PROCEEDINGS OF THE BRITISH CARDIAC SOCIETY

The Twenty-Ninth* Annual General Meeting of the British Cardiac Society was held at the Westminster Hospital, on Thursday, May 25, 1950. Chairman: Sir Arnold Stott. The Chairman took the chair at 9.30 a.m.; 104 members and 34 visitors were present.

PRIVATE BUSINESS

1. The minutes of the last Annual Meeting having been published in the Journal (11, 407, 1949) were taken as read, and confirmed.
2. The balance sheet for 1949–50 was presented, having been audited and found correct by Boyd Campbell and Perry. The credit balance on April 28, 1950, was £156.
3. G. E. S. Ward and K. D. Wilkinson were elected members of the Council in place of Geoffrey Bourne and J. R. H. Towers (terms of office expired).
4. The following were elected Extra-ordinary Members: Hugh Barber, T. F. Cotton, B. T. Parsons-Smith, and A. F. Rook.
5. The following Associate Members were elected as Ordinary Members: C. G. Baker, V. G. M. Hamilton, S. Oram, C. Papp, C. A. Parsons.
6. The following Associate Members were elected:
   - W. H. R. Cook, Liverpool
   - H. A. Dewar, Newcastle-upon-Tyne
   - R. Byron Evans, Cardiff
   - J. F. Goodwin, London
   - F. Jackson, London
   - I. Macpherson, Leeds
   - J. P. Shillingford, London
7. The following Associate Members were re-elected for a further period of three years:
   - D. R. Allison
   - R. G. Anderson
   - R. Hartley
   - Ronald Jones
   - G. Konstam
   - R. W. Luxton
   - W. A. Oliver
   - R. Kemball Price
   - R. W. Turner
   - H. E. S. Pearson
   - J. R. B. Hern
8. The question of guests to the Annual Dinner was considered. It was proposed by Hill and seconded by Maurice Campbell that anyone introduced to read a paper may be invited to the Annual Dinner by the member introducing him, this rule applying only to the individual who actually delivers the paper.
9. Reference was made to Rule 10. "Associate Members shall be elected for three years, and shall be eligible for re-election as Associate Members or for election as Ordinary Members. They shall take part in the scientific business but not in the private business of the Society. They will be expected to resign if they cease to be qualified as Associate Members."

The Chairman said that the Council had reviewed this matter at its last meeting. It considered that re-election of Associate Members should in future be more discriminating and that it would depend on Associate Members showing proof of continued interest and activity in cardiology by, for example, contributions at Meetings or to the Journal.

DISCUSSION ON THE USE OF ANTICOAGULANTS

Opened by Rae Gilchrist, Sir James Learmonth, R. J. Macfarlane, and K. Shirley Smith.

Rae Gilchrist and John Tulloch (introduced). This communication is an extension of the report on anticoagulants in coronary thrombosis given to the last meeting of the Society by Tulloch, October, 1949 (Brit. Heart J., 12, 208, 1950).

* There were 15 Annual meetings of the old Cardiac Club and this is the 14th meeting of the reconstituted Society, including the 1944 meeting under the chairmanship of Sir Thomas Lewis, which was represented by a business meeting only, as the general meeting had to be cancelled owing to the flying-bombs.

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Mortality rates recorded amongst hospital patients during the first six weeks after a coronary occlusion vary greatly in different centres. Our Edinburgh experience is based on two series of patients treated by conventional methods exclusive of the use of anticoagulants. The first group, consisting of 100 consecutive patients admitted immediately preceding our trial of anti-coagulant therapy, had a mortality rate of 43 per cent. The second group, forming the present control series of 84 consecutive patients, observed concurrently during the use of heparin and dicoumarol, had a mortality rate of 41 per cent. This implies that four patients in every ten admitted to hospital on account of this disease may die within the first six weeks. This is the challenge which as cardiologists we must be prepared to face.

Anticoagulant therapy has been employed in 70 consecutive patients. The control group of 84 patients were treated by the usual conventional methods. The choice of treatment for each patient was governed by the day of the week on which he arrived in hospital. This determined his inclusion in one or other medical pavilion, in one of which anticoagulants were in use and in the other, excluded. No patient was admitted to hospital for the specific purpose of anticoagulant therapy. The two groups are therefore unselected.

