PROCEEDINGS OF THE BRITISH CARDIAC SOCIETY

An Autumn Meeting of the British Cardiac Society was held at the Royal Society of Medicine, 1 Wimpole Street, London, on Friday, October 14, 1955. The Chairman, CRIGHTON BRAMWELL, took the Chair at 9.30 a.m.; 123 members and 27 visitors were present.

PRIVATE BUSINESS

1. The minutes of the last Annual General Meeting having been published in the Journal (17, 573, 1955) were taken as read and confirmed.
2. The Chairman spoke of the loss that the Society and British Medicine had sustained in the deaths of the Right Hon. Lord Horder, Dr. A. J. Wilson, and Dr. J. McPherson, and members stood in silence.
3. Sir William Hume was unanimously elected as an Honorary Member of the Society, with acclamation.
4. Sir John Parkinson’s term as President expiring at the beginning of the next Annual General Meeting, he having been elected in May, 1952, Maurice Campbell was nominated as the new President to take office at the Annual General Meeting, 1956.
5. Samuel Oram’s term of office expiring at the beginning of the Annual General Meeting, 1956, Patrick Mounsey was elected as the new Honorary Secretary and Treasurer.
6. The names forwarded to Dr. K. E. Grewin, the Secretary-General of the Second European Congress of Cardiology, to represent this country in the various panel discussions and round-table conferences to be held at the Congress, which is to take place in Stockholm from September 10 to 14, 1956, were reported.

SHORT COMMUNICATIONS

MITRAL VALVOTOMY—A PROGRESS REPORT

By R. W. D. TURNER. Many groups reported their early experience with the surgical treatment of mitral stenosis, but there has been no progress report from this country. We have reviewed the first 250 patients operated upon from one medical unit over a period of 4½ years, none less than 6 months ago.

The cases have been analysed in relation to various factors that may influence the result, including age, rhythm, heart size, heart failure, clot, calcification, and associated mitral incompetence or aortic valvular disease.

Brief reference was made to physical signs, special methods of investigation, instrumental aids to valvotomy, and subsequent re-fusion.


By BERTRAND WELLS. One hundred patients with mitral stenosis have been assessed by measuring the delay in the first heart sound, and the interval between the second heart sound and the opening snap of the mitral valve. An operation of mitral valvotomy has since been performed on each of these patients, and the size of the mitral orifice and the pressure gradient across the mitral valve has been recorded. The correlation between the pre-operative assessment and the operative findings is close, and enables the mechanism of formation of the heart sounds to be clarified.

Further information on the mechanism of formation of the first heart sound and the opening snap is obtained from phonocardiograms taken during operation, with synchronous pressure tracings from the left atrium and the left ventricle.

This study demonstrates that the phonocardiograph is of value in selecting patients suitable for mitral valvotomy.

THE CONTRIBUTION OF TRICUSPID INCOMPETENCE TO RIGHT VENTRICULAR OUTPUT IN CONGESTIVE HEART FAILURE

By PAUL KORNER (introduced) and JOHN SHILLINGFORD. Studies of the right atrial pressure and flow curves in patients with cardiac failure show that with a raised venous pressure considerable tricuspid regurgitation may occur. The presence of this incompetence throws an increased load on the right ventricle for
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a given cardiac output. With the development of the dye dilution technique for the quantitative estimation of regurgitant flow, it has now been possible to gain further knowledge of the effect of incompetent valve lesions on the cardiac output and its relation to heart failure.

A quantitative estimation of the amount of backflow through incompetent tricuspid valves has been correlated with the clinical and necropsy findings in 14 patients, 13 of whom were in congestive heart failure. With the development of functional tricuspid incompetence in congestive heart failure the right ventricular stroke output remains higher than in normal subjects in spite of a low forward cardiac output, the discrepancy being due to regurgitant flow.

Exercise tends to increase the regurgitant flow at the expense of the forward cardiac output although the total right ventricular output may remain the same or rise; rest has the opposite effect.

These results correlate with the clinical and necropsy findings and offer an explanation for the phenomenon of "low fixed cardiac output" in some patients with congestive cardiac failure.

