PROCEEDINGS OF THE BRITISH CARDIAC SOCIETY

The ELEVENTH ANNUAL GENERAL MEETING of the British Cardiac Society was held at Guy's Hospital, London, on Tuesday, May 6, 1947. Chairman: MAURICE CAMPBELL. The Chairman took the chair at 9.30 a.m.; 62 members and 19 visitors were present.

PRIVATE BUSINESS

1. The minutes of the last meeting having been printed in the Journal (8, 233, 1946) were approved and signed.
2. The Secretary reported that a balance of £8 16s. 8d. had been handed over at the transfer of the Secretaryship and said that subscriptions amounting to £78 were due in 1947–48. It was decided that the subscription should be increased to 20s. for Ordinary members and 10s. for Associate members.
3. Boyd Campbell (Belfast) and McMichael (London) were elected members of the Council for the years 1947–51.
4. The following new members were elected:
   
   **Ordinary Members**
   
   Wyn Jones
   Lovibond
   Sharpey-Schafer
   Suzman
   Swan

   **Associate Members**
   
   D. Rhodes Allison
   Roland E. Anderson
   Ronald Hartley
   Ronald Jones
   Geoffrey Konstam
   R. W. Luxton
   Wilfred Oliver
   R. Kemball Price
   Richard Turner

   Five Associate members were re-elected for a further period of three years.
5. The following changes in the Rules were carried, *nem. con.*, on the recommendation of the Council:
   
   Rule 8. Substitute " Rules 12 and 22 " for " Rule 12 ".
   
   Rule 17. Add at the end " and shall take over office from his predecessor at the start of the meeting ".
   
   Rule 21. Insert before " the Chairman-Elect ", " the Chairman ".
6. The Secretary reported that as a result of representations to the President of the Royal College of Physicians, a Committee on Cardiology had been constituted by the College. The Report of this committee, dealing with the training of specialists in cardiology, the practice and teaching of cardiology, and the planning of cardiac services under the National Health Scheme, had been published and copies were available. The Secretary also announced the progress of arrangements for the International Conference of Physicians at which the Section of Cardiology would be under the Chairmanship of Sir Maurice Cassidy.
7. The Council of the Society was authorized to decide on the election of new Honorary Members after the conclusion of the International Conference of Physicians.
8. The Surgical Treatment of Hypertension was chosen as the main discussion at next year’s meeting.

DISCUSSION ON CARDIAC EMERGENCIES

1. **Acute Right and Left Heart Failure**: opened by BRAMWELL and McMicheal.
2. **Cardiac Infarction**: opened by BOYD CAMPBELL and HILL.
3. **Loss of Consciousness**: opened by MAURICE CAMPBELL and LENDRUM.
4. **Pulmonary Embolism**: opened by PAUL WOOD.

CRIGHTON BRAMWELL said that the two most important precipitating causes of *acute heart failure* were coronary occlusion and pulmonary embolism, both of which would be dealt with by later speakers. He also mentioned the possibility of rupture of a cusp of the
aortic valve and of sudden occlusion of the mitral orifice by a ball thrombus in the auricle. He then referred to two causes of acute heart failure that had received less general recognition. Sudden failure of the left auricle in association with severe mitral stenosis might occur unexpectedly, and he had reported two cases seen during pregnancy. Secondly acute heart failure due to vitamin B1 deficiency, though rare in this country was important since the treatment was specific and lesser degrees of vitamin deficiency might be a contributing factor in the production of heart failure.

J. McMichael divided his remarks into acute left and acute right heart failure.

**Acute Left Heart Failure.** Opportunities had occurred of observing attacks of acute orthopnea with the cardiac catheter in situ. The arterial pressure rose above the previous level even in hypertensive subjects. The right auricular pressure invariably rose in the attack and, as the lungs became congested, presumably the left auricular pressure also rose considerably. Blood returning to the right heart contained less oxygen than before the attack, but owing to the increased oxygen uptake resulting from the struggle for breath the cardiac output was found to be slightly increased.

In treatment the most effective and reliable remedy seemed to be morphine, under the influence of which right auricular pressure, metabolic rate, and arterial pressure fell. It was not yet possible to say what was the exact sequence of events either in the precipitation of the attack or in the mechanism of its relief. After morphine the next most reliable remedy was theophylline-ethylene-diamine which often reduced the right auricular pressure within 5 minutes. Venesection should also be recognized as a most effective, though less convenient, measure. Intravenous digoxin may not be satisfactory in these acute seizures. An attack had actually been observed to come on following intravenous digoxin and perhaps precipitated by the pressor action of this drug on the arterial system.