In respect of sex and age distribution, the pre-existing state of the heart muscle as determined by the usual clinical methods, the severity of the attack of coronary occlusion as judged by the degree of shock, and the duration of the acute illness before the patient came under treatment by one or other method, the two groups on analysis are found to be sufficiently homogeneous to warrant comparison.

In the treated group, heparin was given by the interrupted, intravenous injection method, the interval between doses being eight hours. Each injection consisted of 10,000 units of heparin. Its efficacy was assessed by estimation of the clotting time one hour after injection. Dicoumarol was started on the first day of admission, the initial dose being 300 mg. in the first 24 hours, 200 mg. in the second and 100–200 mg. on the third day, all subsequent doses of dicoumarol being determined in relation to the daily estimation of the prothrombin time. Heparin was usually discontinued on the third day, by which time the prothrombin estimations indicated that the dicoumarol was proving effective. Treatment with dicoumarol was maintained for three weeks, the prothrombin time being kept in the neighbourhood of 28 seconds.

In the control group of 84 persons, 34 died within six weeks of admission, giving a mortality of 40 per cent. This contrasts with the 16 deaths amongst 70 persons in the treated group, with a mortality rate of 23 per cent. We do not claim that these figures as they stand are necessarily statistically significant for such small groups, but when the men in the control and treated groups are compared, it is found that the mortality rates respectively were 33 and 15 per cent—a difference of statistical significance. We conclude that in men efficient anticoagulant therapy during the first six weeks after coronary occlusion is capable of reducing the death rate by half. It remains to be established whether women obtain as much benefit.

Of the control series of patients, 29 per cent developed thrombo-embolic complications while under observation, as a result of which 13 of the 84 patients died. Ten of these deaths were directly attributable to the thrombo-embolic complications in one form or another. On the other hand, in the treated group, 9 of 70 patients developed thrombo-embolic complications, of whom 4 died, death being attributable to the embolic episode. The incidence of complications in the treated group was 13 per cent—less than half that observed amongst the controls. When the control and treated groups are compared, there is a statistically significant difference in the incidence of complications.

It is too early to conclude that anticoagulants used during the acute phase of the disease process influence the ultimate outcome. The immediate prognosis is undoubtedly improved, the mortality rate being reduced by half during the first six weeks of treatment. If anticoagulants limit the extent of the infarcted area in the heart muscle, then it may be that patients treated in this way may ultimately have a better expectation of life.

Anticoagulant therapy cannot succeed in the absence of strict attention to the established methods
of treatment. It does not shorten the stay in bed. It has its limitations and contra-indications, but it should be considered for every sufferer from coronary thrombosis. This implies an extension of laboratory facilities and a greater number of hospital beds set aside for this purpose.

Sir James Learmonth (introduced) gave an account of his experience with anticoagulants in the treatment of peripheral vascular diseases. In arterial emergencies, the object is to prevent the occurrences of consecutive thrombosis; in venous emergencies, to stop the extension of the clot. The procedure should be the same in both: intravenous heparin immediately, in a dose of 12,500 units, followed by doses at 8 a.m. (12,500), 12 noon (10,000), 4 p.m. (10,000) and 8 p.m. (12,500), and as soon as possible one of the newer coumarin preparations (tromexan, pelantel) by mouth, the dose being 1-2 g. on each of the first two days, followed by 0-3 to 0-6 g. according to the prothrombin readings. In 80 per cent of cases the prothrombin level falls to under 50 per cent of the normal within 36 hours. When it reaches 40 per cent of normal, heparin can be discontinued, and the dosage of coumarin adjusted to keep the prothrombin between 20 and 40 per cent of normal. If for any reason heparin is used alone, it is important not to stop the drug suddenly, and in cases of venous thrombosis to continue it until the patient is up and about. It is possible to continue the administration of tromexan or pelantel for months while the patient continues at work (e.g. in treating thromboangiitis obliterans) without more than an occasional (fortnightly) estimation of prothrombin, once a level has been established.

