SUCCESSFUL RESUSCITATION AFTER CARDIAC ARREST DUE TO A PENETRATING WOUND OF THE HEART

A Case Report

BY

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SUMMARY

A case is presented of a 26-year-old man who was admitted to hospital having sustained a penetrating wound of the heart followed by cardiac arrest. When resuscitative measures had led to clinical improvement thoracotomy was performed, the myocardial wound sutured, and blood clots removed from the pleural cavity and pericardial space. After 15 days of treatment, first in the intensive therapy unit and afterwards in the surgical department, the patient was discharged home in a satisfactory physical and mental condition. The patient resumed his previous work (truck driver) two months after injury.

The purpose of this report is to draw attention to an interesting and rare case. As far as can be ascertained there has been no similar published report.

CASE REPORT

A 26-year-old man was admitted at 6.20 p.m. to the emergency room having received a penetrating wound of the left parasternal region in the fifth intercostal space. A police officer who had been present reported that the patient had been wounded with a knife at 6.10 p.m. about 200 m from the hospital. Absence of breathing and reflexes, unconsciousness, impalpable carotid and femoral pulses, inaudible heart sounds, dilated pupils, pale skin and lips, and clothes soaked in blood, led to the diagnosis of cardiac arrest due to haemorrhage, due to penetrating wound of the heart. External cardiac massage and artificial ventilation with an Ambu resuscitator, first by facepiece and then through a Magill endotracheal tube, were started immediately using air and oxygen. Adrenaline 2 mg was given into the heart.

While awaiting group O Rh negative citrated blood and blood group typing of the patient, two Portex intravenous cannulae were quickly inserted into both saphenous veins (cut-downs) and a disposable cannula was placed in the superior vena cava. Thereafter Rheomacrodex with 6 per cent glucose 500 ml, Macrodex in 6 per cent glucose 1000 ml, 7.5 per cent sodium bicarbonate 400 ml were infused rapidly. Hydrocortisone 2 g was injected intravenously. When O Rh negative citrated blood was available, 1350 ml (each unit cross-matched) was infused as fast as possible. When it was learned that the patient's blood group was O Rh positive, citrated blood of this group was infused rapidly (2700 ml before and during operation). In the pre-operative period 10 per cent calcium gluconate 30 ml was given intravenously in divided doses. After some minutes slight clinical improvement was observed. The colour of the face was less pale and the pupils were smaller. At this stage electrocardiography showed electrical activity arising in several foci (fig. 1). In view of the signs of improvement resuscitation was continued, and when a femoral pulse was felt (6.45 p.m.) the patient was moved to the operating theatre (7.00 p.m.) and the lungs ventilated with 100 per cent oxygen (8 l./min flow) from the anaesthetic apparatus. Cardiac massage was continued until the moment of preparing the operation field. With each compression of the thorax and each inflation of the lungs, blood emerged from the wound. At about 7.00 p.m. the

electrocardioscope now showed regular sinus rhythm with upright T waves. The rate being approximately 100 beats/min. The pupils were small and reacted to light. Arterial pressure was unobtainable in the radial artery until 8.10 p.m. but from then on exceeded 100 mm Hg systolic. At 7.30 p.m. atropine 0.4 mg was given intravenously. Ventilation was maintained with nitrous oxide (3 l./min) and oxygen (3 l./min), and because the patient showed motor activity tubocurarine 30 mg was given intravenously; operation started at 7.40 p.m.

Operation.

Anterolateral thoracotomy was performed via the fifth left intercostal space (the site of the knife wound). Large quantities of dark blood were removed from the pleural and pericardial cavities. The pericardial wound was enlarged and in the ventral apical surface of the left ventricle a penetrating wound could be seen. Temporary haemostasis was obtained by thumb pressure. The myocardial wound was closed with two thick silk sutures and catgut. When haemostasis was sufficient, the heart was beating strongly. The pericardium was also sutured but an aperture was left.

The lung tissue was undamaged. After inserting a pleural cavity catheter and inflating the lungs, the chest wall was closed by the usual technique.

During anaesthesia 10 per cent calcium gluconate 20 ml in divided doses was given intravenously. To aid the surgeon in locating the bleeding point the cuff of the endotracheal tube was deflated and the tube was advanced into the right main bronchus. The left lung then partly collapsed and operating conditions were markedly improved.

