.01$).  

Results suggest that minor changes in QRS configuration consistent with the development of minor areas of myocardial infarction or “micro-infarction” may be detectable in about one third of cases of unstable angina.

Distribution of receptors for calcitonin gene related peptide (CGRP)—a potent vasodilator—in human and guinea pig heart  
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The factors which control coronary artery tone are incompletely understood but is likely that several recently described peptides have a role. One such peptide, CGRP, is a potent vasodilator that has been shown by immunohistochemistry to be abundant in nerves around coronary arteries and is also found in the circulation. CGRP acts on specific receptors located on smooth muscle. Using autoradiographic techniques we have demonstrated the receptor for CGRP in both guinea pig heart and human heart removed at transplantation and have compared the distribution of these receptors in various cardiac tissues. Human cardiac tissue was removed from three patients undergoing transplantation for end stage ischaemic disease. The receptor density in various areas of the heart was compared by counting the binding sites (BS) in 12 separate highly magnified areas from each region studied. In the guinea pig heart the concentration of receptors (> 250 BS/1000 $\mu$m$^2$) in the major coronary arteries and veins was high. Lower concentrations (50–150 BS/1000 $\mu$m$^2$) were found on arterioles, great vessels, and endocardium and no receptors were detected on myocardium or stroma. Similar values were found in human tissue. In a single atheromatous artery the concentration of CGRP receptors resembled that of a normal artery from the same patient but in a thrombosed vessel the concentration was considerably reduced.  

This technique permits localised changes in
Proceedings of the British Cardiac Society

receptor function to be studied in situ and it showed that specific receptors for CGRP occurred in large numbers in the coronary arteries. This evidence may support a role for CGRP in regulation of coronary tone although further studies are necessary.

**Sustained activation of cardiac sympathetic tone in unstable and post-infarction angina**

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The cardiac sympathetic nervous system has a profound effect on the susceptibility of the ischaemic myocardium to serious ventricular arrhythmias, and alterations in cardiac autonomic tone have been implicated in sudden ischaemic cardiac death. We used 1-[3H] noradrenaline (NA) kinetics to evaluate sympathetic activity in 20 patients with ischaemic heart disease: 10 with recent (<12 weeks) unstable or post-infarction angina (group U) and 10 with chronic stable angina (group S). Plasma NA (333 pg/ml in group U, 210 (30 pg/ml in group S) and whole body NA spillover (375 (56 ng/min in group U, 286 (23 ng/min in group S) tended to be higher in group U but not significantly so. In contrast, coronary sinus NA (515 (117 pg/ml in group U, 171 (24 pg/ml in group S, p < 0.001) was increased in the patients with unstable angina. Cardiac NA spillover, which reflects cardiac sympathetic tone, was also greatly increased in the patients with unstable angina (24.5 (6.9 ng/min in group U, 6.1 (1.3) ng/min in group S, p < 0.001). Coronary sinus plasma flow and cardiac NA clearance were similar in both groups.

This study shows that patients with unstable ischaemic symptoms, who are known to be at increased risk of sudden cardiac death, have evidence of a sustained and specific increase in cardiac efferent sympathetic tone. This supports the theory that sympathetic overactivity is an important factor in causing serious ventricular arrhythmias in ischaemic heart disease.

**Do the investigation for and treatment of oesophageal abnormalities improve the outcome of patients with angina pectoris, normal coronary angiograms, and normal left ventricular function?**

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A postal questionnaire was used to assess the symp- toms, use of medical facilities, and employment state of patients with angina pectoris and normal coronary angiograms after cardiac catheterisation. In a retrospective study of 187 patients, 66 had left ventricular dysfunction (LVD) shown by abnormal regional wall motion and 121 had normal left ventricular function (NLV). At follow up 12–46 months after catheterisation 89% of patients with LVD and 82% of those with NLV continued to experience chest pain. There was no significant change in the rate of admission to hospital because of chest pain or in the proportion of patients who were working after compared with before catheterisation in either group. In a prospective study of 63 patients detailed investigation of oesophageal function was performed. Twenty two patients had LVD. Most of the 41 patients with NLC had oesophageal abnormalities (gastro-oesophageal reflux or oesophageal dysmotility, or both), which were treated appropriately. At follow up 6–24 months after catheterisation significantly fewer patients with NLV than patients with LVD continued to experience chest pain (67% v 90%, respectively; p < 0.05). The rate of admission to hospital fell significantly after catheterisation in patients with NLV (mean 0.068 (SD 0.076) to 0.008 (0.022) admissions/month; p < 0.01) but not in those with LVD (0.065 (0.077) to 0.047 (0.047) admissions/month). The proportion of patients working after catheterisation increased significantly in those with NLV (from 49% to 74%; p < 0.05) but not in those with LVD (from 52% to 48%).

The findings suggest that investigation for and treatment of oesophageal abnormalities improves the symptoms and employment state of patients with angina pectoris, normal coronary arteries, and NLV and reduces their use of medical facilities.

**Free radical generation during pacing induced myocardial ischaemia**

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Free radical generation occurs in experimental models of myocardial ischaemia and reperfusion and may cause cellular damage by peroxidation of membrane phospholipids. To determine whether free radical generation occurs during transient myocardial ischaemia in man we measured aortic and coronary sinus concentrations of malondialdehyde (MOA), an end product of lipid peroxidation. Twenty two patients (16 male) undergoing diagnostic coronary arteriography were studied during incremental atrial
(or ventricular) pacing until the development of severe chest pain, a 2 mm ST segment depression, or a paced rate of 180 beats/min. Group 1 (n = 11) produced lactate during pacing, whereas group 2 (n = 11) continued to extract lactate throughout the study. All patients in group 1 and four in group 2 had coronary artery disease. Coronary sinus MOA values (median (interquartile range)) in group 1 were significantly increased from baseline at peak pacing rate (0.116 (0.112 to 0.126) v 0.120 (0.117 to 0.134); p = 0.035); at 30 seconds after pacing (0.129 (0.115 to 0.131); p = 0.032); and at 5 minutes (0.134 (0.126 to 0.143); p = 0.036). No differences with pacing were observed in the coronary sinus MOA values in group 2 or in the aortic MOA values in either group.

These results suggest that intramyocardial free radical generation occurs during transient myocardial ischaemia in man.

Relative efficacy of various physical manoeuvres in termination of re-entrant supraventricular tachycardias

D Mehta, K Channer, I Crozier, M Griffith, D E Ward, A J Camm
St George’s Hospital Medical School, London

Physical manoeuvres are often attempted to terminate supraventricular tachycardia (SVT). We studied the relative efficacy of four different manoeuvres in terminating induced SVT and related the findings to the changes in heart rate caused by the manoeuvres in sinus rhythm. Twenty five patients (16 men) of mean age 36 years (range 16–69) with inducible SVT were studied. SVT was induced by programmed stimulation. The response to right and left carotid sinus massage for 5 s each, face immersion in water at 5–19°C, and the Valsalva manoeuvre (VM) for 15 s (blowing at pressure of 35 mm Hg) were measured with continuous electrocardiographic recording at 50 mm/s during both sinus rhythm and SVT. If SVT was terminated it was reinitiated and the manoeuvre repeated two times to evaluate consistency. VM was most successful in terminating SVT (13 out of 25), especially when the patients were supine (13 out of 25 compared with two out of 25 standing; p < 0.001). Right carotid massage was effective in four patients, left carotid sinus massage in one, and face immersion in three. VM was more effective in younger patients (median = 29 years (16–44) v 44 years (17–69) conversions and non-convertions, respectively, p < 0.02) and in atrioventricular tachycardia (12 out of 18) compared with atrioventricular nodal tachycardia (one out of seven; p < 0.03). During sinus rhythm the RR interval increment expressed as a ratio was greatest with the VM (1.74 (0.38)) and progressively less with face immersion (1.26 (0.27); p < 0.001 when compared with VM), right carotid massage (1.25 (0.32), and left carotid massage (1.1 (0.17)). The VM ratio was higher in those in whom the manoeuvre successfully terminated SVT (1.88 (0.39) v 1.58 (0.32); p < 0.06).

In conclusion, VM was the most successful physical manoeuvre in terminating induced re-entrant SVT. It was more commonly effective in younger patients and in those with atrioventricular re-entrant tachycardia. The heart rate response to the VM during sinus rhythm related weakly to its effectiveness in terminating SVT.

Identification of major arrhythmias by retrospective patient activated monitor after negative conventional Holter recordings

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The problem of identifying (or excluding) a cardiac arrhythmia as the cause of transient symptoms has not been solved by 24 hour Holter electrocardiographic tape recordings (HTR) except in those patients in whom the episodes are frequent enough to occur during the recording. Moreover, asymptomatic arrhythmias detected on HTR may be treated, often without relieving the symptoms and sometimes leading to complications. A new alternative, the retrospective patient activated monitor (RPAM), can be used continuously for two weeks or longer if the battery is charged. The electrocardiogram (ECG) is stored in digital form by continuously overwriting a circular solid state memory. This stops after the device is activated by the patient so that 150 seconds of retrospective and 10 seconds of prospective ECG is saved and can be replayed on a standard ECG machine. We have used RPAM on 27 patients in whom HTR had failed to capture a symptomatic episode. In 10 patients no attack occurred during 2–8 weeks of investigation. In six patients the typical symptoms were associated with normal sinus rhythm, mild sinus tachycardia (up to 120 beats/min), or appropriate VDD (atrial synchronous) pacing. In a further five patients minor arrhythmias (atrial or ventricular extrasystoles) were recorded that required no treatment apart from reassurance. Major arrhythmias were identified in six patients: sinus arrest or severe bradycardia, or both, in two; paroxysmal supraventricular tachycardia in
three; and ventricular tachycardia in one. Repeat studies were performed in five patients: in two because the lead or electrode, or both, had become disconnected; in one because of inadvertent activation; and in three because the attacks were considered to be not quite typical. In all five patients the second result was the same. Diagnostic information was thus gained in 17 out of 27 patients (63%).

We conclude that the RPAM is a valuable tool and may displace HTR as the primary investigation for patients with suspected paroxysmal arrhythmia.

Rapid exhaustion of atrial natriuretic peptide during sustained tachycardia

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To assess the ability of the atria to maintain raised plasma concentrations of atrial natriuretic peptide (ANP), the temporal changes in plasma ANP concentrations were studied during four hours of sustained rapid cardiac pacing in seven dogs anaesthetised with chloralose. Heart rate increased from (mean (SEM)) 124 (26) to 278 (28) beats/min during the four hours of rapid cardiac pacing. At 30 minutes pulmonary wedge pressure increased from 3-6 (1-8) to 17-4 (7-1) mm Hg (p < 0-01) and mean right atrial pressure rose from -1-7 (1-9) to 2-0 (2-8) mm Hg (p < 0-01). Both pressures remained constant at these raised values for the entire 240 minutes of rapid pacing. Arterial ANP concentrations increased from 87 (11) pmol/l to a maximum of 1263 (592) pmol/l at 30 minutes (p < 0-01), falling to 411 (42) pmol/l after 60 minutes and 146 (70) pmol/l after 240 minutes of rapid continuous pacing (p < 0-01 compared with value at 30 minutes). Coronary sinus ANP concentrations showed a similar pattern, rising from 241 (79) to a maximum of 1837 (203) pmol/l after 30 minutes (p < 0-01). These peak values likewise were not sustained, falling to 962 (198) pmol/l after 60 minutes and 297 (41) pmol/l after 240 minutes of rapid pacing (p < 0-01 compared with value at 30 minutes).

We conclude that atria are unable to maintain the peak concentrations of ANP reached after 30 minutes of rapid pacing despite persistently raised atrial pressures. Sudden sustained increases of atrial pressure, as occur during acute myocardial infarction, may rapidly exhaust stores of ANP with the loss of its natriuretic, vasodilator, and renin inhibiting actions.

Successful treatment of supraventricular tachycardia with an antitachycardia pacemaker: European results with Orthocor II

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As part of a prospective study conducted at 10 European evaluation centres, 24 patients (6 men, 18 women; age 35–78 years) with resistant supraventricular tachycardia (SVT) were treated with Orthocor II, an implantable antitachycardia device with additional “on board” Holter and non-invasive electrophysiological facilities. Devices were implanted between June 1984 and January 1987 after full electrophysiological study, with at least 20 tachycardia inductions and terminations by electrostimulation. Tachycardia was also repeatedly initiated and terminated automatically by the pacemaker before discharge. Data for these procedures and for up to 24 months’ follow up were collected.

Mechanism of SVT was atrioventricular nodal re-entry in eight patients, Wolff-Parkinson-White syndrome in four, accessory bypass tract in four, and not further specified in eight. Rate of SVT was 130–240 (mean 181) beats/min with an estimated 1–300 (mean 5-3) occurrences a week. Endocardial lead position was atrial in 21 patients and ventricular in three. Tachycardia recognition criteria were 4–15 (mean 6-3) beats counted, at 120–180 (mean 145) beats/min. The automatic antitachycardia mechanism selected was burst rate scanning (20 patients) and automatic burst (one), with 1–10 (mean 5-9) extrastimuli used. Automatic overdrive was used in the remaining three patients. After implantation, the duration of tachycardia was reduced from 5–500 (mean 126) minutes to 6–130 (mean 31) seconds. The patient’s condition was judged to have improved in 18 out of 24 (not specified in the six others), with drug treatment being reduced or eliminated in 17 (not specified in five patients). Discriminating sinus tachycardia from SVT was a problem in one patient with a low rate of tachycardia. No episodes of acceleration of tachycardia were reported; there were no explantations and no pacemaker related deaths.

This study confirms that implantable antitachycardia pacemakers provide a highly effective means of treating resistant SVT and that Orthocor II is itself a safe and reliable device.

Does the site of origin of ventricular tachycardia predict the presence of late potentials?

M J Griffith, D Mehta, A J Camm
St George’s Hospital Medical School, London
In 20–40% of patients with inducible ventricular tachycardia (VT) no late potentials are detected in sinus rhythm. This may be owing to the early activation of the VT origin in sinus rhythm and the late potentials being hidden. This study examines whether the site of origin of the tachycardia, as determined by its axis and morphology, is associated with the detection of late potentials. Thirty two patients (mean age 54 years) with VT were studied. Patients with bundle branch block (BBB) in sinus rhythm were excluded. Late potentials were recorded in sinus rhythm with the standard Simpson technique using orthogonal leads (X, Y, and Z). A test was positive if the filtered QRS was greater than 120 ms or the root mean square of the last 40 ms was less than 25 μV, or both. Late potentials were detected in 10 out of 16 patients with ischaemic heart disease, seven out of 14 with morphologically normal hearts, and one of the two patients with congestive cardiomyopathy. Late potentials were present in eight out of 17 patients with left BBB pattern VT and 10 out of 15 with right BBB pattern VT. Multiple morphologies were present in eight patients, four of whom had late potentials. None of these differences approached significance, but 11 out of 13 patients with a superior axis and six out of 17 with an inferior axis during VT had late potentials (p < 0.01). Both axes were present in two patients, one of whom had late potentials. This pattern persisted when the patients with and without ischaemic heart disease were analysed separately, though it only remained significant for patients with ischaemic heart disease (p < 0.05).

To conclude, this study shows that patients with an inferior axis during VT, especially if they have ischaemic heart disease, are much less likely to have late potentials detected in sinus rhythm. This suggests that the site of origin of VT, whether superior or inferior, is important in determining the presence of late potentials.

