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CARDIAC ARREST AND VENTRICULAR FIBRILLATION*

A METHOD OF TREATMENT BY ELECTRICAL SHOCK

BY

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Cardiac arrest during operation is a subject of importance to all surgeons. During intra-thoracic operations cardiac arrest and ventricular fibrillation can be differentiated by direct observation of the heart.

Ventricular fibrillation is an incoordinated type of contraction which produces no useful beats. The typical rippling movements of the ventricular muscle are unmistakable when seen or felt with the hand directly on the heart. The treatment of a heart that has stopped is cardiac massage and adequate oxygenation. The treatment of ventricular fibrillation which we recommend is, first, massage to maintain the oxygen supply to the heart through the coronary arteries, followed rapidly by electrical shocking to restore normal rhythm.

In abdominal operations, however, or during operations elsewhere in the body, once the pulse becomes undetectable most surgeons waste no time in performing a quick midline upper abdominal incision and massaging the heart through the diaphragm. At the same time adrenaline is given either intravenously, intramuscularly, or more usually directly into the ventricle. The latter is a most dangerous procedure, as it is now known that one of the effects of adrenaline on a heart which is already anoxic may be the production of ventricular fibrillation. Until recent years ventricular fibrillation was almost always fatal.

The purpose of this paper is twofold : (1) to emphasize once again the dangerous effects of adrenaline ; (2) to describe the experiments leading up to the development of a satisfactory electrical instrument for defibrillating the heart.

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VENTRICULAR FIBRILLATION

The treatment of ventricular fibrillation by electrical shock therapy is not new and was first tried experimentally in 1899 (Prevost and Battelli). In recent years this method has been extensively investigated in the United States and France (Kouwenhoven and Kay, 1951 ; Johnson and Kirby, 1951 ; Santy and Marion, 1950).

The treatment of ventricular fibrillation by electrical shock gives encouraging results in the experimental animal, and may be a useful adjunct in the operating theatre during cardiac and general surgery. Under these conditions shock therapy can be rapidly applied. Provided that adequate oxygenation is maintained and direct cardiac massage promptly initiated and maintained, shock therapy can be applied without undue haste.

METHOD

The principle of the method is to apply a powerful shock across the ventricles and produce complete arrest of the fibrillating heart. Once asystole occurs a regular ventricular beat may start spontaneously or cardiac massage may be needed to initiate it.

The apparatus comprises a double-wound transformer having a tapped secondary capable of supplying 1 ampere from any tapping. The primary circuit is supplied from 50 c/s mains via a foot-operated switch and a Londex motor-driven interrupter whose contacts close every 1.5 seconds for 0.5 second.

The output voltage tappings are selected by means of a rotary switch on the front panel. Available voltages are 85, 125, 185, 250, 270, and 300. A buzzer operated from an auxiliary winding on the transformer gives an audible warning that the electrodes are alive. Closure of the mains switch merely sets in motion the interrupter. In order to administer a shock the foot switch must be depressed.

The electrodes consist of two large curved plates which are shaped to fit the curve of the ventricles.

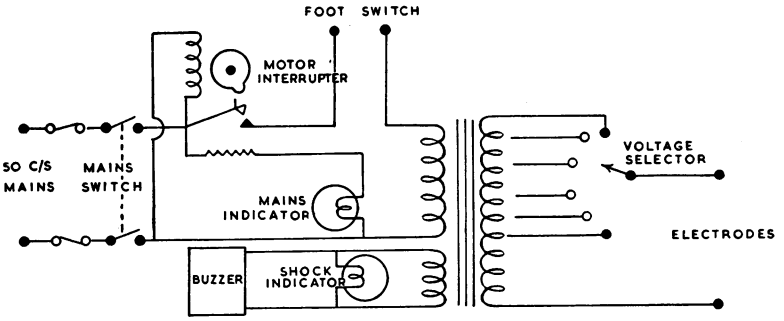

FIG. 1.—Circuit diagram.

The voltage required varies with the size of the heart and its electrical resistance, and this latter factor is also governed by the efficiency of the contact between electrodes and heart surface. Single shocks may be sufficient to stop ventricular fibrillation or, if they are insufficient, the voltage may be increased and further single shocks applied. If these are still inadequate a series of about six shocks at 1.5-second intervals may be given (Wiggers, 1940).

RESULTS IN DOGS

All dogs were under nembutal anaesthesia.

