CARDIAC ARREST AND RESUSCITATION

Hunterian Lecture delivered at the Royal College of Surgeons of England
on
26th January 1956
by

B. B. Milstein, F.R.C.S.
Senior Surgical Registrar, Brompton Hospital

CARDIAC ARREST IS an emergency which may face any surgeon at any time. However, the complexity of methods of diagnosis and treatment which are used in all branches of medicine and surgery to-day is such that this emergency may face anaesthetists, obstetricians, radiologists using intravenous radioopaque materials or air injections, cardiologists during cardiac catheterisation and physicians during aspirations and injections. Stephenson, Reid, and Hinton (1953) have shown in 1,200 collected cases that the emergency arises most often during abdominal surgery, and that only one-third of the cases occur when the chest is open.

Treatment of this condition, to be successful, must be immediate. There is no time to call in an expert, because the delay that may occur is likely to result in cerebral damage even though the heart may recover its function. Anybody concerned with the management of a patient in whom cardiac arrest might occur should be familiar with the methods of treatment and be capable of applying them himself. The maximum time for which the brain can withstand anoxia is three minutes, but preexisting anoxia before the cardiac arrest, or cerebral vascular disease, may mean that the brain has much less margin than this. There is need therefore for the utmost speed in initiating treatment if the patient is to recover.

Incidence

It is difficult to determine the incidence of cardiac arrest in this country to-day, as the figures are not available. The Ministry of Health report for 1954 states that in 1954 an estimated 2,860,000 operations were performed on in-patients, and in 1953 there were 562 anaesthetic deaths. The average number of anaesthetic deaths per annum in recent years was 609 (Registrar-General, 1955). Beecher and Todd studied the deaths associated with 600,000 anaesthetics given in ten American University Hospitals in the period 1948-1952. There were 384 deaths associated with anaesthesia of which 199 were fatal cases of cardiac arrest, *i.e.*, half the total.

It seems probable that the incidence of cardiac arrest amongst total anaesthetic deaths will be the same in this country so that of the 600 anaesthetic deaths per annum, about 300 are due to cardiac arrest.

Assuming a mortality from cardiac arrest of about 50 per cent. at the present time it is likely that about 500 or 600 cases of cardiac arrest occur every year in this country. Table I shows the incidence of cardiac arrest in recently reported series. The figure varies between 1 : 1,000 and 1 : 5,000 operations.

The mortality from cardiac arrest was in the region of 60 to 70 per cent. 50 years ago. Table II shows that with one or two exceptions the mortality

has remained unchanged. This is a challenge to all who encounter the emergency, for there is no doubt that with prompt treatment, nearly all these lives could be saved.

TABLE I

INCIDENCE OF CARDIAC ARREST

Author	Year	Incidence
Bonica	1952	1 : 4,950 Operations
Miller, *et al.*	1952	1 : 858* Operations
Stephenson, *et al.*	1953	1 : 2,384 Operations
Snyder, *et al.*	1953	1 : 2,504 Operations
Cole	1953	1 : 1,200 Operations
Registrar-General	1953	1 : 4,850* Operations (estimated)
Beecher and Todd	1954	1 : 3,000* Operations
Lewis and Stanley-Brown	1955	1 : 1,463* Operations

* Fatal cases.

TABLE II

COLLECTED CASES OF CARDIAC ARREST

Author	Year	Number of Cases	Mortality per cent.
Green	1906	40*	78
White	1909	50*	80
Bost	1923	75*	76
Lee and Downs	1924	23*	65
Bailey	1941	40	90
Negovski	1942	290*	48
Nicholson	1942	7	71
Cooley	1950	48	75
Johnson and Kirby	1951	20	60
Dale	1952	12	84
Bonica	1952	13	62
Lahey	1953	24	33
Snyder, *et al.*	1953	35	86
Stephenson, *et al.*	1953	1,200*	72

* Collected cases.

Historical background of cardiac resuscitation

This problem is one which we might expect would have attracted the attention of John Hunter and he applied his experimental approach to it. He reported his findings in his " Proposals for the recovery of people apparently drowned " in 1776.

Hunter performed tracheotomy on animals and introduced bellows into the trachea for artificial respiration. By stopping the action of the bellows he produced cardiac asystole. He showed that when artificial respiration was begun again within ten minutes the heart would often recover. He concluded " it appears that anything salutary to life applied to the lungs would restore the heart's action after it had been at rest some time."

The greatest advance, cardiac massage, was made by Moritz Schiff, Professor of Physiology in Geneva (Fig. 1).

In 1874 Schiff published an article on an investigation on dogs into deaths caused by chloroform and ether. Schiff stated categorically that resuscitation was ineffective with the chest closed. He tried insufflating air and the application of electrical currents without success. Then follows the original description of cardiac massage. Simply, " One makes rhythmic movements with the hand holding the whole heart." Schiff made it clear that the response of the heart was not due to mechanical stimulation but from the filling of the coronary vessels which resulted from cardiac compression. He also suggested clamping the abdominal aorta in order to

