Proceedings of the British Cardiac Society

THE 62ND ANNUAL GENERAL MEETING of the British Cardiac Society was held at the University of Bristol Students Union on Wednesday and Thursday, 13 and 14 April 1983. The President, M F OLIVER, took the Chair during private business. At the scientific sessions the Chair was taken by D W BARRITT.

The following were elected to the British Cardiac Society.

New Ordinary Members: D A Brennand-Roper (Kent); D C Cumberland (Sheffield); I A S Davis (Stafford); F G Dunn (Glasgow); P J Ell (London); G F George (Southampton); J D Hearse (London); M M Khan (Belfast); R Leanage (Leicester); G P McNell (Dundee); G W Morrison (Yorkshire); J R Pepper (London); M T Rothman (London); G R Sutherland (Southampton); R D S Watson (West Midlands); R P Havelock Wilde (Bristol).

Corresponding Members: W Bleifeld (West Germany); K Chatterjee (USA); F H Epstein (Switzerland); V Fuster (USA); D C Harrison (USA); S A Ismail (Sudan); S Lenzi (Italy); F Loogen (West Germany); J Ross (USA); B Sobel (USA); M Sznajderman (Poland).

New Overseas Member: C Ilsley (New Zealand).

M F Oliver re-elected President for following year. C P Aber, M J Godman, and M Stephens elected to Council.

The following died during the past year: W H Craib, G J Gavey, J S Glennie, Sir Ian Hall, Kt, CBE, and R E Steen.

Abstracts of papers

Electrocardiographic abnormalities associated with oxygen desaturation in sleep apnoea syndrome
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Recent studies have shown that morbidly obese subjects develop sleep disordered breathing and nocturnal hypoxaemia. This study was undertaken to investigate the occurrence of electrocardiographic abnormalities and arrhythmias in five obese men, weighing between 76 and 172 kg (mean 129 ± 38, SD). Complete sleep studies, ear oximetry, and ambulatory cardiographic monitoring were carried out before and after weight reduction. Multiple episodes of central and obstructive apnoea occurred in all and during a single night of monitoring oxygen saturation fell to 50% in three subjects. During the period of oxygen desaturation, the QTc was lengthened from 0.35 to 0.52s in two and they both had multiple episodes of sinus tachycardia and premature ventricular contractions. All patients had frequent runs of sinus arrhythmia during apnoeic episodes. After a mean weight reduction of 6.2 ± 3 kg there was a remarkable reduction in the frequency of apnoeic episodes (< 5% of the initial figure), the oxygen saturation improved to over 90% and the QTc remained within normal limits. These findings show that life threatening electrocardiographic abnormalities occur in association with sleep disordered breathing in otherwise healthy obese men, and that even a modest degree of weight reduction may prevent these.

Ambulatory electrocardiographic ST segment changes in normal individuals

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Changes in the ST segment during ambulatory electrocardiographic monitoring are often interpreted as ischaemic episodes in patients with coronary artery disease, but the frequency and magnitude of similar changes occurring in normal subjects have not been established.

We studied 120 normal volunteers: 50 men and 50 women below 40 years and 20 men between 40 and 60 years. Twenty-four hour ambulatory monitoring was performed with a frequency modulated recorder and precordial leads V2 and V5 were recorded. Twenty-eight subjects (22 men, six women) developed ST segment elevation (range 1 to 3 mm) which occurred almost invariably at night, with a slow heart rate (62-4 ± 10-4 bpm). ST segment elevation occurred gradually over several hours in the majority, with superimposed shorter bouts of further elevation, and was not found in subjects over 37 years of age.

Ten subjects (age 31.7 ± 8.6 years) developed episodes of horizontal or downsloping ST segment depression (range 1 to 2 mm) in lead V5. ST segment depression usually occurred with a tachycardia (135 ± 19.5 bpm). None of these subjects were exercised on a bicycle ergometer and widespread ST segment depression occurred in eight.

