ABSTRACTS OF CARDIOLOGY


Leads I, II, III, CF2, CF4, and CF5 were used for electrocardiography, and 48 patients were divided into four groups: (1) 12 with no evidence of cardiovascular disease; (2) 16 with various types of cardiovascular disease other than infarction; (3) 14 with acute infarction; (4) those whose cardiograms showed a digitalis effect. In groups 1 and 2 all cardiograms taken at successively lower prothrombin levels were substantially the same. An occasional tracing showed minimal changes in amplitude of the T wave in lead I, and more rarely in the amplitude of the P wave, commensurate with the associated changes in rate. In group 3 the expected progressive changes of healing infarction were observed, and the rate of progress did not differ materially from that obtained in patients not receiving dicumarol. Withdrawal of the drug during convalescence did not cause significant cardiographic change. All tracings in group 4 retained patterns which were unchanged by effective doses of dicumarol. It is concluded that in none of the four groups of subjects was significant electrocardiogram deviation attributable to dicumarol. S. Oram


Diabetics develop arteriosclerosis early, amongst other sites in the coronary arteries. Anichkov ascribes this to a disturbance of the cholesterol metabolism.

The authors have aimed at estimating the functional capacity of the coronary arteries of patients up to 50 years of age with glycosuria. They chose the electrocardiographic method, taking records during rest and immediately after physical exertion. This last consisted of walking up a three-step ladder (presumably revolving) to a distance equivalent to a climb to the third floor of the Institute. Twenty-eight patients were examined, 5 with diabetes of mild, 14 of medium, and 9 of marked severity; there were 22 controls. In 26 there were no clinical signs of cardiovascular changes; 2 had hypertension. The electrocardiograms before exercise were normal in 14 cases; there was left axis deviation in 9 (including the 2 with hypertension) and right axis deviation in 2. The remaining 3 showed slight depression of the ST portion in leads I and II. The control group were all normal, clinically and cardiographically.

After the patients' exercise, 9 electrocardiograms showed changes corresponding to coronary insufficiency (depression of ST in leads I and II), but there were no changes in controls. Other alterations in the cardiogram, such as changes in height of the T wave, with depression of the PQ segment, were observed with equal frequency in diabetic and control subjects. The authors conclude that in diabetes mellitus there is an especial tendency to early coronary changes.

L. Firman-Edwards


The effect on the heart rate and blood pressure of tilting normal subjects was studied. Previous investigations have yielded widely divergent findings, which, the authors state, are due to the fact that it is impossible to obtain accurate readings of a rapidly changing blood pressure with an ordinary sphygmomanometer, and because the position of the arm during the tilting has varied in different experiments.

They recorded the intra-arterial blood pressure by means of a needle within the brachial artery at the elbow or radial artery at the wrist. The arm was maintained in a horizontal plane. Tilting was controlled by an electrically operated table, the subjects being supported so that muscular activity was not required to maintain the position. They were tilted head down to 45 degrees and erect to 20 degrees "at a moderate rate." An electrocardiogram was used to record the heart rate.

Blood pressure rose immediately in all subjects during the tilt from 20 degrees erect to 45 degrees head down, and fell immediately during the return tilt. In the 15 healthy young adults investigated these primary alterations of blood pressure were followed by a characteristic pattern of depressor response in the head down position, and of pressor response in the erect position, whereby the blood pressure returned in from 8 to 18 seconds to a level that was approximately the same regardless of the position of the body. The heart rate always slowed in the head down position and increased in the erect position. In some subjects, whilst in the head down position, the P wave gradually decreased in size and finally disappeared for several beats. The authors consider that, as these changes were not associated with shortening of the P–R interval, the resultant rhythm was not nodal.

S. Oram


The author describes the pharmacological actions of digoxin, g-strophanthsin (ouabain), theophylline, and mersalyl on the failing human heart. Forssmann's technique of right-hand catheterization was used in observing the action of digoxin and g-strophanthin.

Generalized venous congestion is seen in conditions that demand an increase in cardiac output as well as
in conditions which impair the cardiac mechanism and thus reduce its output. The former is regarded as compensatory to maintain the increased flow. On this basis the author classifies the congestive type of heart failure into two groups: (a) failure with high output due to conditions demanding an increase in the output such as anemia, emphysema, Paget's disease, and metabolic disturbances; and (b) failure with low output due to ischemia, hypertensive and valvular heart disease. In both groups the venous pressure is high, and the author believes that digoxin produces its beneficial effects by lowering this pressure. He states that, whereas in the first group this reduction in pressure causes diminution in cardiac output, in the second group it increases it.

