CARDIAC ARREST

By ERIC M. NANSON, F.R.C.S.

Lecturer in Surgery, University of Bristol

Sudden death in the operating theatre is a distressing event which is fortunately infrequent. In most large hospitals two such cases a year may be expected to occur, and throughout England and Wales the Registrar General's reports show that there were 728 cases of anaesthetic deaths in 1947. In the Bristol Royal Infirmary there have been 17 such deaths in the last five years. Death is due to cessation of the circulation, but cessation of an effective heart beat need not necessarily mean irrevocable death, provided prompt, vigorous and purposeful action is taken. This question has been the subject of numerous articles in the American literature in the last few years, and these have been well reviewed in a recent article by Dale.¹ In this country, Bailey^{2,3} has been a strong advocate of prompt action. The purpose of this communication is to discuss the condition and to outline the necessary line of treatment which has now outmoded that recommended by Bailey.

Sudden cardio-respiratory arrest occurs most commonly during anaesthesia and operations, but it can occur during cardiac catheterisation and angiocardiology. It is also the mechanism of death in electrocution, drowning and inhalation of noxious gases.⁴

The condition to be discussed is really cardio-respiratory arrest because the two vital functions of respiration and circulation are so closely interwoven. Sometimes respiration may cease first, sometimes the circulation, but neither can continue without the other for any length of time. This concept is most important from the point of view of treatment.

There are many causes of sudden cardio-respiratory arrest, but the most common in order of frequency are:

- (1) Anoxia.
- (2) Haemorrhage.
- (3) Overdose of anaesthetic agent.
- (4) Drug idiosyncrasy.
- (5) Toxaemia
- (6) ? Vaso-vagal reflex.

(1) *Anoxia* is a most important cause. It is seen under many conditions such as obstructed air way, inhaled vomitus, spill over of secretions from bronchiectasis, and in particular in the surgery of cyanotic heart disease. It produces slowing of the heart and diminution of the force of contraction. This is an urgent danger signal which calls for immediate correction of the anoxia by temporary cessation of the operation, release of retractors, and giving free play to the anaesthetist to inflate the lungs with 100 per cent. oxygen. If the anoxia is uncorrected the heart will stop in 'standstill.'

(2) *Haemorrhage* is another potent factor, especially if torrential and catastrophic, the mechanism again being that of tissue anoxia due to diminished blood volume and lack of oxygen-carrying capacity. The treatment is, of course, rapid blood replacement, preferably by intra-arterial transfusion into the aorta if the chest is open.

(3) *Overdose of anaesthetic.* Nowadays the commonest offender is probably thiopentone, with cyclopropane, trilene and nitrous oxide in second place.

(4) *Drug idiosyncrasy.* Examples of this are cocaine and its related compounds, used in local anaesthesia, and some of the compounds of iodine used in radiography as opacifying media.

(5) *Toxaemia.* It is well known that the vital centres and the myocardium are severely and adversely affected by a profound toxaemia such as occurs in a diffuse peritonitis. A patient in such a condition is liable to sudden death during operation.

(6) *Vaso-vagal reflex.* This is a term used as a convenient cloak for ignorance to cover those cases of cardio-respiratory arrest in which no sound pathological explanation is to be found at autopsy. There is no sound experimental evidence that reflex vagal stimulation alone will produce cardiac arrest, but there is evidence that hypoxia plus vagal stimulation may produce reflex slowing of the heart rate.^{6,7}

Diagnosis

The immediate diagnosis of sudden cardio-respiratory arrest usually falls to the anaesthetist unless the heart happens to be under direct view. The patient becomes suddenly flaccid and unresponsive; there is rapid blanching or mottling of the skin; there is absolute loss of pulse and blood pressure and dilatation of the pupils. The anaesthetist should at once ask the surgeon to confirm the diagnosis by palpating the nearest large artery, *e.g.* the aorta if he happens to be operating in the abdomen, or the femoral, carotid, or axillary arteries if they are the vessels in the operative field. The use of the ECG, or even the stethoscope, is a waste of precious time and entirely unreliable.⁵

Treatment

Once the diagnosis is made, and this should take but a few seconds, treatment must be immediate. Two objectives are paramount:

- (1) To maintain adequate oxygenation of the blood.
- (2) To maintain adequate circulation of this oxygenated blood.