**Relative relevance of right and left ventricular biopsies in patients with ventricular tachycardia and clinically normal hearts**

D Mehta, J R Dawson, D E Ward, M J Davies, A J Camm
St George’s Hospital Medical School, London

A proportion of patients (10–15%) with ventricular tachycardia (VT) have clinically normal hearts. It has been suggested that endomyocardial biopsy may help to identify the underlying pathological process in such patients. Although right ventricular (RV) and left ventricular (LV) biopsies are usually performed, both procedures are associated with an unmeasurable risk. We have examined the relative relevance of performing RV and LV biopsies in patients with VT and clinically normal hearts. Twenty two patients (15 men, median age 45 years (range 19–70)) with documented VT were studied. The results of a chest x ray film, the QTc interval, left ventricular wall motion from angiography, and coronary arteries were normal in all patients. The configuration of the clinical VT was left bundle branch block pattern in 16 patients, right bundle branch block pattern in four, multiform in one, and indeterminate in one. Biopsy specimens were obtained by means of a Cordis biotome, and an attempt was made to obtain three specimens from each ventricle. They were reviewed by a pathologist unaware of the configuration of the VT. The average number of biopsies was 2.6 from the right and 2.7 from the left ventricle. Nine patients (40%) showed abnormalities in the RV biopsy specimens, seven of the nine also showing abnormalities in the LV biopsy specimens. No patient showed abnormalities in the LV biopsy specimen alone. The pathological findings from light microscopy were non-specific and included endocardial thickening; subendocardial interstitial and perivascular fibrosis; and cellular hypertrophy, atrophy, and vacuolation. Of the four patients with RBBB configuration of VT, three showed no abnormality in both biopsy specimens, and one showed abnormalities in both RV and LV biopsy specimens.

We conclude that a substantial proportion (40%) of patients with VT and clinically normal hearts show abnormalities in endomyocardial biopsy specimens and that LV biopsy in this group of patients confers little more information than that which can be obtained from RV biopsy.

**Preoperative and intraoperative colour flow mapping in the definition of high velocity continuous shunting in the heart**

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Continuous high velocity intracardiac shunting of blood is a comparatively rare finding in cardiology. Physical examination, duplex scanning, and angiography are all likely to give suboptimal information in such cases. Potentially, colour flow mapping (CFM) can determine the origin and extension of such a disturbance of flow by encoding turbulence,
thus providing optimal diagnostic information in these complex lesions. Eighteen patients (seven ventricular septal defect/aortic regurgitation; five acquired Gerbode defects; two coronary sinus fistulas; four coronary artery/right heart communications) were studied. Seventeen patients had correlative angiography and 12 had had subsequent surgical confirmation of the defect. Cross sectional echocardiography alone showed the morphology in only seven patients. Continuous wave Doppler recorded high velocity continuous flow in all 18. Pulsed Doppler subsequently failed to predict the exact nature of the intracardiac shunt in nine patients. In contrast, CFM correctly defined the nature of the complex flow disturbance in all 18 patients (multilevel shunts being clearly shown in 13 patients in whom this was present). Angiography failed to adequately define the precise nature of the shunt in nine out of seventeen patients, being notably poor in distinguishing ventricular septal defect/aortic regurgitation from coronary sinus fistulas. Of the 12 patients undergoing corrective surgery, five had intraoperative CFM studies carried out to confirm the repair of the defect. In every patient surgical repair was shown to be effective by the absence of a significant residual flow disturbance.

We conclude that CFM is the investigative technique of choice in defining complex high velocity continuous intracardiac shunts and for the intraoperative assessment of their repair.

Improved ultrasound diagnosis of coarctation with colour Doppler flow mapping

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A duplex echo/Doppler system with colour Doppler flow mapping (CDFM) (Vingmed CFM 500) was used to determine the site and nature of the lesion in a consecutive series of 26 patients (seven newborns, four older infants, 15 older children) who presented with coarctation of the aorta and were being referred for operation. The site was clearly shown in 10 of the 11 infants but in only eight of the 15 older patients. CDFM showed increased velocity at the site of the coarctation in 24 of the 26 patients. In patients aged less than 1 week the increased velocity was usually from flow into the descending aorta through the ductus and not the coarctation site; in patients in whom the coarctation had already been shown CDFM confirmed the site; and in four of the most recent patients, in whom coarctation was not clearly shown, the superimposition of the colour display on the ultrasonographic image allowed the position and nature of the lesion to be appreciated and surgery to be undertaken without catheterisation.

CDFM adds considerably to the diagnostic capability of ultrasonography in coarctation of the aorta and obviates the need for angiography in many patients in whom imaging echocardiography fails to delineate the exact site and nature of the lesion.

Indium-111 labelled monoclonal antimyosin antibody for the localisation of transmural myocardial infarction

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The role of indium-111 labelled monoclonal antimyosin antibody (AMA) binding fragment in localising myocardial necrosis was assessed in 13 patients with transmural myocardial infarction, eight of whom had received intravenous streptokinase. AMA was injected within 48 hours after the onset of chest pain. Images were obtained at 24 and 46 hours in three projections, and uptake of AMA was graded in intensity (0–4) by two independent observers. In all patients the images correlated with the site of Q waves on the electrocardiogram. Four images showed more widespread myocardial necrosis than indicated on the electrocardiogram. Eleven images showed no change in extent or intensity between 24 and 48 hours. Three "doughnut" images occurred with anterior myocardial infarction. The eight patients who received streptokinase (one million units intravenously) within 4 hours of the onset of pain nevertheless developed transmural myocardial infarction by electrocardiographic criteria. The image grade for those receiving streptokinase (mean 3±1 (SD 0.6) v 2±2 (0.4); p < 0.05) was greater than for those who did not. In a further five patients with chest pain, normal cardiac enzyme activities, and normal results from serial electrocardiograms the AMA images were negative. There were no adverse effects from injection of AMA.

In conclusion, AMA is an effective agent for localising myocardial infarction and in some patients indicates the presence of myocardial necrosis beyond the electrocardiographic changes. In those patients with a myocardial infarction despite receiving streptokinase the areas of myocardial necrosis produced more intense uptake of AMA. AMA images were clearly interpretable and provided a new way of
assessing the site and extent of myocardial infarction.

Magnetic resonance imaging of coarctation of the aorta in infants

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The aortic arch was studied in eight infants (aged 5 days to 10 months) with coarctation of the aorta by magnetic resonance imaging. A 1.5 Tesla, high field strength, imaging system was used. Infants were positioned inside the adult head coil and multiple, simultaneous, 5 mm thick sections were acquired, which were gated by the electrocardiogram. A spin echo sequence was used. The imaging plane was, if necessary, oblique to standard planes of the body. Imaging was successful in every infant. Sedation with chloral hydrate was required in most of the infants. The area of narrowing was imaged in at least two, and usually three, planes in each infant. The relation of the coarctation to the left, or in one infant the right, subclavian artery was clearly shown, as was the aortic isthmus. All patients have subsequently undergone operative correction and the diagnosis confirmed. In a further four infants coarctation was suspected clinically and not excluded by cross sectional and Doppler echocardiography, but it was excluding by magnetic resonance imaging.

Magnetic resonance imaging is an important advance in the non-invasive imaging of aortic coarctation.

Acute effects of disopyramide, mexiletine, and flecainide in patients with impaired ventricular function assessed by nuclear angiography

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Antiarrhythmic drugs may precipitate cardiac failure in patients with severely compromised ventricular function. Many patients with arrhythmias have moderately reduced cardiac function. To assess the acute effects of class 1 antiarrhythmic drugs on cardiac function in patients with impaired left ventricular function we performed a double blind crossover study on nine men and one woman (mean age 57 years). All 10 of them had suffered myocardial infarction and arrhythmias and four had coronary artery bypass surgery. First pass radionuclide ventriculography was performed with either gold-195 m or technetium-99m before and after each drug. Disopyramide, mexiletine, and flecainide, 1-5 mg/kg, were given to each patient by intravenous infusion in random order on three separate occasions. Electrocardiograms and blood pressure were monitored throughout each study. Mean resting left ventricular ejection fraction (LVEF) was 37-0 (7-7) (range 27-48%). After disopyramide mean LVEF was reduced to 29-0 (7-2) (p < 0-01 compared with control); after mexiletine it was 33-3 (7-4) (p < 0-05) and after flecainide 31-6 (9-6) (p < 0-025). In the three patients with initial LVEF values of less than 30% the fall in LVEF was similar in magnitude after each drug. No drug had any significant effect on heart rate or blood pressure, and no patient developed overt heart failure. Mean serum concentrations of the drugs were disopyramide, 3-3 (0-7) mg/l; flecainide, 288 (132) µg/l; and mexiletine, 0-31 (0-29) mg/l.

Thus disopyramide and flecainide produce a similar reduction in resting LVEF values, whereas mexiletine has a significant but less pronounced effect with the same dose.

Can pulsed Doppler replace direct measurement of left ventricular end diastolic pressure?

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It has been claimed that simple measurements from pulsed Doppler recordings of left ventricular (LV) diastolic filling allow accurate and reliable estimates of left ventricular end diastolic pressure (LVEDP). This has important implications for acute coronary care, where decisions about treatment are often based on indirect pressure measurements. Simultaneous pulsed Doppler recordings of left ventricular pressure and diastolic inflow velocities were made in 32 patients with ischaemic heart disease. A multichannel recorder provided superimposed pressure, pulsed Doppler, electrocardiogram, and phonocardiogram traces, which allowed accurate timing and unambiguous identification of the events in each diastolic period. Micromanometer tipped catheters were initially used to calibrate the fluid filled pressure recording system. The range of LVEDP was 7-25 mm Hg. Computer assisted analysis of the Doppler flow patterns showed no correlation between the early (E) or late (A) diastolic flow velocity peaks or the E to A ratio (range 0-27-1-9) with the
simultaneously recorded LVEDP. A weak positive correlation was found, however, between the mean deceleration from the peak initial velocity and the LVEDP (r = 0.51). Although characteristic abnormalities of left ventricular filling are detected by pulsed Doppler recording in ischaemic heart disease, they do not relate in a simple quantifiable way to LVEDP, which in itself correlates poorly with other indices of impaired ventricular function.

Pulsed Doppler recording of left ventricular filling as an isolated measurement does not provide a clinically useful alternative to direct pressure measurement in patients with ischaemic heart disease.

Follow up of patients after balloon dilatation of the aortic valve

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The long term outcome of balloon dilatation of the aortic valve (BAV) for aortic stenosis is uncertain, the development of symptomatic restenosis being a significant hazard. We followed up nine patients in whom BAV had been performed for relief of severe aortic stenosis for up to one year (mean nine months) using Doppler velocity measurements to assess aortic valve gradient. All patients had acquired aortic stenosis. Two patients had bicuspid aortic valve, one rheumatic aortic stenosis, and six senile calcific aortic stenosis. Mean transvalvar gradient at BAV was reduced from 80 mm Hg (range 50–120) to 51 mm Hg (range 20–70). Non-invasive follow up assessment of aortic gradient was made at six weeks and repeated at up to 12 months after BAV. Mean gradient at six weeks was 46 mm Hg (range 30–70) and at six months 50 mm Hg (range 35–80). Before BAV all patients were in New York Heart Association (NYHA) class III or IV. After BAV all patients reported improvement in symptoms with incidence of angina reduced from 30% to 20% at 6 months and 10% at 1 year.

Controlled clinical trial of prednisolone in treatment of 240 patients with tuberculous pericardial effusion

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Tuberculous pericarditis is common in Transkei. To assess the effect of steroids as an adjunct to anti-TB treatment in pericardial effusion 240 patients with active effusion were randomised. All received the same six months course of anti-TB treatment. Those willing and suitable were randomised either to open drainage on admission or to closed pericardiocentesis as required, which was also performed as necessary in those unsuitable for open drainage. All patients were allocated to receive either prednisolone or matching placebo for the first 11 weeks of treatment. Mycobacterium tuberculosis was cultured from pericardial fluid in 57% of 188 patients, and characteristic histology was present in biopsy specimens of 72% of 33 patients. As assessed at 24 months, complete drainage on admission abolished the need for subsequent pericardiocentesis (p < 0.01) but did not diminish the risk of death or the need for subsequent pericardectomy for constriction. Among patients who did not have drainage on admission, two out of 76 (3%) given steroids, died of pericarditis compared with 10 out of 74 (14%) given placebo (p < 0.04); six (8%) and eight (11%) required pericardectomy, six (8%) and 17 (23%), p < 0.02) pericardiocentesis, and one (1%) and six (8%) open surgical drainage respectively. Open drainage on admission did not influence favourable state at 24 months, but 73 of the 76 (96%) patients given steroids had a favourable status compared with 62 (84%) given placebo (p < 0.03).

It is therefore recommended that in the absence of specific contraindication, anti-TB treatment should be supplemented initially by corticosteroids in the treatment of tuberculous pericardial effusion.

Captopril has a direct negative inotropic effect on isolated myocardium

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Angiotensin converting enzyme inhibitors (ACE) have been successful in the treatment of heart failure
and are under investigation for use in myocardial infarction. Not all the actions of ACE inhibitors can be explained by their effect on circulating angiotensin II. Isolated strips of human right atrial muscle (H), obtained from patients undergoing cardiopulmonary bypass surgery, and guinea pig papillary muscles (GP) were mounted to contract isometrically at 1 Hz in physiological solution at 37°C. These were exposed to incremental doses of captopril of 100–800 ng/ml. A dose related negative inotropic action was detected (isometric tension with 800 ng captopril as proportion of control in H, 54 (14)% (n = 5); GP, 48 (12)% (n = 7)). The negative inotropic effects of 800 ng captopril were attenuated by 2.5 μM adrenaline (H, 62 (20)% (n = 3); GP, 72 (12)% (n = 5)). In both human and animal muscles atenolol (2 μg/ml) did not affect the negative inotropic action of captopril. A considerable effect on the recirculation fraction was observed when captopril was added to muscles superfused with adrenaline, which suggested an antiadrenergic effect. The recirculation fraction reflects myocardial inotropic state and is consistently increased by adrenaline (H, 0.63 (0.11) to 0.98 (0.21) (n = 3); GP, 0.46 (0.13) to 0.94 (0.30) (n = 5)). At the lowest doses of captopril investigated (100 ng/ml) the increase in the recirculation fraction produced by adrenaline was reversed (H, 0.56 (0.10); GP, 0.67 (0.14)).

These results show that captopril has a direct, non-beta receptor mediated antiadrenergic effect on human myocardium. The mechanism of this action remains to be explained.

Analysis of left ventricular function during follow up after intravenous streptokinase treatment of myocardial infarction

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Thrombolytic treatment during early acute myocardial infarction results in an early improvement in left ventricular function. In this study the long term effects of thrombolysis on left ventricular function were examined. Serial measurements of cardiac function were made on 64 patients treated with streptokinase (1·5 million units intravenously). Two groups of patients were followed up. Group 1 comprised 45 patients who received standard medical treatment after thrombolysis: twenty eight had an open and 17 a closed infarct related coronary artery at postinfarct angiography. Baseline and follow up radionuclide ventriculograms were examined at a mean 2·4 weeks and 11·9 months after treatment, respectively. Patients with anterior infarction and an open artery had a better baseline ejection fraction in the region of the infarct than those with a closed artery (34·1% (SD13) v 24·6% (10)). This initial benefit was lost at follow up (29·9% (10) v 30·7% (14)). No difference in regional ejection fraction was observed in patients with inferior infarction. Group 2 consisted of 19 patients who had successful reperfusion followed by percutaneous transluminal coronary angioplasty to a lesion of the infarct artery (mean stenosis 71·5% (7)). Contrast ventriculography was performed at baseline (6·4 (2) days) and follow up (16·7 (6) months) to evaluate a regional wall motion index. In 10 patients with anterior infarction regional akinesis decreased from 76·3 (84) to 34·3 (68) at follow up. No decrease in akinesis was seen with inferior infarction.