THE MURMURS OF PATENT DUCTUS ARTERIOSUS

By CATHERINE NEILL (introduced) and PATRICK MOUNSEY. A clinical and phonocardiographic study has been made of the murmurs in 100 consecutive patients with patent ductus, in whom the diagnosis was subsequently confirmed at operation. The murmurs were of two types, that of uncomplicated patent ductus and that of patent ductus complicated by severe pulmonary hypertension. In the latter the diastolic element of the murmur tended to disappear while the systolic murmur was often insignificant. A functional mid-diastolic murmur, preceded in some cases by a sound resembling a soft opening snap, was seen in the presence of a large shunt, and both the murmur and sound disappeared after ligation of the patent ductus. This observation suggested that the "snap" as well as the murmur might be functional and due to increased blood flow through the mitral valve.

In the differential diagnosis from other continuous murmurs, the timing of the murmur appeared important, although in most cases the site of the maximum intensity indicated the nature of the lesion. The timing of the murmurs of patent ductus, of bronchial arteries, of systemic and pulmonary arteriovenous fistula, and of venous hums were compared, and it was seen that the murmur became increasingly delayed as the distance of its site of origin from the heart increased. In rarer congenital lesions, such as aortic septal defect or coronary-pulmonary fistula, the site and timing of the murmur were sometimes atypical of patent ductus, but in other patients no certain differentiating points were found.

THE AUSCULTATORY AND PHONOCARDIOGRAPHIC SIGNS OF ATRIAL SEPTAL DEFECT

By IAN GRAY (introduced) and AUBREY LEATHAM. Published in full Brit. Heart J., 18, 1956.

CLOSURE OF ATRIAL SEPTAL DEFECTS—A METHOD OF TREATMENT

By F. RONALD EDWARDS. Atrial septal defects can be closed by suture. A method is described whereby the defect is sutured through the wall of the right atrium, the edges of the defect being directly opposed and the atrial wall fixed over the sutured object as a strengthening patch. The operation is performed under tactile control. No hypothermia is required.

The operation is best performed in the young, and before severe failure and gross cardiac enlargement are established.

Of the 14 cases explored, 13 have had the operation performed, and a satisfactory result obtained in 11 cases. Two died post-operatively; one, an unsuitable advanced case, from cardiac failure, and the other from a technical error producing an aortico-atrial fistula.

CINE-ANGIOCARDIOGRAPHY WITH AN IMAGE INTENSIFIER

By R. ASTLEY (introduced by Clifford Parsons). The conventional method of cine-radiography is photography of the fluorescent screen image: this involves high dosage of radiation to the patient and heavy loading of the X-ray apparatus. The advent of the image intensifier has reduced these hazards and provided a practical method of cine-radiography. Limitations are the restricted field area and the standard of definition, which is inferior to that of an ordinary radiograph. An advantage is that the gain of a sense of movement aids interpretation of the results.

A short 16-mm. film, made up of prints from cine-angiograms, was shown to demonstrate the possibilities of the method.
A PROPOSED CLINICAL METHOD OF INVESTIGATING THE ELASTICITY OF LARGE ARTERIES

By JAMES CONWAY (introduced) and K. SHIRLEY SMITH. A method of determining the state of the elastic tissue of the large arteries is being studied; it depends upon the delivery from the heart at each beat of the same volume of blood at different levels of diastolic pressure, changes in pulse pressure thus produced being an indication of loss of arterial elasticity. If the arterial reservoir is reasonably elastic at the normal diastolic level of pressure, then reduction of pressure without change in stroke volume would leave the pulse pressure unchanged.

In order to produce these theoretical conditions, patients and volunteers have been used in observations in which the blood pressure and pulse pressure was recorded by a condenser manometer from the brachial artery, and the blood pressure was reduced by inhalation of amyl nitrite. It was assumed that amyl nitrite did not have an immediate effect upon stroke volume and did not influence the elasticity of the large arteries while it produced dilatation of the small muscular vessels. Reduction of pressure was thus produced and the pulse pressure could be studied for a limited number of heart beats, until tachycardia ensued and the assumption concerning stroke volume no longer held. Predictions from this theory have been examined and upheld in 10 young student volunteers all of whom have been shown to have elastic arteries. Thirty patients with hypertension have been studied and have been found to fall into two groups: (1) those in whom the pulse pressure does not fall with the reduction of diastolic pressure (i.e. those with elastic arteries), and (2) those with a considerable fall in pulse pressure and according to the test with inelastic arteries. Many of the "inelastic group" have clinical evidence of arteriosclerotic hypertension and many of the "elastic group" are young patients with severe hypertension. There are very few contradictions between advanced clinical signs and the findings in these tests.

