PROCEEDINGS OF THE CARDIAC SOCIETY OF GREAT BRITAIN AND IRELAND

The FIFTH ANNUAL GENERAL MEETING of the Cardiac Society of Great Britain and Ireland was held at the School of Geography, Oxford, on Friday, April 18, 1941.

CHAIRMAN: A. G. GIBSON

36 Members and 8 Visitors were present
The Chairman took the chair at 10.15 a.m.

PRIVATE BUSINESS
1. The minutes of the last meeting were read and confirmed.
2. The accounts, audited by Evan Bedford and Curtis Bain, were presented by the Council and approved, the balance being £66 4s. 11d.
3. The Secretary proposed on behalf of the Council and the Society approved the following addition to Rule 11
   "In exceptional circumstances the Council may release a Member from this rule."
4. The Chairman proposed on behalf of the Council, and the Society agreed
   "that in view of the importance of keeping the Society in being during the war, Rule 23 should be in abeyance and that the Secretary should be asked to continue in office, although he would have completed five years after the 1941 meeting."
5. On the recommendation of the Council, it was decided to ask the following to accept nomination as Honorary Members: J. B. Herrick, Chicago, U.S.A., and Frank N. Wilson, Ann Arbor, U.S.A.
6. Five Associate Members were elected Ordinary Members; two new Associate Members were elected; and ten Associate Members were re-elected for another period of three years.
7. Crighton Bramwell, Manchester, and Henry Moore, Dublin, were elected members of the Council for the years 1941–45.
8. The Secretary reported that:—
   (1) since the last meeting the Council had heard with regret of the death of three Honorary Members—
      Maud Abbot of Canada,
      Karel Frederik Wenckebach of Vienna, and
      Charles Laubry of Paris;
   (2) the Committee appointed at the request of the Ministry of Labour and National Service to advise on the instructions for the examination of the cardiovascular system of recruits had reported to the Ministry, and that the report had been accepted in part;
   (3) the Council had appointed a Committee to consider the question of members from other parts of the British Empire;
   (4) the Journal Board of the British Medical Association had written expressing...
DISCUSSION ON EARLY SIGNS OF CARDIOVASCULAR DISEASE (Morning Session)

CHAMBERLAIN opened the discussion on the early signs of valvular heart disease. Auscultation was still the method of diagnosis of paramount importance, diastolic murmurs in aortic regurgitation and mitral stenosis establishing the diagnosis with certainty in most cases. There were certain other clinical, electrocardiographic, and radiological signs, some of which in combination might be sufficient to establish a diagnosis of valvular disease without the characteristic murmur.

In mitral stenosis the clinical signs usually emphasized were accentuation or reduplication of the mitral first, pulmonary second, or mitral second sounds. An abrupt loud mitral first was the most constant associated clinical sign in mitral stenosis and was of more importance when the heart rate was slow. Accentuation of the pulmonary second was a much less constant phenomenon in early cases of mitral stenosis though common in the more advanced ones, whilst accentuation of the mitral second was rather uncommon. Reduplication, though common in the pulmonary second, was not a constant or a particularly helpful sign. The electrocardiogram frequently showed no changes, but a split P wave, especially of low voltage, was such a common sign in mitral stenosis that it was worthy of consideration in doubtful cases, especially when associated with the less common right ventricular preponderance.

Enlargement of the left auricle, seen best on screening in the right (I) oblique view after a barium swallow, and prominence of the pulmonary arc were important radiological signs; the latter was an earlier and more constant sign than auricular enlargement.

The diagnosis of aortic stenosis was debateable. Some held that the characteristic anacrotic pulse associated with a harsh systolic murmur (with or without a thrill) over the aortic area and conducted into the neck was essential for diagnosis. For others a harsh murmur and thrill were alone acceptable. Some of the latter cases proved eventually to be due to genuine stenosis and when left ventricular hypertrophy was present the presumptive diagnosis might be justified even in the absence of an anacrotic pulse, for such a pulse indicated a well-developed aortic stenosis.