These data suggest that coronary reperfusion produces its main functional benefit in patients with anterior infarction. This improvement is maintained during follow up in patients who receive percutaneous transluminal coronary angioplasty but may be lost in patients who are followed by conventional medical treatment.

Balloon dilatation for pulmonary stenosis in older patients

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Percutaneous balloon dilatation of the pulmonary valve (PBVPV) was performed on four older patients (three men, age range 56–70 years) with congenital pulmonary valve stenosis. All patients presented with dyspnoea (New York Heart Association grade III), including one patient with cardiac tamponade from a pericardial effusion which was relieved before PBVPV. All patients had pulmonary systolic murmurs. Two patients also had ostium secundum atrial septal defects, and one had a membranous ventricular septal defect, although no shunts were shown on oximetry. PBVPV was performed with a single 18 mm or 20 mm balloon and was well tolerated and free of complications. The transvalvar gradient fell from a mean (SD) of 79 (14·1) to 25 (10·8) mm Hg (p < 0·01). At three months all patients were symptom free and the mean transvalvar gradient was 28 (11·5) mm Hg. No increased shunt was shown by oximetry in those patients with
Preparation of human saphenous vein for coronary artery bypass grafting impairs its capacity to release endothelium derived relaxing factor

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Intraoperative damage to saphenous vein grafts may contribute to both their early and late occlusion, and platelet activation may be involved in both processes. We investigated whether routine surgical preparation of saphenous vein led to impaired release of endothelium derived relaxing factor (EDRF), which would favour adhesion and activation of platelets. Veins were perfused at 23°C at 2 ml/min and bradykinin (10^{-6}M) stimulated release of EDRF into the perfusate was measured by its ability to relax a preconstricted (5-hydroxytryptamine 3 x 10^{-6}M) de-endothelialised porcine coronary artery. The maximum relaxation (expressed as a percentage of preconstricted tone) induced by stimulated release of EDRF was 60 (SEM 15%) (n = 5) in freshly isolated vein obtained by a no touch technique as soon as possible after the first incision and stored in heparinised blood for up to 60 minutes. It was 19 (3%) (n = 11) in vein obtained at the end of the surgical procedure (surgically prepared vein) (p < 0.01). Relaxation induced by EDRF from freshly isolated vein that had been stored for 80–160 minutes declined to 25 (5%) (n = 6), which was similar to that induced by EDRF from surgically prepared vein.

This study shows that routine surgical preparation reduced the capacity of the human saphenous vein to release EDRF. It also suggests that prolonged storage in heparinised blood may contribute to this inhibition.

Clinical experience with a minute ventilation sensing rate responsive pacemaker

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Minute ventilation is linearly related to oxygen consumption and is used as an indicator to determine the rate in rate responsive pacing. The Meta pacemaker (Teletronics) senses minute ventilation by measuring thoracic impedance with a standard intravenous bipolar pacing electrode. Programmable rate
responsive variables include rate limits (50–180 beats/min) and slopes of rate response (1–60). This pacemaker was implanted in 11 patients (complete heart block in nine, sinoatrial disease in two) with a mean age of 59 (SD 14) years. Programming was achieved with the help of suggested slopes during a submaximal exercise test (range of slopes programmed, 16–25). Treadmill exercise with the Bruce protocol performed in the rate responsive mode showed improvement in the duration of exercise over fixed rate pacing (608 (177) v 450 (128); p < 0.001).

Cardiac output measured by continuous wave Doppler was improved by 44 (20%). Appropriate rate responses to different workloads such as walking at different speeds and slopes were achieved. Ascending four flights of stairs resulted in a higher rate response than that on descending (110 (24) v 77 (5); p < 0.01). With activities like walking, rate response occurred at 24 (16) s and half of the maximum rate was attained in 92 (30) s. Voluntary effects on respiratory pattern can affect the pacing rate: talking continuously during a three minute treadmill exercise attenuated the rate response (from 123 (26) to 96 (22) beats/min; p < 0.001) and coughing for one minute increased the pacing rate (87 (8) beats/min).

It is concluded that the Meta pacemaker improves exercise capacity and cardiac output during exercise and its rate response is related to workload. Voluntary interference modified the response but excessive rate acceleration was not encountered.

Absent baroreceptor response in haemodialysed patients with chronic renal failure and good ventricular function

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Both autonomic and left ventricular dysfunction have been postulated as the cause of the serious hypotension that is a frequent complication of haemodialysis (HD). In 10 patients with end stage chronic renal failure who were being maintained on HD blood pressure was monitored continuously by an intra-arterial ambulatory technique over 24 hours before HD and continuing during it. Autonomic function was tested with the Valsalva manoeuvre, and technetium-99m gated radionuclide ventriculography was performed at the beginning of the 24 hour recording to assess left ventricular function. On the day of dialysis the ejection fraction was measured immediately before HD and every 30 minutes during it. The day and night variations in blood pressure and heart rate were significantly reduced in nine of the patients with chronic renal failure (blood pressure 12/7 (8/6) mm Hg, heart rate 6 (4) beats/min) compared with a control population (blood pressure 36/28 (10/5) mm Hg, heart rate 23 (9) beats/min) and all 10 patients had a classic "square wave" response to the Valsalva manoeuvre. Despite these indications of baroreceptor failure, which are usually seen in patients with severe cardiac failure, all 10 patients had a normal or high resting ejection fraction (mean 66%, range 55–79%) and peak filling rate (mean 3–1 end diastolic volume, range 2–7 to 5–3). They experienced a progressive fall in blood pressure during HD (mean maximal fall 42/21 (15/11) mm Hg) but their heart rate rose little or not at all (4.5 (5) beats/min) despite the volume depletion and hypotension. Nor was there any significant fall in ejection fraction during dialysis.

These results suggest that hypotension during HD is caused by a failure of the baroreceptor response to volume depletion. There is, however, no evidence of left ventricular dysfunction due to a uraemic cardiomyopathy, as has been postulated.

Why is atrioventricular block an ominous prognostic sign in myocardial infarction?

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Atrioventricular block is associated with a higher short term mortality in acute myocardial infarction, but it is not certain if this effect is independent of other prognostic factors such as infarct size. We studied 705 successive patients admitted with a first Q wave myocardial infarction, of which half were anterior, to determine the factors predisposing towards atrioventricular block and the relation of block to subsequent mortality. Second or third degree block developed in 61 (8.8%) patients. It was commoner in inferior (12.4%) than anterior (4.9%) infarction. A multiple logistic regression identified three factors as being independently correlated with block: inferior infarction, older age, and higher peak creatinine kinase activities (all p < 0.01). Mortality was 9.3% in patients without block and 27.9% in those with block. Mortality was significantly higher in both anterior (47.0% v 11.8%) and inferior (20.4% v 6.7%) infarction. When age, peak creatinine kinase activity, infarct site, and block were analysed simultaneously as predictors of death block
was a significant independent prognostic factor, as were age and site, whereas peak creatinine kinase activity was not associated with death independently of the other three factors. The relative risk of death, corrected for age and creatinine kinase activity, was 4.57 in anterior infarction and 4.5 in inferior infarction.

The relative increase in the risk of death associated with atrioventricular block is as large in inferior infarction as it is in anterior infarction. Its prognostic effect is independent of age and not due to its correlation with infarct size: even in a small inferior infarction block appears to be an ominous prognostic sign.

Importance of stress testing in apical hypertrophic cardiomyopathy

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Apical hypertrophic cardiomyopathy (AHC) is a subgroup of hypertrophic cardiomyopathy characterised principally by unremarkable physical examination and electrocardiographic changes that may simulate myocardial ischaemia or athlete's heart. We studied 20 patients (mean age 50 years, range 24–76; 13 men) with clinical and echocardiographic features of AHC. Six had giant negative T waves (more than 10 mm) whereas 14 had T wave inversion ranging from 3–8 mm. Time course patterns of ST segment and T wave changes that occur with angiographically normal epicardial coronary arteries were shown. In nine patients the ST segment depression and T wave inversion improved immediately after exercise and returned to the pre-exercise appearances 3–5 minutes after exercise. In another nine patients the ST and T wave changes worsened immediately after exercise but returned to control values 3–5 minutes after exercise. Neither of these time course patterns is compatible with ischaemia, but the former pattern is also encountered in athlete's heart. Coronary arteriography performed in 15 of the 18 patients yielded normal results. The remaining two patients showed a time course pattern associated with ischaemia in that the ST and T wave changes worsened immediately after exercise and remained abnormal for 10–12 minutes. Coronary angiography showed significant right coronary artery lesions in both patients.

We conclude that stress testing can differentiate AHC from ischaemia and that coronary artery disease associated with AHC may be detected. Evaluation of the postexercise time course patterns of ST segment and T wave changes will also differentiate athlete's heart from some cases of AHC.

Development of a current based defibrillator

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As current rather than energy is considered the more important factor in successful defibrillation we studied 37 consecutive patients (29 men, eight women; mean age 60 (range 41–81 years)) with 41 cardiac arrests (ventricular fibrillation) who received a first shock of 200 J and if required a second of 200 J and then 360 J delivered thereafter to assess peak current (Ip) at defibrillation. With self adhesive pregelled 12/12 cm pads in the anterior–anterior position a first shock of 200 J was successful in 33 of the 41 (81%) cardiac arrests and a first or second shock in 39 (95%) (mean Ip for successful defibrillation, 32 (SD 6.3) A (range 22–48); mean predicted transthoracic impedance (TTIp) 70 (SD 16) Ω (range 41–122 Ω)). We then developed an Ip based defibrillator in which a microprocessor uses TTip and preprogrammed defibrillator characteristics to estimate the energy required to produce a preselected Ip for each patient. We studied a further 45 consecutive patients (30 men, 15 women; mean age 62 (range 19–81 years)) with 45 cardiac arrests where the first shock was 30 A and if unsuccessful the second shock was 30 A and then 40 A if required. For safety, manual charging to the energy required was used. Component tolerance and operator variability led to small variations in Ip. A first shock of 30 A was successful in 36 of the 45 (80%) cardiac arrests and a first or second shock was successful in 44 (98%) (mean Ip for successful defibrillation, 29 (SD 1.4) A (range 25–31); mean TTip, 69 (SD 17) Ω (range 38–104); and mean energy per shock was 154 (SD 66) J (range 63–304)). Thus the mean energy per shock of 154 J delivered using an Ip system was significantly less than using 200 J (p < 0.001) and the mean Ip per successful shock was also reduced (29 A v 32 A; p < 0.05). First shock success, however, was not significantly different (80% v 81%).

Mechanisms for the early mortality reduction produced by β blockade in acute myocardial infarction

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In the First International Study of Infarct Survival (ISIS-1)\(^1\) the mortality was 15% lower in the group randomised to receive atenolol in the early hours after the onset of suspected myocardial infarction than in the control group. An unexpected finding on retrospective analysis of the data was that this benefit was mainly confined to deaths during the first two days of treatment, there being little further divergence in the mortality of the two groups subsequently. To determine the mechanisms responsible for this reduction in mortality, the case notes of patients in the British Isles and Scandinavia who died during days 0–1 of the trial were scrutinised. Adequate records were available for 193 of the 217 patients. Of the 79 patients given atenolol who died, necropsy confirmed rupture in five; a further 15 had electromechanical dissociation, but no necropsy had been performed. Comparable figures for the 114 patients in the control group were 17 and 37. As rupture was probably responsible for most of the cases of electromechanical dissociation the difference in fatality in the two groups can largely be explained by this mechanism. Additional findings included a slightly higher frequency of fatal ventricular fibrillation and aortic dissection in the control group, and of bradycardia and asystole in the atenolol group.

Limitation of infarct size and the prevention of reinfarction do not seem to have played a part in the reduced mortality.


Changes in neutrophil activation and free radical activity after thrombolysis for myocardial infarction in man

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Thrombolytic treatment after myocardial infarction is widely practised but reperfusion injury, with free radical production and amplification of the inflammatory response, remains a possibility. We previously showed increased free radical activity (non-peroxide diene conjugate of linoleic acid (octadecadienoic acid)) and neutrophil activation with release of neutrophil elastase in myocardial infarction. We have since measured these activities sequentially over 48 hours in 16 patients with acute infarction: eight who were given intravenous thrombolytic treatment (age 52.5 (13-4) years) and eight who were treated conventionally (age 57.9 (11-9) years). Octadecadienoic acid concentration increased in the thrombolytic (34.83 (4.46) µmol/l; p < 0.001) and non-thrombolytic (30.79 (6.0) µmol/l; p < 0.01) groups compared with normal values (19.45 (1.09) and fell over the 48 hours. There was no difference between the two groups. Neutrophil elastase values increased above normal values (21.2 (2.0) ng/ml) in the thrombolytic (49.58 (8.41); p < 0.001) and non-thrombolytic (37.25 (9.76) ng/ml; p < 0.02) groups. The non-thrombolytic group showed a biphasic increase in neutrophil elastase values with a significant second peak at 40 hours (55.01 (8.89) ng/ml) that was not seen in the treated group (32.43 (3.72) ng/ml; p < 0.05). The inflammatory response was imaged by indium-111 labelled autologous neutrophils in a subgroup of these patients and seemed attenuated in the group receiving thrombolysis.

Thrombolytic treatment within four hours after onset of symptoms is not associated with greater free radical induced damage and abolishes neutrophil activation in the second day after myocardial infarction, when neutrophils are normally present in the infarcted area. Reperfusion injury may therefore be a theoretical rather than an actual phenomenon.

Early intervention in myocardial infarction with streptokinase at home and in accident and emergency department

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The beneficial effect of intravenous streptokinase in acute myocardial infarction decreases with the delay between the onset of symptoms and intervention. We report our experience of streptokinase given at home, in the accident and emergency department, and in hospital (CCU). Between August 1985 and April 1986, 38 patients in the CCU received intravenous streptokinase within four hours according to conventional criteria. Thereafter unit policy was to give intravenous streptokinase at home or in the accident and emergency department when practicable. Since then, of 138 admissions, 43%, received streptokinase at home, 24% in the accident and emergency department, and 33% in the CCU. Complications outside hospital were few: two patients had ventricular fibrillation, four transient hypotension, and one transient asystole; one patient bled from the forehead and another died in shock. Of patients given intravenous streptokinase outside hospital, two had ventricular fibrillation and one...
haematemesis and one patient developed a saddle embolus in the CCU. The mean delay for patients given intravenous streptokinase at home was 104.5 (SE 7.2) minutes, in the accident and emergency department 127.7 (8.9) minutes, and in the CCU 165.4 (8.6) minutes (all differences significant). Seven patients given intravenous streptokinase at home and three in the accident and emergency department would have been ineligible on delay criteria for such treatment in the CCU.

Intravenous streptokinase given at home or in the accident and emergency department is safe, reduces delay to intervention by up to one hour, and allows the treatment to be given to more patients.