These findings must be considered when using ambulatory monitoring of the ST segment for the diagnosis of evaluation of ischaemic heart disease.
Detection of transient myocardial ischaemia in stable angina by 24 hour continuous monitoring: pathophysiological and clinical implications

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In order to evaluate transient myocardial ischaemia objectively during daily life, 30 patients with "stable angina" (five to 60 attacks a week) and positive exercise tests (heart rate to ST depression 100 to 150) were studied by ambulatory ST segment monitoring, on glyceryl trinitrate alone over four consecutive days (120 tapes). Variability was assessed over 18 months in 20 patients by repeated 48 hour monitoring (four to 14, mean eight times a patient—326 tapes). Findings were related to angina (diaries), heart rate, serial exercise tests (135 tests), and compared with 100 normal subjects (age 20 to 62). Transient ST depression of 1 mm was found in only two normal subjects. In the patients, there were 1934 episodes of ST depression (911.1 mm, 638.2 mm, 385 > 3 mm) of which only 470 (24%) were accompanied by pain. Symptomatic episodes were slightly longer and had greater ST depression than asymptomatic episodes (p < 0.01), despite wide overlap. Heart rate rose by less than 10 beats during the minute preceding ST depression in 1489 (78%) episodes. The heart rate at the onset of ST depression was significantly lower than the rate at the onset of ST depression during exercise testing (98 ± 20 vs 127 ± 15 beats a min, p < 0.01). Over 18 months, there was pronounced variability in the number of ST episodes with remissions (four patients) and exacerbation (six patients). These were consistently underestimated by symptoms and not reflected in the exercise tests. Thus, heart rate increase appears to be an infrequent determinant of myocardial ischaemia during daily life, suggesting that transient impairment in oxygen supply may contribute. Ambulatory monitoring discloses that patients with "stable angina" may have highly variable electrocardiographic evidence of ischaemia, which is not detected by conventional assessment.

Effect of prehospital coronary care on community mortality from myocardial infarction

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The majority of deaths from myocardial infarction occur within the first few hours before possible admission to hospital. The apparently obvious value of early prehospital coronary care, however, has never been established. This study compares the mortality from myocardial infarction in two similar communities, Omagh (population 52 224) and Ballymena (population 111 339) served by hospitals with similar hospital, coronary care and, in Ballymena, by mobile coronary care.

During one year definite or possible myocardial infarction (WHO criteria) was documented in 222 and 429 patients in Omagh and Ballymena, respectively. The incidence per 1000 population was similar in these two areas. The median delay from onset of pain to initiation of coronary care, either in hospital or by the mobile unit, was four and a half hours in Omagh compared with one and a half hours in Ballymena. In Omagh 53% of patients reached hospital alive, while in Ballymena 70% were admitted to hospital or received prehospital coronary care. For patients who received coronary care the three month mortality rates were similar in the two areas, but the total community mortality was 61% in Omagh and 49% in Ballymena (p < 0.01). This suggests that early prehospital coronary care produced a significant reduction in the early mortality from myocardial infarction.

Failure of intravenous prostacyclin to influence initial progress of patients with unstable angina

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It has been suggested that the combination of coronary vasospasm and platelet aggregation induced by thromboxane release may have an important and deleterious role in patients with unstable angina. The therapeutic value of prostacyclin with its vasodilator and antiplatelet aggregatory actions was therefore investigated intravenously in seven patients with unstable angina. Serial haemodynamic, metabolic, and electrocardiographic (16 point precordial electrocardiographic mapping and vectorcardiography) observations were made over a 24 hour period. Prostacyclin was given as an incremental infusion up to a maximum tolerated dosage followed by a maintenance infusion. The range was from 2.5 to 7.5 ng/kg per min.

During dose titration, potent systemic vasodilatation was observed, with a 28% fall in systemic vascular resistance (p < 0.01) at the peak dose (7.5 ng/kg per min), but systolic pressure was unchanged. Cardiac output and heart rate increased from 4.7 to 6.3 l/min and 71 to 82 beats/min, respectively (p < 0.01).
Coronary flow increased from 117 to 174 ml/min (p < 0.01), because of an increase in oxygen consumption (12.9 to 19.1 ml/min, p < 0.02). Associated with these effects four patients developed further angina, mean lactate extraction ratio fell from 19.4 to 4.2% (p < 0.02), and the sum of negative ST segments increased from -283 to -524 µV (p < 0.05). Similar haemodynamic effects were maintained during 24 hours maintenance treatment but recurrent chest pain occurred in three of the five patients who completed this phase.

These results do not indicate an initial beneficial effect of prostacyclin given at maximum tolerated dosage in patients with unstable angina.