THE HEART IN DISSEMINATED LUPUS ERYTHEMATOSUS

By W. BRIGDEN, E. G. BYWATERS, and (introduced) M. LESSOF and I. ROSS. The cardiac manifestations of disseminated lupus erythematosus were investigated in 30 patients, and 11 of these were examined at necropsy. Acute or subacute pericarditis was recognized clinically on some occasion during the disease in 50 per cent, but electrocardiographic changes compatible with pericardial disease occurred in 85 per cent, and evidence of past pericarditis occurred in all of the necropsy cases. Myocarditis was difficult to recognize clinically, but there was evidence that extensive myocardial disease was a factor in the development of heart failure in patients who had systemic hypertension. Definite clinical evidence of valvulitis occurred in only 5 cases and two of these developed mitral systolic and diastolic murmurs while under observation. One patient died of bacterial endocarditis. At necropsy 5 cases had endocarditis; the mitral valve was affected in all, the tricuspid valve in three, and one had evidence of slight aortic valvulitis.

THE EFFECTS OF PENICILLIN TREATMENT ON THE HISTOLOGICAL LESIONS OF SYPHILITIC AORTITIS

By B. E. TOMLINSON (introduced by W. G. A. SWAN). The criteria used in this communication for the diagnosis of active syphilitic aortitis were stated.

The histological changes in the aorta in 23 cases of syphilitic aortitis treated with varying amounts of penicillin were described. These findings were compared with those of a control series of cases of syphilitic aortitis in which penicillin had not been given. Slides of the lesions were shown. The changes in the treated cases in relation to the period of survival after treatment, and the time needed for syphilitic lesions to resolve, were discussed.

The conclusion was reached, that in this series, there was no convincing evidence that penicillin is usually effective in the treatment of established syphilitic aortitis.

CORONARY HEART DISEASE IN THE PRESENCE OF PULMONARY DISEASE

By ARTHUR J. THOMAS. Pulmonary disease and coronary artery disease are frequently found together in necropsies on coalworkers. To study this problem in the population it is necessary to verify the clinical diagnosis of coronary heart disease. To this end a correlation of the clinical and post-mortem findings has been made in 51 coalworkers with severe pulmonary disease, either pneumoconiosis or chronic bronchitis and emphysema.

27 cases had clinical and post-mortem evidence of coronary heart disease.

17 cases queried clinically as having other heart disease in addition to pulmonary disease were not confirmed at necropsy as having coronary heart disease.
7 cases with severe coronary artery disease at necropsy had not been so regarded during life, but had severe pulmonary heart disease.

<table>
<thead>
<tr>
<th>Necropsy findings</th>
<th>Cardiac infarction: 11</th>
<th>Severe coronary artery disease: 16</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart weight (mean)</td>
<td>455 g. (340–700 g.)</td>
<td>305 g. (220–400 g.)</td>
</tr>
<tr>
<td>Cardiac pain</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>B.P. over 160/100</td>
<td>3</td>
<td>2</td>
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</tbody>
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**Electrocardiographic analysis of the 27 cases**

<table>
<thead>
<tr>
<th>Pulmonary heart disease</th>
<th>Cardiac pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac infarction</td>
<td>6</td>
</tr>
<tr>
<td>Right bundle-branch block with S–T segment change</td>
<td>4</td>
</tr>
<tr>
<td>S–T trough depression I and V6</td>
<td>5</td>
</tr>
<tr>
<td>Right axis with S–T trough depression in III and V6</td>
<td>2</td>
</tr>
<tr>
<td>QIII abnormal</td>
<td>2</td>
</tr>
<tr>
<td>aVL abnormal (also auricular fibrillation in I)</td>
<td>2</td>
</tr>
<tr>
<td>Left axis deviation and auricular fibrillation</td>
<td>3</td>
</tr>
<tr>
<td>L.A.D. and auricular tachycardia</td>
<td>1</td>
</tr>
<tr>
<td>L.A.D. and low blunt TI</td>
<td>1</td>
</tr>
<tr>
<td>L.A.D. only</td>
<td>1</td>
</tr>
</tbody>
</table>