Kinetic changes in coagulation, fibrinolysis, and plasma viscosity associated with coronary artery reperfusion with tissue plasminogen activator in acute myocardial infarction

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Twenty one patients (15 men, six women, age range 38–68) with acute myocardial infarction (<4 hours; 13 anterior, eight inferior) received either placebo (n = 11) or 100 mg tissue plasminogen activator (rtPA) (n = 10) in a double blind randomised design. Serial samples were taken for profiles of creatine kinase (CK) release and at 0, 30, 45, 60, 90, 120, 180, and 240 minutes to measure serial changes in coagulation, viscosity, and fibrinolysis (with and without monoclonal antibody to rtPA (Ab)). An early (12 hours) peak in CK activity suggestive of reperfusion was seen in eight of the ten patients receiving rtPA and in only one of the 11 receiving placebo; mean values (SEM) 2852 (1246) iu/l (rtPA), 1590 (1087) placebo at 12 hours and 2404 (1268) (rtPA), 2108 (1045) placebo at 24 hours. Fibrinogen concentration fell within 30 minutes from 2.8 (0.3) g/l to a nadir at 45 mins of 1.1 (0.2) g/l (p < 0.01) and plasminogen values fell from 98.7% to 47% (8) at 30 minutes. The addition of Ab inhibited in vitro activity reducing the fibrinogen fall from 3.2 (0.3) (t = 0) to 2.4 (0.4) at 30 minutes (p < 0.02) and plasminogen from 47 (8) (rtPA) to 65 (9) (Ab) at 30 mins (p < 0.05). Plasma viscosity fell from 1.14 (0.02) m pasq.s to 1.09 (0.01) (p < 0.02) and was sustained for four hours. There were no changes in the placebo group. Fibrin clot lysis (D-dimer) showed initial similar values (99 (4.5) mg/ml (rtPA), 114 (24) placebo; NS) but rose in the active group at 30 minutes to 687 (166) (p < 0.01), which was sustained for four hours. These results show that rtPA has a rapid onset of activity, a sustained fibrinolytic effect, and decreased plasma viscosity and is associated with an early high peak of CK release. Accurate measurement of in vivo fibrinolytic activity is obtained by the addition of Ab to blood samples for assay.

Does chronic pre-infarction angina affect in hospital outcome in patients with a first acute coronary event?

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Several studies have reported an adverse short term prognosis after myocardial infarction associated with previous chronic angina of effort. To determine if this effect was independent of other prognostic factors we studied 955 consecutive patients of all ages, of whom 329 (34.5%) were women, 750 (78.5%) were admitted to coronary care with a first myocardial infarction, and 205 (21.5%) were admitted to coronary care with an episode of unstable angina. A history of angina of effort dating at least three months before admission was present in 211 (22.1%) patients. These patients with previous angina were an average of 4.2 years older than those with no such history (p < 0.001). They had higher cholesterol concentrations (p = 0.016) and did not smoke as much (p < 0.001). The two groups did not differ in either the occurrence of hypertension or high density lipoprotein cholesterol concentrations. The mortality in hospital of patients with previous angina was 12.3% whereas that of patients without previous angina was 7.7% (p = 0.036). Patients with previous angina also had a higher rate of heart failure and cardiogenic shock (28.9% v 22.2%; p < 0.04). When multiple logistic regression was used to correct for the difference in age between the two groups previous angina correlated neither with mortality (p < 0.289) nor with complications (p = 0.432). Patients with previous angina had smaller infarctions, as assessed by peak cardiac enzyme activities (p = 0.013), which may reflect better collateral circulation.

We conclude that the reported adverse effect of previous angina on early prognosis after an acute coronary event results from the difference in age between those with and without angina.
Thrombolysis in acute myocardial infarction: why the delay?
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Interest in early thrombolysis for acute myocardial infarction (AMI) led us to conduct a prospective audit of delays occurring during the management of emergency patients with chest pain both in the accident and emergency department of this teaching hospital and during transfer to the cardiac care unit (CCU). Over a period of 100 days, 235 patients attended the accident and emergency department with chest pain, but we excluded three who discharged themselves from the department and 45 in whom neither the time at which the doctor first saw them nor the time of leaving the department were recorded in the boxes available on the casualty card. As all intervals fell on a skewed distribution medians and ranges are given. The median delay between arrival at the accident and emergency department and first seeing a doctor was 32 (0–215) minutes for the 155 patients for whom this measure was available. In 49 of these patients who were brought by ambulance the delay was 17 (0–175) minutes, while in 18 who were later transferred to the CCU it was 11 (0–75) minutes. The median stay in the accident and emergency department (difference between arrival and departure times) was 78 (19–510) minutes for the 126 patients in whom this measure was available. In 22 patients who were transferred from the accident and emergency department to the CCU the median stay was also 78 (25–422) minutes. The median stay was related to the number of investigations performed (no electrocardiography or chest x ray, 48 (19–133) minutes (n = 21); both electrocardiography and chest x ray, 91 (33–510) minutes (n = 10), and to different doctors (medians 66 to 91 minutes). In a second study the median differences between the arrival of 29 consecutive patients with AMI or unstable angina at the accident and emergency department and their admission to the CCU was 115 (42–406) minutes.

These timings raise questions about the management of patients with AMI in accident and emergency departments, the role of the CCU in early management ofAMI, and the feasibility of early thrombolysis.

Out of hospital resuscitation in Brighton 1981–6
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The resuscitation ambulances in Brighton practise a system of immediate care that depends wholly on ambulance crews who have received extended training in a range of disciplines. From January 1981 to December 1986, 143 patients were successfully resuscitated from cardiac arrest out of hospital and survived to be discharged alive. Of these, 125 (87%) were victims of heart attacks or aborted sudden cardiac death. Seventeen patients (12%) suffered a primary respiratory arrest, which was secondary to acute asthma in eight, pulmonary oedema in one, overdose in five, respiratory obstruction in two, and hanging in one. Hypovolaemia after having been stabbed was the cause of circulatory collapse in one patient. In 85 patients (59%) cardiorespiratory arrest preceded the arrival of the ambulance. Eighty-three patients (56%) recovered after only basic life support manoeuvres and defibrillation. In 60 patients (42%) additional advanced skills—namely, endotracheal intubation (52) and administration of drugs (eight)—were judged instrumental in achieving a successful outcome.

Instruction in basic life support and defibrillation deserves priority in extended training of ambulance crews because it can be achieved readily and saves lives. But many attempted resuscitations can be successful after prolonged cardiorespiratory arrest only when other techniques of advanced life support are available. Multidisciplinary training for ambulance crews is valuable and should be provided whenever possible.

Do all patients receiving intravenous thrombolytic treatment require aggressive invasive management?
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There is uncertainty about the need for aggressive investigation and management of patients with myocardial infarction (MI) after fibrinolytic treatment. Early invasive intervention is difficult in hospital without catheter laboratory facilities. Between August 1985 and July 1987, 170 patients with suspected MI were treated with intravenous streptokinase by this hospital's coronary care unit backed by a mobile unit. Catheterisation after treatment with streptokinase was performed acutely only on recurrence of chest pain or electively, depending on the patient's clinical and follow up treadmill electrocardiographic results. Medical treatment included long term aspirin and beta blockade unless these
were contraindicated. Criteria for intravenous streptokinase included a delay from the onset of pain of four hours or less and ST segment elevation or considerable ST segment depression. Conventional contraindications applied. Q waves and clinical heart failure were not contraindications. Coronary arteriography was carried out in 15% of patients, usually electively after the first outpatient review. Three per cent of patients had percutaneous transluminal coronary angioplasty and 3% had coronary artery bypass graft. Some patients are currently awaiting either percutaneous transluminal coronary angioplasty or coronary artery bypass graft. The mortality at mean follow up over 12 months was 4.7%, which is lower than expected from coronary prognostic index.

Comparison with other centres advocating early invasive investigation with view to percutaneous transluminal coronary angioplasty or coronary artery bypass graft is difficult as comparability of patients is impossible to establish. Our results suggest, however, that intravenous streptokinase followed by a conservative investigational approach is justified. Fibrinolytic treatment is appropriate in a district general hospital without catheter facilities.

Balloon dilatation of the aortic valve for congenital aortic stenosis beyond infancy

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Experience in infants suggests that balloon dilatation of the aortic valve (BAV) can provide effective relief of aortic stenosis after the neonatal period. We attempted BAV as the primary procedure in 29 consecutive children beyond infancy (aged 16 months to 17 years, median 7 years) from June 1985 to November 1987. On echocardiography, the aortic valve appeared to have three cusps in 20 (70%) patients, two cusps in eight (27%), and no commissures in one (3%). The aortic valve was crossed in all but one patient. In 28 patients the mean peak to peak systolic pressure gradients decreased from 72 (SD 31) to 31 (20) mm Hg (p < 0.001). The reduction in gradient was 57%. New aortic regurgitation occurred in seven patients (grade I in six, grade II in one) and was exacerbated (from grade I to II) in two out of six patients with pre-existing mild aortic regurgitation. There were no other complications apart from femoral artery occlusion in one (3%) patient who required treatment with intravenous streptokinase. In 10 patients who were restudied three to 14 months later there was no significant change in gradient from that immediately after BAV (48 (16) vs 48 (22) mm Hg). The two patients with the highest residual gradient after BAV showed substantial improvement at restudy (70 to 42 mm Hg and, 70 to 40 mm Hg), while the patent with the unicusp aortic valve showed an increase in gradient from 37 to 99 mm Hg over nine months and required aortic root replacement. Surgical valvotomy was performed in the child in whom the aortic valve could not be crossed, but no other patients underwent aortic valve operation.

The short term results of BAV suggest that it offers safe alternative palliation to valvotomy.

Cavopulmonary anastomosis with preservation of pulmonary artery continuity in patients with univentricular heart

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Since 1967 the condition of 27 patients with univentricular heart (UVH) and severely reduced pulmonary blood flow has been palliated by constructing a side to side cavopulmonary anastomosis (CPA) with ligation of the superior vena cava at the right atrial junction. The continuity of the pulmonary artery was thus preserved. There were 17 patients with tricuspid atresia (group A, mean age 32 months, range 16 days–108 months) and 10 patients with other complicated UVH (group B; mean age 48 months, range 5 weeks–180 months). In two patients with non-communicating left and right superior vena cava bilateral CPAs were performed. A further two patients had azygos continuation of the inferior vena cava. There was one death related to a procedure in a 5 year old. Clinical improvement occurred in all survivors, and in 15 no further intervention was required (mean follow up 8 years, range 2–15). In 11 patients further surgery was performed because of increasing hypoxia in spite of angiographically proven shunt patency and low pulmonary artery pressure. There were three Fontan procedures, two peripheral arteriovenous, four Blalock-Taussig, and two aortopulmonary shunts.

CPA may have advantages over systemic to pulmonary artery shunts. Venous blood is directed to the lungs without increasing the volume load on the ventricle or significantly increasing pulmonary artery pressure. The anatomy of the pulmonary artery is preserved, which improves the anatomical
survival in complete transposition of the great arteries: a 10 year review

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Until recent years the standard management for the infant with uncomplicated ventriculoarterial discordance has been balloon atrial septostomy (BAS) followed by Mustard’s or Senning’s correction. In some centres an anatomical correction in the neonate is now preferred despite an increased operative mortality. In the light of this change in direction we reviewed our experience in Belfast. The records of 35 patients with uncomplicated ventriculoarterial discordance who presented from January 1978 to December 1987 were studied. All patients underwent BAS at 1–18 days (mean 5 days), which produced satisfactory palliation in 26 (74%). Six of these 26 patients (23%) died while awaiting corrective surgery. Fifteen patients were successfully corrected: 10 by Senning’s and five by Mustard’s procedure. There was no operative mortality, but two late deaths occurred among the patients treated with Mustard’s procedure. Five infants currently await correction. In the nine patients (26%) with an unsatisfactory initial BAS, the procedure was repeated in five. Two underwent a Blalock-Hanlon septectomy and one an open septectomy, who did not survive the operation. Six had early correction (at less than 6 weeks of age): four by Senning’s and one by Mustard’s procedure. The remaining patient had an anatomic correction but did not survive. The mortality for the patients overall was 10 (28%). Seven (20%) died before corrective surgery with one death related to an open atrial septectomy and none directly related to BAS. There were 22 survivors of corrective surgery (8 aged less than 5 years), with no operative mortality for Mustard’s or Senning’s procedure irrespective of age, but a late mortality in two (9%).

In summary, in our experience atrial redirection for uncomplicated ventriculoarterial discordance carries negligible operative mortality. There is, however, a significant mortality before definitive repair despite apparently successful palliation by BAS.

Fate of patients with transposition of the great arteries in the Republic of Ireland 1979–87

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All cases of suspected congenital heart disease in the Republic of Ireland are referred to this hospital. There were 104 patients with transposition of the great arteries (TGA) born during this period of eight years. Subgroups with associated patent ductus arteriosus (PDA), ventricular septal defect (VSD), left
ventricular outflow obstruction (LVOTO), alone or in combination, were defined. The largest of these was isolated TGA (51%). The male to female ratio was 1:8:1, the mean birth weight was 3410 g (1800–4900 g), and the mean age at referral 5.3 days (0.2–84 days). Altogether 99 patients had balloon atrial septostomy (BAS) at a mean age of 5.9 days, 35 through the umbilical vein and 25 under echocardiographic guidance. One patient died during BAS. Other complications of BAS were infrequent (7%). After BAS and before definitive surgery complications included hypercyanotic spells, 13 patients (13%); cardiac failure, 11 (11%); and cerebrovascular accidents, four (4%). Six patients died before definitive surgery: one with profound hypoxia at 4 weeks, one after PDA ligation at 10 weeks, two with sepsicaemia at 5 and 12 months, one with CVA at 11 months, and one at home at 17 months. Palliative surgery was required in 10 patients (10%): Blalock-Taussig shunt in six (6%), pulmonary artery banding in three (3%), PDA ligation in two (2%). Eighty-two patients had definitive surgery. The subgroup with isolated TGA had an operative mortality of 4.3%. The overall operative mortality was 19.5%. In the first half of the study period (1979–83) the operative mortality was 24% as compared with 7.5% for the second half (1984–87). There were three late deaths (3.6%). Fifty-eight patients were followed for a mean of 2.5 years after operation (mean 0.2–5.8 years). Forty-six (75%) were in sinus rhythm. Ten patients (17%) were in nodal rhythm alone or alternate with sinus rhythm. None has required pacing. Four patients (7%) had residual hemiparesis and one (1.7%) motor developmental delay. Two patients (3.4%) had systemic ventricular failure. None has shown evidence clinically or at repeat cardiac catheterisation (17 patients) of pulmonary or systemic venous pathway obstruction.

This represents the first study of patients with TGA in the Republic of Ireland. These results compare favourably with other reported series.

Postnatal management of supraventricular arrhythmias diagnosed in utero

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Twenty-two out of 23 children with supraventricular arrhythmias in utero (12 hydropic) required resuscitation at birth; four of nine infants had complications of prematurity. Postnatally, supraventricular tachycardia occurred in eight, atrial flutter in eight (two with atrial septal defects), and atrial tachycardia in two. Five children with arrhythmia control in utero remained in sinus rhythm from birth. Digoxin was administered within 48 hours of birth in 19 children and at 3 weeks in another. Three received additional anti-arrhythmic agents (verapamil in two, propranolol in one). DC cardioversion was necessary in five. One premature child with partially controlled atrial flutter died. Pre-excitation was present on electrocardiography in three. Digoxin was later (16 months) successfully withdrawn in two of the eight with supraventricular tachycardia (mean follow up 52 months). Five of the six currently taking digoxin are aged less than 1 year; the other (aged 3) with a preexcitation syndrome had recurrent arrhythmias for 15 months. It was possible to withdraw digoxin in four of the eight children with atrial flutter (mean age 8 months) with no recurrence on follow-up (mean 40 months). Atrial tachycardia was more resistant to treatment, requiring both amiodarone and digoxin in one child who later died of meningitis aged 23 months. Another with atrial tachycardia failed to respond to medical treatment.