The first of these objectives devolves upon the anaesthetist, the second upon the surgeon.

The anaesthetist must give 100 per cent. oxygen by means of positive pressure from an anaesthetic machine, giving artificial respiration by manual compression of the rebreathing bag. Should an anaesthetic machine not be available, then mouth-to-mouth insufflation is the next best procedure. The oxygen may be delivered to the patient by means of an intratracheal tube, if this happens to be already in position; if it is not then a well-fitting face mask is satisfactory as an initial procedure to be replaced later, when opportunity presents, by an intratracheal tube. It is wrong to waste time looking for an intratracheal tube and a laryngoscope as an initial procedure when a face mask can be 'clapped on' and oxygen delivery started immediately thereby. Of course, if any excess secretions or inhaled vomitus are present in the tracheo-bronchial tree, these must be sucked out, or if any mechanical obstruction exists this must be cleared before oxygenation can be begun.

The surgeon must maintain adequate circulation by beginning cardiac massage at once. The use of the time-honoured term 'cardiac massage' is really a misnomer—it should be called cardiac squeezing or cardiac pumping, looking on the heart as a Higginson's syringe. Indeed, it is *the* most perfect example of such a syringe.

The only satisfactory way of performing adequate cardiac pumping is to grasp the heart in the hand or between the two hands, and to squeeze

FIG. 1.—Site of anterior thoracotomy incision in 4th intercostal space.

it vigorously at the rate of 40 to 60 per minute. This can be done only if both the chest and the pericardium are opened. A left anterior intercostal incision through either the third or fourth intercostal space is quickly made with a scalpel, cutting the adjacent costal cartilages (Fig. 1). The pericardium is freely incised, avoiding the phrenic nerve, and the heart is grasped firmly in the whole hand and squeezed rhythmically and purposefully (Figs. 2 and 3). If this is being done properly the anaesthetist should be able to feel a carotid pulse and it should be possible to maintain a blood pressure of 60 to 70 mm. Hg.^{8, 9, 10}

Once these conditions have been established, *viz.* oxygenation by artificial respiration with 100 per cent. oxygen and artificial circulation by manual squeezing of the ventricles, the immediate crisis is over and the principal participants, the anaesthetist and the surgeon can, so to speak, sit back and consider the next move. The patient is now artificially alive because the two vital circulations (the cerebral and the coronary) are being adequately maintained. Authorities differ as to the length of time available to establish these conditions, but they generally agree that it is between 3½ and 5 minutes from the time of cessation of

FIG. 2A.—Cardiac squeezing—using one hand diastole.

FIG. 2B.—Systole.

FIG. 3A.—Cardiac squeezing—using two hands diastole.

FIG. 3B.—Systole.

FIG. 4.—Injecting 4 cc. calcium chloride 10 per cent. solution into the cavity of the left ventricle.

the heart beat, if the integrity of the brain cells is to be maintained.^{4, 5, 10, 11} It is, of course, possible to revive a patient whose heart has stopped for

longer than 5 minutes, but irrevocable cerebral damage will occur so that a decerebrate animal results—the body is alive but the mind is dead. Furthermore, it is obviously necessary to re-establish the coronary circulation if the cardiac muscle is to be expected to resume its normal rhythmic contractions. Once begun, cardiac massage will maintain the circulation indefinitely and Beck¹² has recorded a recovery after 70 minutes of asystole.

With the heart under direct vision, the type of cardiac arrest can be seen. It is either

- (a) Cardiac standstill or
- (b) Ventricular fibrillation.