Seven dogs were given ventricular fibrillation electrically by means of an induction coil. Three dogs have been given ventricular fibrillation by the application of aconitine (one drop 0.2% solution in benzene) to the auricle, which causes auricular fibrillation and later ventricular fibrillation. To defibrillate the heart of a 20-kg. dog 185 volts was sufficient when given in a series of five shocks.

Nine dogs have been successfully defibrillated at periods of up to five minutes after the onset of ventricular fibrillation, but in all cases cardiac massage has been continued through the intervening period. Of the three dogs chemically stimulated, two were defibrillated with a voltage of 300. The third, due to other procedures on the heart, was in very poor condition and inadequately oxygenated. Ventricular fibrillation was not arrested.

Another dog in which ventricular fibrillation occurred during a thoracic sympathectomy failed to revert to normal, probably because of inadequate oxygenation owing to collapse of both lungs.

E.C.G.s taken after defibrillation in one dog showed a regular rhythm (Fig. 3). After electrical shocking sections of ventricular muscle show some superficial coagulation but no burning. Ventricular fibrillation is a terminal event, and any procedure with a chance of success is worth attempting in spite of possible minor damage. Later experiments with large electrodes showed no coagulation or burning.

RESULTS IN MAN

Case 1.—Mrs. T., aged 65 years, who had been attending as an out-patient, had gross left ventricular enlargement, anginal attacks, and multiple ventricular extrasystoles. During a visit to the hospital she collapsed while climbing a flight of stairs. She was found to be pulseless and respiration had almost stopped. She was put on a couch, and within three minutes the upper abdomen was opened and subdiaphragmatic cardiac massage started. The heart then started to beat again, but weakly, and soon faded away. She was intubated and oxygen given by means of controlled respiration from an anaesthetic apparatus. Next the diaphragm was incised, the pericardium was entered, and direct cardiac massage started. A few minutes later, as the strength of the beat was poor, adrenaline (1 ml. of 1/1,000) was given intracardially. This produced a strong beat temporarily, but was rapidly superseded by ventricular fibrillation. Procaine amide hydrochloride ("Pronestyl") was injected intravenously without effect. Cardiac massage was continued, and 10 minutes later the defibrillator was used. The electrodes were

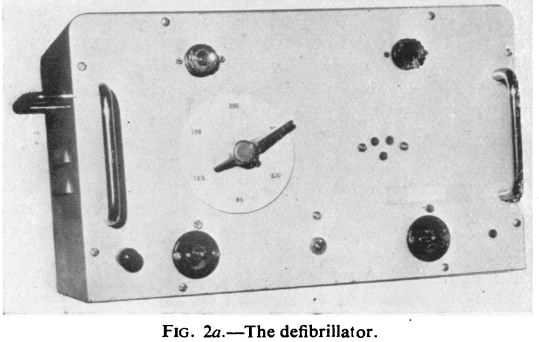

FIG. 2*a*.—The defibrillator.

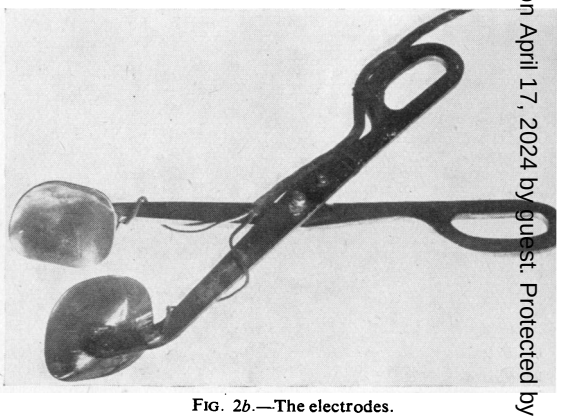

FIG. 2*b*.—The electrodes.

