PROCEEDINGS OF THE BRITISH CARDIAC SOCIETY

THE AUTUMN MEETING of the British Cardiac Society was held at the Royal College of Physicians, London, on Friday October 30, 1964. The President, Shirley Smith, took the Chair at 9.15 a.m. during Private Business before handing over to the Chairman, John McMichael.

PRIVATE BUSINESS

1. The Minutes of the Annual General Meeting having been published in the Journal (1964, 26, 697) were taken as read and confirmed.

2. The President reported with regret the deaths of Blalock, Fraser, and Weitzman.

3. The Treasurer reported that the Society's finances had not altered since the Annual General Meeting, and reminded Members that an increase in subscription had become inevitable owing to the increased costs of the Society resulting from two-day meetings, increased secretarial expenses, and other factors. Last year, for the first time, expenditure exceeded income, and the former was expected to rise substantially in the next year, to £1,000 or more. Since the present income of the Society was in the region of only £650 per annum, the Council felt that the Society needed approximately an extra £600 per annum to maintain investments and the Congress Fund. Furthermore, the number of issues of the Journal in the year would very likely increase in the near future. It was proposed therefore to increase the subscription to the Society to 8 guineas per annum for Ordinary Members and 5 guineas per annum for Associate Members. This increase would include any future extra issues of the Journal and no further increase in subscription was intended in the immediate future.

4. The Secretary reported that the 1965 Annual General Meeting would be held at Liverpool on April 8 and 9 at the invitation of Chamberlain.

5. Future foreign meetings:
   The Secretary reminded Members that the First World Congress of Ballistocardiography and Cardiovascular Dynamics was to be held at Amsterdam on April 12 to 14, 1965, and the V World Congress of Cardiology was to be held in New Delhi in 1966.

6. The Secretary reported that he had heard from the World Health Organization about Swedish Research Fellowships in Cardiology, and further details could be obtained from him on application.

7. The Chairman announced that the Council had decided to add the Members' dates of entry to the Society in the booklet. He also requested that Members should advise the Secretary immediately of any change of address.

8. One hundred and fifty-five Members attended the Meeting.

   The Society dined at the Royal College of Physicians with Shirley Smith in the Chair.

THE EFFECT OF A LOW FAT DIET ON THE PROGNOSIS OF MYOCARDIAL INFARCTION


(1964, 26, 697 introduced)

264 men under 65 who had recently recovered from a first myocardial infarction were placed at random on to either a low-fat diet (40 g. daily) or on to a normal diet. No attempt was made to alter the nature of the fat consumed. The diet, which was carefully supervised by a dietitian, was also controlled by regular
weighings of the food and by serial serum cholesterol estimations. The mean fat intake calculated from the dietary weighings was 45 g. daily (low-fat) and 110–130 g. daily (control). After 6 months the serum cholesterol had fallen from 260 to 223 mg./100 ml. (low fat) and from 266 to 251 mg./100 ml. (control), a significant difference between the two groups.

The patients were followed up from 2½ to 6 years and each clinical relapse was assessed by a physician who did not know the patient’s régime, using strict criteria. After 4 years the relapse rate was 41 per cent in the low-fat and 39 per cent in the control group.

It is concluded that the low-fat diet had no effect on the relapse rate of patients following a myocardial infarction in this series.

ANGIOCARDIOGRAPHY IN THE DIAGNOSIS OF PERSISTENT PATENCY OF THE DUCTUS ARTERIOSUS IN INFANCY AND CHILDHOOD

M. S. Gotsman (introduced), Roy Astley, and Clifford Parsons

Between January 1959 and July 1963 ligation and transsection of the ductus arteriosus was undertaken in 100 patients. These patients have been reviewed to assess the relative value and importance of angiocardiography in establishing the diagnosis. In 54 the clinical picture was typical; in the remainder atypical features were present.

Venous angiocardiography alone was a simple and safe method of demonstrating the presence of a patent ductus arteriosus in very small infants in heart failure, without subjecting them to the hazards of cardiac catheterization (15 patients). It was also useful in older children in whom a ductus was suspected but in whom one or more signs were atypical (13 patients). It provided no information about intracardiac pressures or absolute pulmonary blood flow, but always demonstrated a shunt between aorta and pulmonary artery. Right heart catheterization was reserved for children with atypical physical signs, severe pulmonary hypertension, or additional abnormalities (19 patients). Multiple selective angiocardiography with "withdrawal" angiocardiography demonstrated the length, diameter, and shape of the ductus. Retrograde aortography was undertaken in only 2 patients to demonstrate additional lesions, e.g. coarctation of the aorta or aortic stenosis. Countercurrent aortography was performed once.