(a) *Cardiac Standstill.* The heart is flaccid in diastole. Frequently this condition will respond to two or three minutes of cardiac massage, when a rhythmic beat will return. Should this not occur, 3 to 4 cc. of 1/10,000 adrenalin are injected into the cavity of the left ventricle, from whence, by massage, it is carried quickly to the coronary circulation, where it can act on the myocardium.⁹ Massage is stopped only long enough to make the injection and then immediately resumed. If this fails, Blalock and Kay¹³ have shown the great value of an intra-ventricular injection of 3 to 4 cc. of 10 per cent. calcium chloride solution, again using the left ventricle (Fig. 4). This drug has, in many instances, brought back a forceful rhythmic beat. It is also extremely useful when the heart action is becoming slow and weak, as it may do in operating for congenital cyanotic heart disease; such a flagging heart will revive in a most dramatic manner. It is stressed that the ionisable soluble salt of calcium, *viz.* calcium

FIG. 5.—Defibrillator circuit.

FIG. 6.—Cardiac emergency set. 1. Foot switch. 2. Defibrillator. 3. Sterile pack containing gloves, scalpel, syringes, needles, calcium chloride 10 per cent., normal saline. 4. To mains. 5. Electrodes. 6. Procaine 2 per cent. 7. Adrenalin 1:1000.

FIG. 7A.—Electrodes covered with gauze and soaked in normal saline solution.

FIG. 7B.—Electrodes applied along the long axis of the heart. Cardiac defibrillation.

chloride, must be used. Calcium gluconate is useless.

(b) *Ventricular Fibrillation.* The heart is 'writhing' or 'shimmering' in diastole. Massage again must be instituted at once and maintained, but before normal rhythm can be restored the ventricles must be defibrillated. Two methods of doing this are available:

(i) By electric shock.

(ii) By procaine.

There is a considerable amount of literature on the subject of electric defibrillation^{9,10,12,14,15,16,17,18,19}, and some complicated pieces of apparatus have been designed for the purpose. But a very simple defibrillator can be made up in any surgical department at very little cost. The circuit is shown in Fig. 5 and the complete apparatus is shown in Fig. 6. An alternating 60-cycles current of 2 to 5 amperes at 110 to 130 volts is delivered by two plate electrodes about 1 in. in diameter, and covered with cloth or felt well saturated with saline. They are placed along the long axis of the heart (Fig. 7). Two or three short, sharp shocks are given. These will cause the whole patient to go into momentary violent tetanic spasm, each shock lasting about half a second. This, with good fortune, will stop the fibrillation and leave the heart flaccid in diastole. Massage must then be immediately resumed, and normal rhythm may result. If this does not occur, then the treatment of cardiac standstill already detailed must be instituted, although adrenalin should be used with caution after defibrillation as it may reprecipitate it. The rationale behind electric defibrillation is that the strong current will cause all the muscle fibres of the heart to go into systole at once. They will therefore all pass into refractory period together and so cardiac standstill will result.

Many writers^{16, 17, 20, 21, 22, 23} recommend the use of procaine for defibrillation. If an electric

defibrillator is not available, then this is a useful measure. Procaine 2 per cent. (3 to 4 cc.) is injected into the cavity of the left ventricle, whence massage will convey it to the coronary circulation and so to the fibrillating muscle fibres which it will paralyze. The disadvantage is that the resulting cardiac standstill may be refractory to treatment until the procaine effect has worn off, so that massage must be continued for a long time.

Once a normal rhythmic beat of good force has been restored, the pericardium may be loosely closed with a few interrupted stitches, the chest drained with an under-water seal, and the intercostal incision closed with some pericostal catgut stitches, followed by muscle and skin closure.

