A REPORT ON TWO CASES OF CARDIAC ARREST

BY

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CASE 1. Circulatory Collapse following I.P.P.R.

This incident occurred at the Royal Liverpool Children's Hospital. The patient was a mature 14-year-old girl, weighing approximately 8 stone (50 kg).

She was admitted on February 26, 1958, with severe burns sustained when her nightdress caught alight. Forty per cent of her body surface area was affected, half of this being third-degree burns. Plasma and later whole blood were transfused, and dressings were applied under morphine sedation. She passed through a critical 36 hours, showing some signs of peripheral circulatory failure, but then began to improve. The relevant biochemical investigations were carried out, and normal levels were achieved by oral and intravenous replacement therapy. On March 12 she came to theatre for the first time for skin grafting and redressing of the burns.

Premedication consisted of quinalbarbitone 120 mg 1½ hours before operation, and then morphine 10 mg and atropine 0.6 mg 1 hour before operation. A test dose of d-tubocurarine 4 mg was given and anaesthesia was induced with thiopentone 200 mg. A further 20 mg of d-tubocurarine was given. Intubation was carried out with a plain No. 8 endotracheal tube and a mouth pack was inserted. Anaesthesia was maintained in a closed circuit using 3 l. nitrous oxide and 1 l. oxygen per minute, with carbon dioxide absorption being effected in a Water's canister (adult type). Controlled respiration was maintained throughout.

During the course of the operation which lasted 2 hours, the patient was transfused with 2 pints (1 l.) of whole blood and given two supplementary doses each of 3 mg of d-tubocurarine. When the anaesthesia was discontinued, the curarization was terminated by atropine 0.8 mg followed some minutes later by neostigmine 4 mg. The patient was fully conscious when she left the theatre.

On March 18 a similar operation was carried out, the same anaesthetic being used.

On March 31 a third grafting and redressing was planned. The same premedication was given, the morphine and atropine being administered 75 minutes before operation. Since admission, the patient's condition had remained fair, apart from bouts of fever with accompanying tachycardia resulting from sepsis in the ungrafted areas. The pre-operative estimation of electrolytes was normal and the child did not look toxic.

Anaesthesia was again induced with thiopentone 200 mg, but 40 mg of suxamethonium was now used for intubation since it was planned to keep the patient on spontaneous respiration with a mixture of nitrous oxide, oxygen and trichloroethylene. Intubation, preceded by spraying of the larynx with lignocaine 2 per cent presented no difficulty and no hypoxia occurred. Approximately 1.5 ml (i.e. 30 mg) of lignocaine were used.

Controlled respiration by pulmonary inflation with a mixture of 6 l. of nitrous oxide and 2 l. of oxygen only was commenced, since spontaneous respiration had not yet begun. No trichloroethylene was at any time added. At this point the patient's colour, which until that moment had been a "healthy pink", suddenly became a deathly white. The pulse was impalpable. The pupils were dilated. No heart beat could be heard on direct auscultation of the chest wall. At 4.32 p.m. the surgeon was informed that cardiac arrest had
occurred, and the patient was immediately wheeled into the theatre on a trolley. The sequence of events was then as follows:

P.M.
4.32. Head of the trolley lowered and inflation with pure oxygen commenced.
4.33. No response. Surgeon requested to open chest and start cardiac massage. Inflation with oxygen continued.
4.34. Surgeon's hand within the thorax, and the heart massaged against the sternum.
4.38. Pericardium opened after chest wall retraction and bimanual massage instituted. Ventricles not fibrillating but no return of beat.
4.39. Adrenaline 6 ml of 1:3,000 solution injected into the left ventricle and massage continued. Within seconds, three forceful contractions occurred, followed by arrest and ventricular fibrillation. Whilst the defibrillator apparatus was being set up, massage was continued. It was noted that the patient's colour was now a little better, the pupils were less dilated and the carotid pulses could be palpated with each massaging stroke.
4.42. Delay due to technical hitch with defibrillator apparatus. Calcium chloride 5 ml of 10 per cent solution injected into left ventricle. No visible effect.
4.48. Defibrillator apparatus ready. Cardiac muscle tone now poor. A further injection of adrenaline 6 ml given into the left ventricle and massage continued. Two contractions, arrest and coarse fibrillation followed. Electrodes applied and three shocks delivered successively, each of 110 volts for 0.2 seconds. After several more massaging strokes, the heart started beating spontaneously at 4.51 p.m.
5.00. B.P. 70/50 mm Hg. An infusion of intravenous noradrenaline 2 mg in 540 ml of dextrose 5 per cent solution commenced. Marked improvement of force of heart beat, but severe slowing of rate noted. Intravenous atropine 0.32 mg given, with subsequent return to normal rate.
5.15. Spontaneous respiration returned. Controlled respiration instituted with suxamethonium in intermittent doses, and nitrous oxide introduced to maintain anaesthesia.
5.20. Electrocardiographic tracings from limb leads only showed normal rhythm but marked depression of the ST segment, which improved markedly during the succeeding 20 minutes.
5.45. Chest closed with waterseal drainage. Noradrenaline discontinued and blood transfusion commenced. Blood pressure maintained at 100/70 mm Hg.
7.00. Hypothermia considered to counter possible cerebral oedema, but not undertaken since the pupils were now normal in size.
7.15. Patient returned to the ward.
8.15. Consciousness returned. Patient recognized people and complained of feeling tired.
Next day. Patient felt well and a chest radiograph revealed no signs of postoperative complication. Convalescence was uneventful, and no cerebral changes have since been detected.

