SUCCESSFUL RESUSCITATION AFTER CARDIAC ARREST DUE TO HAEMORRHAGE

Case Report

BY

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SUMMARY

A case is reported of successful resuscitation after cardiac arrest due to haemorrhage. Certain aspects of management are discussed, such as the value of aortic compression to conserve blood, when loss is likely to exceed replacement by a substantial margin. The hazards of this procedure and the risks attached to massive blood transfusion are also briefly discussed.

Cardiac arrest due to blood loss is, fortunately, a rare event in an operating theatre, but when it occurs the problems faced by the clinical team are formidable and the situation is sometimes regarded as irreversible. A case is presented here in which this accident occurred and in which resuscitation was successful. Some of the problems involved are briefly discussed.

CASE REPORT

A 29-year-old male patient was admitted to a medical ward following a haematemesis. He had been admitted on four occasions in the preceding three years for treatment of similar episodes, and was on the waiting list for surgical treatment of a radiologically proven duodenal ulcer. There was no evidence of concurrent illness.

On admission he was in a state of obvious hypovolaemia and required blood transfusion, which produced clinical improvement. For this reason a further trial of medical treatment was undertaken. During the next two days the general condition of the patient remained satisfactory but continued bleeding was indicated by the marked fall in haemoglobin level and multiple melaena stools. Further blood transfusion was required and it was decided to perform a gastrectomy the next day.

On the morning of operation a further haematemesis occurred, and the operation was postponed for 2 hours to permit further resuscitation. After the administration of 1 litre of blood, the patient's blood pressure was 120 mm Hg systolic and pulse rate 90 beats/min; a significant deficit in blood volume did not appear to be present and he appeared fit for operation. A further 2 litres of blood were cross-matched and available for use when required.

Anaesthetic management

Premedication was confined to atropine 0.6 mg administered 1 hour pre-operatively, since morphine 10 mg had been administered during the haematemesis. After pre-oxygenation, induction of anaesthesia was effected with thiopentone (125 mg) and suxamethonium (50 mg), followed by rapid intubation with a cuffed endotracheal tube (size 9.5). After the return of spontaneous ventilation, tubocurarine chloride 30 mg was administered, relaxation being maintained with increments of approximately 10 mg/hour. A light plane of anaesthesia was maintained by the administration of nitrous oxide and oxygen from a Boyle apparatus; several increments of thiopentone were also administered for this purpose and ventilation was sustained using a Cyclator ventilator (British Oxygen Company). Further relevant anaesthetic details are given during the description of the operation.

Operation

Following induction, the patient's condition was satisfactory and he was moved into the operating theatre. Immediately thereafter his condition deteriorated, presumably because bleeding had restarted. The rate of blood transfusion was increased to the maximum possible by applying intermittent manual pressure to the drip chamber of the plastic giving set in use (Baxter BR I), and the table was tilted into a steep Trendelenburg position. As a result of the angle at which the table was now placed, the patient regurgitated about 2 litres of blood. In spite of the rapid rate of transfusion, the pulse became impalpable and the pupils dilated. Cardiac arrest due to haemorrhage was diagnosed.

Oxygen inflation was begun immediately and external cardiac massage performed (Kouwenhoven, Jude and Knickerbocker, 1960). Transfusion of blood was continued using pressure and the long saphenous vein incised to set up a second infusion. At this point, some 30 seconds after the arrest, the chest was opened and the heart observed to be in asystole, flaccid and empty. Internal massage was begun and the thoracic aorta compressed manually (Wiggers, 1940).

As these manoeuvres were being carried out, the second surgeon present opened the abdomen and the entire gastro-intestinal tract was observed to be distended with blood. After the administration of 2 units of blood, 20 ml of 10 per cent calcium gluconate solution in divided doses and 500 ml of low molecular weight dextran, spontaneous cardiac action recommenced. The administration of 5 per cent sodium bicarbonate solution was now started and e.c.g. monitoring with an oscilloscope established. The cardiac rate was then 100 beats/min (sinus rhythm) and
systolic blood pressure in the radial artery 90 mm Hg. As soon as a further unit of blood had been administered with a further 10 ml of calcium gluconate and 250 ml of bicarbonate solution, manual compression of the aorta was stopped. The period of cardiac arrest was about 10 minutes and that of aortic compression 15 minutes.

The bleeding point in the gastro-intestinal tract was located in the duodenum and clearly visualized, bleeding briskly. The duodenal tissues proved friable and the bleeding impossible to control even with digital pressure. In spite of the fact that two intravenous infusions were being used with manual compression to replace loss, it was obvious that bleeding was once again outstripping replacement, and the condition of the patient rapidly deteriorating. It was feared that a second cardiac arrest might occur, either from unreplaced blood loss or selective refrigeration of the heart (Boyan and Howland, 1961), though an attempt was being made to warm the blood by placing the drip tubing in a basin of warm water at 40°C. The supply of blood-bank blood for the patient was temporarily nearly exhausted and there was difficulty in cross-matching a further supply.