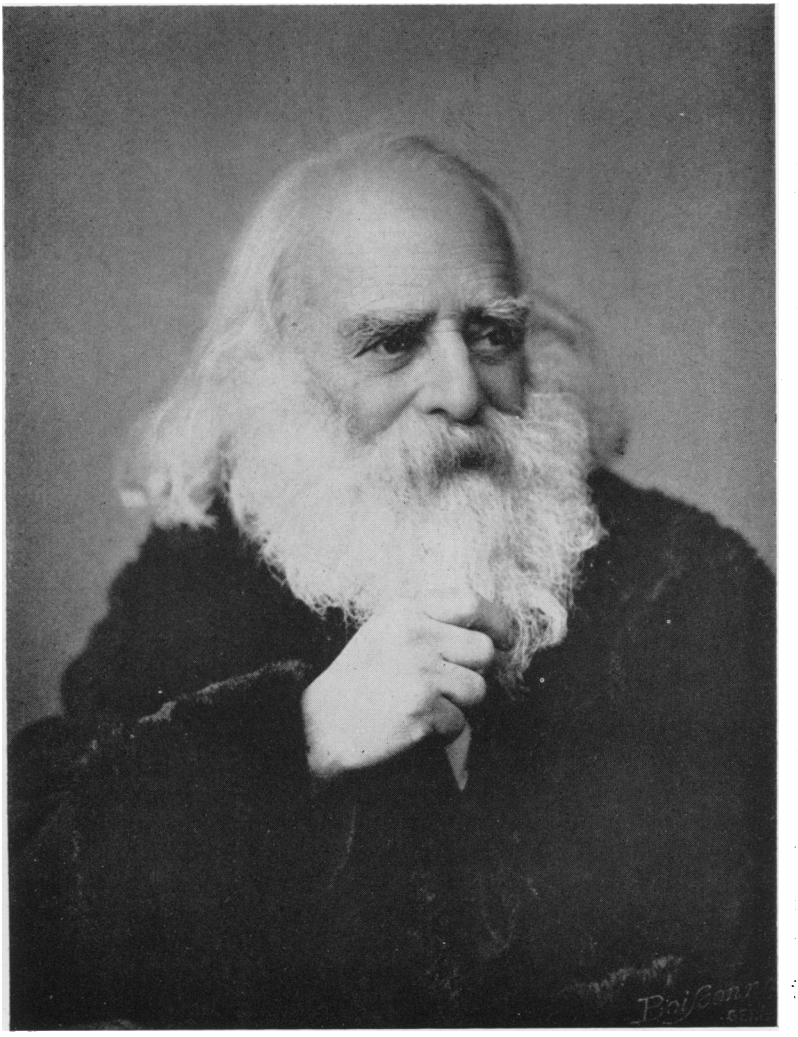

Fig. 1. Moritz Schiff, who described cardiac massage in 1874.

increase the peripheral resistance, a method which was revived by Wiggers (1940) who suggested clamping the thoracic aorta. Schiff found that the heart could be resuscitated even after 11½ minutes of arrest and described assistance of the circulation by further massage after the heart had recovered a feeble beat.

Towards the end of the nineteenth century, attempts were made to treat cardiac arrest in man by cardiac massage. The first of these was by Niehaus of Berne in 1889 although this was not reported.

Tuffier in 1898 reported an unsuccessful case in which he had performed cardiac massage through an incision in the chest on a man of 24 who developed cardiac arrest in the ward. Cardiac action and respiration were restored, but only for a short time.

The first successful case was treated by Igelsrud in 1901 but this was not reported until 1904. He resected the third and fourth ribs on the left side and massaged the heart, which beat after one minute. The patient recovered completely.

The first successful case to be reported was by Sir William Arbuthnot Lane in November, 1902 (Starling and Lane, 1902). Cardiac arrest occurred during an appendicectomy. The heart restarted after compression through the intact diaphragm.

Green reported nine successful cases in 1906 and by 1909 Von Cackovic was able to collect forty-six cases.

Pathology

Cardiac arrest occurs in two forms : as cardiac asystole or as ventricular fibrillation. Cardiac asystole is usually the result of pure anoxia of the myocardium or of vagal reflexes especially in the presence of anoxia. Ventricular fibrillation occurs when the myocardium is stimulated or when stimulation is combined with myocardial anoxia. With the chest closed it is not possible to say which of these states is present although this may be detected by means of the electrocardiograph—the only time when the electrocardiograph is of value in the management of this condition. Outside the sphere of cardiac surgery, it is most unlikely that an electrocardiograph will be attached to the patient when this emergency occurs. This is one reason why it is necessary to use an approach which will enable the heart to be seen. These conditions cannot be diagnosed through the intact pericardium because feeble fibrillation is not visible as movement under such circumstances and because coarse fibrillation cannot be differentiated from ventricular tachycardia.

The asystolic heart is relaxed, soft, and blue, the coronary arteries are flattened and the veins stand out because of their tense content of black blood. Ventricular fibrillation shows itself as a fine or coarse, irregular unco-ordinated twitching or writhing of the whole heart muscle. The heart is pale and cyanotic giving it a lavender hue.

If ventricular fibrillation responds to treatment, the fine movement first becomes coarser and more vigorous and this is accompanied by an

increase of tone which gives a sense of resistance when the heart is compressed. When this occurs the coronary arteries become distended, and pink lines of oxygenated myocardium appear alongside them. From these, pink areas extend out along their branches and gradually link up until the whole myocardium is pink.

Aetiology

Cardiac arrest may be caused by a wide variety of mechanical and electrical stimuli and by toxic doses of many drugs. The usual cause, however, is myocardial anoxia. This may be part of a generalised anoxia during anaesthesia, as a result of respiratory obstruction from failure to maintain an adequate airway or from the inhalation of vomit or other material. It may also result from inadequate ventilation or from the administration of too low a concentration of oxygen. A respiratory acidosis caused by carbon dioxide accumulation during anaesthesia increases the likelihood of the development of ventricular fibrillation.

General anaesthesia of course is not an essential prerequisite. Cardiac arrest may develop in any unconscious patient from respiratory obstruction or the inhalation of vomit and is commonly the cause of death in drowning. Another important cause of generalised anoxia which may cause cardiac arrest is sensitivity to, or reactions to, toxic doses of local anaesthetic agents such as cocaine and amethocaine. These drugs may cause convulsions of central origin which result in anoxia due to spasm of the respiratory muscles. This combined with a central effect on the heart causes cardiac asystole of a pure variety, which responds to cardiac massage very readily. More often in cardiac surgery, but less commonly in other branches, anoxia is local, affecting the heart only, as the result of impaired coronary flow. This may be due to temporary acute obstruction of the circulation or to coronary air embolism. The direct effect of chloroform on the heart is of course well known, but all anaesthetic agents given in excessive dosage will depress cardiac function.

Massive arterial haemorrhage also not uncommonly precipitates cardiac arrest. The effect cannot be immediately counteracted by intravenous infusion because cardiac contraction may have become impaired and blood may not be transferred to the left side of the heart with sufficient speed. Slow continued blood loss in a long operation without adequate replacement, combined with operative trauma, may result in a steadily falling blood pressure and ultimately, inadequate coronary flow.

Many drugs apart from anaesthetic agents may cause cardiac arrest. Two of these, which may cause ventricular fibrillation, demand further consideration. Probably the most important is adrenaline. This is frequently used in the treatment of cardiac asystole and often converts it to ventricular fibrillation. Table III shows the precipitating factors which were responsible for the onset of ventricular fibrillation in thirty-one patients during operations on the heart. Adrenaline converted cardiac asystole into ventricular fibrillation on seven occasions. This is a strong

argument against the treatment of cardiac arrest by intra-cardiac injection of adrenaline through the intact chest wall, for the state of the heart cannot be known.