**Acute Right Heart Failure.** The most striking instances occur in acute pulmonary embolism. This subject is dealt with by Paul Wood. Patients with emphysema heart, however, are liable to develop attacks of congestive failure during acute exacerbations of bronchitis. In these attacks the cardiac output is high and the raised venous pressure should be regarded as to some extent a compensating mechanism. Reduction of venous pressure by venesection or digoxin often brings down the cardiac output, and these measures are thus of little benefit as a rule (Clin. Sci., 1947, 6, 187). The best treatment at this stage is an oxygen tent to improve the oxygen content of the blood, and measures to overcome the infection.

S. B. Boyd Campbell discussed the possible etiology of coronary infarction. Case histories of 169 hospital and 100 private patients were investigated. Age groups, occupation, fat consumption, and previous infections were noted; 85 per cent of the private and 92 per cent of the hospital cases occurred in the 40 to 70 age group with 37 per cent and 31 per cent in the 50 to 59 group. Over-indulgence of fatty foods has been prevented for many years by rationing and seems to have no bearing on the increase in infarction. Occupation in both private and hospital cases showed similar findings. No special occupation seemed to be associated with the occurrence of infarction. Occupations found included civil servants, clergymen, clerks, farmers, skilled workmen, labourers, housewives etc. Stress and strain seemed to have no bearing on the etiology though the actual onset in some cases was associated with exertion and in others followed a heavy meal.

It was difficult to find any definite infection in the history of previous illnesses. Some cases had had some feverish illness within a few weeks of the infarction. Case histories were of little help in solving the etiology.

He was indebted to J. H. Biggart, M. G. Nelson, and Florence McKeown for the lantern slides illustrating their recent work on coronary artery disease. First slides were shown from
cases dying of acute rheumatism. The initial change was an acute arteritis affecting the media followed by intimal changes with healing fibrosis and then secondary degeneration. The intimal fibrosis may almost occlude a large coronary artery. Similar changes were shown in coronary arteries of rabbits injected with horse serum. The final stages was demonstrated in a series of lantern slides of sections taken from the site of the coronary thrombosis. Intramural intimal hæmorrhage with fibrinoid degeneration preceded the thrombosis and was shown to be the precipitating factor. Secondary atheromatous changes were demonstrated. While hypertension plays a part in initiating the intramural hæmorrhage there seems to be evidence that all the changes from the initial medial arteritis to the fibrinoid degeneration and intramural hæmorrhage are an allergic reaction in patients sensitized to some protein resulting from an infection or possibly ingested.

I. G. W. HILL spoke on the electrocardiographic aspects of cardiac infarction.

MAURICE CAMPBELL said that even if loss of consciousness is rarely an emergency in the sense of needing urgent treatment, the anxiety caused and the difficulties of diagnosis justify its inclusion. The same word, syncope, is used for trivial fainting attacks and for a method of death and should, therefore, be banished from scientific discussion. It might be useful if it was recognized that syncope meant a sudden loss and fainting a gradual loss of consciousness, and he had sometimes used the terms in this sense. There is, however, no such agreement and it would at present be wrong because syncope is by definition (O.E.D.) "a failure of the heart's action, resulting in loss of consciousness or sometimes in death."

A more accurate knowledge of attacks of unconsciousness will be difficult to acquire without more accurate descriptions and this should be helped by more precise definition of terms. The speed with which consciousness is lost seems one of the most fundamental distinctions; and I suggest that the word tachy-a-psychia should be used for the sudden loss of consciousness, seen in most Stokes-Adams attacks or in epilepsy, and brady-a-psychia for the gradual loss of consciousness seen in most fainting attacks in young people. Lipo-psychia is used most often by Hippocrates for swooning, but apsychia is an alternative that makes the compound words euphonious. I am indebted to Mr. Yorke, Classical Tutor at New College, for coining these words for me.

Loss of consciousness concerns the physiologist and neurologist even more than the cardiologist; but there is, perhaps, more knowledge of the provoking mechanism of unconsciousness in disorders of the heart than of the nervous system. The common factor in the former group is the production of cerebral anoxæmia—whether by changes of rhythm, including ventricular standstill as in many Stokes-Adams attacks; by standstill of the heart as in a few fainting attacks or during anaesthetics; by the combination of a slow heart and a fall of blood pressure or by one of these alone, as in most fainting attacks and in many cases of the carotid sinus syndrome; or by temporary arrest of the cerebral circulation by venous stasis or difficulty in auricular filling during violent spasms of coughing or after pulmonary embolism. Anoxæmia without a change in the blood flow must also be remembered.