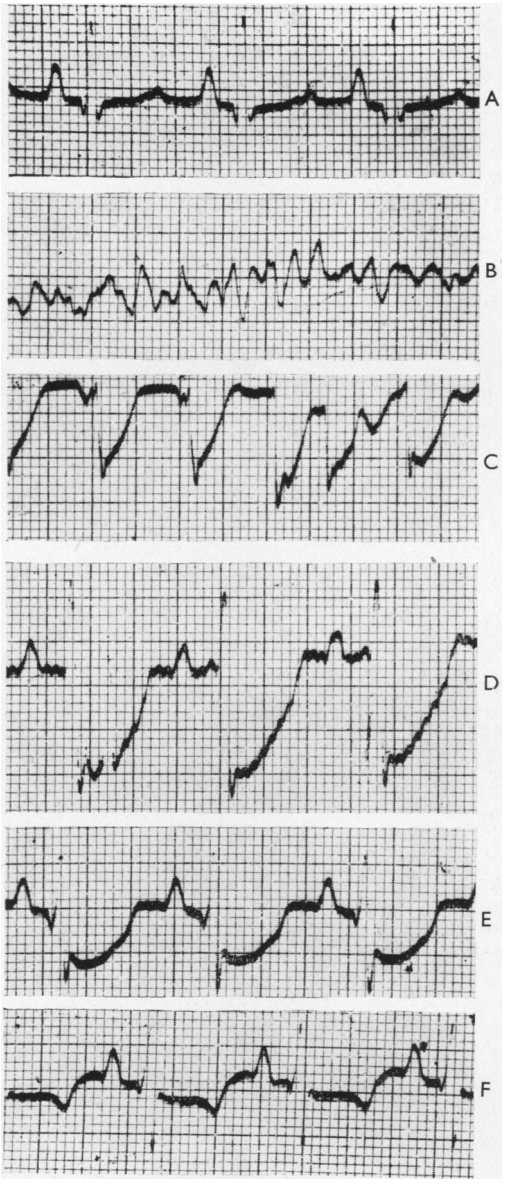

FIG. 3.—Electrocardiograms taken from Dog 3 to show effects of defibrillation: (A) before shock normal; (B) ventricular fibrillation; (C) 1 min. after shock; (D) 10 min. after shock; (E) 15 min. after shock; (F) ½ hour after shock.

inserted through the abdomen and up through the diaphragm and one placed on each side of the ventricle. The heart was given four shocks at 1.5-second intervals, each shock lasting 0.5 second, of 300 volts (this voltage was based on the voltage required in large dogs). This abolished fibrillation, and on massage a regular ventricular beat started. The heart beat regularly for the next hour when aided by massage and showed no sign of returning to fibrillation. The strength of the beat, however, was poor and the heart would only beat spontaneously for about two minutes following massage.

After an hour the strength of the beat was getting progressively weaker and asystole followed the cessation of massage. Further measures were considered useless. She had not had a spontaneous respiration for half an hour and the heart was no longer filling following inspiration.

The shock produced only slight localized muscle contraction outside the heart.

Necropsy confirmed the above findings, and section of the ventricles showed no evidence of damage due to electrical shocks or burns.

Case 2.—A man of 62, suffering from polycythaemia vera, was taken to the theatre for the evacuation of a haematoma following a rib biopsy. After 60 mg. of pethidine he was given gas, oxygen, and a little cyclopropane. During this minor procedure respiration ceased, and at about the same time the pulse could not be felt.

At once the patient was turned on to his back, a midline upper abdominal incision was made, and the cessation of the heart beat confirmed. A transverse slit in the diaphragm opposite the pericardial attachment was then made, four fingers thrust through this, and direct cardiac massage started. A regular but weak beat was started without difficulty. In order to strengthen this 25 m 1/1,000 adrenaline was given intracardially. Almost at once the rhythm changed from normal to ventricular fibrillation. Intracardiac "procaine" was then given without effect. Massage was continued and the defibrillator was sent for. This arrived, from another hospital, and was set up ready for use within 45 minutes of the start of cardiac massage. During the whole of this period massage was maintained and oxygen was given. The patient made spontaneous respiratory efforts and a corneal reflex was present for the whole of this time.

The electrodes of the defibrillator were introduced through the wound under the diaphragm and pericardium and applied to the ventricles.

Two shocks of 270 volts were given with only momentary cessation of fibrillation. Accordingly four shocks of 300 volts at 1.5-second intervals lasting about 0.5 second each were given and again fibrillation was only momentarily stopped. Four more shocks were then given and this produced asystole which lasted about three minutes. During this time massage was continued, and after three minutes the heart started spontaneously and beat fairly strongly. Massage was continued, and after about half an hour a steady beat persisted at a rate of about 50 per minute. At this stage many beats were coupled and appeared to be ventricular in origin. Blood pressure by a cuff was unrecordable, the corneal reflex was present, the pupils were fixed, central, but not dilated, spontaneous respirations were present, and the colour was less cyanosed than previously.