Of 22 cases of sudden arterial occlusion in which anticoagulants were given from the first, six required early exploration or embolectomy; two required amputation (late cases); two died of further multiple emboli; and 12 showed immediate and continuing improvement, although one, an elderly patient, died on the ninth day from cerebral haemorrhage. Heparin also relieved the pain of embolism, probably through its vasodilator effect. In sudden arterial occlusion, conservative treatment should be adopted in the first instance—anticoagulants, morphia if necessary, and reflex vasodilatation: except when an embolus has lodged at the aortic bifurcation, or when an embolus has previously lodged in the same limb. Anticoagulants will not alter the level of a line of demarcation already clinically evident. Heparin is not used post-operatively after suture operations on blood vessels. The treatment of acute venous occlusions requires in addition elevation of the limb until œdema has subsided, and after a further ten days or so light massage.

In 157 cases there have been 16 complications, none being serious: haematuria (7), haemoptysis (1), haematemesis (1), epistaxis (2), haematoma (2), and cutaneous petechial haemorrhage (3). Four patients died during treatment. In two necropsy showed only myocardial failure, in one massive cerebral haemorrhage (prothrombin level 20 per cent normal); in the fourth necropsy was refused, but the probable cause of death was cerebral haemorrhage.

R. G. Macfarlane (introduced). Anticoagulant therapy is based on the supposition that pathological thrombosis is the direct result of blood coagulation, and that anticoagulants inhibit blood coagulation in vivo. This conception is probably an over-simplification, and other factors are likely to be concerned. Blood coagulation is the result of a disturbance of the normal equilibrium between the coagulant factors and the anticoagulant factors which are present in fluid plasma. Contact with a foreign surface, or the entry of tissue fluid into the blood stream, results in stimulation of thrombin generation, and if the rate of this exceeds the rate at which thrombin is destroyed by antithrombin, clotting is likely to occur. The conditions of blood flow are therefore important in determining whether or not a local concentration of thrombin sufficient to produce clotting can be achieved. Stasis of the blood in a situation where thrombin generation is taking place favours massive coagulation, whereas a more rapid flow will cause the thrombin to be diluted and inactivated by normal blood. In assessing the results of anticoagulant therapy it is essential to take into consideration the effect of modern post-operative treatment which recognizes the importance of maintaining a good circulation.

Many efforts have been made to detect any pathological change in the blood coagulation mechanism which precedes the onset of a thrombotic episode. So far none of the many tests applied has been able to demonstrate any such change which could be considered to be either
aetiologically or diagnostically significant. Thrombosis therefore appears to be the result of comparatively slight deviations of the normal equilibrium in the direction of increased thrombi generation and decreased removal. By restoring this equilibrium the anticoagulant drugs may have considerable clinical effect even though their action on the clotting mechanism in vivo may comparatively small. Two other factors may be involved in this action. Thrombosis is probably favoured by vascular spasm, and evidence is accumulating to show that the anticoagulants have a vasodilatory effect. Secondly, agglutination of the platelets on damaged areas of endothelial is frequently a starting point of massive thrombosis and it is well known that the tendency to such platelet agglutination is reduced by the anticoagulants. A third, and more problematical factor is the extraordinary change in blood viscosity and cell behaviour which follows trauma or infection, and known as "sludging of the blood," which may have a considerable part in the initiation of thrombosis.

K. Shirley Smith said that the assessment of the value of anticoagulants in the treatment of cardiac infarction must depend upon a knowledge of the incidence of thrombo-embolic complications in infarction not treated with anticoagulants. Such information might be derived from clinical and post-mortem studies. The results of five large clinical series were compared. The incidence of thrombo-embolic complications in the large composite series (1605 cases not treated by anticoagulants) of Hellerstein and Martin (Amer. Heart J., 33, 443, 1947) was seen to be the same as that in the series of 432 patients reported by Wright, Marple, and Beck (Amer. Heart J., 36, 801, 1948) treated by anticoagulants, namely, 11 per cent. Reviewing these statistics it could hardly be claimed that the general incidence of thrombo-embolic complications, judged clinically, in cardiac infarction was greater than 15 per cent.

When post-mortem statistics were compared there was again a great disparity of results, and it was remarkable that there was a greater lack of uniformity in the statistics derived from post-mortem series than in the clinical series, whereas the reverse might reasonably have been expected.