The following case of successful cardiac resuscitation after 22 minutes' arrest is reported as illustration of the effectiveness of the measures advocated above:

Brian O—, a male aged five years, had a left anterior thoracotomy performed for tetralogy of Fallot on July 13, 1952. The pericardium was opened and a stenosis of the pulmonary artery, 1 cm. distal to the valve, was found. It was decided to make a direct attack upon the stenosis. Accordingly an incision was made in the infundibular area of the right ventricle and a sound passed up through the stenosis. The smallest Brock valvulotome was then introduced through the stenosis, followed by a dilator. There was moderate blood loss, about 100 cc., and the cardiac beat faltered and stopped. The incision into the right ventricle was rapidly closed with two silk sutures and cardiac 'squeezing' begun at once at a rate of between 60 and 80 to the minute (timed by watch). Dr. Woolmer, the anaesthetist, gave 100 per cent. oxygen by an intratracheal tube. He reported that a blood pressure of 70 to 80 mm. of mercury systolic was maintained by the squeezing. After five minutes no regular beat had returned, therefore 3 cc. of

Backed by Franklin's outstanding experience of over 80 years in the Surgical Rubber Industry, we now have to our credit **over 40 years of continuous production of Surgeons' Gloves**

J. G. FRANKLIN AND SONS LIMITED

Head Office: Birbeck Rubber Works
DALSTON LONDON, E.8

1/10,000 adrenalin was injected direct into the left ventricle and squeezing resumed. The heart was then observed to be in ventricular fibrillation. Accordingly, serial defibrillation by electric shock at 110 volts and 1.5 amperes was used. This was not successful and squeezing was resumed. Further defibrillation using 130 volts and 2 amperes was used and this was successful. Cardiac standstill resulted. Cardiac squeezing was resumed and a weak rhythmic beat returned, but when squeezing was stopped this beat was ineffective, therefore 4 cc. of 10 per cent. calcium chloride were injected into the left ventricle and squeezing was resumed. A forcible regular beat now resulted and was satisfactorily maintained. Twenty-two minutes of cardiac arrest had been overcome. The pericardium was closed, the De Pezzer intercostal catheter was inserted into the 5th intercostal space and the chest closed. The patient recovered consciousness in two hours and moved all his limbs. Next day he was reasonably well with no evidence of any cerebral damage. He made a satisfactory recovery and was discharged three weeks after operation.

Fortunately, cardiac arrest is not a common condition, but when it does occur it is sudden, often unexpected, and calls for prompt diagnosis and immediate planned action. The treatment is simple and straightforward to carry out, and requires no great technical dexterity or skill. The greatest over-riding difficulty is for the anaesthetist and the surgeon to overcome their natural inertia and to act promptly and boldly. The anaesthetist must make the diagnosis swiftly and the surgeon open the chest fearlessly, even though he has never done so before. After all, there is nothing to lose and everything to gain. The patient is dead if nothing is done, and whatever is done cannot make him more dead than he is; whereas, with fortune favouring the brave, especially in the case of a young patient, success may be expected and the patient revived with no greater disability than a chest scar which he did not expect.

Summary

(1) The subject of cardiac arrest has been discussed.

(2) Some of the causes have been briefly reviewed.

(3) The objectives of treatment have been described. The single most important factor is to establish an artificial circulation with oxygenated blood.

(4) Cardiac massage through a thoracotomy incision and an open pericardium is the basis of treatment.

(5) Cardiac standstill should be treated by massage, plus adrenalin or calcium chloride.

(6) Ventricular fibrillation should be treated by electric shock or procaine.