For the diagnosis of aortic regurgitation auscultation still remained the principal method. The basal diastolic murmur might long precede other clinical or instrumental methods of diagnosis. The aortic second sound was rarely abolished until regurgitation was free and the vascular phenomena were late in appearance.

TERENCE EAST discussed the early signs of hyperpneic disease.

Changes in the arterial system and in the heart could only be regarded as late signs, so the early diagnosis would depend on the interpretation of the readings of the sphygmomanometer.

The chief difficulty lay in distinguishing the transient rise in blood pressure due to nervousness from the true early high blood pressure. On the whole the diastolic pressure was a safer guide than the systolic because it was less labile. Nervousness might cause rise in pulse rate and not in pressure, and vice versa. The diastolic pressure might sometimes be unreadable. In doubtful cases raising the arm might give a clearer diastolic reading. In any borderline case the more readings the better.

Even quite a long time spent waiting recumbent would not always bring a nervous rise down. The way the heart beats was perhaps the most constant indication of the
confirmed by overweight, by of degree in the remaining 17 patients they were not seen to better advantage in this lead than causes. An estimation of the often remain must of this was exhibited.

42 in difficulty great

In acute rheumatism, the mitral regurgitant murmur due to the dilated mitral ring was the usual first evidence of myocarditis followed rapidly by evidence of cardiac enlargement. In the other types of heart disease, thyrotoxic, hypertensive, and senile, the first evidences of involvement of the myocardium were usually the symptoms of cardiac insufficiency. Here the importance of dyspnée was paramount, and it became of considerable value to be able to distinguish dyspnée arising from cardiac insufficiency from that resulting from other causes. An estimation of the arm–tongue circulation time had proved of considerable value in this, though this was generally an indication of the onset of failure. The great difficulty in accepting minor changes in the electrocardiogram or in the X-ray picture of the heart as early evidence of myocardial damage lay in the wide variation met with under normal conditions.

**SHORT COMMUNICATIONS**

**THE RIGHT PECTORAL ELECTROCARDIOGRAM IN AURICULAR FIBRILLATION**

William Evans showed how well the auricular movements in auricular fibrillation are displayed in the right pectoral electrocardiogram. When this lead was recorded in 42 cases of auricular fibrillation conspicuous auricular waves were seen in 25, but in the remaining 17 patients they were not seen to better advantage in this lead than in limb leads. A study of the favourable tracings showed that the form of the auricular waves, although not so completely regular as the waves of flutter, was remarkably uniform. In more than one-half the cases the wave recurred regularly, and in the others the irregularity was slight; it occurred at intervals and was sometimes associated with ventricular systole. The rate of auricular contraction in the majority was slower than 400 a minute which was the average for all the cases. Occasionally the right pectoral lead showed typical flutter in patients previously regarded as instances of fibrillation following clinical examination and limb-lead electrocardiography. Further the application of this chest lead in patients believed to have simple paroxysmal tachycardia will sometimes demonstrate the arrhythmia to be auricular flutter, and an example of this was exhibited.

(Published in full on p. 247)

**THE COLLABORATIVE CIRCULATION IN COARCTATION OF THE AORTA**

Bramwell Crighton and A. Morgan Jones

(Published in full: see p. 205)

**THE HÆMATIC FACTOR IN ANGINA PECTORIS**

B. T. Parsons-Smith described some cases illustrating the hæmatic factor in angina pectoris, and discussed the factors involved. Coronary sclerosis was the basis of myocardial ischaemia in 80–90 per cent of
cases. Myocardial anoxæmia might be produced also by various types of anæmia and possibly also by polycythaemia, and these syndromes might be curable by treatment. He recounted the details of three patients suffering from chronic hemorrhagic anæmia, hypochromic anæmia, and pernicious anæmia. In the last case anginal symptoms had developed, and in each case the repair of the anæmia had abolished the angina. The importance of blood counts in difficult cases of angina pectoris was illustrated by these clinical examples. The existence of pernicious anæmia might remain unsuspected until the later stages of its development.