A considerable source of error in clinical material undoubtedly arose through difficulty and uncertainty of diagnosis of thrombo-embolic complications. In the series of 150 consecutive cases of cardiac infarction reviewed in collaboration with C. Papp, 20 patients were thought to have embolisms of one kind or another but of these, three were disproven at necropsy. For example, what was clinically thought to be pulmonary infarction was, in fact, pulmonary oedema and congestion. In general, signs developing in the lungs a few days after cardiac infarction were due to consolidation of a congested area, as right ventricular infarction was so uncommon, while lung signs after the tenth day were probably embolic from crural venous thrombosis. Another diagnostic difficulty arose with ischemic limbs; if the symptoms developed within a day or two of cardiac infarction the condition was not caused by embolism but by the sudden fall of blood pressure in extremities previously threatened.

The speaker held that the advantageous results of anticoagulant therapy were exaggerated by inclusion of the lesser grade of cardiac infarction. These were not liable to thrombo-embolic complications. In a series of 109 consecutive cases of cardiac infarction closely observed in collaboration with Papp, 33 were classed as "slight": that is, they were unaccompanied by shock or failure, showed persisting cardiographic signs but no cardiographic or laboratory evidence of extensive myocardial necrosis. These patients showed a favourable clinical course, and in no case was there a thrombo-embolic complication. This was in accordance with the proven localized nature of the lesions in such cases, mural thrombosis being most unlikely. It was probably unjustifiable to submit these patients to anticoagulant treatment.

The indications for the use of anticoagulants and the aims of this treatment in cardiac infarction might be summarized as follows: (i) in infarction accompanied by shock, failure, sustained fall of pressure, and cardiographic evidence of an extensive lesion; (ii) with the aim of preventing cardiac infarction when pain of increasing severity or frequency seems to threaten this development; and (iii) with the object of prevention of venous thrombosis in the legs during the period of rest and impaired circulation, since this was a common cause of pulmonary embolism.
If pulmonary infarction had already developed it was doubtful whether the anticoagulants were of avail. An important corollary in treatment arose from these considerations; it was that therapy in cardiac infarction, whether by anticoagulants or not, should be supplemented by efforts to augment the respiration and the movement of the legs once the stage of shock had passed, in order to minimize the risk of thrombo-embolic complications.

**SALICYLATE AS AN ANTICOAGULANT**

By J. L. Lovibond. The anticoagulant properties of salicylate have been studied in relation to clinical signs of salicylism in an attempt to assess its potential value in the treatment of coronary thrombosis. Patients were selected for this investigation who gave a short history of effort angina and for whom a few weeks of bed rest and anticoagulants seemed justifiable therapy. Twenty cases were investigated, of whom 10 were the subject of more detailed study. Few were able to tolerate sodium salicylate or disprin in the massive daily dose of 200 grains for many days. This produced a satisfactory lengthening of prothrombin time in most cases, but reduction of dosage caused unpredictable variations. It was only possible to maintain a therapeutic prothrombin level with doses of salicylate that caused symptoms of salicylism. There was a considerable personal variation in response, but no dangerous complication occurred. The clinical degrees of salicylate intoxication were found to bear no constant relation to the fluctuations in prothrombin time, and only within their wider limits could they be used as a guide to the action of the drug on the clotting time. It was concluded that salicylate is an unpleasant but safe anticoagulant for domiciliary use.

**INTRAMUSCULAR HEPARIN**

By D. G. Abrahams (introduced). The possibility of using heparin therapeutically by intramuscular injection has been investigated. An aqueous solution of heparin 25,000 units per ml. was used and results showed that satisfactory anticoagulation could be obtained with twelve-hourly injections of 1 ml. There was some evidence that the dose could be decreased after two to three days. Local pain frequently attended the injection, but this was completely abolished by simultaneous administration of procaine. The advantages of the intramuscular route were discussed.

**A PERIPHERAL ACTION OF HEPARIN**

By D. G. Abrahams and Sheila Howarth (introduced). With a plethysmograph the fore-arm blood flow was measured in subjects before and after injection of heparin. Following therapeutic doses intravenously, there was a significant rise in the fore-arm blood flow. Further investigation seemed to show that this rise in peripheral flow was occasioned by a direct vasodilator action of heparin. The significance of this finding in connection with the clinical application of heparin was discussed.