We conclude that postnatal control of supraventricular arrhythmias diagnosed in utero is achievable, often with digoxin alone, and treatment can later be withdrawn in most patients by the age of 1 year. The rarer atrial tachycardias are more resistant to treatment.

Magnetic resonance imaging of ventricular septal defects in infants

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Electrocardiogram gated cardiac magnetic resonance imaging was performed in 10 infants (aged 6 weeks to 4 months) with ventricular septal defects. A high field strength (1.5 Tesla) imaging system was used with the infant, sedated if necessary, placed in an adult head coil. A spin echo sequence was used, multiple, synchronous, 5 mm thick sections being acquired in various imaging planes. Sections that were related to the long and short axis of the heart were used. The angles of the imaging planes were chosen individually in each patient to show the anatomy of the ventricular septum optimally. Imaging
was successful in every patient. Six patients had perimembranous defects, the relation of the defect to the central fibrous body was readily determined from the images. One patient had a doubly committed, juxta-arterial defect. The area of fibrous continuity between the arterial valves, which was roofing the defect, was clearly shown. Another patient had a bilateral subarterial infundibulum in association with the defect, which was excellently shown. Two further patients had muscular defects. In one of them the defects were multiple. In all patients the anatomy of the ventricular septal defects was more clearly shown in the magnetic resonance images than by cross sectional echocardiography or, in four patients, cineangiography.

Our early experience suggests that magnetic resonance imaging will become the optimum technique for imaging ventricular septal defects.

Paediatric supraventricular arrhythmias: age at presentation, nature, and treatment

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Of 97 children referred with supraventricular arrhythmias, 70 had paroxysmal re-entry supraventricular tachycardia (PRSVT), 25 of these had an overt pre-excitation syndrome (Wolff-Parkinson-White syndrome (WPW)), 20 had atrial fibrillation or flutter (AF), and seven atrial tachycardia. Of the 70 with PRSVT, structural heart disease was present in twelve (17%). Twenty three (51%) without overt WPW presented before the age of 6 months and 14 (31%) between 7 and 12 years. Twelve (27%) received at least three antiarrhythmic drugs; electrophysiological studies were performed to aid treatment in 13 (29%); one child received an antitachycardia pacemaker and one operation. Of the 25 with overt WPW, 10 (40%) presented before the age of 6 months and 12 (48%) between 7 and 12 years. Ten (40%) of these received at least three antiarrhythmic drugs; electrophysiological studies were performed in 15 (60%), which led to one child receiving an antitachycardia pacemaker and two operation. Ten of the 20 children with AF (50%) had structural heart disease, and in 6 AF occurred post-operatively associated with sinus node dysfunction. Of the remaining 10, nine presented before 1 month of age. Arrhythmia control was obtained in 12 (60%) with one antiarrhythmic drug and three (15%) required no antiarrhythmic drugs; electrophysiological studies were performed in two (10%) and two received antitachycardia pacemakers. Two of the seven children with atrial tachycardia had cardiomyopathies and one child congenital heart disease; ages at presentation varied widely. Four children (57%) received at least three antiarrhythmic drugs, electrophysiological studies were performed in three (43%), one received an antitachycardia pacemaker, and one operation.

In conclusion, PRSVT was the most common paediatric supraventricular arrhythmia and had a bimodal age presentation. Half of those with AF have structural heart disease. Medical management achieved control in most, although surgery or a pacemaker was occasionally required for refractory arrhythmia.

Long term follow up after coronary thrombolysis in acute myocardial infarction

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Between 1982 and 1985 113 patients (mean age 55 years) who presented at a median time of 2-6 hours (range 0.5-6) after the onset of symptoms of acute myocardial infarction were treated with an intravenous or intracoronary thrombolytic agent. One hundred had angiographically proven coronary recanalisation. One hundred and five patients survived to leave hospital, and 100 of these have been followed up to the end of November 1987 (minimum follow up 23 months, median 35 months). A consistent follow up policy was adopted, consisting of early anticoagulation with heparin (controlled by the calcium thrombin time) followed by warfarin. Angioplasty or coronary grafting were used only when there was post-infarct angina or a strongly positive exercise test. Reinfarction (confirmed by electrocardiographic and enzyme criteria) occurred in nine hospital inpatients, seven of whom were not treated with anticoagulants at the time. Nine other patients sustained a myocardial infarction during the first year after treatment and four of them were treated with warfarin. After one year only two more patients had a myocardial infarction. Twenty six patients (25% of those alive at time of hospital discharge) had an intervention during the subsequent 24 months because of persistent or recurrent angina. Intervention was undertaken within three months of discharge in 13 cases. In hospital mortality was 7-1% and was due to pump failure in five cases and to cardiac rupture, papillary muscle rupture, and sudden death in one case each. A further six patients
died during the first year—all these deaths were “sudden”. Three of the late deaths were in patients in whom reperfusion had been unsuccessful.

Pacing termination of spontaneous ventricular tachycardia

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Pacing techniques to terminate ventricular tachycardia (VT) induced during electrophysiological testing are well documented but require specialised equipment. We attempted pacing termination of spontaneous VT in the coronary care unit with a standard external pulse generator with $3 \times 3$ rate multiplier. Temporary pacing lines were inserted on 27 occasions in 15 patients (13 men) aged 38–76. Twelve had previous myocardial infarction, one recent (48 h), and two had cardiomyopathy. There were 68 separate episodes of spontaneous VT. Intravenous antiarrhythmic treatment terminated VT in only seven (22%) of 32 attempts, while pacing termination was successful in 31 (54%) of 57 attempts. Underdrive and overdrive pacing were used when appropriate and were successful in 13 (34%) of 38 and 18 (46%) of 39 attempts respectively. Acceleration, with a subsequent need for DC cardioversion occurred on four occasions during pacing and once during catheter manipulation (9%).

The success rate of pacing termination of spontaneous VT at the bedside is similar to that obtained with induced VT in the laboratory. This technique minimises the need for repeated DC cardioversion or the use of multiple intravenous antiarrhythmic drugs.

Human defibrillation thresholds with biphasic waveforms and contoured epicardial patches

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Implantable defibrillators are becoming established as important devices for the management of life threatening ventricular arrhythmias. Present devices are bulky because they require large batteries and capacitors; so reduction of energy requirements is an important goal. We studied the defibrillation thresh-

old (DFT) with contoured large surface area epicardial patches (60 cm and 58 cm) in 21 patients (18 men, three women) undergoing coronary artery surgery. An incremental energy protocol was used in nine patients and a “searching” modified Bourland protocol in 12. The pulse consisted of a biphasic impulse (6.5 ms +ve/3.5 ms –ve), timed to discharge 10 seconds after the onset of induced ventricular fibrillation. Clinical details, left ventricular function, and coronary anatomy were evaluated and were correlated with the DFTs. The mean age was 58 years (range 46 to 71). Mean left ventricular ejection fraction was 59%. Eight had previous infarction, 16 were taking β blockers, and 16 were taking calcium blockers before operation. The mean DFT was 2.6 (2.3) (0.7 to 9.3) J, (174 (69) (96 to 357) V, with a mean measured impedance of 63.2 (14.6) (42.6 to 100.7) Ω. Sixteen patients had DFTs of <3 J, and 16 patients had DFTs of <175 V. There were no significant predictors of DFT based on clinical and anatomical details. Two patients with fatty hearts had notably higher DFTs but unremarkable impedances. There was no significant difference between the two different protocols.

Very low DFTs are possible if large contoured epicardial patches and biphasic pulses are used. DFTs were not significantly influenced by clinical and structural variables, except pericardial fat; none the less these patients did not have severely impaired left ventricular function. These findings have implications for optimising the design of implantable defibrillators.

Laser probe design: features of the optimum probe for coronary angioplasty

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Thermal angioplasty with a percutaneous laser probe (LP) is now used as an adjunct to balloon angioplasty in peripheral arteries. We evaluated techniques of delivering LP energy to coronary arteries by three different LPs in 11 perfused coronary arteries in seven intact cadaver hearts suspended in a water bath at 37°C. A guiding catheter was used for angiography, passage of the laser probe, and simultaneous perfusion with 60 ml/min during lasering. The most rigid LP (2.0 mm tip) used for peripheral angioplasty was unable to pass more than 2–3 cm into the major coronary vessels. A more flexible 1.5 mm LP with a guide wire through the tip could be passed to distal vessels but only by unacceptable
displacement of the coronary arteries. Incorporation of the optical fibre into a catheter with a 2 cm floppy spiral metal coil proximal to the tip (1-7 mm) and a central guide wire channel permitted access to the distal vessels with less lateral distortion but also with reduced axial force that was necessary to advance the LP through atheromatous stenoses. Application of laser energy (10 W for 5–15 s) to the stationary probes resulted in lateral adherence to the vessel wall in 20/25 coronary segments. There was macroscopic charring of the vessels in 8/13 segments not perfused during lasering and 0/12 segments that were perfused (p < 0-05). Three segments in which the vessel diameter to probe diameter ratio was < 1:5:1 were perforated. Adherence and charring did not occur in any of the segments in which the vessel to probe ratio was > 2:1:1.

Flexible, small diameter, LPs are most appropriate for coronary laser angioplasty because they give vessel to probe diameter ratios which reduces lateral damage while retaining sufficient strength to avoid their being welded to the artery wall as they are advanced through the vessel.

Comparative survival at twelve months of patients admitted by mobile coronary care unit and ordinary ambulance

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The mortality in an area served by a mobile intensive coronary care unit was compared with that in a similar neighbouring area without such a unit. Mortality was 13% less in the area with a mobile unit. After a mobile unit was established in the other area survival at three months and 12 months and coronary prognostic indices were compared in patients admitted to the district coronary care unit by ordinary ambulances and by the mobile intensive coronary care unit. Three hundred and forty three patients with 385 cardiac events entered the study (mean age 63 (19–89), 63% men and 37% women). The distribution of infarcts by type and site was similar in both admission transport groups (χ² = 2.93, df = 5, p = 0.70). The average coronary prognostic index was 3-7 for patients admitted by the mobile unit and 4-0 for patients admitted by ordinary ambulance. Twelve month survival for those under 65 years was 66% for the mobile unit and 53% for the ordinary ambulances. Twelve patients were resuscitated outside hospital by the mobile unit and 10 (83%) of them were alive at 12 months. No successful resuscitations were carried out by ordinary ambulance.

Balloon dilatation of the aortic valve for critical aortic stenosis

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Balloon dilatation of the aortic valve (BAV) has been suggested as an alternative to aortic valve replacement in elderly or infirm patients with aortic stenosis (AS). We report our initial experience with this technique in patients who were considered to be unsuitable for operation because of severe left ventricular failure (3 patients) or coexisting disease (nine patients). Thirteen procedures were performed on 12 patients (mean age 65-5 yr (range 50–75). All had pure AS and were in New York Heart Association (NYHA) class III or IV. The mean transvalvar gradient was reduced from 81 mm Hg (range 40–140 mm Hg) to 48 mm Hg (range 20–80 mm Hg) (p = 0-008). In one patient symptomatic restenosis required repeat dilatation. Fatal arterial thromboembolism occurred in two patients two days and seven days after BAV. Both had symptomatic peripheral vascular disease. Total procedure related mortality was three out of 13 procedures. All patients who survived BAV reported an improvement in symptoms of at least one NYHA class. Doppler assessment of the gradient after BAV correlated well with directly measured values. The high procedure related mortality may be attributed to the presence of severe coexisting medical disease or left ventricular failure in these patients.

We conclude that though BAV is an effective means of palliation for critical AS, it has serious risks for those most likely to benefit from it.

Syncope in athletes: the role of the Valsalva manoeuvre

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Syncope and near syncope have been reported in trained athletes. Although such episodes are often associated with bradycardias, some athletes, without detectable bradycardia complain of near syncope, particularly during high resistance training. We evaluated beat to beat changes in left
ventricular volume associated with isometric exercise, dynamic exercise, and the standard and maximum Valsalva manoeuvres in five olympic athletes by means of a nuclear stethoscope. In one subject simultaneous intra-arterial blood pressure was also recorded. Changes were qualitatively similar in all subjects. Mean resting ejection fraction at rest was 66-6%. During isometric exercise (maximum hand-grip for 10 seconds) the ejection fraction fell by an average of 4% when subjects breathed normally and by 18-6% with simultaneous Valsalva manoeuvre. During maximum Valsalva manoeuvre maintained for 10-15 seconds (simulating the manoeuvre performed during weight training) dramatic falls in the ejection fraction were seen such that in two mesomorphic subjects the left ventricular volume time-activity curve showed a flat response. Moreover, an arterial trace in one of these subjects showed a mean pressure of 50 mm Hg at the end of the procedure with a pulse pressure of 15 mm Hg. Background counts (representing blood in the pulmonary vascular bed) also decreased during maximum Valsalva.

We conclude that a dramatic fall in cardiac output occurs in trained athletes during the Valsalva manoeuvre, which may be partly responsible for near syncope observed in some athletes during heavy resistance training.

Early (24 hour) electrocardiographic stress testing in the evaluation of acute chest pain

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Altogether 231 patients with severe chest pain were admitted to this hospital during 1986. Within 24 hours 108 confirmed recent myocardial infarcts were identified; 38 patients were considered to exhibit unstable angina, and 76 patients had posed a diagnostic problem. Forty nine apparently stable patients proceeded to electrocardiographic stress testing (treadmill and Bruce protocol) within 24 hours after admission. Ten showed strongly positive results with significant atheroma on subsequent angiography. Five showed equivocal stress performance. All had normal or minimally diseased coronary vessels. There were no false positive results. Thirty four patients showed normal results on stressing (max heart rates 85%) at 24 hours and were discharged. All were reviewed after one year. No clinical events were recorded; three patients experienced variable chest pain, but all stress tests were again normal.

Thus early (24 hour) stress testing showed serious potentially threatening disease in patients with clinically mild unstable angina and also reliably identified many patients suitable for quick discharge.
In hospital delay in administration of thrombolytic treatment

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Administration of thrombolytic treatment to patients outside hospital in the early phase of acute myocardial infarction remains impractical or impossible in many health districts; but there may be scope for in hospital delays to be reduced. We analysed 50 consecutive patients entering the APSAC-Swift study who received thrombolytic treatment by simple intravenous bolus injection within 180 minutes from the onset of symptoms. Details of the study were well publicised in the recruiting hospitals. Electrocardiographic criteria for entry were ST segment elevation of 0·1 mV in two standard or 0·2 mV in two precordial leads. We found a delay in hospital of 25–150 minutes (median 77·5). This contrasted with a delay for opiate treatment (if given) of only 5–139 minutes (median 30) in the same patients. The delay out of hospital was 15–120 minutes (median 56), with a total delay of 60–180 minutes (median 150). The in hospital delay was less for those with strikingly abnormal electrocardiographic changes (ST elevation >2 mV and reciprocal changes), those seen in the accident and emergency department by senior house officers on cardiology rotation, and those in whom life threatening arrhythmia occurred before thrombolytic treatment. In 14 patients treatment was delayed because of non-diagnostic changes in the initial electrocardiogram. A further 24 patients with diagnostic electrocardiographic changes were transferred to the coronary care unit before treatment because of lack of awareness about thrombolytic treatment, because of physicians’ concern about reperfusion arrhythmias, or for administrative convenience (delay for patients treated in the accident and emergency department 25–75 minutes (median 35): delay for patients treated in the coronary care unit 65–150 minutes (median 105)).