**Lack of correlation between ventricular late potentials and inducibility of malignant ventricular tachyarrhythmias after experimental canine myocardial infarction**

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The detection of latent electrical instability by induction of ventricular tachyarrhythmias or by recording ventricular “late potentials” from highly amplified, signal averaged electrocardiograms has been advocated as a method of identifying patients at high risk of sudden death after myocardial infarction. We have compared the presence of delayed ventricular activation in epicardial recordings (late potentials) with the inducibility of ventricular tachyarrhythmias in 15 conscious dogs after experimental myocardial infarction.

A total of 57 programmed ventricular stimulations was performed three to eight days after infarction. Ventricular fibrillation occurred in four, sustained ventricular tachycardia in 18, and non-sustained tachycardia in 28. Two dogs with sustained tachycardia on day 3 were stable by day 7, while five dogs progressed from non-sustained to sustained ventricular tachycardia between days three and seven. There was no significant difference in the overall duration of ventricular activation in stimulations resulting in fibrillation (108 ± 31 ms, mean ± SEM), sustained tachycardia (87 ± 21 ms), non-sustained tachycardia (88 ± 6 ms), or no tachycardia (78 ± 2 ms). Daily variations in duration of ventricular activation occurred, independent of the changes in inducible tachyarrhythmias.

The predictive value of inducible ventricular tachycardia or the presence of ventricular late potentials for subsequent sudden death is likely to be poor in the early postinfarction period.

**Digital subtraction angiography in diagnosis of left ventricular aneurysm**

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In twenty five patients diagnostic left ventriculograms were obtained after intravenous injection of contrast medium using the Technicare DR 960 digital subtraction angiography system. All patients had previously been investigated by conventional left ventriculography. Using this new technique ectopic free ventriculograms were obtained in all cases. All investigations were carried out on an outpatient basis with no complications. The digital left ventriculograms obtained in nine normal patients showed symmetrical contraction patterns. In the abnormal cases there was a clear distinction between globally poor left ventricular function (10 patients) and those with left ventricular aneurysms (six patients). In each case there was agreement between the diagnosis obtained by conventional ventriculography and that obtained by digital subtraction.

This new technique allows relatively simple, ectopic free, diagnostic investigation of left ventricular wall movement without the need for arterial puncture. It permits serial investigation on an outpatient basis. The results of this study suggest that digital subtraction angiography is a potentially valuable technique allowing discrimination of those patients with a resectable left ventricular aneurysm from those with global impairment of wall movement.

**Evaluation of coronary artery bypass graft patency by computerised tomography**

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In order to evaluate the sensitivity of computerised tomography in identifying coronary artery bypass graft patency we have studied seven patients with 18 grafts by computerised tomography and angiography. The two procedures were performed within a 10 day period. Four dynamic scans were performed at the level of the aortic root after an intravenous bolus injection of contrast medium. The injection was timed to provide left heart opacification during the second and third scans. Selective angiography demonstrated 11 patent grafts, two grafts showed distal retrograde filling at
coronary angiography (one internal mammary artery and one vein graft not entered selectively), four grafts were occluded, and one graft was not demonstrated.

Computerised tomography demonstrated 14 patent grafts. Four grafts were not visualised and were assumed to be occluded. All 11 angiographically patent grafts were shown by computerised tomography. The two grafts showing retrograde filling were also visualised by computerised tomography. Of the four grafts not visualised, three were occluded and one was not demonstrated angiographically. One graft was thought to be patent by computerised tomography: angiography four days later, however, showed it to be occluded. These results suggest that computerised tomography is as accurate as angiography in demonstrating graft patency.

Two dimensional subtraction encoding: method of increasing two dimensional diagnostic versatility

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We describe a simple colour encoding system for use with two dimensional video images which has increased our diagnostic accuracy. Sixty-four grey levels are converted to eight colour codes with variable window and gain controls. Unlike other systems any colour or set of colours can be subtracted. There is no grey level superimposition.

The system has proven most valuable in tissue characterisation and boundary definition especially with poor quality images, as unwanted echo return can be eliminated.

The system enables differentiation between myxomatous tissue and vegetative material, the latter tending to show uniform higher density echogenicity. Myxomata tend to have a "patchy" encoded appearance. Left ventricular thrombus can be more readily indentified and determined to be fresh or organised. The appearances are quite different from tumour or vegetative tissue.