Two subsequent operations, on April 8 and April 25, for further skin grafts, using the thiopentone d-tubocurarine technique previously described, were uneventful.

In retrospect, it would appear that the cardiac arrest in this case was due to a combination of factors.

The blood volume of the patient was probably subnormal due to the constant slight plasma loss from the old burned areas, and the injection of thiopentone caused a very severe fall in blood pressure. The final insult was the introduction of positive pressure ventilation impeding the already reduced venous return.

Anoxia was avoided at all times during induction and the small amount of lignocaine used in spraying the larynx was unlikely to have any general effects. Relaxation was complete during easy intubation and reflex cardiac arrest was most improbable.

Immediate diagnosis of the cardiac arrest followed by rapid efficient cardiac massage were all important in the successful management of the case described.

Case 2. Striking Effect of Intracardiac Adrenaline

This incident occurred in the treatment of a case of barbiturate poisoning.

On July 10, 1958, a 65-year-old woman was admitted to the casualty department of the Liverpool Royal Infirmary. According to her husband she had swallowed an overdose of sleeping tablets. On admission she was unconscious and respirations were slightly depressed. When the casualty officer attempted to pass a stomach tube, her respirations ceased altogether. Oxygen was immediately administered through a Boyle's machine, but her pulse became imperceptible and no heart sounds could be heard. The casualty officer thereupon injected adrenaline 2 ml of 1:1,000 solution through the chest wall into the left ventricle, and the heart restarted.

At this stage, the patient's colour was fair and her blood pressure reading 260/120 mm Hg. While an endotracheal tube and a Water's circuit and canister were being procured, inflation was maintained with oxygen and nitrous oxide at a high flow rate and an intravenous drip of Noradrenaline was prepared. Whilst this was
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being done, the heart again stopped beating. A further intracardiac injection of adrenaline was given and the heart again restarted. The endotracheal tube was now passed and inflation continued, but the heart again stopped and it was noticed that the noradrenaline was running into the tissue. The drip was discontinued and adrenaline injected into the heart for the third time. Again the heart started. The noradrenaline drip was now established satisfactorily and the blood pressure level maintained at about 100/70 mm Hg by varying the rate of flow of noradrenaline. By controlled hyperventilation it was hoped to washout any excess carbon dioxide which had been accumulated. During this phase the pupils, which had been widely dilated, started contracting.

Gastric lavage was now performed and numerous capsule remnants washed out. Bemegride 50 mg and amiphenazole 30 mg were injected alternately at intervals through the noradrenaline drip until the patient began breathing spontaneously about 45 minutes later. A total of bemegride 1 gram and amiphenazole 600 mg was given. By that time she had been transferred to the operating theatre and the intermittent positive pressure respiration continued with the Aintree respirator. Since it appeared that the blood pressure level could be maintained only by vasopressor drugs, it was considered desirable to introduce a longer acting drug and 10 mg of methoxamine was injected intravenously and continued so that it was possible to wean the patient off noradrenaline within a few hours.

In order to minimize the possible brain damage resulting from the anoxic episodes occurring during cardiac arrest, two injections of 50 per cent sucrose (20 ml) were injected intravenously to counter any cerebral oedema. As a further precaution, reduction of the cerebral metabolism by means of hypothermia was considered worth while. The difficulty was, of course, that the noradrenaline and methoxamine had produced peripheral vasoconstriction, which would hamper the process of cooling and markedly increase the chance of local frostbite. Hypothermia might also delay the excretion rate of barbiturate from the body, but both these points were considered less important than overcoming the brain damage by reducing the cerebral metabolism. A large dose of chlorpromazine might produce peripheral vaso-

dilatation, but only at the expense of a fall in the still labile blood pressure. Eventually a dose of 25 mg was given.

Hypothermia was obtained by the surface method of cooling and, after a slow fall, a final temperature of 85°F (29.5°C) was obtained.

The patient was returned to a side ward, the cuffed endotracheal and stomach tubes being left in situ. Careful changing of the position in which the patient was lying, tracheobronchial, pharyngeal and stomach aspirations were carried out until consciousness returned. Antibiotics, intravenous fluids and vasopressor agents were given as required, the last being controlled by frequent blood pressure estimation.

After 10 hours the temperature was gradually allowed to rise and the laryngeal reflex returned. By this time, vasopressor drugs were no longer required, the blood pressure being stable, and 36 hours after admission the patient was conscious, recognizing her relatives.

Subsequently, electrocardiographs showed no evidence of myocardial damage. Slight surgical emphysema developed on the left side of the chest wall and radiography showed an underlying partial pneumothorax which cleared up quickly.

According to the patient's relatives, the episode caused no deterioration in her mental powers. She was a manic-depressive who had been treated with electroconvulsive therapy, and had attempted suicide on two previous occasions. It is not known how many tablets the patient consumed on this occasion.

In the above case the cause of the cardiac arrest was central depression due to barbiturate overdosage, causing respiratory failure followed by cardiovascular collapse. Intracardiac injections of adrenaline were successful here, but had the first injection failed, thoracotomy and cardiac massage would have been performed immediately.

With the resumption of spontaneous respiration, treatment was aimed at maintaining body functions as near normal as possible during the period of deep anaesthesia by avoiding or correcting shock, hypoxia, respiratory obstruction and peripheral circulatory collapse. So with the excretion of the barbiturates the coma lightened, during which phase the patient was nursed carefully. Hypothermia was induced for reasons already stated.