There are significant in hospital delays in the administration of thrombolytic treatment that could be reduced by education, reassurance about reperfusion arrhythmias, and avoiding the tendency to wait until the end of any chosen time window. Less stringent electrocardiographic criteria (with a convincing history) would result in earlier treatment of some patients.

Do high risk patients with acute myocardial infarction benefit from early intravenous metoprolol?

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Doppler flow patterns reflect the pulmonary artery pressure in ductus arteriosus

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The Doppler spectral pattern of flow through the ductus arteriosus was studied in 33 patients in whom aortic and pulmonary artery pressure were measured at catheterisation (in 18 simultaneously with two catheters). Flow was continuous throughout the cardiac cycle and exhibited four distinct wave forms: (a) high maximum velocity occurring in late systole with gradual fall throughout diastole; (b) high systolic velocity with rapid fall to a very low value in early diastole and maintained throughout; (c) low velocity throughout with maximum in late diastole; and (d)
bidirectional flow. The appearance of (a) was associated with normal or minimally raised pulmonary artery pressure (systolic below 50 mm Hg, diastolic below 30 mm Hg), that of (b) with raised pulmonary artery pressure (systolic above 50 mm Hg, diastolic above 30 mm Hg, and that of (c) and (d) with pulmonary artery pressure at systemic values. Comparison of the Doppler and catheter measured pressure differences between the great arteries showed a poor correlation for both the peak and trough readings.

The signals were more readily identified with colour Doppler flow mapping but could also be recorded with a stand alone transducer. Doppler ultrasound clearly shows ductal flow; the flow wave form gives an indication of the pulmonary artery pressure, but measurement of pressure by application of the Barnoulli equation to the flow velocities is not reliable.

**Arginine vasopressin abolishes the diuretic effect of atrial natriuretic factor in man**

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Chronic heart failure is characterised by abnormal concentrations of hormones including inappropriately high arginine vasopressin (AVP) concentrations. Patients with this condition also have raised atrial natriuretic factor (ANF) concentrations, yet chronic heart failure is typified by sodium and water retention. Exogenous ANF also fails to stimulate a diuresis in patients with chronic heart failure. To investigate whether AVP might be blocking the effect of ANF in chronic heart failure, AVP alone, ANF alone, and AVP and ANF together were infused into nine normal subjects during steady state maximal water diuresis. A dual placebo (P) infusion day was also performed. The dose of AVP (0.003 pmol/kg/min) and ANF (15 pmol/kg/min) were chosen so as to mimic the circulating concentrations of these hormones found in chronic heart value. The change in urinary sodium excretion (μmol/min (SEM)) was: (a) P + P -0.5 (0.29), 0.46 (0.35); (b) AVP + P -6.22 (0.92)*, -6.78 (0.9)*; (c) P + ANF +1.68 (0.35)*, +0.98 (0.42); (d) AVP + ANF -4.57 (0.96)*, -5.71 (0.97)*. The change in urinary sodium excretion (μmol/min (SEM)) was: (a) P + P -11.8 (3.9), -9.0 (5.3); (b) AVP + P -9.4 (2.6), -2.5 (8.9); (c) P + ANF +29.7 (7.1)*, +23.4 (6.9)*; (d) AVP + ANF +38.5 (11.4)*, +26.0 (11.5)* (*p < 0.01 v baseline).

These results show that AVP abolishes the diuretic but not the natriuretic effect of ANF in man. This suggests that other neuroendocrine influences may be required to blunt the natriuretic effect of ANF in chronic heart failure or that haemodynamic factors may be more important in the control of sodium excretion.

**Characteristics of energy delivery during catheter ablation in man: primary role of voltage in successful ablation**

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The precise mode of action of catheter ablation is unknown, but barotrauma, current, voltage, and heat have all been implicated. We examined recordings of voltage, current, derived energy, and impedance from 39 shocks with the National Heart Hospital (NHH) ablater (capacitive discharge) in 14 patients and compared them with results with a conventional power source and shocks in saline. Changes in impedance were similar in vivo and in vitro and of four types. (1) A short (<50 μs) transient high impedance due to initial polarisation. (2) A subsequent constant impedance for non-arcing shocks, but a fall in impedance for arcing shocks after a mean 0.33 (SEM 0.02) ms, proportional to delivered energy (r = 0.78, p < 0.001); in vitro recordings showed that the fall in impedance coincided with the onset of arcing. (3) A steep rise in impedance after arc extinction. Complete insulation occurred in vitro but not in vivo as the electrode tip remains in partial contact with myocardial tissue. (4) A return to a baseline impedance value similar to that for non-arcing shocks. With the NHH ablator successful ablation of atrioventricular conduction was performed with energy, peak current, and peak voltage of 19.7 (11.2), 18.8 (8.6) A, and 2328 (919) V, respectively; successful ablation with a conventional defibrillator was achieved with 221.4 (77.3) J, 44.9 (14.9) A, and 2093 (600) V. Lower energy and pressure changes with NHH shocks suggest heat and barotrauma are not important for success. Survival curve analysis of atrioventricular conduction showed that energy and peak current were dissimilar for the two power sources but that the voltages were not significantly different, suggesting the primary importance of voltage in terminating outcome.

Thus voltage is the likely mediator of useful cardiac damage during catheter ablation in man. This result has implications for future designs of power source.
Increased rate of first myocardial infarction in immigrant Asians

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Mortality and morbidity from coronary disease in Asians living in Britain is thought to be higher than in whites, but estimating infarction rates from hospital activity analysis records can be unreliable. We prospectively studied 78 consecutive patients presenting with myocardial infarction. All patients were men aged less than 60 and resident in the London Borough of Harrow at the time of the 1981 census. To ascertain the possible existence of ethnic bias with respect to general practitioners' referrals in patients with suspected myocardial infarction a questionnaire survey of 99 local general practitioners was performed. No such bias was found. Asians were younger at presentation (47-6 vs 54-7 years) (p < 0-001). Twenty nine myocardial infarctions occurred in a population of 5188 Asian men versus 49 in 31 308 whites (p < 0-000005). Twenty one Asians were smokers versus 38 whites. Five Asians were known diabetics versus one out of 49 whites. Predischarge glucose tolerance tests showed a further seven patients with diabetes or impaired glucose tolerance in the Asian group (total 12 out of 29) and nine in the white group (total 10 out of 49) (p < 0-01). Twelve Asians had a positive family history versus 21 out of 49 whites (NS).

We conclude that male Asians in Harrow have a much higher rate of myocardial infarction than whites. The first infarction occurred at a younger age in Asians, and the prevalence of diabetes is much higher in Asians with infarction than in whites. Smoking habits and family history were not different between the groups.

Effects of β adrenergic and calcium channel blocking agents on the circadian distribution of silent myocardial ischaemia

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One hundred and fifty patients (mean age 55 years) underwent 6264 hours of ambulatory ST monitoring when no longer taking routine anti-anginal therapy. Altogether 598 episodes of significant ST change (≥ 1 mm) were recorded (447 silent); 77% of silent ischaemic episodes occurred during the waking hours (0730–2230). Two subgroups were studied among these patients. Thirty three patients also had ST monitoring (1313 hours) while taking nifedipine and 41 patients (1581 hours) while taking atenolol (A). In the subgroup who had stopped taking nifedipine 43·8% of 129 silent episodes occurred between 0730 and 1330 (AM) and 31·5% of episodes between 1330 and 1930 (PM) (ratio = 1·39). In the patients taking nifedipine 40·1% of 136 silent episodes occurred AM, and 27·7% PM (ratio = 1·45). In the subgroup who had stopped taking atenolol 34·5% of 237 silent episodes occurred AM and 30·3% PM (ratio = 1·14). In patients taking atenolol 23·0% of 135 silent episodes occurred AM and 36·3% PM (ratio = 0·63). The frequency of silent myocardial ischaemia (SMI) in the six hour AM period was significantly higher than the average of the other three six hour periods in both subgroups who were no longer being treated and in the patients receiving nifedipine (p < 0·01). In contrast, in the patients taking atenolol SMI increased significantly compared with the average of the other three periods.

In summary, atenolol led to a reversal of the distribution of silent ischaemic episodes from the morning to the evening hours. In contrast, nifedipine did not significantly alter the circadian distribution of silent ischaemic episodes. This is of therapeutic importance as the incidence of both myocardial infarction and sudden death is greatest in the morning hours and the circadian rhythm of acute myocardial infarction is reported to be altered by β adrenergic blocking agents.

Initial electrocardiograms and outcome for patients seen by a mobile coronary care unit within four hours of the onset of suspected myocardial infarction

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With the advent of thrombolytic therapy for patients with suspected myocardial infarction analysis of the initial electrocardiogram (ECG) is crucial in decision making. Thus we analysed the initial ECGs of 89 consecutive patients with suspected myocardial infarction who were seen within 4 hours after the onset of chest pain by a mobile coronary care unit (MCCU) and survived to be admitted to hospital. We studied 67 men and 22 women whose mean age was 61 (25–81). MCCU response time ranged from 5–45 minutes (mean 13·0), and mean time from onset of
pain to arrival of the MCCU was 84 minutes (range 15–240). The initial ECG was considered positive if there was evidence of infarction (pathological Q waves or ST segment elevation), ischaemia (ST segment depression or T wave inversion, or both), or left bundle branch block. A diagnosis of myocardial infarction was confirmed in 40 of 89 (45%) patients, 25 (28%) patients had acute ischaemia, and 24 (27%) other diagnoses. Of the 35 patients seen within the first hour, five (14%) with a normal initial ECG had a proved myocardial infarction. Two of the 34 (6%) patients seen in the second hour had a proved myocardial infarction, one of the 10 (10%) seen in the third hour, and none of those with a normal initial ECG seen in the fourth hour. Analysis of the normal initial ECGs showed that in the first hour five out of 11 (45%) patients had a proved myocardial infarction, in the second hour two out of 12 (17%), in the third hour 1 out of five (20%), and in the fourth hour none out of four. Of those with an initial positive ECG for myocardial ischaemia but no proved myocardial infarction there were 12 out of 35 (34%) in the first hour, six out of 34 (18%) in the second hour, four out of 10 (40%) in the third hour, and three out of 10 (30%) in the fourth hour.

We conclude that 9% (eight out of 89) of patients presenting within four hours after the onset of chest pain had a myocardial infarction but normal initial ECG. The highest percentage of normal initial ECGs associated with an acute myocardial infarction were recorded within the first hour after the onset of symptoms.

Restrictive cardiomyopathy and constrictive pericarditis: non-invasive distinction by digitised M mode echocardiography

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Restrictive cardiomyopathy and constrictive pericarditis may be difficult to distinguish on clinical grounds or simple investigation; cardiac catheterisation has been the gold standard for diagnosis but does not always allow distinction. Using a Summagraphics digitiser and Prime 750 computer system we have digitised the M mode echocardiograms of patients with restrictive cardiomyopathy and constrictive pericarditis (n = 10) and those of normal subjects matched for age and sex (n = 20). In restrictive cardiomyopathy restrictive pathophysiology was shown by reduction in the following when compared with controls: (a) fractional shortening 22% (9.8) v 36% (3.6), p < 0.001; (b) peak left ventricular filling rate 7.5 (3.3) v 12 (12.6) cm/s, p < 0.001; (c) left ventricular peak emptying rate −5.3 (1.7) v −8.3 (1.1) cm/s, p < 0.001; (d) percentage posterior wall thickening 24% (12) v 35% (7), p < 0.007; (e) peak left ventricular posterior wall thickening rate −4.1 (2.5) v −5.8 (1.8) cm/s, p < 0.02; and (f) peak left ventricular posterior wall thinning rate 2.6 (0.9) v 3.4 (0.7) cm/s, p < 0.008. When patients with constrictive pericarditis were compared with controls the only significantly different variables were a decreased peak left ventricular filling rate (8.5 (2.5) cm/s, p < 0.004) and an increased peak left ventricular posterior wall thinning rate (6 (2.8) cm/s, p < 0.01). In patients with constrictive pericarditis, however, peak left ventricular emptying rate (−8.5 (2.8) cm/s, p < 0.006), percentage posterior wall thickening (47% (−15), p < 0.008), peak left ventricular posterior wall thickening rate (−6.7 (1.9) cm/s, p < 0.007), and peak left ventricular posterior wall thinning rate (6 (2.8) cm/s, p < 0.004) were all significantly increased compared with patients with restrictive cardiomyopathy.

We conclude that digitisation of M mode echocardiograms, with particular attention to posterior wall function, may be a useful adjunct to cardiac catheterisation in restrictive cardiomyopathy and constrictive pericarditis.

Quantitative echocardiographic tissue characterisation after cardiac transplantation

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Detection of myocardial changes after cardiac transplantation usually requires myocardial biopsy. To assess a possible role for echocardiography single end diastolic frames recorded with 6 bit grey levels with standardised gain and depth compensation settings were stored and analysed off line. Myocardial areas of interest were located in the septum and posterior wall, and mean backscattered amplitude automatically determined in samples of 100–200 pixels after speckle reduction. Results in 40 patients up to seven years after transplantation were compared with results in normal subjects matched for age and expressed in 0-9 dB units above zero. Mean backscattered amplitude was increased after transplantation to 13.6 (3.2) u (normal 6.6 (1.9); p < 0.001) with minimal overlap in individual patients. Mean backscattered amplitude within the first year after transplantation (12.9 (2.7) u) was less...
than that after one year (14.3 (3.7) u; p = 0.03). Mean values of backscattered amplitude greater than 15 u occurred in 18 patients in the septum or posterior wall. In eight patients this correlated with evidence on biopsy grade 1 rejection and in two patients with fibrosis. Greater degrees of rejection were not seen in any patient. In the remaining patients, high values of backscattered amplitude were seen in all at five at risk beyond four years, but only three of 26 before that (p < 0.001).

Thus myocardial backscattered amplitude is greater than normal after transplantation and increases with time. These increases reflect, in part, fibrosis or low grade rejection. This method may be useful in detecting, and in particular in determining, the myocardial distribution of these processes.

**Diurnal variation of pulmonary artery pressure in chronic heart failure**

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Variation in pulmonary artery pressure in patients with chronic heart failure may reflect changes in posture, pulmonary artery tone, and left ventricular function. To investigate the nature of diurnal variation in pulmonary artery pressure in chronic heart failure eight men aged 50–72 years who were free of angina and with treated chronic heart failure caused by ischaemic heart disease underwent continuous ambulatory pulmonary artery pressure recording with a transducer tipped catheter. A frequency modulated signal was recorded over 24 to 48 hours and played back on a paper chart recorder. The systolic and diastolic pressure was measured every 12 minutes by averaging pressures over one minute intervals. The mean daytime systolic pulmonary artery pressure was 29.6 (SD 5.0) mm Hg and diastolic pressure 13.7 (5.6) mm Hg. During the night the systolic pressure rose to 36.0 (4.1) mm Hg (p < 0.005) and the diastolic to 17.6 (2.9) mm Hg (p < 0.001). The mean change in systolic pressure from day to night was 5.1 (3.2) mm Hg and from standing to lying 9.3 (2.3) mm Hg (p < 0.025). The mean change in diastolic pressure from day to night was 3.8 (1.7) mm Hg and from standing to lying 6.4 (2.1) mm Hg (p < 0.025). In six of the eight patients there was considerable diurnal variation in pulmonary artery pressure. In four of them recordings were obtained over 48 hours, and the diurnal variation in the first 24 hours was similar to that in the second 24 hours. In two of the eight patients there was no pressure rise during the night. Although the diurnal variation in pressure might be partly explained by postural change, no correlation was found in the whole group of patients between postural pressure changes and diurnal variation.