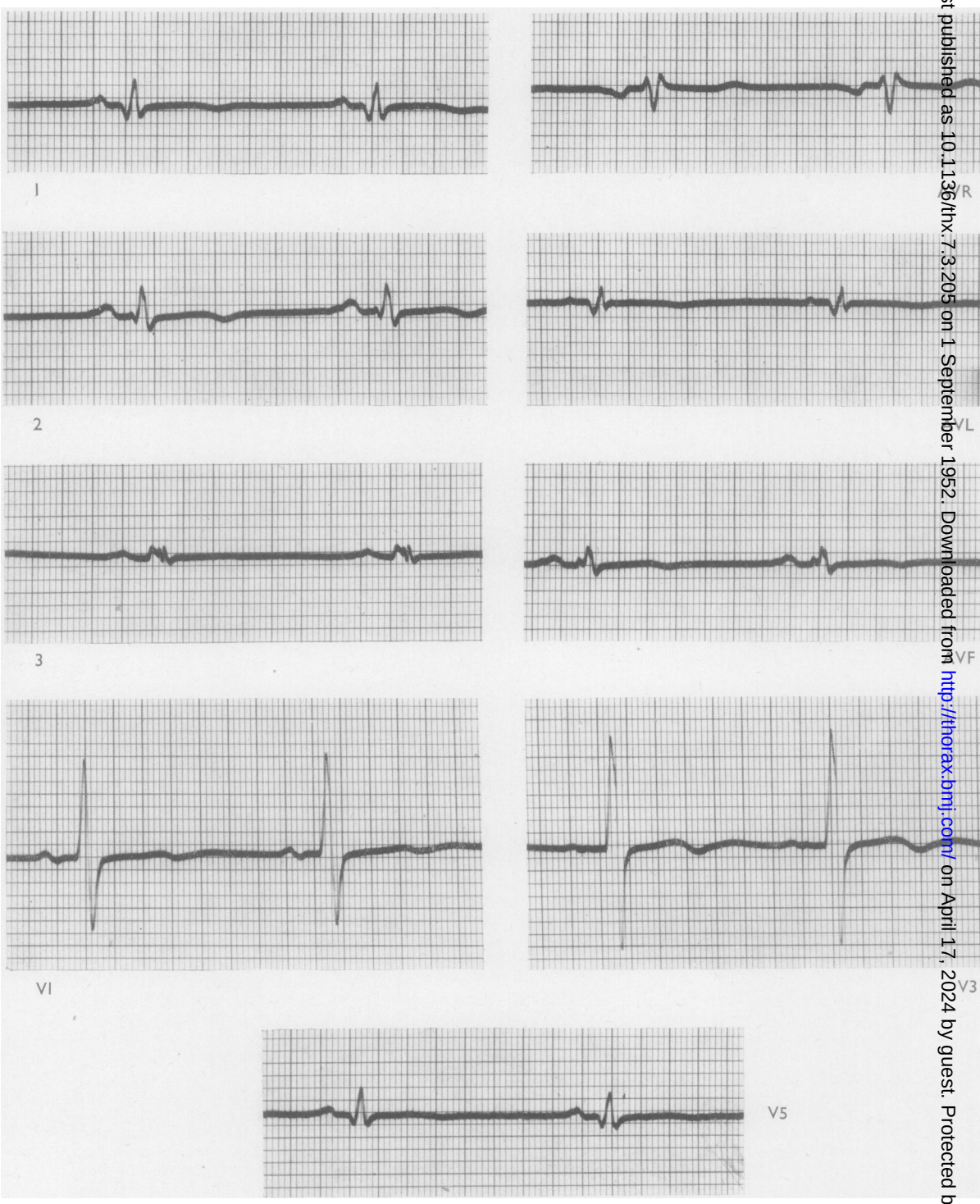

FIG. 4.—Electrocardiographs taken from Case 3, 10 days after defibrillation.