Broadly speaking, these various cardiovascular causes—and there are many more—can be divided into those where there is arrest of the cerebral circulation, often but not necessarily with cardiac or ventricular standstill, and those where there is diminution of the cerebral circulation (as in the severe fall of blood pressure after a large cardiac infarct or with a very rapid paroxysmal tachycardia); this may reach a stage of deficiency severe enough to cause unconsciousness which will therefore generally be gradual.

As a rule, therefore, tachyapsychia will mean sudden arrest of the cerebral circulation and bradyapsychia a gradual diminution of the cerebral circulation: there are, however, exceptions and, of course, many cases of tachyapsychia will be cerebral in origin. Arrest of the circulation
has greater dangers of the heart failing to start again and so tachyapsychia often has a much graver prognosis than bradyapsychia.

Generally, however, a patient who has been unconscious can give the physician no direct indication as to whether the cause was cardiac or cerebral and the decision may be very difficult and may call for much knowledge and experience: the symptoms produced in either way may be almost identical. It is, in particular, essential to be on familiar terms with epilepsy, idiopathic or symptomatic, in both its major and minor forms.

The duration of the attack is of great importance in this connection. A short attack, and this means one of not much more than two minutes, may be either cardiac or cerebral in origin. A long loss of consciousness, and this means one lasting more than 5 or 10 minutes must be wholly or partly cerebral—partly, when a short cardiac arrest started the loss and produced changes in the cerebral cortex that prolonged the unconsciousness.

I shall make some rather dogmatic statements that will help to clarify the discussion. Arrest of the cerebral circulation for from 5–10 seconds by a pressure cuff round the neck (Rossen, Kabat, and Anderson, Arch. Neurol. Psychiat., 1943, 50, 510) causes sudden loss of consciousness. Standstill of the heart or of the ventricle for from 5–10 seconds also causes sudden loss of consciousness, so this is further evidence that both act in the same way by producing sudden arrest of the cerebral circulation.

In unconsciousness due to cerebral anæmia, spasmodic movements or fits are dependent on the depth and duration of unconsciousness and not on its cause; and may, therefore, occur in attacks of all sorts. They follow 15–20 seconds after the arrest of the circulation.

Arrest of the cerebral circulation for 2 minutes in experiments on men or arrest of the circulation for 2 minutes by standstill of the heart in disease or during anaesthetics is compatible with perfect recovery.

Standstill of the heart for 5 minutes is generally permanent, i.e. the patient is dead. In the rare cases where the heart starts again after a longer interval than this, anoxæmia will have caused irreversible damage to the brain, especially to the cortex. The first sign of this cerebral damage will often be a much longer period of unconsciousness. Where the cerebral arteries are diseased as in many cardiac cases and most Stokes-Adams attacks a shorter period than 5 minutes may produce cerebral damage from which it is not possible to make a recovery.

Standstill of the heart for some time more than 2 minutes but less than 5 minutes will produce cerebral damage from which it may be possible to make a perfect recovery. The first sign of this cerebral damage may be a more prolonged period of unconsciousness. This means that even in a case known to be cardiac and known to be unconscious for many minutes, it is of great prognostic significance to know when the circulation returns, i.e. when the heart starts beating. Ultimately this period of between 2 and 5 minutes may be defined more precisely though there will always be great danger about recovery in such cases. In clinical records of heart standstill periods of from 20 to 80 seconds are not often exceeded and these are well within the limits when complete recovery can take place.

R. G. LENDRUM mentioned some of the conditions where a cardiac ætiology is wrongly and not infrequently diagnosed. Cerebro-vascular epilepsy and even hypertensive crises can thus be mistaken, especially where the patient is already under care for cardiovascular disease. Another is idiopathic epilepsy, if beginning in middle age, showing little or no movements and possibly having a prolonged sensory aura. Epilepsy of the chronic alcoholic is aided in joining this group by wrong assessment of the cyanotic complexion.

Patients who take attacks of hysterical hyperventilation with "unconsciousness" are looked on as liable to "heart attacks," this being most likely where the process starts with retrosternal discomfort (globus hystericus).

In nervous, hypersensitive people, consciousness of even a single ventricular extrasystole
than half months were diagnosed as a constriction of the pericardium; the onset. Swelling a woman is obscure, pericardectomy has not yet been undertaken. Cause and six was lead even his doctor, may decide that the heart has been "strained."

Aerogastric bloqué gives discomfort in the chest, faintness, and a premonition of death. This is most likely in someone already affected with cardiac disease and thus is all the more readily diagnosed as a "heart attack."