THE EFFECT OF ATRIAL SYSTOLE ON RIGHT VENTRICULAR STROKE OUTPUT*

D. G. Greene, W. J. Gillespie (both introduced), and G. de J. Lee

We have examined the role of atrial systole in augmenting ventricular function by measuring the changes in right ventricular stroke volume that take place when the time interval between atrial systole and ventricular contraction (P–R interval) is varied.

Right ventricular stroke volume was measured by the N₂O-body plethysmograph method in three groups of patients, studied at rest lying supine.

Group 1 consisted of subjects who complained of no symptoms but who had congenital complete heart block and whose heart rates ranged between 38–50 per minute. Right ventricular stroke volume measurements were made from complexes whose P–R intervals varied from 0·01–0·90 sec. Maximum augmentation of stroke volume occurred when the P–R interval was between 0·2 and 0·3 sec. The increase achieved was approximately 30 per cent. Group 2 were patients with acquired complete heart block. All were restricted by varying degrees of breathlessness on exertion. Right ventricular stroke volume was somewhat lower in this group than in the congenital group. It was also less influenced by the timing of atrial systole. Group 3 consisted of patients with complete heart block and Stokes-Adams attacks whose ventricular rates were controlled by an electrical pacemaker with internal electrodes. Stroke volume was again less influenced by the timing of atrial systole. The profile of lung blood flow was much less pulsatile than in either Groups 1 or 2, which suggests that right ventricular muscle contraction may be taking place abnormally in the artificially paced group.

* This work has been supported in part by the British Heart Foundation and in part by the United States Air Force under grant no AF.61(052)—746, European office, Office of Aerospace Research.
The Effect of Beta-adrenergic Blockade on Exercise Tolerance in Patients with Angina Pectoris

John Hamer, Theo Grandjean, Lee Melendez, and Edgar Sowton (the last three introduced)

The action of a new beta-adrenergic blockade drug, propranolol, in patients with angina pectoris has been assessed by means of an exercise tolerance test. There were 26 patients judged clinically to have stable angina of slight or moderate severity who were exercised on a bicycle ergometer at increasing work loads before and after an intravenous injection of the drug.

Exercise was limited by angina pectoris both before and after the drug in 11 patients. The exercise tolerance of these patients increased, on the average, by 45 per cent. A further 7 patients limited by angina in the control test had no angina at a similar work level after the drug; 8 patients were limited by fatigue both before and after propranolol and showed no change in exercise tolerance; and 7 seriously limited patients were given an injection of normal saline and showed only a 6 per cent improvement in effort tolerance.

Cardiac output was measured during the exercise test in 7 patients, using the Fick method. Pulmonary artery blood was obtained by means of fine polythene tubing (PE 50) passed through an arm vein. There was, on the average, a fall in cardiac output during exercise of 1·5 l./min. after propranolol.

Our findings suggest that treatment with propranolol may be helpful in some patients with angina pectoris.

Ischaemic Heart Disease in Young Women

M. F. Oliver

During the past 12 years, 125 women with ischaemic heart disease have been seen. Their ages ranged from 23 to 44 years. There were unequivocal electrocardiographic changes in 100, and in 25 the diagnosis of angina was made from the patients' clinical history. Of the 100 confirmed cases, 50 presented with effort angina and 50 presented with acute myocardial infarction or acute coronary insufficiency; the mean age of each group was 39 years.

There was commonly present some associated condition that might be expected to contribute to the premature development of ischaemic heart disease, and only 12 of the 100 patients with confirmed ischaemic heart disease were otherwise healthy. In 45, there was diastolic hypertension (diastolic blood pressure greater than 100 mm. Hg) and in 17 of these raised blood pressure of pre-eclampsia had occurred in a pregnancy. In 40, there was hypercholesterolaemia (serum cholesterol greater than 270 mg./100 ml.). In 25, there was a strong family history of ischaemic heart disease. In 20, menstruation had ceased before the development of ischaemic heart disease either spontaneously or as a result of bilateral ovariectomy. In a significantly large number, coronary artery calcification was detected by fluoroscopy using an image intensifier. Obesity was not common and diabetes was present in only 2 patients.

Thus, the majority of women had more than one factor that appeared to contribute to the premature development of ischaemic heart disease. In several many contributory conditions were present emphasizing the multifactorial aetiology of this disease.