The Electrocardiogram of the Stokes-Adams Attack

John Parkinson and Cornelio Papp (introduced)

(Published in full: see p. 171)

"Chronic Myocarditis"

John Hay urged that the words "chronic myocarditis" should not be used as a diagnosis because he considered it misleading. Myocarditis should signify an inflammation of the myocardium, and admittedly there were sound reasons for the term chronic rheumatic myocarditis when referring to the advanced phases of a rheumatic infection.

In the later decades of life, however, myocardial fibrosis was circulatory in origin and degenerative rather than inflammatory. He feared that the teachers in medicine and the writers of text-books were very largely responsible for the persistence of this term and quoted several examples from well-known books, illustrating the loose and confusing use of the term. The pathologists were also to blame; one referred to "so-called fibrous myocarditis." Another wrote that obliteration of a branch or branches of a coronary artery was the "most common cause of cardiac fibrosis, or as it is usual to term it 'interstitial myocarditis,'" Similarly in books dealing with electrocardiography, one finds "Myocarditis, Chronic" used almost as an equivalent of "Myocardial Disease" or "Myocardial Damage."

There was no need to multiply instances. It seemed quite clear that one writer after another had timidly refused to discard this unsatisfactory nomenclature because others were equally timid.

It might be true that in the production of ischemic fibrosis changes occurred that might legitimately be termed inflammatory, but he was convinced that to call such a pathological product myocarditis was to mislead. Cardio-sclerosis was a clearer and more appropriate term.

He felt that the Society as a whole should refrain from perpetuating such a muddling and misleading phrase as chronic myocarditis, except in those cases such as rheumatic carditis, where it might be justified.

Discussion on the Treatment of Coronary Thrombosis (Afternoon Session)

The speakers had been asked to consider if the orthodox treatment was all essential and based on sound principles.

Curtis Bain introduced the discussion and suggested that the period of rest in bed might be reduced to three weeks, provided that

(a) the patient had had no previous attacks;
(b) there had been no significant fall in blood pressure, no fever, and no peri-carditis; and
(c) there were no complications. He recognised that this suggestion would be provocative of discussion.

For treatment of the painful phase intravenous morphia was valuable; it would
stop the pain in about 3 minutes. One sixth of a grain should be injected intravenously, followed at once by one quarter hypodermically and later by another quarter, if required. Other drugs included theophylline-ethylene-diamine (Cardophyllin). It could be given intravenously in emergencies such as pulmonary oedema or complete heart block. Although useless in true shock following coronary thrombosis, respiratory stimulants such as ephedrine might revive certain patients who passed into a condition of pseudo-shock. Oxygen therapy was of undoubted use in those cases where the pain had persisted in spite of adequate doses of morphia and in those in whom cyanosis developed at any stage of the illness. The small open-top type of oxygen tent were well tolerated.

Digitalis should be reserved for those cases in whom cardiac failure with oedema developed. Auricular fibrillation was paroxysmal in type and the paroxysms usually subsided naturally without causing difficulty. In severe cases fibrillation might be present from the outset, but in these it was doubtful whether a myocardial poison such as quinidine would not do more harm than good.

The dietetic regime of under-nourishment was introduced by Masters. It aimed at reducing the work of the heart by lowering the basal metabolic rate with a concomitant fall in the pulse rate. Upward pressure from flatulent distension of the stomach was avoided. The diet was maintained for the whole period during which the patient was in bed and increased gradually thereafter. The diet suggested by Masters had been found to be rather severe, but a diet consisting of carbohydrates 100 g., proteins 45 g., and fats 35 g. has been well tolerated, and the results from it had been satisfactory.

John Hay visualized three stages in the treatment of coronary thrombosis. The first was the stage of onset with pain and shock. The second or intermediate stage was that of healing. Thirdly, at the end of three or four months the softened necrosed area had been replaced by scar tissue, and this was the period in which the cardiac reserve had to be built up and the patients' limitations defined.

A few days after the onset the pain was in abeyance, and the symptoms of shock had disappeared and the patient—when at rest—was comfortable, though often apprehensive.

As the production of a sound scar takes at least three months, so the patients' activities must be supervised for that period and kept well within safe limits, more especially in the first few weeks.