Paul Wood analysed twenty cases of massive pulmonary embolism. Of the clinical features special attention was directed to the frequency of substernal pain (12). Detailed studies of the behaviour of the arterial and venous blood pressure were presented. The arterial pressure fell abruptly and profoundly, reaching its lowest level within a matter of minutes or hours, and thus differed from its behaviour in acute myocardial infarction. The four patients with the lowest pre-embolic blood pressures all died at the time of the profound fall; no others died. The venous blood pressure rose sharply in all but two cases. A graph based on daily readings showed that the maximum elevation occurred within the first 24 hours, and was followed by a steady decline towards normal which was reached in 3–7 days. Maximum levels ranged between 2 and 10 cm. above the sternal angle.

The relationship of the venous blood pressure to the cardiac output was studied in one case. When the venous pressure was lowered by means of cuffs on the thighs, the cardiac output fell. When the cuffs were removed the venous pressure and the cardiac output rose. It was concluded that the raised venous pressure was beneficial and that the heart was not overloaded. The right ventricular pressure in this case, measured by means of cardiac catheterization, was +32 cm. of saline above the sternal angle with the subject nearly horizontal.

The rest of the communication dealt with the electrocardiographic diagnosis. The multiple chest lead pattern previously described by the author (Brit. Heart J., 1939, 1, 49, and 1941, 3, 21) was further amplified. The changing pattern was attributed to transient right ventricular stress, and was met in other conditions besides massive pulmonary embolism.

Short Communications
Constrictive Pericarditis
H. Cookson

Three cases were reported illustrating variations in the onset and in the course of chronic constrictive pericarditis. In the first, a man of 24, the onset was acute with pericardial effusion and six months later the full picture of chronic constrictive pericarditis had developed. Pericardectomy was done one year after the onset, and was partially successful, ascites persisting; because of technical difficulties adequate resection was not possible. The second case, a woman of 22, had an insidious onset with shortness of breath, puffiness of the face and swelling of the legs; there was X-ray evidence of old pleurisy. While under observation ascites developed and there was recurrent bilateral dry pleurisy. Pericardectomy was done 16 months after the onset; result good. The third case, a girl of 16, had abdominal symptoms at the onset. Over a period of 33 months the spleen has gradually enlarged and now fills more than half the abdomen. Gross thickening of pleura on both sides; extensive calcification in the pericardium; short attacks of paroxysmal tachycardia (auricular). The signs of pericardial constriction are moderate and because of this and because of the huge spleen, of which the cause is obscure, pericardectomy has not yet been undertaken.

There was no bacteriological proof of tuberculosis in any of the three cases. Cardiograms were very similar, showing S–T depression and T inversion. In the operated cases pulmonary
shadows, presumably due to pleural thickening, diminished. Little change in cardiac outline occurred after pericardectomy, but the abnormalities of T and S–T diminished.

**CONTINUOUS RECORDING OF SYSTOLIC BLOOD PRESSURE DURING THE UNCONSCIOUSNESS OF SUDDEN ANOXIA**

F. Latham. (Introduced by W. K. Stewart.)

A method of continuous systolic blood pressure recording as employed at the R.A.F. Institute of Aviation Medicine has been described, and also its application to the study of the unconsciousness of sudden anoxia.

The technique utilizes pulse waves passing under an arm cuff to actuate a solenoid valve which automatically maintains the pressure in the cuff at systolic blood pressure level. The pulse waves are detected by means of a photoelectric cell applied to the finger.

During a preliminary series of experiments, subjects were exposed in the decompression chamber to simulated altitudes of 35,000–40,000 feet and continuous blood pressure records taken during the subsequent anoxia, which ensued when their oxygen was cut off. The subjects were given oxygen when they became unconscious and the duration of each anoxia run was between 30–90 seconds. Difficulty was encountered in some of the experiments owing to convulsions occurring prior to unconsciousness.

In the four cases investigated at this stage there did not appear to be any evidence of cardiovascular collapse and the blood pressure remained elevated throughout.

**A CASE OF LONG-STANDING AURICULAR FIBRILLATION**

BY CRIGHTON BRAMWELL. To be published in full.

**FAMILIAL CARDIOMEGLY**

BY WILLIAM EVANS. To be published in full.

**PERIPHERAL AND CENTRAL VENOUS PRESSURES IN TRICUSPID INCOMPETENCE**

BY E. P. SHARPEY-SCHAFFER. To be published in full.