TABLE III

CAUSES OF VENTRICULAR FIBRILLATION DURING 31 CARDIAC OPERATIONS

Temporary circulatory obstruction	3 cases
After the injection of adrenaline for asystole	7 cases
Intracardiac manipulation	5 cases
Sudden acute haemorrhage	3 cases
Pricking or incising of myocardium	3 cases
Hypothermia	3 cases
Injection of diodone	2 cases
Following on asystole (no adrenaline)	3 cases
Doubtful	1 case

Another substance which is not commonly recognised as capable of causing ventricular fibrillation is diodone, which causes a fall of blood pressure when it is injected intravenously. In a healthy individual there is little likelihood of a disturbance of cardiac rhythm, but in the presence of heart disease, especially disease of the coronary arteries, such an injection may diminish coronary flow to a point where cardiac arrest supervenes.

Air embolism may cause cardiac arrest in one of two ways. If the air reaches the right side of the heart, it may fill the right ventricle, which is then unable to propel blood into the lungs. If the air reaches the left side it may enter the coronary circulation giving rise to immediate ventricular fibrillation. The air can be massaged through the coronary circulation, and the removal of air from the coronary arteries can be assisted by pricking them distally and allowing the air to escape.

The part played by vagal stimulation is difficult to determine in man. In our series it was a rare cause, but in Stephenson's (1953) collected cases about 25 per cent. of the 600 in which a precipitating factor was mentioned were attributed to vagal reflexes. It seems certain that a combination of vasovagal reflexes and anoxia may result in cardiac arrest, and this is reflected in the number of cases which have occurred during the insertion or removal of an endotracheal tube, on tracheal suction or bronchoscopy, and on dissection of or traction on the vagus nerve in the chest or upper abdomen.

If any of these precipitating factors is acting, the likelihood of circulatory failure is much greater if there is co-existing disease of the coronary arteries, and in such cases it is more difficult to restore normal rhythm.

Diagnosis and treatment

If during the course of an operation, investigation, or injection under local or general anaesthesia the carotid pulse disappears the diagnosis of cardiac arrest must be made by immediate thoracotomy, and cardiac massage must be started. Once cardiac massage has begun, a circulation adequate to preserve the cerebral function can be maintained indefinitely.

The tragedies of restoration of the circulation in patients who have become decerebrate or who have developed hemiplegia or other neurological sequelae can probably be avoided so long as the circulation of oxygenated blood is maintained by cardiac massage and pulmonary ventilation. Such a tragedy is recorded here.

Case 1. A child of 8 was undergoing bronchography under general anaesthesia. All went well until the anaesthetist performed tracheal suction to remove the opaque material. The child became grey and stopped breathing. Pulmonary ventilation was being performed. A scalpel was sent for. When it arrived, discussion was still continuing as to whether a pulse was palpable or not, and whether heart sounds could be heard. Thoracotomy seemed to those present to be a very drastic step to take, and it was not carried out. Percussion of the precordium was then performed, and after a short time, the pulse could be felt. The child did not die, and is still alive to-day, six years later, completely decerebrate. Complete recovery was certain in this case if prompt thoracotomy and cardiac massage had been performed.

To advocate a policy of immediate thoracotomy means of necessity that the thorax will be opened in some patients in whom the heart is found to be contracting. It can be argued that this is not a very serious matter. In fact in the majority of such cases, it is still proper that the thorax should be opened for the heart, although beating, may be so weak that the cerebral circulation is inadequate and manual assistance of the heart's action is necessary.

In addition to cardiac massage pulmonary ventilation must also be started immediately. Adequate oxygenation of the blood is necessary, first to save the patient's life by ensuring the survival of the brain, and secondly as a preliminary to restoration of normal heart action, which must be preceded by oxygenation of the myocardium. The lungs must therefore be inflated with oxygen and it must be possible to do this under pressure since the pleura is to be opened. Intubation is not essential. We have found it possible to maintain satisfactory inflation with the aid of a face piece only, although this might not be possible in small children, or the edentulous patient.

Case 2. A girl of 10 was suffering from Fallot's tetralogy and transposition of the pulmonary veins. She was unwisely given an intravenous injection of diodone for angiocardiography under general anaesthesia in the presence of a sinus tachycardia. Three and a half minutes later, the pulse disappeared, and the E.C.G. showed ventricular fibrillation.

A left anterior thoracotomy was immediately performed and cardiac massage begun thirty seconds after the onset of fibrillation. No response was obtained and so the pericardium was opened. The presence of ventricular fibrillation was confirmed. Massage was continued and, as an electric defibrillator was not immediately available, 10ml. of 2 per cent. procaine hydrochloride was injected into the right ventricle. After two minutes of massage, good ventricular contractions appeared. The heart was then observed. Seventeen minutes later, ventricular fibrillation recurred. The beat recovered with further massage and procaine injection, and the systolic blood pressure rose to 120mm.Hg. In spite of the open pleura, good ventilation was maintained without the use of an endotracheal tube. The patient regained consciousness after two hours and made an uneventful recovery.

It seems likely that an experienced anaesthetist will have little difficulty in maintaining inflation with a face piece, but an inexperienced anaesthetist might lose valuable time in attempting to pass an endotracheal tube in difficult circumstances. If no apparatus for inflating the lungs is available, ventilation should be started by mouth to mouth insufflation.

Cardiac massage and pulmonary ventilation are the essential and immediate steps which must be taken when cardiac arrest is encountered. It is true that cases have been reported in which percussion of the precordium, pricking of the heart with a needle and the injection of various substances into it have met with success, but success cannot be guaranteed by any of these methods and they must be regarded as time wasting manoeuvres. Once cardiac massage has been instituted there is no further need for hurry and expert assistance can be sent for.

If antibiotics are given postoperatively infection need not be feared, even though no aseptic precautions are taken. None of the cases in this series developed any infective complication.

Access to the heart

Concerning the approach to the heart, when the abdomen is open, two methods are favoured : the first is massage of the heart through the intact diaphragm, between it and the sternum. This method has the disadvantage that the heart cannot be seen and therefore no accurate diagnosis can be made. The method of massage is inefficient and injections cannot be made into specific chambers. The alternative is to incise the tendinous part of the diaphragm through to the pericardium. With this method it is not possible to make a large enough incision to introduce both hands into the pericardium, which is often necessary for efficient massage. Injection, especially into the left side of the heart, is difficult and it is also very difficult to apply electrodes to the ventricles for defibrillation. A third approach, incision of the diaphragm so as to enter the left pleural cavity, has nothing to commend it.