Unlike g-strophanthin, digoxin is said to have insignificant action on the failing myocardium, and its hitherto well-known rate-reducing action is regarded only as an excellent guide to proper maintenance dosage when the drug is given over a long period of time. Experimental evidence is offered in support of these conclusions.

Theophylline is considered to have a direct stimulating action on the healthy heart as well as on the failing heart, increasing the cardiac output and thus reducing the venous pressure. This increased activity of the heart also produces secondary coronary dilatation, and the author adds timely warning as to the indiscriminate use of this drug in coronary thrombosis. Finally, observations made by his colleagues on the circulatory action of mersalyl are quoted. It is thought that mersalyl, by producing diuresis, reduces the venous pressure and thus raises the cardiac output. This action is similar to that of a venesection.

S. Karani


Amongst about 1000 patients in whom 2 or 3 electrocardiograms were taken after exercise (Master's two-step test) paradoxical changes were found in 35. In 16 of these the electrocardiogram abnormal at rest became normal in all leads after exercise, in 14 the abnormalities became less pronounced but did not entirely disappear in any or certain leads, and in 5 an improvement was found in some and deterioration in other leads; 28 patients had angina pectoris (in 10 of these pain occurred during the exercise test) and, of the remaining 7, 5 had valvular lesions and 2 arteriosclerosis. The improvement in the electrocardiogram may be only apparent; the changes may be due to the effect of extracardiac nerves or they may be due to an improvement, due to exercise, of the circulation through small vessels of the heart. The first 2 of these possibilities are rejected; the last one is discussed in more detail, also in relation to the mechanism precipitating anginal pain. [This explanation is highly hypothetical and not entirely convincing.]

A. Schott


Heart failure was produced in dogs by injection into the left ventricle of a 3:3% suspension of potato starch in saline, the aorta being occluded immediately above the heart for a few seconds during the injection. This produced diffuse myocardial damage by coronary embolism, most of the injected small (diameter 12 to 72 µ) particles presumably getting into the coronary arteries. The injections were repeated at 2 to 7 minute intervals. After more than three injections myocardial
failure was produced in most dogs, as evidenced first by a progressive rise in right auricular pressure, then by dilatation of the heart, congestion of the lungs and liver, and pulmonary oedema. The arterial pressure did not fall significantly until congestive heart failure had fully developed. The experiments usually terminated in death. Congestive failure could generally be produced with less starch suspension when the blood volume was first increased by 100 to 250 ml. of blood. In a few animals congestive heart failure did not develop but a precipitous fall in arterial blood pressure occurred, leading to death.

It is noted that whereas in the experiments of Landis et al. coronary ligation alone (without "exercise") produced a fall in venous pressure, coronary embolism in the present experiments produced a progressive rise in pressure. There is a discussion on the changes in cardiac output in relation to right auricular pressure.

H. Pollak


This paper contains a description of a modification of the technique for measurement of the cardiac output with the Fick formula and the carbon-dioxide method of Douglas and Haldane. The apparatus was devised to render the results independent of the co-operation of the subject tested. It enables the operator to conduct the measurement at a distance. Even so, this apparatus does not eliminate all sources of possible errors inherent in the carbon-dioxide method itself. In 12 normal subjects under basal conditions the cardiac output varied from 3.4 to 7 litres per minute, and the cardiac index from 2 to 3.7.

A. I. Suchett-Kaye


This investigation was undertaken to obtain some idea of the ultimate effects of anastomosing a systemic to a pulmonary artery as in the operative treatment of pulmonary stenosis. In necropsies on 67 cases with congenital systemic→pulmonary shunts of various kinds atherosclerosis was greater than in the controls in only 11 cases, and in 9 of these the increase was probably due to rheumatic mitral disease.

D. M. Pryce


From kymographic and hemodynamic findings the authors estimate that in the heart of normal subjects the residual blood volume is about double the stroke volume; in highly trained subjects the amount of residual blood is further increased. The increase in the stroke volume after exercise is associated with a decrease in cardiac volume (estimated radiologically) and is at least partly due to a decrease in the residual blood volume. The inhibitory vagal and stimulating sympathetic effects upon cardiac work are relatively independent of the cardiac volume. It is pointed out that, as a result of sympathetic nervous action, additional energy can be utilized by a mechanism which is independent of length or tension of fibres. The vagal effect, on the other hand, results in increased economy of the cardiac mechanism.

A. Schott


In the study of a case of ventricular septal defect with a large left to right shunt and normal arterial oxygen saturation but a marked pulmonary hypertension, cardiac catheterization is employed to illustrate a possible relation between isolated ventricular septal defect occurring in early life and a subsequent fully developed Eisenmenger complex.