**ANTICOAGULANT THERAPY IN MYOCARDIAL INFARCTION—FURTHER SAFETY MEASURES**

By H. E. S. Pearson and C. H. Greer (introduced). The results in the first 100 of a series of cases of myocardial infarction treated with heparin-dicoumarol or heparin-tromexan have been studied with the aim of reducing the therapeutic risk of anticoagulant treatment in this type of case.

Using Toohey's wet-brain method and expressing the results as the prothrombin time ratio, a therapeutic range of from 1·5 : 1 to 2·5 : 1 was found safe at the upper level and reasonably effective at the lower. Accidental high values (in excess of 3·5 : 1) occurred chiefly in the first 4 days, resulting from the initial "blind" dose, and the degree of sensitivity first shown altered rarely and slightly.

An attempt was made to avoid over-reaction by reducing individually the initial dose in persons of frail physique and poor general condition. Clinical analysis of those cases whose abnormal
sensitivity remained unpredicted by these general considerations showed a high incidence of congestive failure or arterial hypertension among them, and the recognition of these factors as additional indications for precaution would have practically eliminated over-reaction.

It is suggested that congestive failure may increase sensitivity by prolonging the contact time of the drug with the liver cells as well as by causing parenchymatous damage. In our post-mortem records 84 per cent of the cases showed evidence of chronic passive congestion of the liver and 43 per cent had various degrees of ischaemic renal scarring. The prevalence of such changes in this particular type of case imposes the need of special precautions in anticoagulant dosage, and the use of a graduated table for the initial doses is recommended.

Following the opening papers and the short communications on the use of anticoagulants, there was a discussion to which contributions were made by Goodwin, Courtenay Evans, Morgan Jones, Evan Bedford, Turner, Peel, Robertson, Wallace-Jones, Crozier, Hill, Gavey, and Papp.

**Short Communications**

**Electrokymography**

By B. G. Wells. The electrokymograph will record movements of the heart borders or changes in X-ray density of the heart and lung fields. Although introduced by Henny and Boone in 1945 it has only recently become available in this country. For this reason a description of experiences with the instrument and an analysis of its potentialities seems useful.

Tracings are made on a cardiographic apparatus and reveal much more accurately than the X-ray kymogram the minute details of heart movement. The volume changes of the cavities of the heart are, however, confused by two main factors: (1) the complexity of and individual variation in the movements of the heart as a whole; and (2) the change towards the spherical shape that the elongated ventricles assume in systole. Thus, for instance, there are in the normal heart certain portions of the ventricular walls that show an outward movement in isometric systole. Until such factors are carefully studied the significance of movements of the heart borders will remain obscure.

Tracings other than from the borders of cardiac chambers are in contrast comparatively simple to analyse. The borders of the pulmonary artery and aorta show tracings characteristically altered by stenosis or insufficiency of their respective valves. Synchronous tracings of the density changes in the hilus and lung periphery show the rapidity of propagation of the pulse wave in the lesser circulation. If the instrument were slightly modified it would give a tracing showing the quantitative density change over any area of the lung fields. Such information might well prove to be the most important single finding in the evaluation for surgical treatment of congenital malformations of the heart.

**The Radiology of Acute Pulmonary Ædema**

By F. Jackson. X-rays were taken during and after attacks of acute pulmonary Ædema in 17 patients with heart disease. There were 9 cases of hypertensive failure (6 with renal involvement), 3 of aortic and 5 of mitral valve disease; all but one were in normal rhythm.

Dense cloud-like shadows extended from the hila into the central parts of the lung fields obscuring the pulmonary vessels and the lung markings. These opacities were always bilateral, though sometimes greater on one side, especially when there was added lung infection. In severe cases the shadowing reached the chest wall but the apices were usually spared as were the costophrenic angles.

The shadowing often took one of at least two forms, either a dense “blotchiness,” or a “blurred reticular” appearance with a generalized loss of translucency of the lung fields. The changes frequently disappeared quickly, even within as short a time as 24 hours.

Pleural effusions were uncommon, the superior vena cava was seldom dilated, and the right
dome of the diaphragm was not greatly elevated. Cardiac enlargement was often less than expected, and it changed little during the attack.