Thus in patients with chronic heart failure diurnal variation in systolic and diastolic pulmonary artery pressure is common and may have important implications for the selecting and timing of treatment.

**Diagnostic value of comparison of ventriculoatrial intervals during junctional tachycardia and right ventricular apical pacing**

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We postulated that comparison of ventriculoatrial intervals during junctional tachycardia and continuous right ventricular apical pacing may provide similar diagnostic information to that obtained from the insertion of right ventricular apical extrasystoles during tachycardia. Patients with either atrioventricular re-entrant tachycardia (AVRT) (n = 23) by a single atrioventricular pathway or atrioventricular nodal re-entrant tachycardia (AVNRT) (n = 16) were studied. Ventriculoatrial intervals were measured during tachycardia, the ventricular pacing being at the same rate as the tachycardia and following a ventricular extrasystole delivered at the minimum reset interval (minimum prematurity of a ventricular extrasystole that advanced the subsequent atrial complex > 10 ms). The difference between the minimum ventriculoatrial interval during tachycardia and ventricular pacing was closely related to both the minimum reset interval (r = 0.92, p < 0.001) and the difference between the minimum ventriculoatrial interval during tachycardia and following a ventricular extrasystole delivered at the minimum reset interval (r = 0.97, p < 0.001) in the 23 patients in whom the minimum reset interval could be determined. The ratio between the minimum ventriculoatrial interval during tachycardia and ventricular pacing could be determined in all the patients and was 1.53–1.68 in atrioventricular re-entrant tachycardia with right free wall (two patients), 0.94–1.29 in that with anteroseptal defect (three patients), 0.91–1.08 with posteroseptal defect (five patients), and 0.48–0.71 with left free wall pathways (13 patients) while it was −0.32–0.27 in atrioventricular nodal re-entrant tachycardia (16 patients). The ratio was more discriminative when
corrected for ventricular latency and was also useful when calculated from the high right atrial electrogram.

We conclude that comparison of ventriculoatrial intervals during tachycardia and right ventricular apical pacing can discriminate between the mechanism of tachycardia and the site of pathway if present. It provides similar information to that obtained from right ventricular apical extrasystoles during tachycardia with the advantage that it can be determined in all cases.

Technetium-99m-2-methyl isobutyl isonitrile (MIBI): a new imaging agent for localisation of myocardial infarction

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A new myocardial imaging agent (technetium-99m-2-methyl isobutyl isonitrile (MIBI)) was evaluated in locating reduced perfusion in eight patients with recent myocardial infarction that was confirmed by cardiac enzyme activities and serial electrocardiograms. MIBI has advantages over thallium-201, with a higher energy and a slow myocardial clearance of five hours. Resting first pass radionuclide ventriculography was performed with a bolus injection of MIBI followed within two hours by perfusion imaging in three standard projections after a single MIBI injection. Images were reported by two independent observers. One patient had a previous anterior Q wave infarction with no anterior perfusion abnormality and normal wall motion. Seven patients had new Q wave infarctions and one had septal ST segment depression. All patients had regions of abnormal perfusion that corresponded with regional wall motion and the site of electrocardiographic changes. One patient with an anterior myocardial infarction and reduction of R wave amplitude showed anterior and posteroinferior perfusion abnormalities but only apical akinesia. A further patient had posterior hypoperfusion with an inferior myocardial infarction on electrocardiography and first pass radionuclide ventriculography. The extent of hypoperfusion, wall motion abnormalities, and ejection fraction were closely related in all patients.

This agent can be used not only for detecting abnormal myocardial perfusion but also for identifying regional wall motion abnormalities by first pass radionuclide ventriculography in patients with myocardial infarction.

Evidence of subclinical myocardial necrosis in patients with unstable angina detected by indium-III antimonyon antibody imaging

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In a prospective study of 103 patients admitted to hospital for investigation of suspected acute myocardial infarction (AMI) or unstable angina antimonyon antibody (FAB) (In-AM) imaging was performed to evaluate this new scintigraphic technique. Within 48 hours after experiencing chest pain all patients received 0·5 mg In-AM labelled to 74–83 MBq with indium-III. Gamma camera imaging was performed at 24 and 48 hours. Serial cardiac enzyme activities of creatine kinase and creatine kinase MB were measured and 12 lead electrocardiograms (ECG) were obtained daily and during chest pain. Of 98 patients imaged, 64 had AMI (45 had Q and 19 non-Q wave AMI), and 34 showed no evidence of AMI (unstable angina in 13 and no AMI or resting ischaemia in 22). The overall sensitivity and specificity for detecting AMI were 98% and 91%, respectively. Three out of the four “false positive” scans were noted in three patients with unstable angina. All three patients had severe three vessel coronary artery disease, and the uptake of In-Am was confined to the myocardial segments supplied by a critically narrowed artery; one patient died suddenly while awaiting operation one week after imaging.

Our results confirm those of experimental work showing a high degree of specificity of In-AM to bind with the acutely necrotic myocardium. Thus the myocardial uptake of In-AM in the patients with unstable angina may represent subclinical myocardial necrosis in the absence of the typical rise in cardiac enzyme activities and changes in electrocardiograms.

Grading programmed electrical stimulation for ventricular tachycardia

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Programmed electrical stimulation (PES) for ventricular tachycardia (VT) is usually performed in a specific order of increasing numbers of extrastimuli (ES) and increasing drive rates. Because PES is stopped when VT is induced it is not known whether this represents increasing aggression or is a random
order. All eight stages of our PES, with one then two ES from the right ventricular apex in sinus rhythm and three drive rates (100, 120, and 140 beats/min), were performed sequentially regardless of whether VT was induced at any stage. The protocol was stopped if the patient was distressed by the VT or it was difficult to stop with PES. If only non-sustained VT was induced, 3 ES were then added in sinus rhythm and in the three drive rates. Twenty one patients (mean age 57 years (range 27–75)) completed the protocol. Sixteen had ischaemic heart disease and five had morphologically normal hearts. VT was induced in all of them: it was sustained (> 30 s) in 13, non-sustained (≥ 5 beats, < 30 s) in seven, and both types occurred in one patient at different stages. In all 21 patients VT was induced by a 140 beats/min drive rate. In 17 it was induced by a drive rate of 120 beats/min, in 16 it was induced by 100 beats/min, and in six it was induced in sinus rhythm. Two patients had VT induced only by 1 ES, eight patients only by 2 ES, and three patients only by 3 ES. Five patients had VT induced by both 1 and 2 ES, two patients by both 2 and 3 ES, and one patient by 1, 2, and 3 ES. In only three patients was VT not induced at an intermediate drive rate with the same number of ES if the higher and lower rate induced VT. The cycle length of the VT induced at a later stage (366 (95) ms) was not significantly faster than that induced at an earlier stage of the protocol (387 (98) ms).

It seems that a protocol of sinus rhythm followed by increasing drive rates represents a stepwise increase, separately for 1, 2, and 3 ES, in the likelihood of inducing VT. In this study a drive rate of 140 beats/min with 1, 2, or 3 ES was as effective as the entire PES in inducing VT.

Platelet activation in unstable angina

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We investigated the importance of platelet activation in unstable angina pectoris by examining the temporal relation between the occurrence of episodic platelet activation and spontaneous myocardial ischaemia. Continuous electrocardiographic ST segment recording was performed for 24–96 hours in 11 patients with unstable angina treated with intravenous isosorbide dinitrate and oral diltiazem but no cyclo-oxygenase inhibitors. Platelet activation was assessed by radioimmunoassay of the main thromboxane metabolite, 11-dehydro-thromboxane B2 (TXB2), from serial eight hours urine collections. A total of 38 episodes of ischaemia were recorded (23 asymptomatic) and 56 urinary samples collected. The mean peak TXB2 value in each patient was 3389 (SD 2745) pg/mg creatinine (range 697–10 897), which was significantly greater (p < 0.01) than in 23 control patients without unstable angina or myocardial ischaemia (356 (151) (range 80–633)) or 20 patients with peripheral vascular disease (426 (160), (range 160–899), p < 0.01). In 46% of all urine samples TXB2 excretion was increased, but only 12% were associated with ST segment changes. TXB2 was raised in association with half of all episodes of myocardial ischaemia, during 78% of all episodes of chest pain, but also in 37% of urinary collections not associated with ST segment changes of pain.

Although episodes of intense platelet activation may often be observed in unstable angina, the relation between TXB2 excretion and the occurrence of myocardial ischaemia is highly variable; ischaemic episodes are frequently not associated with platelet activation and vice versa. Platelet activation is important but may be only one mechanism involved in the pathogenesis of unstable angina.

How important is pain during exercise induced myocardial ischaemia?

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Angina pectoris remains the main indication for coronary artery surgery, though most episodes of myocardial ischaemia are asymptomatic. We assessed whether the presence or absence of angina pectoris during formal exercise testing was related to the anatomical extent of disease or functional impairment during exercise. Left ventricular ejection fraction (LVEF) was measured every two minutes during symptom limited bicycle exercise in 34 patients with documented coronary artery disease, who were studied when they were not taking antianginal treatment. Eight, 16, and 10 patients had one vessel, two vessel, and three vessel disease, respectively, and the coronary scores for the percentage of myocardium supplied by stenosed vessels were 30 (4%), 52 (3%), and 73 (7%), respectively.
During exercise the limiting symptom was angina (n = 19) or breathlessness (n = 15). The presence of angina was not associated with a lower exercise time (421 (31) v 455 (64) s), ST depression (1-4 (0-3) v 1-1 (0-30) mm), exercise LVEF (47 (3) v 49 (3))% or fall in LVEF (-13 (2) v -12 (2)). Mean vessel disease and coronary score were similar (2-1 (0-1) v 2-1 (0-2) and 53 (4)% v 53 (5)%), respectively. ST depression (> 1 mm) developed in 22 patients. Exercise time was similar (angina v no angina) (452 (45) v 406 (42) s) as was the proportion developing angina (17 of 22 v seven out of 12). ST depression was associated with a lower exercise LVEF (44 (2) v 56 (3) p < 0-005), greater fall in LVEF (-15 (2) v -8 (3) p < 0-02), and more severe vessel disease (2-3 (0-1) v 1-6 (0-2); p < 0-005) and coronary score (62 (3)% v 37 (3)%; p < 0-001). The degree of ST depression correlated with the fall in LVEF (r = -0-5; p < 0-002) and coronary score (r = 0-6, p < 0-001).

Thus the symptom of angina pectoris during formal exercise testing bore no relation to the severity of disease and should not be a major criterion on which to base a management strategy. ST segment depression was a more reliable marker of disease and functional impairment during exercise.

**Infarct size can be measured acutely in developing myocardial infarction**

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In patients with acute coronary artery occlusion flow markers can reliably detect the site of infarction. Gate perfusion imaging with thallium-201 was used to estimate infarct size when the patient was admitted to coronary care. Altogether 120 consecutive patients were imaged at a mean time of 86 minutes from arrival at hospital by a mobile gamma camera after an intravenous injection of 60 MBq of thallous chloride. Infarct size was estimated in the 82 patients who subsequently proved to have myocardial infarction and the size was correlated with clinical (Norris index) and enzymatic (peak creatine kinase and creatine kinase MB release curves) estimates. In patients with first infarction perfusion and enzymatic estimates of infarct size were significantly correlated: r = 0-7 (p < 0-001) for 22 patients with anterior infarction and r = 0-5 (p < 0-001) in 32 with inferior infarction. In these groups there was no correlation between either the perfusion of enzymatic measure of infarct size and Norris index or ejection fraction. In contrast, in 24 patients who died there was a significant correlation between clinical findings (Norris index) and total perfusion defect (r = 0-6; p < 0-001) and ejection fraction and total perfusion defect (r = 0-5; p < 0-009), but not with enzymatic measures of infarct size or any other variable. Eleven patients died within 14 days and the correlation between Norris index and total perfusion defect was 0-8 (p < 0-001).

Gated perfusion imaging on admission reflects the volume of myocardium affected and thus infarct size and should be of value in predicting the clinical outcome in patients with acute myocardial infarction.

**Sublingual nifedipine and coronary flow**

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Sublingual nifedipine is frequently used by patients for the relief of angina. To investigate the mode of action 12 patients were studied at the time of routine angiography. Heart rate was obtained from the electrocardiogram and blood pressure by femoral arterial line. Regional myocardial nutrient blood flow was assessed from the clearance of xenon-133 after direct intracoronary injection. Imaging was performed in the 40° left anterior oblique projection with a mobile gamma camera. Measurements of both coronary arteries were made at rest and 3–7 minutes after 10 mg sublingual nifedipine. There was no significant change in heart rate or mean arterial pressure (98 (3) to 96 (4) mm Hg). In 12 distributions supplied by vessels that were not significantly diseased flow increased significantly by 16%, from a mean of 63.1 (6.3) to 74.7 (8.1) ml/100 g/min (p < 0-02). In 24 regions supplied by coronary vessels that were significantly diseased mean flow increased from 57.9 (4.1) to 60.8 (5.9) ml/100 g/min, representing an overall 5% increase in flow. This is in contradiction to the effects of sublingual nitrate, with which flow fell by 14% (p < 0-01) in distributions supplied by coronary arteries with high grade obstructions. In regions supplied by collaterals, after nifedipine, flow increased from a mean of 38.3 (4.7) to 72.6 (8.9) ml/100 g/min, whereas with sublingual nitrate collateral flow is reduced. These results show that nifedipine taken sublingually acts as a coronary vasodilator, and this may be the mechanism for relief of angina in patients with obstructive coronary disease.
Prospective study of atrioventricular conduction in patients with sinoatrial disease

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Many patients with sinoatrial disease could be effectively treated with atrial demand (AAI) pacing but for the development of atrioventricular block in a few. If this small group could be identified before pacemaker implantation it would prevent unnecessary insertion of two leads. A prospective study of 152 patients paced for sinoatrial disease in the past eight years was undertaken. Sixty (40%) patients were paced in ventricular demand (VVI) mode. The remainder underwent electrophysiological study at the time of pacemaker implantation. If atrioventricular conduction was normal, the patient was paced AAI. If abnormal, both ventricular and atrial leads were implanted and the patients sequentially paced (DVI). Results: 52 patients (34%) were paced in AAI mode. Mean age at implantation was 66-6 years and mean follow up time 29-4 months. No patients developed conduction disease requiring change of pacing mode. Five patients (9-6%) died. Thirty four patients (22%) were paced DVI. The average age at implantation was 66-0 years. Mean follow up was 24-7 months. Two patients (3-9%) died. Average age of patients paced VVI was 72-1 years and mortality was 12 (20%). A remaining six patients were paced in other modes.

Electrophysiological studies at pacemaker implantation in patients with sinoatrial disease can identify a patient group who did not develop atrioventricular disturbance during follow up. Such patients can be safely and economically paced in AAI mode.