At the end of the operation both lungs were carefully inflated. When the operation was completed (8.45 p.m.) the patient was moved to the intensive therapy unit. Artificial respiration was continued with an Engström ventilator using a minute volume of 10 l./min. It seemed prudent to continue intermittent positive pressure ventilation because of the dangers of hypoventilation which might have aggravated the cerebral oedema. It seemed advisable to eliminate the work of breathing to reduce the work load on the heart muscle. The pleural cavity catheter was connected with a suction pump (−15 cm H$_2$O). The arterial pressure was satisfactory. The cardiac monitor showed the presence of regular sinus rhythm with upright T waves. The central venous pressure was 10 cm H$_2$O. After catheterization urine excretion was promoted by administration of 200 ml of 15 per cent mannitol solution. The temperature (axilla) at 9 p.m. was 34.9°C. In the early postoperative period citrated blood 450 ml was infused very slowly, and a further 500 mg dose of hydrocortisone was given. Radiography showed slight collapse in the left lung; no pleural fluid was present. The results of acid-base analysis were not available during the early postoperative period. Normal values were reported on the following day. A blood sample taken next morning showed: Hb 12.8 g/100 ml; Na 144 m.equiv/l; K 3.2 m.equiv/l; Cl 91 m.equiv/l; creatinine 1.3 mg/100 ml. In the intensive therapy unit the following drugs were given: Na G Penicillin 6,000,000 u., streptomycin 1.0 g, tetanus vaccine 0.5 ml. Diazepam 30 mg (in divided doses) was given to assure synchronization with the respirator and to relieve anxiety.

At about 4 a.m. the patient regained consciousness, started to look round and moved his hands. Breathing was not synchronized with the ventilator any more. Sinus tachycardia at about 160 beats/min was present (fig. 2). Neostigmine 0.75 mg was given intravenously and extubation performed, following which the patient was able to breathe satisfactorily and to speak. The heart rate slowed to normal values.

A few days of antibiotic cover, parenteral nutrition and careful observation the pleural drain was removed and the patient was transferred to a surgical department. After 15 days he was discharged fit and well.

**DISCUSSION**

In this case the problems presented were cardiac arrest with heart muscle injury and haemorrhagic shock. Cardiopulmonary resuscitation was started in the usual way together with treatment of haemorrhagic shock. Ideally ventilation should have commenced with 100 per cent oxygen but this was not possible in the circumstances. Operation could not have started earlier because of the dangers of hypoxia. Cardiopulmonary resuscitation was started in the usual way together with treatment of haemorrhagic shock. Ideally ventilation should have commenced with 100 per cent oxygen but this was not possible in the circumstances. Operation could not have started earlier because the mechanical activity of the heart and blood volume had to be restored. When clinical improvement was apparent the patient was moved to the operating room. Despite strong pulsation in the femoral artery and sinus rhythm cardiac massage was continued because blood pressure in the
radial artery was unobtainable. Apart from the restoration of cardiac action, treatment of haemorrhagic shock was also required. Haemorrhagic shock can be considered to include the problems of hypovolaemia, micro-thrombosis, anoxia, hypercapnia, vasoconstriction, metabolic acidosis, cerebral oedema and electrolyte disturbances. Rapid correction of these abnormalities should be attempted without delay and the therapy should be multidirectional.

In severe haemorrhagic shock low molecular weight dextran transfused together with blood improves blood flow by countering intravascular aggregation (Pharmacia, 1968). Massive and rapid transfusion of blood may be dangerous because of potassium excess and citrate intoxication. This problem was minimized by using recently drawn stored blood and administering calcium gluconate. In this particular situation there were difficulties preventing the use of warmed blood. This might have been of great value.

Vasoconstriction which occurs in cardiogenic (Dietzman and Lillehei, 1968), haemorrhagic (Telivuo and Louhimo, 1966), toxic (Bergvist, 1965), shock (Bauer, 1966) was combated in this case by use of large doses of hydrocortisone. It was thought that this treatment might prevent heart muscle exhaustion, oliguria and metabolic acidosis and might protect cells, especially of the cerebral cortex, against anoxia (Trazzi and Gallo, 1967). Cerebral oedema due to cardiac arrest and anoxia was treated with intermittent positive pressure ventilation and mannitol solution. Metabolic acidosis was corrected empirically by sodium carbonate which at the same time corrected any tendency to decreased Na+ level of the extracellular fluid (Brooks, 1967).

REFERENCES