While the long-term prognosis was worse in hypertensive patients, the five-year survival rate was 83 per cent, which is comparable to the rate in men of this age-group.

The Organization of an Intensive Care Unit for Patients Following Acute Myocardial Infarction

John Shillingford

Over 30 per cent of all patients admitted to hospital with acute myocardial infarction die. The factors responsible for death are not always clearly understood but include disturbances of rhythm, myocardial failure, peripheral vascular failure, biochemical changes, embolism, respiratory disturbances, and drugs. In order to investigate these factors more closely in an attempt to reduce the mortality in this condition a special intensive care unit has been established in a side room attached to the main medical ward. Equipment in this room consists of multichannel recording apparatus for the continuous monitoring of electrocardiograms, venous and arterial pressures, cardiac outputs, etc. Closed circuit television connects the room
to a central recording room where data can be continuously recorded on magnetic tape. Facilities for resuscitation including direct current defibrillation are kept immediately available. Examples of hemodynamic data and preliminary results were described.

HEMODYNAMIC CHANGES FOLLOWING ACUTE MYOCARDIAL INFARCTION WITH SPECIAL REFERENCE TO THE EFFECTS OF OXYGEN AND MORPHIA

M. Thomas, R. Malmcrona, and J. P. Shillingford

Hemodynamic measurements have been made in patients with acute myocardial infarction admitted to a special care and study unit at Hammersmith Hospital. Cardiac output was measured by a dye dilution technique using the photoelectric earpiece and Coomassie Blue dye. Arterial blood pressure was measured directly via a fine polythene catheter introduced percutaneously into the brachial artery.

Measurements were made while patients were breathing air, oxygen delivered by face mask, and again while breathing air. When breathing oxygen there was usually a rise in arterial blood pressure. Cardiac output fell due to a fall both in heart rate and stroke volume. Calculated peripheral resistance rose.

Measurements were also made before and after the administration of morphine sulphate intravenously in doses ranging from 3 to 10 mg. The hemodynamic response was variable and unpredictable. In some patients no measurable change in the circulation occurred after an adequate analgesic dose. In one patient a profound fall of blood pressure followed the slow injection of 3 mg. of morphine sulphate. The heart rate fell when the blood pressure was low. Some patients had a small fall in blood pressure with or without a rise in cardiac output.

The possible mechanisms of these changes and their importance in the routine treatment of patients with acute myocardial infarction were discussed.

VENTRICULAR FIBRILLATION IN ACUTE MYOCARDIAL INFARCTION

D. G. Julian, P. A. Valentine, and G. G. Miller (all introduced by R. M. Marquis)

Electrocardiographic monitoring has been used routinely in all patients admitted to Sydney Hospital with the diagnosis of acute myocardial infarction. In the first one hundred consecutive, unselected patients, there were 10 who developed ventricular fibrillation.

In a total experience of 25 patients with ventricular fibrillation in acute myocardial infarction, some of whom were not monitored, certain characteristics were found that indicated the types of patient most at risk. Ventricular fibrillation affects predominantly the younger male patients, and is most prone to occur on the first day of the illness. A number of cases developed during the subsequent ten days, but these were frequently preceded by warning arrhythmias such as ventricular ectopic beats, ventricular tachycardia, or unresolved complete heart block.

Of the 25 patients, 7 are long-term survivors as a result of cardiac resuscitation.

THE HEMODYNAMIC EFFECTS OF CHANGES IN BLOOD VOLUME IN HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY

P. M. Shah, R. Amarasingham, and C. M. Oakley

[Published in full in Brit. Heart J., 1965, 27, 83]

THE CONTRIBUTION OF ANGIOCARDIOGRAPHY TO SURGICAL PROGNOSIS IN THE TETRALOGY OF FALLOT

D. R. Smith, H. Effat, M. A. Hamed, and M. Omeri (all introduced by J. F. Goodwin)

The right ventricular angiograms of 50 patients with the tetralogy of Fallot were analysed with regard to the site and size of the ventricular septal defect, the calibre of the aorta and main pulmonary artery,
the type of outflow obstruction, the degree of pulmonary stenosis and the size of the left atrium. A method for assessing the amount of aortic override is described.

The findings were compared with data obtained at the subsequent operation for complete correction. A good correlation between surgical and radiological assessments was found in all aspects studied where comparison was possible.