Relative contributions of myocardial elastic modulus and cavity geometry in determining left ventricular cavity stiffness in man

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To investigate the relative contributions of geometrical factors and myocardial elastic modulus (E) to overall left ventricular cavity stiffness simultaneous biplane angiograms and micromanometer pressures were recorded in 10 patients with left ventricular disease, and digitised frame by frame. Left ventricular volumes (V) were determined by three dimensional reconstruction. Regional left ventricular wall stresses were estimated by finite element analysis with a 72 brick model, which allowed for normal variation in fibre orientation across the wall. Values of E were determined by matching the volumetric change with incremental pressure on a frame by frame basis, throughout diastasis, from the end of rapid filling until the onset of atrial systole. Left ventricular cavity compliance, expressed as ΔV/ΔP, varied widely, from 0-23 to 189 cm³ mm Hg. In all patients there was a linear relation between E and 1/(cavity stiffness) throughout diastasis (r > 0.95 for all patients). Dimensional analysis indicated its slope to be a function of cavity volume, relative wall thickness, and cavity shape: V(dP/dV) = k(t⁸/r). (L¹²/r), where r = minor axis, L = long axis, t = wall thickness, and k is a constant. Retrospective calculation of cavity stiffness from these data gave r = 0.995.

Thus measured left ventricular cavity stiffness in man depends only in part on myocardial elastic modulus; it is modified to a large extent by the geometrical properties of the ventricle and, in particular, by cavity shape.

Can Doppler assessment of left ventricular function distinguish hypertrophic cardiomyopathy from other causes of diastolic dysfunction?

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Analysis of left ventricular filling by pulsed Doppler reflects the diastolic function of the heart. The specificity of this analysis, however, has not been readily ascertained. To evaluate the incidence of diastolic abnormalities we investigated 15 patients with aortic stenosis, 52 patients with myocardial infarction, 20 patients with hypertrophic cardiomyopathy, and 33 normal subjects. From the diastolic flow velocity profile, peak velocity (cm/s) during the early filling (E wave), the atrial contraction (A wave), and the ratio of A to E wave were measured. Twenty nine of of 52 (56%) patients with myocardial infarction, nine out of 15 (60%) with aortic stenosis, and 14 out of 20 (70%) with hypertrophic cardiomyopathy had at least one of the above diastolic indexes outside the 95% confidence limits of the control group. The E wave was lower in patients with hypertrophic cardiomyopathy than in normals (53 (15) v 61 (11), p < 0.025), but was
similar to the normal subjects in patients with myocardial infarction and aortic stenosis. The A wave was elevated in all three groups of patients when compared with the normal subjects (39 (10); 56 (11) in hypertrophic cardiomyopathy (p < 0.01); 51 (19) in myocardial infarction (p < 0.001); and 67 (25) in aortic stenosis (p < 0.005)). The A to E wave ratio was also raised in hypertrophic cardiomyopathy (1.1 (0.52); p < 0.005) in myocardial infarction (0.98 (0.52); p < 0.001), and in aortic stenosis (1.1 (0.48); p < 0.005) when compared with the normal subjects (0.69 (21)). The A wave was higher than the E wave in 20 out of 52 (38%) patients with myocardial infarction, in seven out of 15 (47%) patients with aortic stenosis, and in 11 out of 20 (55%) patients with hypertrophic cardiomyopathy, but in only two out of 33 (6%) normal subjects.

Pulsed Doppler shows abnormalities of filling in a high proportion of patients with hypertrophic cardiomyopathy, aortic stenosis, and myocardial infarction. The main characteristic of the Doppler inflow pattern in these patients is the significantly increased velocity during the atrial contraction. Such abnormalities of left ventricular filling are more common in patients with hypertrophic cardiomyopathy than in those with aortic stenosis or myocardial infarction but not specific for any of the three diseases.

Clinical significance of painless ST segment depression on exercise after myocardial infarction

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In order to investigate the clinical significance of painless ST segment depression on exercise, 105 patients were studied prospectively six weeks after myocardial infarction. Patients underwent stress testing by a modified Balke's protocol and left ventricular ejection was measured within three days of the stress test. Thirty one patients were symptom free with no ST change (group 1), 28 stopped with angina and/or dyspnoea with significant ST depression (group 2), 25 were symptom free but had significant (> 1 mm) ST depression (group 3), and 21 had symptoms with no ST change (group 4). The total mean (SD) exercise time in group 3 (9.1 (3.6) min) was not significantly different from that in group 1 (10 (4.4) min) or group 2 (7.9 (3.3) min), but it was significantly different from that in group 4 (6 (4.1) min) (p < 0.01). Resting ejection fraction was 53 (11.8%) in group 3 but was not different from group 1, 49 (11.6%) (p = NS), but was significantly higher than in group 2 (45 (13.3%) (p < 0.05) and group 4 (41.6 (17.2%) (p < 0.02)). During a mean follow up of 16 months five patients (20%) in group 3, 11 patients (35%) in group 1 (p = NS), 15 patients (54%) in group 2 (p < 0.02), and 10 patients (48%) in group 4 (p < 0.05) developed cardiac events (angina, congestive cardiac failure, reinfarction, operation, or death).
Painless ST segment depression on exercise six weeks after myocardial infarction does not seem to reflect impairment of functional capacity or left ventricular function, or predict cardiac events.

Dynamic coronary stenosis in patients with single vessel coronary artery disease: implications for therapeutic intervention

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Alteration in coronary vasomotor tone may be important in the genesis of ischaemia in patients with single vessel coronary artery disease (SVD), and the role of β blockers in the treatment of such patients is controversial. We studied 10 patients (nine men) aged 34–75 (mean 50 years) with SVD. To detect the variability in the ischaemic threshold all underwent serial treadmill exercise testing at a similar time on three days while off all antianginal medications. Nine patients also underwent testing with ergometrine off treatment. Patients then entered a double blind, randomised crossover study of a β blocker (atenolol, 100 mg/day) and a calcium antagonist (nifedipine 10 mg three times a day). Assessment was by treadmill exercise testing, ambulatory ST segment monitoring, and angina diaries. All patients had a positive treadmill test when off treatment, eight had a positive test when on nifedipine, and five when on atenolol. Atenolol significantly prolonged the time to 1 mm ST segment change when compared with both nifedipine (8.7 v 5.6 mm, p < 0.01), and no treatment (8.7 v 5.0 mm, p < 0.01). Atenolol also reduced the total duration of ischaemia on ambulatory monitoring significantly more than nifedipine (22.4 v 59.0 min, p < 0.05). Two patients showed considerable variability during treadmill testing in time to ischaemia, and a further four patients had a positive ergometrine test. In this subgroup with objective evidence of altered vasomotor tone atenolol was again an effective treatment.

β Blockade is an effective treatment in patients with SVD, even when there is objective evidence of alteration in vasomotor tone.

An in vivo assessment of infarct size

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There is no reliable method of accurately estimating infarct size in the acute clinical phase of myocardial infarction. To assess the usefulness of perfusion imaging to provide an estimate of infarct size 20 small pigs (11.8–16.7 kg) had gated thallium scans performed before and after medial sternotomy as control. Myocardial infarction was produced by two stage occlusion of the left anterior descending coronary artery. Two, 4, 6, or 8 hours after infarction 60 MBq of thallium-201 was injected intravenously and scans gated to the electrocardiograms were attained in the anterior and 45° left anterior oblique projection. Infarct size was expressed as a percentage of the whole left ventricle as seen on the end diastolic image and as a mean of two views. Hearts were then extracted and counted whole, and the infarct size was estimated. The open left ventricle was counted from both the endocardial and epicardial surfaces and again infarct size was expressed as a percentage of the whole left ventricle. The infarct sizes measured from the opened left ventricle correlated well with those measured in the intact heart (r = 0.93). In vivo infarct size varied from 15% to 30% of the left ventricle. The infarct size estimated in vivo correlated well with both that of the opened left ventricle and the intact ventricle, but correlation with the intact ventricle was better (r = 0.90). Infarcts of greater than four hours' duration were successfully stained with tetrazolium and infarct weights correlated well with infarct scans.

In the pig model infarct sizing by gated thallium scans seems to be accurate. This technique can be performed in patients and promises to be a method of infarct sizing that can be used during the acute stage.

Doppler detected mitral regurgitation after myocardial infarction: relation to site of infarction and left ventricular function

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To assess the frequency and severity of mitral regurgitation (MR) in the chronic phase of myocardial infarction (MI) 80 patients had pulsed Doppler echocardiography with assessment of MR and radio-nuclide ventriculography with measurement of left ventricular ejection fraction (LVEF) both at a mean of 42 days after the onset of MI. MR was found in 14 (35%) of 40 patients in anterior MI compared with 17 (42%) of 40 patients in inferior MI (p = NS). MR was at least moderate in seven patients with anterior MI and in five patients with inferior
MI. Mean LVEF in patients with anterior MI and MR was 0.27 (0.07) and it was 0.38 (0.12) in those without MR (p < 0.005). When MR was at least moderate, mean LVEF was 0.22 (0.06) as compared with 0.32 (0.05) when MR was only mild (p < 0.005). For inferior MI mean LVEF in patients with MR was 0.41 (0.1) vs 0.45 (0.10) in patients without MR (p = NS). There were no significant differences in mean LVEF between patients with at least moderate MR (0.37 (0.11)), mild MR (0.43 (0.11)), or no MR (0.45 (0.10)).

Mitrail regurgitation is not uncommon after MI and its frequency in anterior and inferior MI is similar. Both its occurrence and severity in patients with anterior MI are related to depression of the LVEF. The mechanism of mitral regurgitation may be different in inferior MI since neither its occurrence nor its severity seemed to be related to the LVEF.

Low dose enoximone is haemodynamically effective in refractory heart failure

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The phosphodiesterase inhibitor enoximone has major inotropic and vasodulatory properties. Doses of between 3 mg/kg and 6 mg/kg have been used and were associated with appreciable side effects. We have studied the effectiveness of a lower dose of enoximone (100 mg) three times a day in 14 patients with refractory heart failure. Baseline values of cardiac index (CI), pulmonary capillary wedge pressure (PCWP), and pulmonary artery pressure (PAP) were measured with a thermodilution catheter. Systolic blood pressure (SBP) and heart rate (HR) were also measured. All these variables were measured again 1, 2, 4, 6, and 8 hours after 100 mg of enoximone and again at 24 hours after further doses of enoximone at eight and 16 hours. During the first eight hours, CI increased by 33% from a baseline of 2.07 l/min/m² (p < 0.022), PCWP decreased by 33% from a baseline of 23.28 mm Hg (p = 0.004), PAP decreased by 22% from a baseline of 35.92 mm Hg (p = 0.017), and RAP decreased by 41% from 9.43 mm Hg (p = 0.024). SBP and HR showed no significant change during this period. At 24 hours a 36% improvement in CI (p = 0.02), a 46% improvement in RAP (p = 0.013), and a 14% improvement in PAP were maintained (p = 0.025). PCWP, SBP, and HR showed no significant change from baseline at this stage. One patient had nausea.

Enoximone in low doses is haemodynamically effective and well tolerated.

Influence of platelets on arrhythmias and haemodynamic function during coronary occlusion

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To investigate the possible role of platelets in mediating the adverse effects of coronary occlusion we studied the occurrence of ventricular arrhythmias and systemic pressure during occlusion of the left anterior descending coronary artery (LAD) in rabbits with different states of platelet activation. In untreated controls (n = 10) ligation of the LAD produced immediate hypotension. Blood pressure fell by 17 (3-3) mm Hg at two minutes and ventricular fibrillation occurred in 50%. In animals pretreated with platelet antiserum (n = 8) platelets were reduced by 98% from 510 (14) x 10⁹/l and LAD ligation caused less hypotension (a fall of 6 (0-6) mm Hg (p < 0.05 vs control)) and ventricular fibrillation (25%). When platelet activating factor (acetyl-uglycerol ether phosphocholine) 1 µg/kg was given intravenously 10 minutes after the onset of LAD occlusion in seven separate control animals it produced immediate profound hypotension (a fall of 61 (5-8) mm Hg in all seven and VF in six). Pretreatment with platelet antiserum (n = 8) substantially blunted the hypotensive response to the platelet activating factor (a fall of 11 (2-1) mm Hg) and ventricular fibrillation in two (25%) (p < 0.05).

These results indicate that platelet activation may contribute to the haemodynamic and arrhythmogenic effects seen during early coronary occlusion.

The mechanism of shock in right ventricular infarction

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Shock associated with right ventricular infarction is well recognised and with appropriate treatment the prognosis is good. The mechanism of shock in right ventricular infarction has been debated. We studied seven patients with clinical cardiogenic shock after transmural inferior myocardial infarction that persisted despite appropriate fluid volume replacement. All were in sinus rhythm. No patient had been taking...
β blockers. All had indwelling pulmonary artery flotation catheters and femoral arterial lines. The mean right atrial pressure (21·3 (3·1) mm Hg) exceeded the pulmonary artery wedge pressure (16·9 (3·9) mm Hg). The average mean arterial pressure was 48·6 (12·1) mm Hg, mean cardiac index 1·34 (0·30) l/min/m², mean left ventricular stroke work index (LVSWI) 6·7 (3·5) gm/m², and right ventricular stroke work index (RVSWI) 0·2 (0·4) gm/m².

After treatment with intravenous dobutamine and glyceryl trinitrate and varying combinations, mean arterial pressure increased to 82·7 (15·3) mm Hg (p < 0·01 compared with baseline) and mean cardiac index to 27·6 (9·3) gm/m² (p < 0·01) but mean RVSWI was unchanged at 0·5 (0·5) gm/m² (p > 0·01). There was no significant change in heart rate, mean right atrial pressure (23·0 (2·8) mm Hg), or pulmonary artery wedge pressure (19·0 (2·7) mm Hg) after treatment.

Reversal of shock was thus associated with an increase in LVSWI but without a concomitant increase in RVSWI, which suggests that the probable mechanism of shock in right ventricular infarction is left ventricular dysfunction.

The hyperadrenergic heart syndrome

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Patients with palpitation caused by persistent and excessive sinus tachycardia in response to normal physiological activities have been called “hyperadrenergic” and it has been suggested that these features are the result of increased sensitivity of cardiac β receptors. We have compared the autonomic responses of eight hyperadrenergic (HA) women with those of seven normal women. Heart rate and blood pressure were measured supine and with 90° head-up tilt, and again after propranolol 0·2 mg/kg alone and propranolol with atropine 0·04 mg/kg. The response of heart rate to isoprenaline infusions (0·5–4 μg/min) was measured in supine subjects. Mean supine heart rate was 91·6 (14·3) beats/min in HA patients and 60·8 (4·2) beats/min in controls (p < 0·001). Mean increase in heart rate on tilting was 53·9 (23) in HA patients and 28·8 (8·3) in controls (p < 0·001). There was no significant difference in blood pressure between HA and controls and no significant change in blood pressure after tilting in either group. In the HA women mean plasma noradrenaline was 1·79 (0·77) nmol/l when subjects were supine and 4·72 (1·12) after tilting and 0·80 (0·78) nmol/l in supine controls and 1·83 (0·76) after tilting the controls (HA v controls, p < 0·005, both supine and after tilting; and supine v tilting, p < 0·02 in both groups). There was no difference in plasma concentrations of adrenaline between the groups. The increase in β blocked heart rate on tilting and with the addition of atropine was similar in HA women and controls. The increase in heart rate during isoprenaline infusion was 9·5 to 15·6 beats/min greater in HA women than in controls (p < 0·05 at doses up to 1·5 μg/min).

These results suggest that HA patients have increased resting sympathetic tone and an exaggerated sympathetic reflex response to postural change.