J. L. Lovibond


Twelve out of 17 patients suffering from coarctation of the aorta were investigated before and after operation to determine the renal filtration rate, the renal plasma flow, and the filtration fraction. In most of the cases the successful resection of the constriction and the end-to-end anastomosis produced an increase in the renal blood flow and a decrease of the filtration fraction. The authors believe that the diminished renal blood flow with nearly normal filtration rate in patients with coarctation of the aorta is due to a compensatory efferent arteriolar constriction—a physiological mechanism similar to that present in essential hypertension. The successful operation of the coarctation releases the constriction of the efferent artery.

A. I. Suchett-Kaye


Most of the reported cases of Friedler's myocarditis have been in adults. The disease occurs in children also, and 4 such cases, in patients whose ages ranged from 3 weeks to 13 months, are described. The myocardial lesions were acute in 3 cases and more chronic in the fourth. Histologically the change was inflammatory and had no specific features. Concurrent necrosis and disappearance of muscle fibres were seen. Endocardium and pericardium were unaffected, or only secondarily involved over the area of the muscle lesion. Terminal pneumonia was present in all 4 cases.

L. Crome

A series of 100 patients who survived the acute stage of myocardial infarction was followed up. The patients could be divided into three groups: (a) Those who made a good functional recovery, without angina or cardiac failure. There were 3 women and 54 men in this group, and the ages ranged from 30 to 72 years, with an average of 50 years. In 32 patients hypertension, and in 25 angina, preceded the attacks. Five of the patients died, a new infarct being the cause in 4. (b) Those who suffered from angina following the infarct. In this group were 22 men and 1 woman; the ages ranged from 34 to 72 years, with an average of 49-5 years. Hypertension preceded the infarction in 15 cases, angina in 18 (in 10 associated with hypertension), and cardiac failure in 1 (associated with hypertension). Four patients died while under observation, a further infarct being the cause of death of 2 of them. (c) Patients with subsequent cardiac failure. In this group there were 2 women and 18 men, whose ages ranged from 48 to 65 years, with an average of 56-3 years. Hypertension preceded the attack in 16 patients, angina in 10 (of which 9 had associated hypertension), and cardiac failure in 7 (all associated with hypertension). Seven patients subsequently died of cardiac failure, but none had a further infarction during the period of observation.

Many patients survived the original infarction for more than 8 years. Causes of death were cardiac failure or coronary insufficiency (usually a further infarction). Cardiac failure was more common when hypertension preceded infarction. Recurrence of infarction was unpredictable, and occurred in patients of the first group many years after the first attack.

F. A. Langley


The authors have examined the records of 800 cases of coronary thrombosis treated in 16 hospitals by members of a committee, set up by the American Heart Association. Of this total, 368 patients were treated conventionally, the remaining 432 being treated, in addition, with anticoagulants along the following lines. Heparin was given for the first 48 hours or more if desired. Dicoumarol, 200 to 300 mg. daily, was given until the prothrombin time was 30 seconds, then 40 to 100 mg. daily if the prothrombin time was between 30 and 35 seconds. When the prothrombin time was 35 seconds or longer, dicoumarol was withheld until it had fallen to 30 seconds or under, when the drug was again given cautiously in 100 mg. doses. Prothrombin time was estimated daily by the Link-Shapiro technique or by the Quick method. Treatment was continued for at least 30 days and preferably for 30 days after the last thromboembolic episode, unless contraindicated. The cases in the two groups were unselected and comparable. Of the control group 23%, and of the treated group 30% were classified as having severe attacks.

Mortality in the control group was 24%, and in the treated group 15%—a statistically significant difference. The lower death rate in the treated group was largely due to the lower incidence of thrombo-embolic complications, which occurred in 25% of controls and in only 11% of treated patients; in 5% of the latter group the episode occurred before treatment began or within the first 3 days of anticoagulant treatment. The death rate by age group, the incidence of embolism, and the complications due to treatment are analysed.

D. Verel


The authors studied serial electrocardiograms in 100 cases of myocardial infarction, and attempted to relate prognosis to the progressive electrocardiographic changes. They conclude that the position of the myocardial infarct is of little prognostic significance. Of greater importance is the extent, the larger the infarct the worse being the prognosis. Associated changes in the electrocardiogram, such as low-voltage curves, bundle-branch block and arrhythmias, did not materially worsen prognosis. In patients who made a good clinical recovery a larger proportion of electrocardiograms became normal. However, no close relation between clinical progress and serial electrocardiographic changes could be conclusively demonstrated.