A second period of aortic compression was commenced and continued for 20 minutes. During this period a litre of blood was administered through one of the drips, and through the other a further 250 ml of the bicarbonate solution with 20 ml of 10 per cent calcium gluconate in fractional dosage. As the duodenal bleeding was virtually arrested during this period, it proved possible to under-stitch the bleeding point, and when the aortic compression was released bleeding did not recommence.

A Polya-type gastrectomy was then expeditiously performed. The condition of the patient now appeared satisfactory: systolic blood pressure was 100 mm Hg and pulse rate 90 beats/min. As a deficit in blood volume was still thought to be present, transfusion was continued.

After 15 minutes pulmonary oedema occurred, as shown by a decrease in compliance and the aspiration of pink froth from the endotracheal tube. This was probably due to over-transfusion, though the preceding cardiac arrest may have been in part responsible. Transfusion was stopped and ouabaine 0.5 mg administered intravenously; inflation pressure was increased and periodic aspiration of the bronchial tree performed. These measures, together with the continuation of some operative bleeding, produced rapid resolution of the oedema.

The remainder of the operation was completed without incident, but before the relaxant was reversed and the patient permitted to recover consciousness, a specimen of venous blood was sent to the laboratory for estimation of the electrolytes and evaluation of its clotting factors. A thermocouple-type thermometer was passed into the oesophagus when it was found that the oesophageal temperature was 35°C. A self-retaining urinary catheter was also passed.

As soon as the relaxant was reversed with neostigmine 2.5 mg (preceded by atropine 1.2 mg), spontaneous respiration commenced, and immediately after extrubation consciousness was regained. The blood pressure was then 120 mm Hg systolic, pulse rate 90 beats/min and the e.c.g. showed normal rhythm. The patient was moved to the recovery room and administration of mannitol begun. As the patient soon began to secrete urine 200 ml of the 20 per cent solution was administered in the next 2 hours.

When the result of the laboratory's investigations of the venous blood became available, it was seen that there was no significant abnormality in serum electrolytes and that bleeding and clotting time were normal, but that the platelet count was 190,000/cu.mm and the plasma fibrinogen level 100 mg/100 ml. A solution of fibrinogen was then administered followed by a unit of reconstituted plasma. During the first 2 postoperative hours the patient passed 300 ml of urine, and during the next 4 hours the output was 700 ml. Before he was returned to the ward it appeared likely that he had not sustained serious cerebral or renal damage. The remainder of his stay in hospital was without interest except that this view was confirmed.

**Transfusion at operation.**

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<td>Bicarbonate 5 per cent solution</td>
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<td>Calcium gluconate 10 per cent solution</td>
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**DISCUSSION**

The value of aortic compression in the management of severe haemorrhage must be stressed (Wiggers, 1940). It is unlikely that transfusion without this measure would have enabled maintenance of an adequate cerebral and cardiac circulation in the 3 minutes assumed to be available before the onset of irreversible cerebral damage, particularly as a substantial portion of the infused blood would have been lost from the circulation. The amount of blood required for this purpose is also substantially reduced, and the problem of producing what could have been a virtually unlimited supply, had this measure not been adopted, eliminated.

Massive transfusion is of itself dangerous and the biochemical derangement associated with this measure may produce a cardiac arrest (Leveen et al., 1960). The adverse factors associated with this measure, unless corrected, may render an arrested heart refractory to massage and replacement therapy (Argent, 1957). The potassium content of stored blood may reach 24 m.equiv/l. after three weeks storage (Leveen, Schatman and Falk, 1959).

This level of potassium would probably not produce an arrest even under conditions of extremely rapid delivery (Howland, Jacobs and Goulet, 1960) if it were not for the depression of ionized calcium induced in the recipient by the excess of citrate in the donor blood (Ludbrook and Wynn, 1958), and by the temperature
and acidity of this substance. Citrate reduction of the level of ionized calcium is particularly liable to occur when the perfusion of the bones is reduced by hypovolaemia (Geddes, 1962). Cardiac action depends on the $K^+/Ca^{++}$ ratio. It would also appear possible that in marked hypovolaemia the serum potassium rises (Zwemer and Scudder, 1938). Under the extreme conditions of the case described the addition of ionized calcium would appear essential and for this reason the patient received 70 mg of 10 per cent calcium gluconate (Burton and Holderness, 1964).

The infusion of large amounts of cold blood can produce selective refrigeration of the heart and lower the contractile efficiency (Boyan and Howland, 1961). In the absence of an efficient means of raising the temperature of the blood we were forced to adopt the expedient of running the drip tubing through a basin of hot water (40°C). Under conditions of massive transfusion this measure is not effective in raising the temperature of the donor blood more than at most a few degrees. The realization of this fact was among the considerations which were responsible for the inception of a second period of aortic compression.

Aortic compression above the renal arteries must seriously reduce renal blood flow and there is a marked risk of renal damage if this period of renal ischaemia exceeds 30 minutes (Wylie and Churchill-Davidson, 1960).