THE ESOPHAGEAL LEAD

By C. W. C. Bain. A series of oesophageal leads have been taken with a Ryle's tube, the tip of which had been cut off and an electrode of German silver substituted. This was connected by wires running through the bore of the tube to the exploring electrode from the galvanometer. The Wilson "V" connection was used as the remote electrode. The tube can either be swallowed or passed through the nose. Most patients prefer to swallow it. The tube is marked at intervals of 2 cm., from 24 cm., at which level the tip will be above the auricle, to 56 cm. when it will be in the stomach. No attempt was made to verify the position of the electrode by screen observation before taking records as this adds to the time required, and inspection of any record will show if the tube has coiled in the throat. When the tube has been swallowed to the desired level, usually about 50 cm., a strip is exposed and records are made at 2-cm. intervals until the complexes are supra-auricular in type, which is usually above 30 cm.

The normal oesophageal record at the ventricular level resembles lead F. There is a positive P, a ventricular R and a positive T. As the tube is withdrawn the P waves become peaked, a ventricular Q appears, and T becomes negative. Above 40 cm. the auricular intrinsicoid deflection begins. Normally, this has first an auricular R. Later R becomes small and is followed by an S. As the supra-auricular levels are approached, the complex becomes W shaped, and then merges into the small broad negative P deflection of lead R.

The auricular T wave is seen in cases of heart block, since normally it is buried in the ventricular complex. At the lower auricular levels the T wave is a broad shallow negative deflection. When the auricular S predominates it changes to an upward dome-shaped deflection.

Oesophageal tracings at auricular levels of auricular fibrillation resemble lead V I, except when the deflections are larger. There is no sharp intrinsicoid deflection. The appearances are what might be expected if the auricle were activated seriatim by impulses originating in a circulating wave. In auricular paroxysmal tachycardia the auricular rate and rhythm can always be established beyond doubt. The auricular intrinsicoid deflections are of sufficient size to be obvious, even when they coincide with the ventricular complexes. In auricular flutter the same sharp intrinsicoid deflections occur as in auricular tachycardia and the records are quite unlike those of fibrillation. They do not favour the idea of a circus movement in flutter. The independent auricular rhythm in ventricular tachycardia is well seen.

The results at the ventricular levels have been disappointing. Some posterior infarcts have shown up well, but only when they were obvious in lead III and F. When the signs were equivocal in these leads, they were also equivocal in the oesophageal lead. There are probably two reasons for this. High posterior infarcts are near the transitional zone in which a ventricular Q and a negative T are normal findings. Secondly, records taken from patients with bundle branch block show that in most cases the oesophagus faces the right ventricle or the septum at its lower end.

THE EFFECT OF DIGOXIN ON THE RIGHT VENTRICULAR PRESSURE IN HYPERTENSIVE AND ISCHAEMIC HEART DISEASE

By M. Etheridge (introduced). Published in full, Brit. Heart J. 1950, 12, 317.

VENOUS PRESSURE PULSES IN CLINICAL HEART FAILURE

By E. P. Sharpey-Schafer.
COMPARISON OF FICK AND DYE METHOD OF CARDIAC OUTPUT ESTIMATION AND OBSERVATIONS ON CIRCULATION TIME

By H. Kopelman and G. d. J. Lee (introduced). Time concentration curves have been obtained from arterial blood following injection of the dye T, 1824. From these curves the minute cardiac output and the intrathoracic blood volume have been calculated. Comparison has been made between this method of calculating cardiac output and that using the Fick principle at the same time. This shows a satisfactory correlation.

The rise and fall in taste sensation following saccharine solution injection was compared to the dye time concentration curves.

FIRST EFFORT ANGINA

By R. Kemball Price. First effort angina may be defined as that which develops soon after a particular effort begins, but which disappears despite continuation of that effort. A brief pause or curtailment on account of the pain is usual but not invariable, and thereafter the particular effort may be completed or even extended without further discomfort. This form of angina pectoris has been compared in respect of pain with "second wind" in respect of dyspnœa.

While few anginal patients voluntarily describe it, one out of five patients presenting with anginal pain might admit that they have experienced this modification of it, if questioned. No evidence has been found to suggest that the prognosis differs from that of angina pectoris generally. In course of time it gives place to the usual form which compels relinquishment of the effort. It is possible in a proportion of cases to prevent the first effort pain by nitroglycerine taken before starting.