The system has shown differences between normal myocardial tissue and that seen in cardiomyopathies, particularly hypertrophic cardiomyopathy.

The subtraction technique has been of value in improving endocardial boundary definition, therefore giving a better appreciation of left ventricular regional wall motion.

We feel the subtraction facility offers greater versatility in two dimensional encoding, frequently improving the original image record in a manner not available in other encoding systems.

Re-evaluation of echocardiographic criteria for diagnosis of hypertrophic cardiomyopathy

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The diagnosis of hypertrophic cardiomyopathy is based upon the demonstration of left ventricular hypertrophy in the absence of a cause. The most widely accepted current diagnostic criteria are echocardiographic; these emphasise the asymmetrical nature of the disease and features associated with an outflow tract gradient. In our 289 patients we diagnosed the disorder on the basis of clinical and angiographic criteria in 285; in only four was echocardiography the primary diagnostic tool. To determine the incidence of diagnostic echocardiographic findings we have performed simultaneous M-mode and two dimensional echocardiography in 89 of these 285 patients. At least one of the segments of the left ventricular wall was thickened (> 1.4 cm) in all, but conventional M-mode diagnostic criteria (asymmetrical septal hypertrophy (ratio ≥ 1.5), systolic anterior motion of the mitral valve, or mid-systolic closure of the aortic valve) were seen in only 42. The pattern of hypertrophy as assessed by the two dimensional echocardiogram was asymmetrical (41), symmetrical (41), or predominantly distal ventricular (seven). These echocardiographic findings confirmed the presence of left ventricular hypertrophy but showed that asymmetrical septal hypertrophy was less frequent than expected (45%). Current echocardiographic diagnostic criteria therefore require modification, and should emphasise the presence of hypertrophy rather than rely on the demonstration of asymmetrical septal hypertrophy or features of "obstruction".

Accurate prediction of regional distribution of coronary artery disease using R wave adjusted ST segment depression

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Previous attempts to correlate the distribution of coronary disease with the pattern of ST segment depression in the 12 lead exercise electrocardiogram have been unsuccessful. Based on the observation that there appears to be a close relation between the depth of ST depression in any lead and the amplitude of its R wave we have devised a method of relating ST segment changes to R wave amplitude. Using this technique an accurate correlation has been obtained between assessments of regional ischaemia judged...
angiographically and electrocardiographically.

Data from 28 selected patients were analysed. All had normal resting electrocardiograms with ≥ 2 mm ST depression on exercise in at least one lead. The most ischaemic myocardial region was predicted (a) from the lead(s) showing the greatest ST segment depression and (b) from the lead(s) showing greatest ST depression relative to the amplitude of its R wave. The coronary angiograms were analysed using a scoring system which relates the severity of disease to specific myocardial regions.

In 16 of 28 patients the most diseased region was confidently identified from the angiogram. Seven were correctly predicted using absolute ST depression, four were incorrectly assigned, and five were indeterminate. Inferoposterior disease was wrongly predicted in four patients with anteroseptal disease. R wave adjusted ST depression correctly predicted all 16 regions.

Hence this simple technique of relating ST depression to R wave amplitude led to greater accuracy in predicting the distribution of coronary disease.

Quantitative assessment of severity of coronary artery disease using ST segment/heart rate relation during atrial pacing

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Unlike other, conventional exercise tests, the maximal rate of progression of ST segment depression relative to increases in heart rate (maximal ST/HR slope) accurately predicted the presence and severity of coronary artery disease in 230 consecutive patients with chest pain. The usual atrial pacing tests, using conventional criteria, have not been reliable. The ability of the ST/HR slope during atrial pacing to predict severity of coronary artery disease has now been examined in 22 patients with angina.

The maximal ST/HR slope during atrial pacing averaged 21.0 mm/beat per min per 10^3 (range 15 to 24) in five patients with one vessel disease, 46.2 mm/beat per min per 10^3 (range 37 to 60) in 12 patients with two vessel disease, and 92.3 mm/beat per min per 10^3 (range 70 to 116) in five patients with three vessel disease. In the same patients, the maximal ST/HR slope during exercise were 17.7 mm/beat per min per 10^3 (range 14 to 21), 42.7 mm/beat per min per 10^3 (range 34 to 56), and 92.5 mm/beat per min per 10^3 (range 74 to 112), respectively. The maximal ST/HR slopes obtained by the two methods correlated closely (r = 0.9, p < 0.001). For each method, the values within each group were significantly different from each other (p < 0.0005, unpaired t test) but not significantly different from those previously reported.