**RIGHT AURICULAR DILATATION WITHOUT VALVE DISEASE**

J. L. Lovibond described the case of a man of 28 who for the past 4 years had suffered from symptoms of chronic right heart failure with swelling of the neck, abdomen, and legs. An intracardiac thrombus was diagnosed during life but the underlying pathology was not revealed until necropsy. There was no history of rheumatic or other infection but, at the age of 14, his heart had been questioned because of cyanosis on swimming. At 21 tonsillectomy was performed for sore throats. At 24 his abdomen began to swell and he was found to have ascites, a big liver and spleen, radiological enlargement of the right auricle and superior vena cava, and auricular fibrillation. Failure was treated appropriately and normal rhythm restored by quinidine. From now on he became increasingly short-winded and ascitic, requiring regular abdominal paracenteses. For most of the next 4 years he remained in hospital. At 26 he had a lung infarct and later in the year underwent omentopexy without relief. At 27 venous pulsation in the neck and a striking apical triple rhythm were noted, but no murmurs. There was sinus rhythm with wide, bifid P waves in leads II and III and right axis deviation. There was no response to diuretics and his abdomen now required fortnightly taping. Although a few months later he reverted to fibrillation and also suffered a further lung infarct his general condition continued to be well maintained. The absence of clinical evidence of tricuspid or other valvular disease and the exclusion of cardiac tamponade by aspirations of a small pericardial effusion without beneficial effect, together with the history of lung infarcts and the radiological finding of a dense inert shadow in the position of the right auricular appendix, prompted the diagnosis of right auricular thrombus. In the face
of constant requests for surgical relief from the patient, an intelligent salesman, operation was
eventually agreed to and an adherent laminated thrombus (6·2×5·0×1·2 cm.) was removed
from a tensely dilated right auricle. Death occurred a few minutes later from pulmonary
embolus. The heart (500 g.) showed great dilatation and hypertrophy of the right auricle
and moderate hypertrophy of the left, but the ventricles, valves, aorta, coronary and pulmonary
arteries were normal. Microscopically there was diffuse myocardial fibrosis with great muscle
hypertrophy and focal endocardial thickening of both auricles and, to a lesser degree, both
ventricles. The liver (1485 g.) and spleen showed “sugar icing” capsular thickening with
advanced congestive reticular fibrosis of chronic back pressure. The pericardium and
peritoneum were unaffected, and the lungs normal apart from old and recent infarcts. The
condition was regarded as a healed generalized myocarditis of unknown origin, affecting
the whole heart but principally the right auricle, causing its inordinate dilatation with
resulting back pressure effects. The resemblance of the cardiac pathology to Fiedler's isolated
myocarditis and to cases described by Kugel (1939), Levy and von Glahn (1944) and Bedford and
Konstam (1946) were briefly discussed. A second case was shown of aneurysmal right
auricle with fibrosis and focal stretching of its wall, due to thrombotic occlusion of the right
auricular branches of the coronary artery.

A NEW SIGN IN COARCTATION OF THE AORTA

S. SUZMAN demonstrated a new sign in coarctation of the aorta by means of which the
superficial collateral arteries of the back and sides of the chest are caused to become visible
when not visible previously, or to become much more prominent. The sign is best produced
by causing the patient to stoop or bend forward and with the arms hanging down vertically.
An equally good method is for the patient to lie prone on a couch with a pillow under the
chest and the arms hanging down over the edge of the couch. A good light is advantageous.
The explanation for this phenomenon is that there is partial compression and obstruction
(in cases where the sign is positive) of the subclavian artery in the costo-clavicular space,
and any change of posture that widens this space will thus release pressure on the subclavian
artery and so cause a sudden filling up of superficial arteries so that they suddenly appear
in places where none were visible before or become more prominent.

In three cases out of four so tested, the sign was positive (subsequently three more cases
tested showed this sign). He suggested that this sign should be looked for as a routine in all
cases of suspected coarctation of the aorta and in fact all cases of high blood pressure, for by
this means there might be less likelihood of missing a superficial collateral circulation.

THE RETINAL VESSELS IN HYPERTENSION

BY A. LEATHAM. (Introduced by William Evans.) To be published in full.

PARTIAL SUPERIOR VENA CAVAL BLOCK

BY SHEILA HOWARTH. (Introduced by Sharpey-Schafer.) To be published in full.

MASS THROMBUS OF THE LEFT AURICLE

BY R. BENSON. (Introduced by William Evans.) To be published in full.

LONE AURICULAR FIBRILLATION

BY S. PRICHARD. (Introduced by William Evans.) To be published in full.