To obtain a leisurely heart rate with no sudden stresses or cardiovascular excitement (leaving to nature the business of repair), mental excitement, restlessness and apprehension are to be mitigated; veganin, theominal, or bromides are useful in producing mental calm, and a more philosophic outlook. For the first three or four days the patient needs only fluids such as weak tea, orange juice, lemonade, and drinks containing glucose or honey. Later, a light mixed diet can be given, but each meal should be small and appetising, and any food tending to produce flatulence avoided.

For the first two weeks the patient should be fed by the nurse. If pyrexia is present or if congestive cardiac failure has developed the nourishment should be limited to one and a half pints of skimmed milk per diem, and as the indications of cardiac failure diminished a gradual return to light, mixed, easily digested meals was indicated.

The fairly large doses of morphia required to relieve the pain at the onset, and the excessive sweating would almost certainly cause constipation and upset the digestion, but it was generally agreed that it is best not to worry the patient with laxatives or enemata until the third or fourth day. At the same time it was important to avoid flatulent distension; a tight belly hampering the diaphragm was an unfair and avoidable handicap to a disabled heart.

In the later stages of the illness from three months onward when the patient would be gradually finding his feet, a new difficulty might present itself, namely, the danger
of his becoming unduly apprehensive about his cardiac condition and drifting into a condition of invalidism. Conversely, the active type of patient, resenting restrictions, might return prematurely to full work and run the risk of overstraining his damaged myocardium with disastrous results.

He agreed with Bain as to the value of oxygen in treatment. Drugs had their place in the alleviation of pain, as mental sedatives, and in the treatment of cardiac failure if and when it appears; but the essential fact to be borne in mind is, that beyond everything, physical rest is the most important factor in enabling the damaged heart to recover.

Gilchrist (in absentia) said that for the relief of pain and for the promotion of mental and physical rest there was no drug to compare with full, and if necessary repeated, doses of morphia. In spite of the relief of pain some patients surviving the initial infarct died in the course of a few days from shock. This aspect of treatment has tended to be neglected, and though there was no specific remedy for this peripheral vascular failure it is possible that more might be done to safeguard the patient. On the other hand, a very large number of patients did very well, after the pain has been relieved, with no active drug treatment. Complete rest in bed might do much more than it was sometimes given credit for, particularly if the rest in bed were complete in every detail.

In the severer cases of shock there was, for instance, a natural hesitation to employ intravenous saline solutions and transfusions of whole blood, although these were commonly given in shock from other causes. In the early stages a plentiful supply of hot water-bottles or preferably a "shock cage", fitted up with more than the usual electric bulbs and continued for 24 or 48 hours, should be provided. Clinical studies in other related forms of peripheral vascular failure indicated that benefit was likely to be obtained from the use of the active principle of the suprarenal cortex. It had been used after extensive burning injuries and in virtue of its power to regulate water-balance and cellular permeability, a potent extract of suprarenal cortex was worthy of a more extended trial in the severer cases of cardiac infarction. The value of intravenous therapy was doubtful, but concentrated blood serum might be preferable to massive saline infusions or whole blood transfusions.

The development of an abnormal rhythm such as a ventricular tachycardia, with the subsequent development of ventricular fibrillation, was probably more common than supposed, particularly as the patient himself might be unaware of any change in heart rhythm. Quinidine might be used as a preventative measure when the indications were those of an extensive infarct: 0-2 g. at eight-hour intervals was harmless and might be of real benefit. The continuous drip method for the intravenous administration of quinidine as proposed and used by Hepburn and Rykert, was valuable in the treatment of paroxysmal tachycardia. By vigorous shaking, 50 to 60 grains of quinidine sulphate could be dissolved in 500 c.c. of 5 per cent glucose solution; after filtering and warming, the sterile solution should be run at a rate of 1 to 2 c.c. per minute until normal rhythm returns or until cinchonism is induced.

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After luncheon Dr. H. W. Garrod showed the Society round the College, and in particular the Library where there were interesting books and records of Harvey.

Dinner was served in Merton Hall, through the kindness of the Warden, Sir John Miles. Fraser proposed the health of the Society and recalled the early history of the Cardiac Club, founded after the last war.