An anterior incision through the left fourth, fifth or sixth intercostal spaces can be made in a few seconds and provides a direct approach to the heart. The incision should extend from the left edge of the sternum at least as far as the mid axilla. The intercostal muscle is incised, the deeper fibres cautiously, so as to avoid damaging the lung when the parietal pleura is incised. The space is then opened widely and the intercostal incision extended as far back as possible. The internal mammary artery should be avoided. If it is injured it will of course not bleed at this stage, but the ends must be secured before the chest is closed. The ribs are then retracted manually. The opening thus made is large enough to enable one or both hands to be introduced. The heart is under direct vision and the various chambers are easily accessible for injection of drugs. This approach has great advantages over any abdominal approach, even in cases in which the abdomen is already open. It is often stated that this advice is reasonable for the thoracic surgeon but unsuitable for the

general surgeon who is unfamiliar with thoracotomy incisions. However, the approach is simple and skill in making it can be rapidly and easily acquired in the post mortem room.

Technique of cardiac massage

The chest having been opened, the heart should be compressed for a short time through the intact pericardium. If there is no response the pericardium should be opened widely to admit both hands, as massage can be performed much more efficiently in this way. If the heart is large it is best to compress it between two hands. If it is so small that only one hand can be used counter pressure should be not with the thumb but with the thenar eminence. Similarly the palmar surfaces of the fingers and not the tips must be used for compression, otherwise the heart may be perforated. Such an event was recorded by Hurwitt and Seidenberg (1953) and by Cameron Haight and Sloan (1955). Another danger is that massage may result in some traction on the heart which may rupture at the only inferior fixed point, the junction with the inferior vena cava.

The rate of massage has also been a controversial subject. Johnson and Kirby (1949) showed by experiment on the normal dog heart that the rate of blood flow in the aorta increased as the rate of cardiac compression was raised from 30 to 120 times a minute. In this series the rate of cardiac compression was fifty times a minute. Fatigue prevents the maintenance of more rapid rates for more than a few seconds. In any case the heart must fill before it can be emptied and in massaging a large heart it is necessary to pause between each compression to allow diastolic filling to take place. This diastolic filling can be easily appreciated by the hands. By this form of massage, blood pressure sufficient to maintain a palpable radial pulse can be generated. Figure 2 shows the electromanometric blood pressure recording from the radial artery during cardiac massage in two of our cases and was published by Deuchar and Venner (1953). In another case an aortic pressure of 98/58mm. Hg. was recorded. However, an adequate cerebral blood flow can be maintained even when no peripheral pulse can be felt.

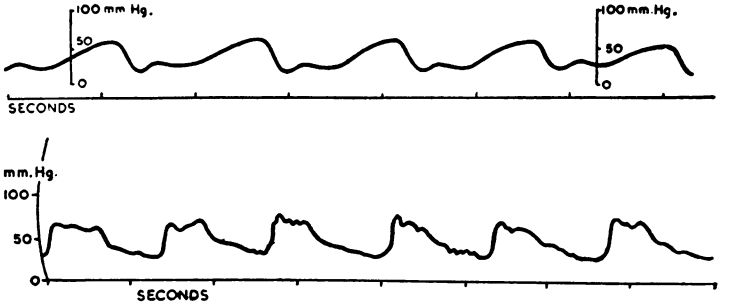

Fig. 2. Electromanometric pressure recordings made from the brachial artery during cardiac massage. The upper record shows a pressure of 55/20mm.Hg.; the lower a pressure of 70/30mm.Hg.

Before any other method of treatment is used, massage should be continued for sufficient length of time for the whole myocardium to become well oxygenated. Observation of the heart will show when this has been achieved, for the whole myocardium will become pink and it will usually regain its tone. If normal rhythm does not then return other methods of treatment should be instituted. The danger of prolonged cardiac massage is myocardial damage. However carefully massage is performed, after a time haemorrhage into the myocardium occurs (Figs. 3 and 4). The effect of extensive haemorrhage is likely to be persistent hypotension in the immediate recovery period and late cardiac failure, with possibly permanent damage. There are, therefore, good reasons for taking, as soon as possible, any steps which may shorten the period of massage.

One helpful measure in any case is to increase the coronary blood flow. This can be done by the simple method of increasing the venous return by tilting the operating table into a head down position, which often produces immediate improvement where the heart action is weak but arrest

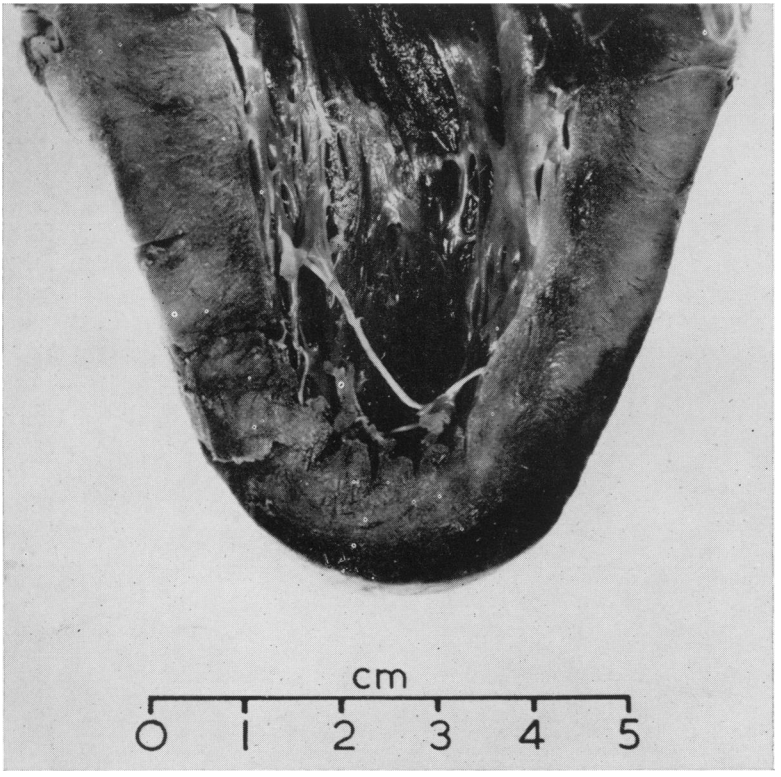

Fig. 3. The left ventricle from a patient in whom cardiac massage was continued for 100 minutes. The dark areas are sites of intramuscular and subendocardial haemorrhage.