The tubular type of infundibular stenosis, a small pulmonary valve ring, a small ratio of pulmonary artery to aortic calibre, and a small left atrium seem to be associated with an increased operative mortality.

The significance and diagnosis of the bulbo-ventricular type of defect, and the syndrome of “cusp elongation” in the tetralogy of Fallot are discussed.

**Audible Murmurs Arising at the Site of Interatrial Defects Proven by Intracardiac Phonocardiography**

Jane Somerville and Leon Resnekov

An immediate diastolic murmur following the usual systolic murmur at the left sternal edge was heard in 16 per cent of 122 patients with ostium primum atrial septal defect. This murmur has always been attributed to pulmonary or aortic regurgitation and has caused diagnostic errors. The genesis of this murmur has been investigated by intracardiac phonocardiography in 20 patients with atrio-ventricular defects, 10 of whom had an immediate diastolic murmur. In all 10 patients the immediate diastolic murmur was shown by the intracardiac phonocardiogram to be the diastolic element of a continuous murmur generated at the site of the atrial septal defect. Aortic and pulmonary regurgitation were excluded as a cause of the immediate diastolic murmur. Following closure of the defect the immediate diastolic murmur previously heard at the left sternal edge was no longer audible.

In other forms of atrial septal defect, murmurs may arise at the site of the defect under certain circumstances and be audible externally. The higher incidence in primum atrial septal defects is probably explained by the presence of mitral regurgitation which increases left atrial pressure.

The clarification of this physical sign by intracardiac phonocardiography is discussed and the importance of recognizing its significance is shown.

**Further Evidence for Arterial Contracture in Chronic Hypertension**

David Short

The nature of the increased vascular resistance in chronic hypertension has been the subject of much investigation. One of the mechanisms that has been considered is an inability of the arterioles to dilate completely.

Evidence in support of this theory has been obtained by injecting the arteries of the small intestine with a radio-opaque suspension containing gelatin at a pressure of 150 to 250 mm. Hg immediately after death, and comparing the size of arterioles in the hypertensive and non-hypertensive patients. Examination of the arteriograms suggested that the arterioles in the hypertensive cases were diffusely narrowed (Short, D. S. and Thomson, A. D. (1959)). The arteries of the small intestine in systemic hypertension. *J. Path. Bact.*, **78**, 321). Direct measurements of diameter and wall thickness have now been made on over 200 arterioles from hypertensive and control patients, and these measurements confirm the conclusions drawn from the arteriograms. The hypertensive cases were injected at a considerably higher pressure than the control cases, so that the presence of arteriolar narrowing in the former must indicate a decreased distensibility.

If this decreased distensibility proves to be widespread (and not confined to the small intestine) it could be an important factor in the maintenance of an increased vascular resistance in chronic hypertension.

**The Mechanism of the Hypotensive Action of Guanethidine During Upright Exercise**

S. H. Taylor, G. J. MacKenzie and H. P. Staunton (both introduced), and K. W. Donald

The haemodynamic mechanisms responsible for the development of hypotension during upright exercise,
which is observed in some hypertensive patients taking guanethidine, have been explored. Serial observations on the systemic arterial pressure, cardiac output, and oxygen uptake have been made in four groups of hypertensive patients.

A. Untreated patients: (i) without symptoms of cardiovascular disability; (ii) with moderately severe symptoms of cardiac insufficiency.

B. Patients under treatment with oral guanethidine: (i) without symptoms of cardiovascular disability; (ii) with moderately severe symptoms of cardiac insufficiency.

The chief conclusions that may be drawn from the results are:

1. During upright exercise the systemic blood pressure is maintained or raised even in patients with moderately severe cardiac insufficiency. In the absence of a normal cardiac output response, the blood pressure is maintained in these patients predominantly by vasoconstriction of the non-exercising regional territories; in patients without such disability vasoconstriction is less evident and the blood pressure is predominantly maintained by the increase in cardiac output.

2. The predominant pharmacological activity of guanethidine is sympathetic blockade with little effect on cardiac output. Patients with cardiac disability, relying largely on sympathetically mediated vasoconstriction for maintenance of the blood pressure during exercise, are severely affected by the drug and develop hypotension in direct relation to the level of exercise undertaken.

3. The arterial pressure during upright exercise in fit hypertensive patients is little affected by the drug except during abrupt increases in exercise level when transient but symptomless hypotension may occur.

4. It is concluded that when guanethidine is administered to hypertensive patients they should be instructed in its use with due regard to their previous exercise tolerance.