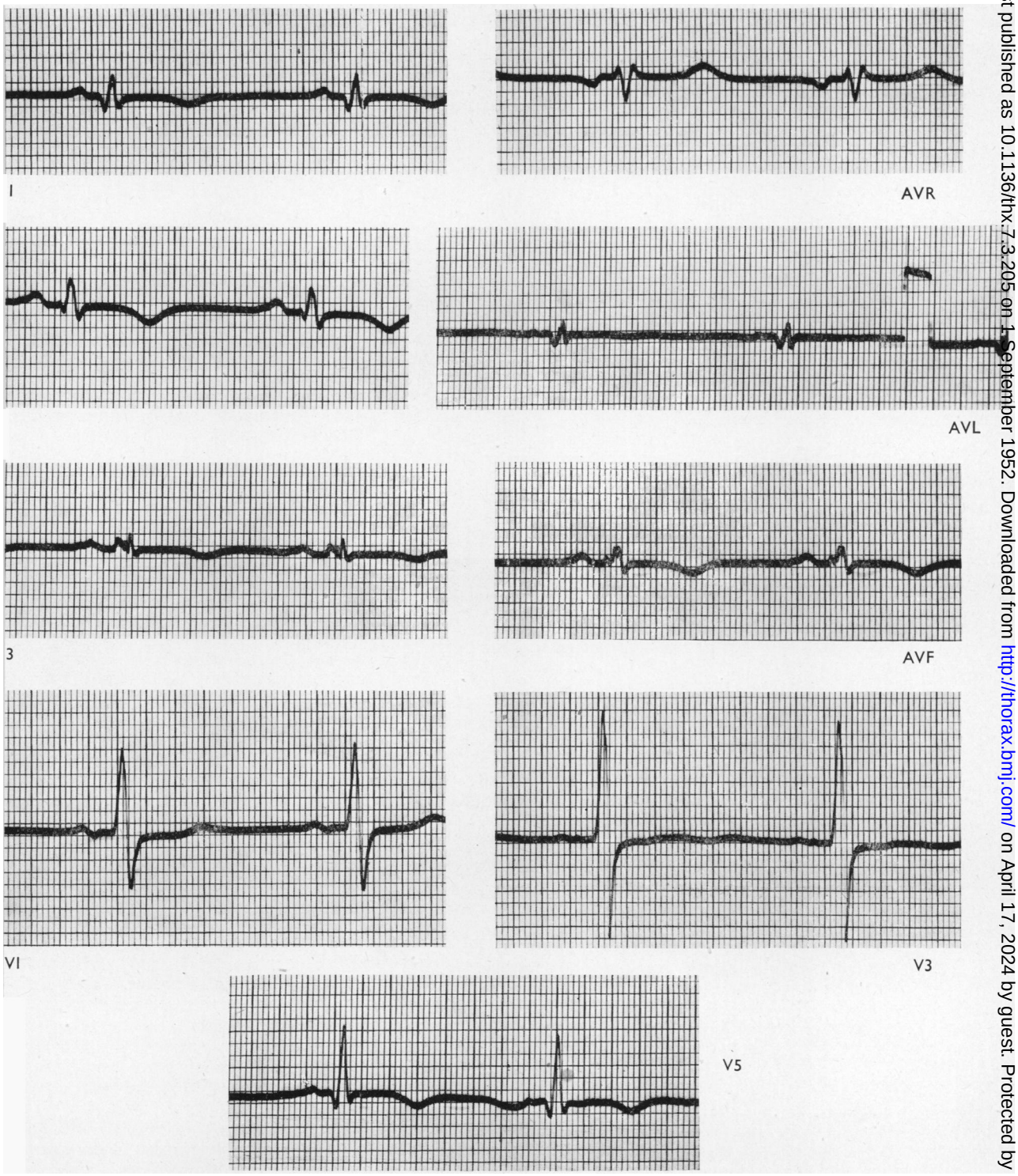

FIG. 5.—Electrocardiographs taken from Case 3, 15 days after defibrillation.

As the heart seemed to be well started it was decided to close the wounds in the diaphragm and abdomen. During the sewing-up procaine amide hydrochloride ("pronestyl") 500 mg. was given intravenously in order to reduce the number of ectopic beats if possible. After the incision had been closed the original biopsy wound was closed. At this stage the carotid pulse rate was 44 per minute. It was decided to leave the patient on the table and continue giving oxygen.

During the next half-hour the heart rate slowly fell to about 30 per minute, and at the end of this period cardiac arrest occurred again. Cardiac massage was tried once more, and apart from a few beats produced no effect, but at no stage after defibrillation was ventricular fibrillation observed.

Post-mortem section of the heart showed no evidence of damage, such as superficial coagulation, which could be associated with the application of electrodes.