These findings suggest that the ST/HR slope during atrial pacing may be used as a reliable alternative to the exercise measurement for accurate prediction of severity of coronary artery disease.

Are “long acting nitrates” effective in effort related angina pectoris?

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There is considerable renewed interest in “long acting nitrates” as routine oral treatment in effort related angina pectoris; but there is little information about their efficacy compared with the standard treatment, beta blockade. We have investigated the antianginal effects of isosorbide dinitrate 40 mg four times daily, atenolol 100 mg per day, alone and in combination, in 15 patients with effort related angina pectoris in a double blind placebo controlled clinical trial, each treatment period lasting four weeks.

Although atenolol reduced the total number of episodes of angina by 40%, only the combination reduced these significantly and reduced glyceryl trinitrate consumption from 218 episodes on placebo to 117 (p < 0.02) and from 189 tablets to 96 (p < 0.02), respectively. The area of exercise induced ischaemia on 16 point precordial electrocardiograph mapping fell from 418 points on placebo to 160 (p < 0.01) and 141 (p < 0.01) on atenolol and the combination, respectively. Similarly, the number of episodes of ST segment depression ≥ 1 mm on ambulatory electrocardiographic monitoring fell from 101 episodes per 48 hours to 51 (p < 0.05) and 40 episodes (p < 0.01) on atenolol and the combination, respectively.

We conclude that in effort related angina pectoris isosorbide dinitrate is not an alternative treatment to beta blockade but the effect of the latter is enhanced by its combination with isosorbide dinitrate. In addition, the variability in anginal frequency and glyceryl trinitrate consumption emphasises the importance of exercise testing and ambulatory monitoring in the assessment of drug responses.
The “retrospectroscope”

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Continuous monitoring of the electrocardiogram is of
great practical value in the diagnosis of paroxysmal
arrhythmias and transient ischaemic ST changes. The
limitation of the surveillance time to 24 to 48 hours
means that positive results will only be achieved in
patients with frequent episodes. In addition, the
analysis of long periods of electrocardiogram is time
consuming, and yields negative results in a high propor-
tion of cases.

We have devised a small portable solid state recor-
der which is responsive to patient activation, storing
the 150 seconds of electrocardiogram immediately
before activation with the succeeding 10 seconds of
the electrocardiogram. It can also be programmed to
respond automatically to bradycardia, tachycardia, or
detection of a pacing spike. It is relatively inexpensive
and can run continuously for up to three weeks. At
the end of this time, only positive findings are shown
from memory and these are limited to single 160 sec-
ond period or to four 40 second periods.

This new simplified approach to ambulatory moni-
toring of the electrocardiogram has been of con-
siderable value in the investigation of infrequent near
syncopal and syncopal episodes and in the detection of
intermittent cardiac pacing. The latter application
may be useful in the assessment of the need for pacing
in selected patients and in the evaluation of pacemaker related symptoms.

Transtelephonic arrhythmia diagnosis: an advance?

C A Bucknall, I Broomen, G Dunkley, P V L Curry
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Conventional diagnostic methods often fail to capture
paroxysmal cardiac events and effective management
may be delayed. The comparative diagnostic value of
the clinical history, a single period of 24 hours elec-
etrocardiographic monitoring, and a patient activated
“symptomatic” electrocardiographic recorder was
studied prospectively in 20 patients (10 male, 10
female, age range 11 to 67 years).

The suspected diagnosis from the history was
selected from: paroxysmal tachycardia regular;
paroxysmal tachycardia irregular; extrasystoles;
hypotensive episode; neurological event; anxiety
state. Patients then received the pocket-sized car-
diomemo recorder until (i) an abnormal rhythm
responsible for the attack was captured, (ii) three
“attacks” had been recorded without an abnormality
of cardiac rhythm or rate, or (iii) until two weeks had
elapsed.