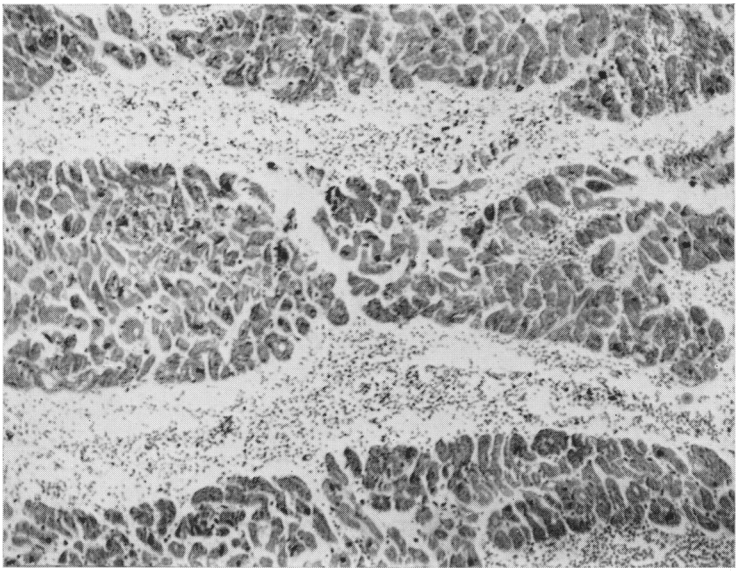

Fig. 4. Section from the myocardium seen in Fig. 3. Numerous red cells are seen in the connective tissue planes and between the muscle fibres. x 110.

has not yet occurred. It is also valuable in that it increases the cerebral circulation. Another device which has the same object is compression of the descending aorta, thereby increasing the peripheral resistance. This method, although simple in theory, is difficult to carry out in practice, for the aorta is lax, and when compressed against the vertebral column tends to slip away. Theoretically the most effective way of increasing coronary flow is by means of an aortic transfusion, preferably of oxygenated blood. Although this sometimes causes a temporary improvement in the tone of the heart we have not found it very effective in helping to restore normal rhythm and have now abandoned it, although it may be essential for counteracting haemorrhage when this is the cause.

Restoration of cardiac action

The method of restoration of normal rhythm depends on the condition of the heart after oxygenation has been achieved by massage. Massage alone is sometimes all that may be required and normal rhythm may be restored whether the condition is one of cardiac asystole or one of ventricular fibrillation.

Case 3. A woman aged 42, was to undergo bronchoscopy under local anaesthesia for recurrent haemoptysis. About 200mg. of amethocaine was used. At the conclusion of this she apparently had a tonic convulsion and became anoxic. She was pulseless and deeply cyanosed, but the pulse soon returned. She then began to have clonic convulsions. Thiopentone was injected into the external jugular vein and the patient was intubated. Shortly afterwards the pulse disappeared.

Thoracotomy was performed. The heart was in asystole, dilated and atonic. It was massaged through the intact pericardium, and after about two minutes, regular contractions were seen. The heart was kept under observation for half an hour. During this time tonic convulsions recurred and more thiopentone was given. Towards the end of the operation the patient began to breathe spontaneously, and at its conclusion she became conscious and began to talk. She made an uneventful recovery.

This case illustrates the ease with which some hearts will respond to cardiac massage alone, especially when the cause is sensitivity to the cocaine group of drugs.

If cardiac asystole persists several drugs may be used to restore normal rhythm. Adrenaline in a dose of 5 to 10ml. of 1 in 10,000 solution may be injected. If the heart is well oxygenated and obviously pink this is the drug of choice but if anoxia persists the effect of adrenaline is likely to be the onset of ventricular fibrillation. The safer drug to use in these circumstances is a 1 per cent. solution of calcium chloride in a dose of 5 to 10ml. Only if this drug fails to restore normal rhythm should adrenaline be used.

When ventricular fibrillation is present the first essential is cardiac massage to induce oxygenation of the myocardium. The fibrillation may then become coarser and more vigorous and if so, the tone of the myocardium can then be felt to be much increased so that more force is required on cardiac compression. The heart is then in a favourable state for electrical defibrillation which is dealt with later. If the fibrillation remains feeble and the myocardium soft an injection of adrenaline should be given, for this often converts the fibrillation to a coarser variety and increases myocardial tone. Sometimes adrenaline fails to work in this way and then calcium chloride may be tried. Ventricular fibrillation may also be treated by means of the injection of 50 to 200mgm. of procaine hydrochloride. However, in our own cases, it was only effective in nine out of twenty-three episodes of ventricular fibrillation in which it was used, and when it is unsuccessful it may make defibrillation by other means more difficult because it diminishes myocardial tone.

It appears from the study of human cases that procaine and adrenaline are antagonistic in their effect on the heart, and in the absence of an electrical defibrillator it may be reasonable to treat ventricular fibrillation by alternate injections of adrenaline and procaine.

Case 4. A child aged 6 with a persistent ductus arteriosus was undergoing tonsillectomy. The operation was almost completed when the anaesthetist noticed that the heart had stopped and the child was grey. The surgeon performed an immediate thoracotomy through the fifth intercostal space and massaged the heart between two fingers and the sternum. The lungs were inflated via a mask and the child became pink. A thoracic surgeon was sent for and took over the cardiac massage at five and a half minutes. The incision was enlarged so as to admit both hands. As there was no response after a minute's massage the pericardium was widely opened. The heart was blue and toneless with feeble fibrillation. After vigorous massage for another minute, tone returned to the myocardium and fibrillation became stronger. 100mg. procaine was then injected into the left ventricle. This did not reverse the fibrillation, and as a

result of it the tone began to diminish. 0·5ml. of 1 : 1,000 adrenaline was therefore injected and massage continued. Tone rapidly returned. The electric defibrillator had now arrived and was first used fifteen minutes after the onset of arrest. Three rapid shocks were given without effect, but after further massage, the defibrillator was effective and sinus rhythm was restored. The child regained full consciousness after two hours and there was no neurological abnormality. The ductus was subsequently closed without event.

This case demonstrates the importance of immediate cardiac massage, which although it was not very vigorous, was sufficient to maintain the cerebral blood flow until an expert could deal with the situation. The use of alternate procaine and adrenaline injections is also demonstrated. This case also emphasises that in spite of the danger of endocarditis or endarteritis in the presence of foci of infection, the cardiovascular lesion should be dealt with first in such cases because of the risk of cardiac arrest during anaesthesia for minor operations.

Other drugs which have some part to play in the treatment of cardiac arrest are methedrine and noradrenaline. They are not of value when cardiac arrest has occurred. Their place is in its prevention by administration to the heart which is beginning to fail. Once cardiac arrest has been successfully overcome they are seldom required because usually the heart, having recovered, beats very vigorously. As long as the blood pressure remains in the region of 80mm. Hg. systolic, it seems unnecessary to impose an added strain on the heart by forcing it to work against a higher pressure.