A high degree of left axis deviation, a QR in aVL with T inversion, and trough depression of S–T in V6 were not present in comparable cases of pure pulmonary disease. The lesser electrocardiographic signs of myocardial injury (Evans and McRae, 1952) have been valid in this series, and were present in 8 cases, 5 without recognized cardiac pain. Auricular fibrillation has been extremely rare in pure pulmonary heart disease, but was present in four cases with both diseases. It has been possible to distinguish cardiac pain from the sharper and more variable pleuro-pulmonary pain correctly 12 times but incorrectly once. The more severe the ischaemic heart disease the heavier the left ventricle (Harrison and Wood, 1948). Left ventricular prominence at cardiography may be supporting evidence of coronary heart disease.

Analysis of 17 cases of severe pulmonary disease with minimal coronary artery disease at necropsy shows that 5 without other electrocardiographic abnormality were pure pulmonary disease; the 12 with unexpected cardiac abnormality had respectively, myocardial fibrosis of unknown origin (2), pericarditis (3), hypertensive disease (2), paroxysmal tachycardia (2), heart block (? digitalis effect) (1), right bundle-branch block (1) and one unexplained. The mean heart weight was 412 g. (300–572 g.). Three of these only, viz. myocardial fibrosis (2) and right bundle-branch block (1), could appear incorrectly in the first group on clinical findings.

These methods can be applied to the detection of coronary heart disease in a group of people without producing a false high incidence. The results in a group of 110 coalworkers not seriously ill but complaining of some respiratory disability was shown.

An analysis of the relationship of pulmonary disease and coronary heart disease did not show any direct relationship between them.

**The Anatomical Changes in Rheumatic Tricuspid Valve Disease**

By Arthur Hollman (introduced by Dr. Kenneth Harris). The normal anatomy and function of the tricuspid valve were described. The orifice points towards the nearby outflow tract of the right ventricle being set roughly at right angles to it. Valve closure is effected mainly by apposition of the atrial surfaces.
of the cusps. The mitral orifice by contrast is comparatively flush with the wall of the left ventricle and does not point towards the outflow tract. Mitral valve closure is more efficient than tricuspid valve closure.

Twenty-one pathological specimens of rheumatic tricuspid valve disease have been studied with the main objects of determining the probable function of the stenosed tricuspid valve and the feasibility of valvotomy. Five specimens had well-marked stenosis with orifices 1.5 cm. in length or less and of these probably only one was a competent valve. The detailed anatomy of these specimens was described with particular reference to commissural fusion and the probable function of the valves after valvotomy.

Of the sixteen larger valves, also fully described, several were grossly incompetent.

In rheumatic disease of the tricuspid valve incompetence is frequently associated with stenosis. There are anatomical reasons for this.

THE ELECTROCARDIOGRAM IN INDUCED HYPOTERMIA

By Peter R. Fleming (introduced by Charles Baker). The study comprises an analysis of electrocardiograms recorded during experimental hypothermia in dogs and during eighteen operations performed using hypothermia at Guy's Hospital.

The changes seen were essentially similar in dogs and in men except that the dogs were resistant to the production of auricular fibrillation which occurred frequently in the human patients. This arrhythmia rarely occurs during cardiac surgery at normal temperatures. In this series its occurrence seemed to indicate a good prognosis.

In all cases changes due to the anaesthetic or the operation have been eliminated as far as possible by comparison with records at similar operations at normal temperatures.

All cases showed bradycardia. This was associated with a lengthening of the S–T interval, greater than would have been expected from the change in rate alone. The P–R interval (where present) and the QRS were not consistently prolonged, but in some cases a characteristic deformity of the QRS–ST junction appeared. This was seen more frequently in dogs and consisted of a notch on the downstroke of the R wave which deepened until the whole complex became biphasic; the secondary wave sometimes exceeded the R wave in height. It is not certain whether the second wave is part of the QRS complex and represents impaired conduction or whether it is the first part of the S–T segment and represents a current of injury. This point was discussed.