Eighteen patients used the cardiomemo recorder: 16
achieved a good recording and transtelephonic
transmission. In 12 patients, the diagnosis obtained
from the 24 hour tape and cardiomemo were the same
(supraventricular tachycardia one; ventricular
extrasystoles two; sinus tachycardia five; sinus
rhythm four). In four patients the cardiomemo
confirmed and clarified the suspected diagnosis,
whereas the 24 hour tape was normal (atrial flutter
one; supraventricular tachycardia one; sinus
tachycardia two). The 24 hour tape failed to make a
diagnosis both in the two who did not use the recorder
within two weeks and in the two who did not achieve a
good recording.

The control of frequent daily episodes of atrial
tachycardia in a patient receiving initial high dose
amiodarone was conveniently monitored with daily
transtelephonic electrocardiographic strips both of
symptomatic events and sinus rhythm for QT meas-
urement.

For patients with paroxysmal cardiac symptoms
the cardiomemo is a more reliable diagnostic tool (16/16)
than the clinical history alone (12/16), 24 hour elec-
trocardiographic recording (12/16), or their combina-
tion (14/16).

Is there a difference between sick sinus syndrome
and carotid sinus syndrome?

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It has been suggested that patients with carotid sinus
syndrome (CSS) have sinoatrial disease as the basis of
their symptoms. Fifty-five paced patients with iso-
lated CSS and 55 age matched paced patients with
isolated sick sinus syndrome (SSS) were compared at
presentation and followed up for a mean of 30
months. At presentation the corrected sinus node
recovery time in the CSS group was 328 ± 106 ms and
in the SSS group 1878 ± 2058 ms (p < 0.01). Resting
heart rate was 70 ± 15 bpm and 63 ± 14 bpm in the
SSS group (p < 0.01). There was a pronounced male
predominance in the CSS group (CSS 85%, SSS 40%,
p < 0.001). At follow up there has been a 25% inci-
dence of paroxysmal tachycardias in the SSS group
compared with 2% in the CSS group (p < 0.001).
Intrinsic heart rate has been measured in 21 patients
by autonomic blockade with 0.2 mg/kg propranolol
and 0.02 mg/kg atropine intravenously. Intrinsic heart
rate in the CSS group was 79 ± 5 bpm (n = 9) and 51
± 10 bpm (n = 12) in the SSS, p < 0.001. Maximum

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exercise heart rate (at comparable work loads) was 134 ± 16 bpm in the CSS group (n = 11) and 90 ± 24 bpm in the SSS group (n = 7) p < 0.001. In 18 patients sinus node recovery times could be remeasured non-invasively using atrially programmable pacemakers over rates 80 to 130 bpm. Corrected sinus node recovery time in CSS patients was 405 ± 207 ms (n = 8) and it was 1703 ± 1098 ms in the SSS group (n = 10) p < 0.01; these values were not significantly different from those measured before pacing. Repeat carotid sinus massage in 76 patients was positive in 80% of the CSS group (n = 40) and was negative in all the SSS patients (n = 36) p < 0.001. Carotid sinus syndrome may of course exist alongside either SSS or atrioventricular block in patients with symptomatic bradycardia. In 78 patients seen at this centre with CSS 5% have had additional evidence of atrioventricular block, 23% have evidence of additional SSS, and 5% have SSS and atrioventricular block. These results suggest that CSS can exist as an isolated entity and does not evolve into sinus node disease.

Implantable pacemakers for tachycardia termination: experience in 28 patients

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St Bartholomew’s Hospital, London

Since 1972, 28 patients, aged 12 to 73 (mean 47) years, have had tachycardia reversion pacemakers implanted. Thirteen had atrioventricular nodal tachycardia, 12 atrioventricular tachycardia, and three ventricular tachycardia. Ten originally had patient activated rapid atrial pacing systems (Medtronic 1515/5998), 12 had Telectronics 4151 automatic scanning pacemakers, one in the atrium (double stimuli) and 11 in the ventricle (three single, eight double stimuli), three had patient activated rapid ventricular systems (Cordis 239A, Lucas, Medtronic 5998), two had magnet operated ventricular underdrive devices (Arco B, CPI Microlith) and one a Telectronics Optima MP dual demand pacemaker. Tachycardia cycle lengths varied from 260 to 430 (mean 339) ms. All had electrophysiological studies before and after implant when the pacing mode chosen was shown to be repeatedly effective.