Electric defibrillation

The treatment of choice for ventricular fibrillation, which does not respond to cardiac massage alone, is electric defibrillation. This method was first demonstrated by Prevost and Batelli in 1899. The first case successfully treated in this way was reported by Beck, *et al.*, in 1947. It is based on the fact that a shock of 1 to 3·8 amps. renders all the muscle fibres of the heart refractory, so that contraction ceases, and resumption of activity is followed by normal rhythm. Wiggers (1940) advised serial defibrillation with weak repeated shocks because sometimes each shock renders only part of the muscle refractory, and this method seems to be more successful. The domestic supply of 110 to 240 volts will give the correct current of 1 to 2 amperes. Two large electrodes, at least 7cm. in diameter, are placed firmly one on each side of the ventricular mass, and a single shock, as short as possible, is given. If this is not effective, series of three to five short shocks are given. The heart goes into asystole, and after ten or fifteen seconds, regular beats are seen. If asystole persists, further massage often restores the beat.

In healthy hearts electric defibrillation is nearly always possible, but success cannot be expected uniformly in man. In our cardiac cases it was only effective in six out of fourteen episodes in which it was used. When it fails, it is still possible to defibrillate by the use of procaine or calcium chloride.

Although in our successful cases defibrillation was usually achieved within five to fifteen minutes, the prognosis may still be good even when the duration of fibrillation is prolonged, as the following example shows :

Case 5. A woman of 47 had mitral stenosis with some aortic stenosis. During a difficult mitral valvotomy a moderate haemorrhage occurred and ventricular fibrillation developed. Massage was begun and 10ml. one per cent. procaine followed by 5ml. one per cent. calcium chloride was injected without effect. The pressure gradient across the aortic valve was measured and was found to be 25mm.Hg. An aortic valvotomy was therefore rapidly performed. There was an immediate improvement in the heart's action and a blood pressure

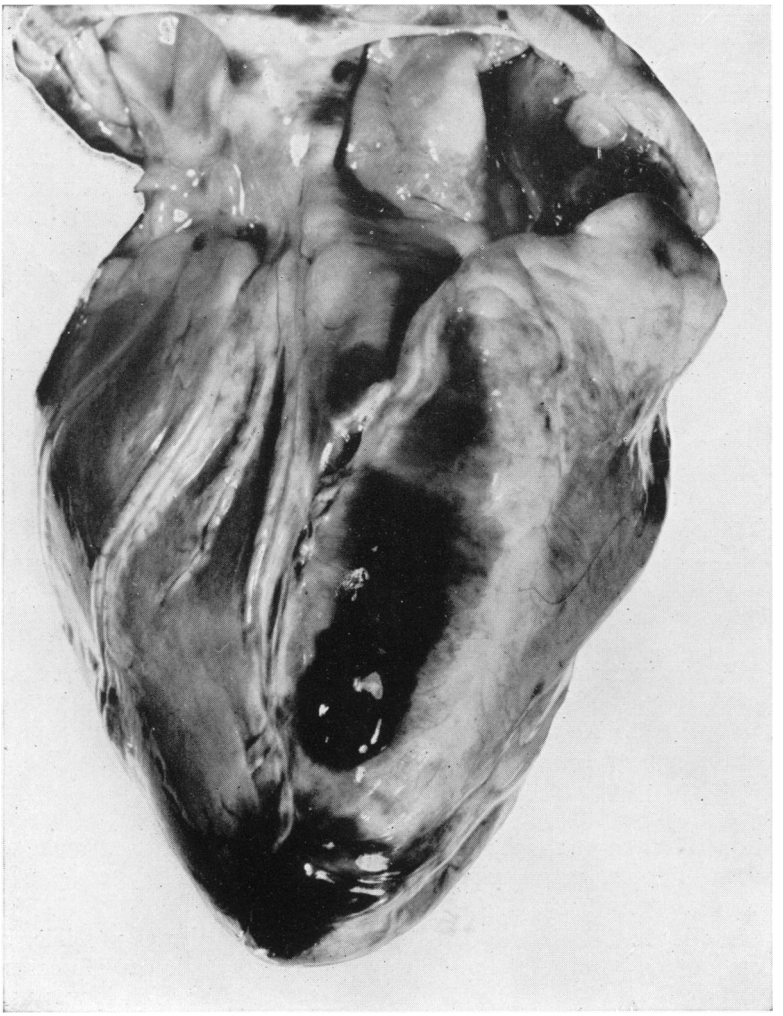

Fig. 5. A burn on the posterior surface of the right ventricle caused by inadequate contact of a metal electrode.

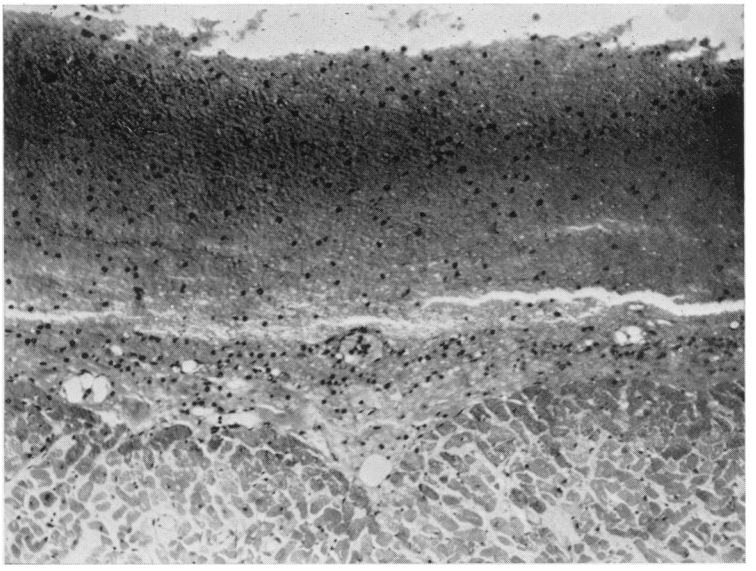

Fig. 6. Section through the burnt area shown in Fig. 5. There is coagulation necrosis of the pericardium, which has extended to involve the superficial muscle fibres. x 110.

of 140/100 was recorded in the aorta. After a few minutes ventricular fibrillation recurred. There were nine episodes of ventricular fibrillation altogether lasting 22, 4, 15, 4, 5, 12, 1, 1 and 18 minutes. Each time after normal rhythm had resumed, a tachycardia would develop and proceed to fibrillation. Massage and repeated doses of procaine and calcium chloride were used to restore the beat. During one episode electric defibrillation was attempted three times but it was ineffective. The heart action finally became stable after 123 minutes, during which ventricular fibrillation was present for eighty-two minutes. There had been pauses of only fractions of a minute during which the circulation was not maintained. One hour later the patient was breathing spontaneously and talking. She made an uneventful recovery.