E. G. Sita-Lumsdén


This deals with the occurrence of arterial pulmonary hypertension in a series of cases comprising 8 patients with ventricular septal defect, 5 with auricular septal defect, 2 with patent ductus arteriosus, 6 with coarctation of the aorta, and 3 with partial transposition of the great vessels. Direct measurement of pressures in the pulmonary artery by means of an intracardiac catheter gave mean values ranging from 25 to 102 mm. of Hg. There are three possible mechanisms that could operate to produce this rise in arterial pulmonary pressure: (1) Increase in pulmonary blood flow exceeding the functional capacity of the left auricle; (2) increase in left auricular pressure; and (3) increase in pulmonary peripheral arterial resistance; in 2 illustrative cases there was a large overall right-to-left intracardiac shunt. This increase in pulmonary peripheral arterial resistance has been found to be due in some cases to functional and in others to arteriosclerotic changes in the pulmonary vessels.

A. I. Sachett-Kaye

Four patients with angina pectoris and normal electrocardiograms at rest developed frequent premature ventricular systole immediately following an exercise test. After treatment with quinidine, 0.3 g. thrice daily, the effort tolerance increased and no premature systoles occurred during the exercise test. Two of the 4 patients died soon afterwards, and it is suggested that premature systoles occurring during the tachycardia induced by exercise have a grave prognostic significance. Since death is probably due to ventricular fibrillation in these patients, it seems reasonable to give them quinidine.

C. W. C. Bain

ABSTRACTS


In 14 cases of coarctation of the aorta angiocardiography was performed. Of these 11 were operated upon and in 6 post-operative angiocardiograms were taken. Robb and Steinberg’s technique was used; 50 ml. of 70% diodone was injected into the cubital vein in 2 seconds. All patients were tested beforehand for iodine-sensitivity, either by the skin test or by an intravenous injection of 2 ml. of the contrast medium. Two cases were rejected on these grounds. A serial cassette changer capable of holding 6 cassettes was used, and films could be taken at the rate of almost one per second. The arm-to-tongue circulation time was estimated first, and in most cases the initial film was taken at the number of seconds after injection corresponding to half the circulation time; subsequent exposures were made at intervals of 1 to 2 seconds.

The main features searched for in the radiographs were: (1) the location of the stenosis relative to the left subclavian artery; (2) the length of the stenosed portion; and (3) the degree of the stenosis. Post-operative angiograms were found useful in assessing the results. It was noted that the lumen of the stenosed part of the aorta was never restored to normal, but was usually very much enlarged.

J. W. D. Bull


The authors report a case in which there was notching of the left 5th, 6th, 7th, and 8th ribs in the absence of coarctation of the aorta, associated with Fallot’s tetralogy and cystic disease in the left upper lobe. Histological study of the affected arteries revealed no evidence of congenital weakness or developmental anomaly to account for the unusual distribution.

L. G. Blair


The authors claim that this syndrome should be recognized clinically. They describe 2 personal cases in detail and give the findings in 27 reported cases.

Its characteristics are contrasted with those of pulmonary stenosis without septal defects, Fallot’s tetralogy, and Eisenmenger’s complex. The syndrome of pulmonary stenosis with patent foramen ovale belongs to the cyanotic group of cardiac abnormalities. The cyanosis, due to a right-to-left shunt, is usually less severe than in the tetralogy of Fallot but more pronounced than in Eisenmenger’s syndrome; pulmonary stenosis with closed septa is essentially a non-cyanotic disease. Other features of the syndrome are polycythemia, clubbing, systolic murmur at the pulmonary area, second pulmonary sound decreased or absent; radiological investigation shows dilatation of the pulmonary artery but without hilar pulsation. Electrocardiography reveals right ventricular hypertrophy, prominent P waves, and occasionally right bundle-branch block due to hypertrophied right ventricle.

The main interest of this clinical identification of the syndrome of pulmonary stenosis with patent foramen ovale lies in the fact that in cases of this cardiac defect benefit may be expected from surgical intervention.

A. I. SUCHETT-KAYE


The different principles of pressure recording in catheterization of the heart are discussed. Resistance-wire manometers have certain advantages over the other methods and have shown their instrumental superiority both in static and dynamic tests.

J. L. LOVEBOND


Two sisters aged 21 and 25 years with Lutembacher’s syndrome are reported; no previous report of this syndrome in brothers or sisters has been found up to 1947. Fifty-four cases of Lutembacher’s syndrome have been reported.

The history and physical findings are described and radiographs, electrocardiograms, and phonocardiograms reproduced. Criteria for the diagnosis are mentioned. Fluoroscopy should show signs of: (1) generalized enlargement of the right heart; (2) marked prominence of the pulmonary conus; (3) increased hilar markings with “hilar dance”; (4) a small left ventricle; (5) a hypoplastic aorta. To these signs are added a characteristic apical diastolic murmur to complete the findings in the syndrome.

John Anderson