During follow-up of 0.25 to 10 (mean three) years, five rapid atrial pacemakers were changed to automatic scanning pacemakers (one single, two double ventricular stimuli, two double atrial stimuli) because of pacing induced atrial fibrillation in three, ineffectiveness in one, and tachycardia induced confusion in one. A patient with a ventricular underdrive pacemaker was changed to a rapid atrial pacemaker and later required His bundle section. One patient with cardiomyopathy died from cardiac failure. One automatic scanning pacemaker has been disabled after successful accessory pathway ablation at the time of coronary bypass surgery. Nine patients have required antiarrhythmic medication (seven with automatic scanning pacemakers, one with rapid ventricular pacemaker, one with dual demand pacemaker). Of the 25 patients currently using tachycardia reversion pacemakers, 18 have automatic systems which, together with simple antiarrhythmic drug therapy in eight, provide them with complete arrhythmia control.

Clinical use of new automatic antitachycardia pacemaker

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A new fully automatic antitachycardia pacemaker has been implanted in four patients and used externally in a further four. The pacemaker will not attempt to terminate a physiological (for example exercise induced) tachycardia, but has several possible responses to pathological tachycardias. The simplest is to deliver preprogrammed stimuli with fixed timing, but the apparatus is also capable of “learning” the most effective stimulation sequence to terminate individual tachycardias. This memory function is updated after each tachycardia whether successfully terminated or not by assigning a ranking number to the successful or unsuccessful sequence. The least successful sequence is dropped from the memory after each event, so that as the episodes of tachycardia accumulate the pacemaker automatically responds with the stimulation sequence most likely to be effective.

The first implanted unit has been followed since late November 1982 and in all eight patients repeated tachycardias have been successfully terminated.

Non-automatic programmed stimulation can be performed after implantation and the pacemaker has a fully programmable anti-bradycardia capability.

Ablation of atrioventricular conduction by high energy shock through electrode catheter: method of treating refractory supraventricular tachycardias

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Eight patients aged between 37 and 69 years were referred for treatment of recurrent supraventricular tachycardias resistant to antiarrhythmic therapy. Three patients had failed to respond to external cardioversion. Three patients had associated illnesses which would have made surgical ablation a high risk. Transvenous ablation was therefore considered appropriate. With a ventricular pacing electrode in situ, His potentials were recorded using a bipolar pacing electrode. When large amplitude spikes were recorded, one pole was connected to standard DC cardioversion apparatus with the second paddle connected to a backplate. A shock of 275 Watt-seconds was delivered through the electrode. Two patients developed complete atrioventricular block immediately and three after a second shock (one after 48 hours). In one patient, transient atrioventricular block reverted to sinus rhythm after 24 hours and repeat shocks were not effective. In two patients persistent atrioventricular block could not be produced. All patients were restudied before permanent pacemaker implantation. In four patients with complete atrioventricular block an escape rhythm with QRS complexes indetical to the previous conducted QRS was noted. Atropine accelerated this rhythm in one patient. The procedure was without side-effects and four patients remain in atrioventricular block at two to 14 months. Two patients have intermittent atrial fibrillation. Another patient has no further attacks of intranodal tachycardia. One patient died with pneumonia 48 hours after the procedure. It is concluded that this method is safe and effective in controlling otherwise refractory supraventricular tachycardias in selected patients.

Management of critical aortic stenosis in infancy


Critical aortic valvular stenosis presents in infancy with severe congestive heart failure. Clinical assessment and electrocardiography are of value, but cardiac catheterisation with angiography has been considered mandatory before surgical treatment. With cross-sectional echocardiography an accurate diagnosis of aortic valve stenosis and associated lesions is possible. Over the past two years, we have established a procedure, where if a clinical diagnosis of critical aortic stenosis is confirmed by cross-sectional echocardiography in the absence of major associated cardiac anomalies, infants are submitted for aortic valvotomy under inflow occlusion without invasive studies. Eight infants with critical aortic stenosis have been operated on, five without prior cardiac catheterisation. Age at operation ranged from two days to seven months, with six children less than two weeks. The non-invasive diagnosis was in each case confirmed at operation. There was one early post-operative death and one late death. No death has been related to the technique of inflow occlusion. Accurate assessment of sick infants with critical aortic stenosis by two dimensional echocardiography can be followed immediately by valvotomy under inflow occlusion. This technique provides excellent exposure and adequate time for valvotomy and has been our technique of choice for the past two years.