This case also shows that if there is an obstruction in the circulation such as a valve stenosis, it must be relieved before cardiac arrest can be overcome.

There are two dangers of electric defibrillation. The first is the production of an electric burn of the heart (Figs. 5 and 6). This may result from inadequate contact of the edge of an electrode. The accident can be avoided by using large electrodes covered with thick cloth which is moistened with saline, ensuring close contact.

The second danger is heat production sufficient to cause bubble formation in the coronary vessels. This is probably due to an excessive duration of each shock, which should be as short as possible, and not longer than half a second.

In thirty of our cases of ventricular fibrillation occurring during cardiac operations, defibrillation was achieved in twenty, and nine recovered

completely (Milstein and Brock, 1954). Nearly all the deaths were due to recurrent fibrillation. Only one patient had residual neurological damage, and this was very early in the series.

Electrocardiographic changes

The electrocardiographic changes associated with cardiac arrest are of considerable interest although their significance is not fully understood.

There are two types of abnormality, although in some cases both of these may be seen. The initial changes, which may occur in any type, consist of persistent S-T segment depression, and partial bundle branch block (Fig. 7A). These phenomena are not rare during anaesthesia and are common during cardiac operations but usually they do not lead to any more serious disturbance.

The first serious type of abnormality is increasing myocardial depression. This is the result of gross anoxia. The bundle branch block and S-T depression increase, and then a sinus or ventricular bradycardia (Fig. 7B) is seen, which is likely to be followed by cardiac asystole. In the case illustrated, the asystole was followed by ventricular fibrillation, at first coarse, and then feeble (Figs. 7, D and E). Regular rhythm was restored after five minutes' massage and two hours later, the electrocardiogram was normal (Fig. 7G).

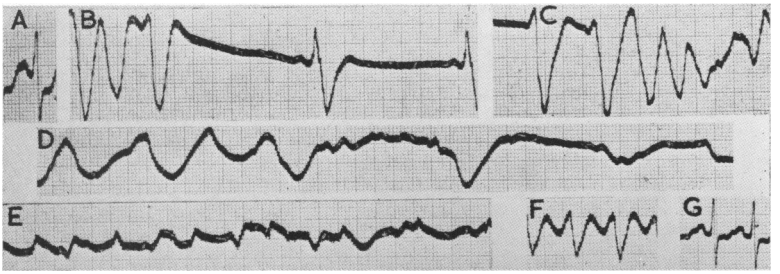

Fig. 7. Electrocardiograms showing increasing myocardial depression. A : S-T depression and partial bundle branch block. B : Lengthening of QRS and sinus bradycardia. C : Cardiac asystole followed by ventricular fibrillation. D : Coarse ventricular fibrillation. E : Feeble ventricular fibrillation. F : After defibrillation. G : Normal ventricular complexes two hours later. Lead II.

The second type of electrocardiographic change is a picture of increasing irritability, as shown by ventricular extrasystoles and paroxysmal ventricular tachycardia (Fig. 8A). This type is more likely to be followed by ventricular fibrillation (Fig. 8B).

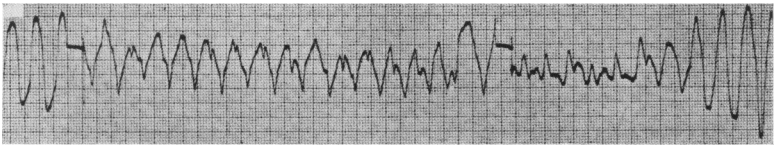

Fig. 8A. Ventricular tachycardia and multiple ventricular extrasystoles. Lead II.

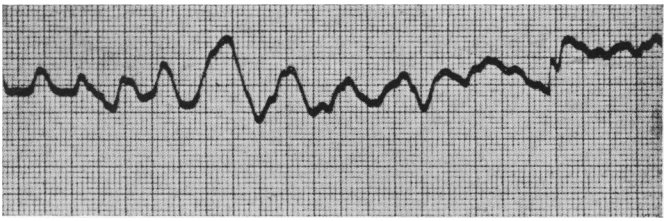

Fig. 8B. Five seconds later. Ventricular fibrillation. Lead II.

If ventricular fibrillation is present, when the tonus of the heart muscle has increased as the result of massage, the rate of fibrillation is seen to diminish and some recognisable ventricular complexes may appear (Fig. 9). Observation of the heart then shows that fibrillation and co-ordinated contractions are both occurring. This is a favourable sign and indicates that the time has arrived when defibrillation is likely to be successful.

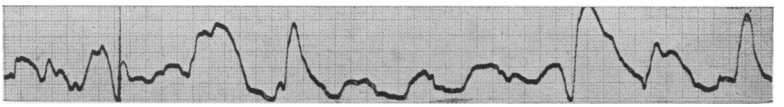

Fig. 9. Coarse ventricular fibrillation with occasional coordinated ventricular complexes. Lead II.

After restoration of heart action an abnormal ventricular rhythm may persist for a short time with atrio-ventricular dissociation, bundle branch block, and severe S-T deviation (Fig. 10). In another case a complete left bundle branch block persisted for five days (Fig. 11).

It is not easy to explain these changes. McMillan, Cockett and Styles (1952) suggest that they are due to myocardial infarction, and this may be so, but it is difficult to see why localised myocardial damage should occur, except from burning by an electrode. Diffuse interstitial myocardial haemorrhage certainly occurs as a result of cardiac massage, but the changes are not localised. Bundle branch block may represent anoxia of the bundle itself. It is probable that this tissue is more susceptible to anoxia than the rest of the heart, and that recovery from the injury may be delayed.

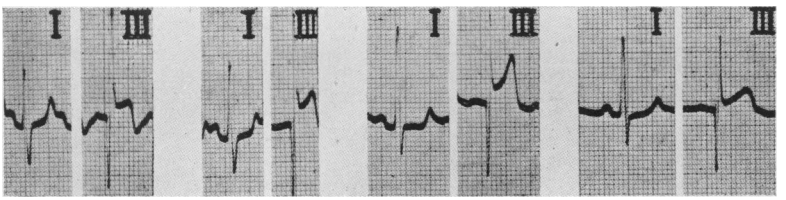

Fig. 10. Leads I and III on the first, second, third and tenth days following cardiac asystole and ventricular fibrillation which responded to treatment. There is gross elevation of S-T III with a return towards normal on the tenth day.