Case 3.—Mr. W., aged 32, on June 23, 1952, was undergoing a left lower lobectomy for bronchiectasis under a general anaesthetic.

Dissection of the left lower lobe was difficult owing to adhesions, and there had been free oozing but no gross haemorrhage. His general condition was satisfactory until the hilum of the lower lobe was approached and one of the lower lobe arteries had been ligated. At this point the anaesthetist reported that the pulse was impalpable.

The heart was examined and found to have stopped, but was not fibrillating. The pericardium was opened. Cardiac massage was started and the patient respired with 100% oxygen. Noradrenaline, 40 γ, was given intravenously. Although as a result of cardiac massage no pulse could be felt, there was good capillary filling after pressure on the forehead.

Cardiac massage was continued for eight or nine minutes without effect and then ventricular fibrillation started.

The defibrillator was applied and three shocks of 250 volts given. This produced asystole again and massage had no effect. Then 1 ml. of 1/1,000 adrenaline was given into the left ventricle, and two minutes later (11 minutes from the start) ventricular fibrillation appeared again. No blood pressure or peripheral pulse was recordable.

Pulmonary ventilation had been carried out all this time.

After 12½ minutes, as the heart was still fibrillating, two further shocks of 250 volts were given. This produced complete asystole once more and cardiac massage was continued. After 15 minutes, however, the heart started with a regular rhythm. A blood pressure of 80/60 immediately became recordable and bleeding started from two vessels in the hilum.

The general circulation was very rapidly re-established, and within half a minute hilar blood was bright red.

The pulse and strength of the heart beat improved steadily until the end of the operation.

After this the lobectomy was completed and the wound closed with temporary drainage.

The patient was kept in the operating theatre for the next five hours. During this time his blood pressure fluctuated considerably and could not be maintained without frequent doses of noradrenaline and methedrine until about nine hours after defibrillation, when it remained steady at about 90/60. At that time an electrocardiogram showed sinus rhythm with numerous extrasystoles. The T waves were upright. He regained consciousness two hours after defibrillation, but was found to have a complete right hemiparesis, including aphasia. At this time he responded to simple commands but was confused.

After five hours the right leg began to twitch and he was a little less confused.

The next morning his general condition was good, with no evidence of cardiac failure. He was moving the right leg a little, the right facial palsy had improved, but speech was still very slurred. The right arm was spastic and immovable. His understanding was better, and he could talk a little. His pulse was regular and blood pressure steady at 90/60.

After 48 hours his condition had further improved. He moved the right leg and face well. The right arm was still immovable.

He made very steady progress after this, and by the tenth day was up and walking slowly. His speech was much better but still slurred, and he appeared to follow a simple conversation but had echolalia. The right arm showed no improvement, and his blood pressure was still 90/60.

An E.C.G. 14 days after operation showed evidence of anterior myocardial infarction. Movement in the right arm started to return about the fourteenth day and improved rapidly, but at this time there was partial motor aphasia and echolalia.

An electroencephalogram was within normal limits.

Twenty-two days after operation screening showed no evidence of aneurysm of the ventricles and movement of the ventricular borders was normal.

Discussion

From the experience gained with these three cases in which the defibrillator was used, certain conclusions and suggestions concerning the surgical treatment of acute cardiac arrest can be put forward.

The Action of Adrenaline.—In all three cases described ventricular fibrillation started soon after the intracardiac injection of adrenaline, given to potentiate the weak, irregular beat of the failing heart. This dangerous action of adrenaline, particularly when the heart muscle is anoxic, or in the presence of chloroform or cyclopropane, is being increasingly recognized by surgeons and anaesthetists. This action is not prevented or reversed by procaine.

The important thing, once a regular beat has been established after cardiac arrest, is to ensure that the coronary blood flow is maintained in

order that the heart muscle may pay off its oxygen debt accumulated during the period of arrest. Coronary flow depends mainly on the level of the diastolic blood pressure. Although cardiac massage may keep up an efficient peripheral circulation, the blood pressure may be unrecordable during the procedure. We have been unable to raise the systolic blood pressure of dogs above 40 mm. of mercury by cardiac massage alone. For the purpose of raising the blood pressure noradrenaline is probably the drug of choice. Barcroft and Konzett (1949) and Swan (1949) have recently described its properties, and Churchill-Davidson (1951) has reported on its clinical possibilities. In brief, it has a powerful peripheral vasoconstrictor action, no action at all on the heart muscle, and, if anything, a vasodilator action on the coronary arteries. Thus it raises the blood pressure as effectively as adrenaline but does not have the dangerous cardiac side-effects. From experience with these three cases we feel that the administration of intracardiac adrenaline in cases of acute cardiac arrest is very likely to precipitate ventricular fibrillation. In this hospital ampoules containing 2 ml. of noradrenaline (strength 20 μg. per ml.) are being issued to all theatres for trial in anaesthetic emergencies instead of adrenaline.