Challenge of cardiac arrhythmias in children

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The management of cardiac arrhythmias in children differs at almost every stage: for example the clinical presentation, available history, logistics of investigation and treatment, and the nature of associated cardiac diseases – a conclusion based on experience of 43 children (aged 1 month to 15 years; mean 9 years 4 months) attending a paediatric arrhythmia clinic between March and December 1982.

Symptoms in 39 were caused by cardiac arrhythmias; four others had epilepsy (two) or vasovagal attacks (two). The presentation of paroxysmal supraventricular tachycardia arrhythmias complicating an overt WPW syndrome (six), the sick sinus syndrome (three), atrial tachycardia (two), ventricular tachycardia (four), and complete heart block (four) varied from palpitations or heart failure to collapse or vomiting. Eight children were found to have minor arrhythmias (for example extrasystoles, pronounced sinus arrhythmia, or second degree atrioventricular block). It was not always possible to obtain a reliable history from the child nor from the parents if attacks were not witnessed (19%).

Investigations included: (a) 24 hour tape electrocardiographic recording in 72%, repeated in 30%; (b) exercise testing in 35% to recreate attacks; (c) intracardiac pacing study in 9% specially adapted for the neonate; and (d) cardiomegaly recordings in 12% capturing symptomatic paroxysmal arrhythmias when used by child, parent, or guardian.

Forty eight per cent required new or modified treatment. Digoxin remained the preferred treatment for nine out of 20 children with paroxysmal supraventricular tachycardia and atrial arrhythmias. Amiodarone was the only effective treatment for three with supraventricular tachycardia and two with ven-
tricular tachycardia. Beta blockers were used in only five children. Plasma levels of all drugs were carefully monitored. The transvenous technique has been adapted for permanent pacing in all age groups.

Intra-atrial baffle function in immediate postoperative period after a Mustard procedure

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Serial two dimensional echocardiograms were recorded to examine the intra-atrial baffle in 17 patients during the first week after a Mustard procedure. Two dimensional echo consistently visualised both the Dacron baffle and the morphology of both pulmonary and systemic venous atria. Partial baffle dehiscence was accurately located in two patients. Inferior baffle limb obstruction was not encountered but superior limb obstruction was directly visualised in three patients. In addition, all 17 patients demonstrated persistent discrete narrowing in the systemic venous atrium where the baffle crossed the primum septal remnant. In 10 patients this mid-atrial narrowing appeared severe. In 11 patients, mid-diastolic prolapse of an initially mobile baffle into the mitral valve orifice was consistently associated with signs of systemic venous obstruction which subsequently resolved as the baffle became a fixed immobile structure. Acute obstruction to pulmonary venous return (nine patients) was consistently associated with a hypercontractile posterior portion of the pulmonary venous atrium. Two dimensional echo did not reliably image the cause of this obstruction. Serial contrast echo studies showed persistent but diminishing bidirectional atrial shunting in all patients. This was not associated with baffle dehiscence and was presumably related to the properties of Dacron.

We conclude that serial two dimensional echo studies have allowed an accurate assessment of the complex changing properties of the intra-atrial baffle in the immediate post-Mustard period.

Deficient "natural" cytotoxicity in patients with congestive cardiomyopathy

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The aetiology of congestive cardiomyopathy is unknown. There is some evidence for the involvement of viruses and abnormalities of immunological variables have been demonstrated. These include the presence of serum autoantibodies and defective T-suppressor cell function. Of particular interest is a report that spontaneous cytotoxicity is deficient in these patients. Spontaneous cytotoxicity is mediated by natural killer cells, a lymphocyte subpopulation which may play a role in innate resistance to viral infections and in immunosurveillance. The natural killer cell activity of a population of peripheral blood lymphocytes varies greatly between individuals and also within the same individual as a consequence of many factors. The final common pathway of activation appears to be alpha-interferon, which increases cytolytic potential and recruits available precursor cells. In order to standardise measurements of natural killer activity, we have tested both peripheral blood lymphocyte and the interferon inducible natural killer activity of lymphocytes from normal individuals (14) and with congestive cardiomyopathy patients (10) against a conventional natural killer target, the K562 cell line, in four hour 51Cr release assays. Contrary to a previous study, we found no significant difference in peripheral blood natural killer activity between patients and controls. Lymphocytes from 6 to 10 patients with congestive cardiomyopathy showed a significantly reduced ability to respond to the induction of natural killer mediated cytotoxicity by interferon.