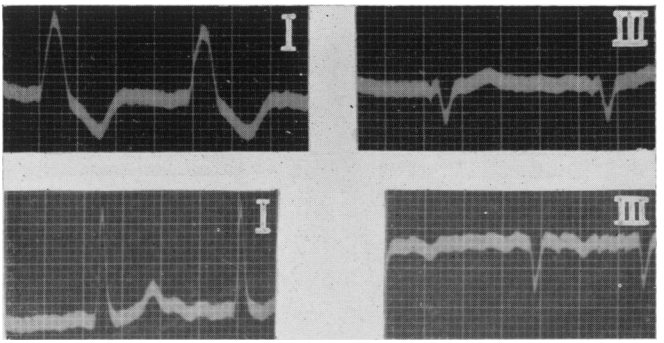

Fig. 11. Complete left bundle branch block following recovery from ventricular fibrillation. Upper tracings immediately after defibrillation; lower tracings five days later show that the block has disappeared. Atrial fibrillation is also present.

The conclusions which have been reached in this paper are based on the study of over fifty cases of cardiac arrest which have occurred mainly during cardiac surgical operations. These conclusions, however, are applicable not merely to cases of cardiac arrest occurring with the thorax open but to the management of this condition under any circumstances.

These cases illustrate the possibilities of a high recovery rate if action is prompt, whatever the circumstances under which the emergency occurs. In John Hunter's words, " the suspension of activity will more frequently last forever, unless the power of life is restored to action by some applications of art."

SUMMARY

(1) In cardiac arrest people die because the brain is deprived of oxygenated blood. If they are to survive when their heart stops, cardiac massage and pulmonary ventilation must be instituted at once.

(2) The indication for cardiac massage is disappearance of the carotid pulse.

(3) A left anterior thoracotomy is the best approach to the heart for cardiac massage, even when the abdomen is already open.

(4) The technique of cardiac massage and resuscitation of the heart is described in detail, with illustrative cases. Infection is not a problem, even if no aseptic precautions are taken, so long as antibiotics are given postoperatively.

(5) Normal rhythm can almost always be restored to the normal heart under these circumstances. If cardiac massage is performed with promptitude there is no reason why neurological damage should ever occur, since once massage has started an adequate cerebral circulation can be maintained indefinitely.

I would like to express my gratitude to Sir Russell Brock, under whose care most of these patients were admitted, for his encouragement and advice in the preparation of this paper.

REFERENCES

BAILEY, H. (1941) *Brit. med. J.* **2,** 84.
BECK, C. S., PRITCHARD, W. H. and FEIL, H. S. (1947) *J. Amer. med. Ass.* **135,** 985.
BEECHER, H. K., and TODD, D. P. (1954) *Ann. Surg.* **140,** 1.
BONICA, J. J. (1952) *Curr. Res. Anesth.* **31,** 1.
BOST, T. C. (1923) *Surg. Gynec. Obstet.* **36,** 276.
COLE, W. (1953) In discussion of paper by Snyder *et al.*, see below.
COOLEY, D. A. (1950) *Ann. Surg.* **132,** 930.
DALE, W. A. (1952) *Ann. Surg.* **135,** 376.
DEUCHAR, D. C., and VENNER, A. (1953) *Brit. med. J.* **2,** 134.
GREEN, T. A. (1906) *Lancet*, **2,** 1708.
HAIGHT, C., and SLOAN, H. (1955) *Ann. Surg.* **141,** 240.
HUNTER, J. (1776) *Phil. Trans.* **66,** 412.
HURWITT, E. S., and SEIDENBERG, B. (1953) *Ann. Surg.* **137,** 115.
IGELSRUD, K. (1901) Reported by W. W. Keen in *Ther. Gaz.* (1904) **28,** 217.
JOHNSON, J., and KIRBY, C. K. (1949) *Surgery*, **26,** 472.
——————————— (1951) *Ann. Surg.* **134,** 672.
LAHEY, F. H. (1953) In discussion of paper by Stephenson *et al.*, see below.
LEE, W. E., and DOWNS, T. M. (1924) *Ann. Surg.* **80,** 555.
LEWIS, K. M., and STANLEY-BROWN, E. G. (1955) *Surg. Gynec. Obstet.* **100,** 397.
MCMILLAN, I. K. R., COCKETT, F. B., and STYLES, P. (1952) *Thorax*, **7,** 205.
MILLER, F. A., BROWN, E. B., BUCKLEY, J. J., VAN BERGEN, F. H., and VARCO, R. L. (1952) *Surgery*, **32,** 171.
MILSTEIN, B. B., and BROCK, R. C. (1954) *Guy's Hosp. Rep.* **103,** 213.
NEGOVSKI, V. A. (1945) *Amer. Rev. Sov. Med.* **2,** 303.
NICHOLSON, J. C. (1942) *Brit. med. J.* **1,** 385.
PREVOST, J. L., and BATELLI, F. (1899) *C. R. Acad. Sci., Paris*, **129,** 1267.
REGISTRAR-GENERAL (1955) Personal communication.
REP. MINIST. HLTH, Lond. (1954) pp. 172, 247.
SCHIFF, M. (1874) *Recueil des Mémoires physiologiques, Lausanne.* **3.**
STARLING, E. A., and LANE, W. A. (1902) *Lancet*, **2,** 1397.
STEPHENSON, H. E., REID, L. C., and HINTON, J. W. (1953) *Ann. Surg.* **137,** 731.
SNYDER, W. H., SNYDER, M. H., and CHAFFIN, L. (1953) *Arch. Surg. (Chicago)*, **66,** 714.
TUFFIER, T., and HALLION, L. (1898) *Bull. Soc. Chir. Paris*, **24,** 937.
VON CACKOVIC, M. (1909) *Arch. klin. Chir.* **88,** 917.
WHITE, C. S. (1909) *Surg. Gynec. Obstet.* **9,** 388.
WIGGERS, C. J. (1940) *Amer. Heart J.* **20,** 413.

OVERSEAS VISITORS TO THE COLLEGE

THE COLLEGE HAS recently been honoured by visits from Dr. Amin El Sayed, Minister of Health, Sudan, and Dr. Ahmed Ali Zaki, O.B.E., Director of Medical Services, Sudan, who were on an official visit to this country; Professor George W. Corner, M.D., F.R.S., Historian of the Rockefeller Institute of Medical Research, New York, and formerly Director of the Department of Embryology, Carnegie Institution of Washington at Baltimore; and Professor Charles H. Best, C.B.E., M.D., F.R.S., Professor of Physiology, University of Toronto.

DONATIONS

£52,500 The Simon Marks Charitable Trust for the "Simon Marks Research Fund." (See Page 114).

ANATOMICAL MUSEUM

THE special display for the month of August consists of a selection of John Hunter's osteological specimens.