REFERENCES

- DALE W. A. (1952), 'Cardiac Arrest,' *Ann. Surg.*, **135**, 376-393.
- BAILEY, H. (1941), 'Cardiac Massage for Impending Death Under Anaesthesia,' *Brit. Med. J.*, **ii**, 84.
- BAILEY, H. (1947), 'Impending Death under Anaesthesia,' *Lancet*, **i**, 5.
- WIGGIN, S. C., SAUNDERS, P., and SMALL, G. A. (1949), 'Cardiac Resuscitation,' *New England J. Med.*, **241**, 370-378.
- JOHNSON, J., and KIRBY, C. K. (1949), 'Cardiac Resuscitation,' *Surg. Clin. N. Am.*, **29**, 1745.
- SLOAN, H. E. (1950), 'The Vagus Nerve In Cardiac Arrest,' *Surg. Gynaec. & Obst.*, **91**, 257.
- YOUNG, W. G., SEALY, W. C., HARRIS, J., and BOTWIN, A. (1951), 'The Effects of Hypercapnia and Hypoxia on the Response of the Heart to Vagal Stimulation,' *Ibid.*, **93**, 51.
- JOHNSON, J., and KIRBY, C. K. (1949), 'An Experimental Study of Cardiac Massage,' *Surg.*, **26**, 472.
- KAY, J. H. (1951), 'The Treatment of Cardiac Arrest,' *Surg. Gynaec. & Obst.*, **93**, 682-690.
- WIGGERS, C. J. (1936), 'Cardiac Massage followed by Countershock in the Revival of Mammalian Ventricles from Fibrillation due to Coronary Occlusion,' *Am. J. Physiol.*, **116**, 161-162.
- WEINBERGER, L. M., GIBBON, M. H., and GIBBON, J. H. (1940), 'Temporary Arrest of the Circulation to the Central Nervous System: I. Physiological Effects,' *Arch. Neur. Psychiat. Clin.*, **43**, 615-634.
- BECK, C. S., PRITCHARD, W. H., and FEIL, H. S. (1947), 'Ventricular Fibrillation of Long Duration Abolished by Electric Shock,' *J.A.M.A.*, **135**, 985-986.
- KAY, J. H., and BLALOCK, A. (1951), 'The Use of Calcium Chloride in the Treatment of Cardiac Arrest in Patients,' *Surg. Gynaec. & Obst.*, **93**, 97-102.
- JOHNSON, J., KIRBY, C. K., and DRIPPS, R. D. (1951), 'Defibrillation of Ventricles by Electric Shock with Complete Recovery,' *Ann. Surg.*, **134**, 116.
- JOHNSON, J., and KIRBY, C. K. (1951), 'The Surgical Treatment of Ventricular Fibrillation,' *Ibid.*, **134**, 672-682.
- BECK, C. S., and MAUTZ, F. R. (1937), 'The Control of the Heart Beat by the Surgeon with Special Reference to Ventricular Fibrillation occurring during Operation,' *Ibid.*, **106**, 525-537.
- BECK, C. S. (1941), 'Resuscitation for Cardiac Standstill and Ventricular Fibrillation during Operation,' *Am. J. Surg.*, **54**, 273-279.
- BECK, C. S., and RAND, H. J. (1949), 'Cardiac Arrest during Anaesthesia and Surgery,' *J.A.M.A.*, **141**, 1230-1233.
- WIGGERS, C. J. (1940), 'Physiological Basis for Cardiac Resuscitation from Ventricular Fibrillation,' *Am. Heart J.*, **20**, 413-422.
- LAHEY, F. H., and RUZICKA, E. R. (1950), 'Experiences with Cardiac Arrest,' *Surg. Gynaec. & Obst.*, **90**, 108-118.
- BURNSTEIN, C. L., MARANGONI, B. A., DE GRAFF, A. C., and ROVENSTINE, E. A. (1940), 'Laboratory Studies in the Prophylaxis and Treatment of Ventricular Fibrillation,' *Anaesthesiology*, **1**, 167-186.
- LAMPSON, S. R., SCHAEFFER, W. C., and LINCOLN, J. R. (1948), 'Acute Circulatory Arrest from Ventricular Fibrillation for 27 Minutes with Complete Recovery,' *J.A.M.A.*, **137**, 1575.
- RUZICKA, E. B., and NICHOLSON, M. J. (1947), 'Cardiac Arrest under Anaesthesia,' *Ibid.*, **135**, 622.
- JOSEPH, S. I., HELRICH, M., KAYDEN, H. J., ORKIN, L. R., and RAVENSTINE, E. A. (1951), 'Procaine Amide for Prophylaxis and Therapy of Cardiac Arrhythmias occurring during Thoracic Surgery,' *Surg. Gynaec. & Obst.*, **93**, 97.