It seems likely that the difficult start is due to failure of the coronary arterioles to dilate soon enough on first effort. Narrowing of a large coronary vessel from arterial disease might predispose to transient spasm of the arterioles it serves, and might also tend to reduce the influence of the rise of aortic pressure at the beginning of exercise on coronary flow.

THE CORONARY ARTERIES IN CARDIAC HYPERTROPHY

By J. P. Shillingford. The study of the coronary arterial circulation by injection and radiography at necropsy has shown that the changes of the arterial vascular pattern in left ventricular hypertrophy tend to fall into three groups; those in malignant hypertension show an increase in the size of the main coronary vessels with an attenuation of the smaller branches; in aortic valve disease all the vessels, large and small, are increased in size; in benign hypertension, where atheromatous narrowing of the larger vessels has occurred, there is a great increase in the finer vasculature.

Histologically the increase in the finer vasculature tends to accompany focal ischaemic myocardial degeneration and fibrosis.

SOME OBSERVATIONS ON THE ACTION OF TRINITRIN ON VENOUS PRESSURE AND OTHER CIRCULATORY FACTORS

By Wallace Brigden. The effects of trinitrin on the circulation have been studied in twenty patients using the technique of cardiac catheterization. There is a fall of right auricular pressure which is not due to an increase in heart rate. The cardiac output tends to fall in patients with a relatively normal circulation. These effects are probably due to a pooling of blood in the periphery.

HORIZONTAL TOMOGRAPHY OF THE CHEST

By J. J. Stevenson (introduced). The purpose of this form of tomography is to take body sections at right angles to those taken with the conventional type of equipment. It necessitates a special and somewhat complicated type of apparatus.
The cross-section views show considerable detail of the mediastinal structures and give an outline of the heart.

The aorta is shown throughout its course in the chest and congenital and other abnormalities are clearly seen. Among other cases, double aortic arch, right-sided aorta, and coarctation, are demonstrated.

A full account of the apparatus and the results obtained are given in the British Journal of Radiology of June, 1950.

**The Electrocardiogram of Syphilitic Heart Disease**

By G. O. Storey (introduced). The cardiograms in 72 cases of cardiovascular syphilis were examined. They were often abnormal (46 out of 72); this was commoner in those with cardiac pain (22 out of 26). Cardiographic evidence of cardiac ischaemia was found in 14 cases, but the classical evidence of cardiac infarction shown by the development of Q waves was seen in one case only.

A changing cardiogram, although not specific of cardiovascular syphilis, was present in 9 cases. From the examination of pathological material, the underlying changes in the myocardium appear to be necrosis with subsequent fibrosis, usually confined to small scattered areas but occasionally larger. Large areas of frank cardiac infarction were not found, unless there was coincident coronary atheroma, and in these cases there was usually little or no stenosis of the coronary arteries from the aortitis.

**Unipolar Electrocardiography in Pulmonary Stenosis**

By R. M. Marquis (introduced). The electrocardiographic features of seven cases of congenital pulmonary stenosis with intact ventricular septum are reported in illustration of the pattern of extreme right ventricular hypertrophy common in this malformation. The chief characteristics of this pattern are seen in the chest leads which show tall R waves, S–T depression, and deep inversion of the T waves extending far across the left side of the chest.

The diagnosis was supported by necropsy findings in four of the cases. It is suggested that the right ventricle hypertrophies in proportion to the degree of the stenosis in relation to the right ventricular output, and that unipolar electrocardiography, by recording the degree of the hypertrophy, affords an accurate measure of the severity of the stenosis.

Evidence is presented to show that the development of the electrocardiographic pattern described precedes clinical deterioration, change in the radiological appearance of the heart, the onset of right ventricular failure, and diffuse fibrosis of the myocardium. This pattern is characteristic irrespective of patency of the foramen ovale, post-stenotic dilatation of the pulmonary artery, the exact site of the stenosis, and of heart size on radioscopy.

The significance of this electrocardiographic pattern is discussed in relation to diagnosis, prognosis, and surgical treatment of the malformation.