In Case 3 noradrenaline was given into a peripheral vein in a subject with a very poor peripheral circulation. In these circumstances it is not likely to be very effective. It should be given directly into a vena cava near the heart if feasible; it may also be given into a pulmonary vein or an auricle. It is possible that ventricular fibrillation is more likely to follow intraventricular injection. A dose of up to 5 ml. ($2\frac{1}{2}$ ampoules, or 100 γ) can be given by this route in an emergency.

A further point arises from Case 3. Autoregulation of the blood pressure did not return until about six hours after cardiac arrest. During this time the blood pressure had to be maintained by the continuous intravenous infusion of noradrenaline and methedrine. This emphasizes the importance of the after-care of these cases. The blood pressure fell away to 30 mm. Hg systolic if these drugs were discontinued during this period.

This patient was the only one who had a normal heart pre-operatively.

The treatment of ventricular fibrillation, once established and recognized, is a practical possibility with the defibrillator. Whether it occurs *de novo* or as a sequel to cardiac arrest, this method is readily applicable.

The approach through the upper abdomen and then straight into the pericardium by incising the diaphragm enables the operator to appreciate without doubt the onset or presence of ventricular fibrillation. It is not always possible to be sure whether the ventricles are fibrillating if they are only felt through the diaphragm. The diaphragmatic incision can be easily enlarged to insert the electrodes of the defibrillator.

In such cases a direct transthoracic approach has been recommended for the exposure of the heart, but we have found the abdominal exposure described quite adequate, and possibly easier as an emergency measure in general. Where the chest is already open, however, as in Case 3, the approach is no problem.

Most of the apparatus for defibrillation described has been designed to work from United States mains of 110 volts 60 cycles A.C. This machine is designed to work from English mains of 220 volts 50 cycles A.C. It appears from our results that higher voltages are required than those reported by United States workers.

In the operating theatre, where, if the machine is at hand, a delay of no more than a minute is necessary, we feel that it has a definite place, together with cardiac massage, in treating this hitherto irreversible condition. These results are necessarily incomplete, but in view of the work already done by other workers we feel that knowledge of the method and the relative simplicity of the machine should encourage others to try it.

It should be emphasized that in the first two cases there was a long and unavoidable delay before defibrillation could be initiated which undoubtedly reduced the chance of permanent recovery. If the interval is short, complete recovery is possible, as shown by American workers. Case 3, where the time interval was very short but the total time of arrest was about 15 minutes, led to recovery with cerebral damage; but the period of arrest was longer than is usually considered compatible with survival.

SUMMARY

A machine for giving electric shocks sufficient to defibrillate hearts is described

Results on experimental animals and three human cases are discussed.

The danger of giving large doses of intracardiac adrenaline to cases of acute cardiac arrest is pointed out, and the alternative use of noradrenaline is suggested.

The importance of maintaining the blood pressure post-operatively, if necessary with drugs, is emphasized.

We should like to acknowledge the interest and stimulation of Mr. N. R. Barrett, at whose instigation this work was started and who carried out the defibrillation in Case 3; also the help and suggestions of Dr. R. Daley and Dr. M. B. Matthews, from the Cardiac Department; also F. R. Scholefield, who assisted with all the animal experiments, A. J. Cronin for technical help, to A. L. Wooding for photography, to Miss F. Hunter for secretarial work, and to Miss S. Davies for the electrocardiographs in Figs. 4 and 5.

Addendum to Case 3

Two months after operation (August 22, 1952) the patient's condition is as follows:

He has a left parieto-temporal thrombosis, and as a result he has aphasia, mainly motor, which is improving. There is some intellectual deterioration and memory impairment. He has weakness and spasticity of the right arm of the cortical type.

These lesions are all steadily improving, and the cardiac condition is satisfactory. He is up and walking about and can look after himself, and is to be transferred to a convalescent home.

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