In the case reported neither period was in excess of this time but it is possible that two periods of reduced blood flow might carry an increased risk. Mannitol administration was carried out to reduce the risk of postoperative renal failure (Powers et al., 1964) and also because of its value in preventing secondary cerebral oedema following an anoxic episode (Cope, 1960).

Bicarbonate solution was administered empirically on the basis of recommendations of Stewart (1964) to treat the metabolic acidosis induced by the cardiac arrest, aortic compression and exsanguination.

REFERENCES


REANIMATION COURONNEE DE SUCCES APRES ARRET CARDIAQUE CAUSE PAR UNE HEMORRAGIE: COMPTE-RENDU D'UN CAS

On rapporte un cas de reanimation couronnee de succes apres arrêt cardiaque cause par une hémorragie. On discute certains aspects des soins, telle que la valeur de la compression aortique pour retenir le sang lorsqu'il est vraisemblable que la perte dépasse le remplacement disponible par un écart trop considérable. On discute brièvement des hasards de ce procédé et des risques inhérents à une transfusion sanguine massive.

ERFOLGREICHE WIEDERBELEBUNG NACH HERZSTILLSTAND BEI BLUTVERLUST: EINE FALLBESCHREIBUNG

ZUSAMMENFASSUNG

CLINICAL EXPERIENCES WITH COMBINED HALOTHANE-TRICHLOROETHYLENE ANAESTHESIA

Sir,—The idea of giving halothane and trichloroethylene together is not original and was described by Pilberg and Vellacott in 1966. I have been using this method quite independently for some three years and, because it is so successful in practice, feel its description in an anaesthetic journal together with some additional viewpoints is justified.

This method is used for a wide range of adult anaesthetics where spontaneous respiration is maintained and muscular relaxation not required. Intubated patients "settle" well and the method can be used for major operations such as radical breast amputation and thyroidec- tomy.

Following intravenous induction, anaesthesia is maintained with nitrous oxide, oxygen, 0.5 per cent halothane and minimal trichloroethylene delivered from a standard Boyle apparatus via a semiclosed circuit. A Fluotec vaporizer is used, this being next to the Rotameters. The trichloroethylene vaporizer has just enough agent in it to cover the bottom of the bottle (about 15 ml); the plunger is fully up and the lever half-way. This is a "standard setting" for the majority of patients. It differs slightly from the method described by Pilberg and Vellacott, who recommend 0.25 per cent halothane and fill the trichloroethylene bottle with 50 ml agent, i.e. they give less halothane and more trichloroethylene. In the old, ill and young they recommend nitrous oxide, oxygen and trichloro- ethylene only for long periods. But as a sole agent halothane is surely better, its main disadvantage being its poor analgesic effect. I feel, therefore, that the opposite method is indicated and turn off the trichlo- roethylene in good time before the end of the anaesthetic. The prolonged analgesic effect of trichloroethylene will last and this method aids rapid return to con- sciousness. Further, performance values below 0.5 per cent halothane are not given on the plastic cards attached to the 4 per cent Mark II Fluotec vaporizers; the trend of these curves would suggest inaccuracy below gas flows of 6—7 l/min.

The simultaneous administration of these two agents raises the question of the order of giving them. The introduction in the mid-1950s of safety mechanisms to prevent trichloroethylene being introduced into the closed circuit led the Fluotec being moved to its present position next to the Rotameters. It is a physical fact that the lower the boiling point of a liquid, the more it cools on vaporization. As the boil- ing point of halothane is 50.2°C and of trichloro- ethylene it is 87°C, the former will reach a lower tempera- ture during an anaesthetic. If the trichloroethylene bottle preceded the Fluotec vaporizer, then the vapour derived from the warmer agent (trichloroethylene) may become oversaturated when passing over the colder agent (halothane) and so recondense. This was demonstrated by Foster and Todd (1956) in the case of ether (boiling point 34.6°C) and trichloroethylene. There is an obvious risk that if the vaporizers were so placed, trichloroethylene vapour remaining in the Fluotec could be passed into a closed circuit. Further, to properly clean a Fluotec vaporizer would be a factory procedure. It is thus imperative for the anaes- thetist to pass first through the halothane and then through the trichloroethylene.

The economics involved are impressive. At contract rate halothane costs about £5 per 250-ml bottle, while a 500-ml bottle of trichloroethylene costs 6s. 9d. Thus, volume for volume, halothane is about thirty times more expensive than trichloroethylene. In foreign countries, where halothane costs more, the difference will be greater. By using this method, about half the quantity of halothane will be required per unit of time, thus halving the cost of the volatile agents used. Many large hospitals spend today £2,500 to £3,000 per annum on halothane alone.

The features of this method can be appreciated by examining the different properties of halothane and trichloroethylene, which are, in many respects, complementary to each other. Using them together seems to bring out the best of each agent, while their respective disadvantages all but disappear. Thus, the undesirable features of halothane (postoperative shivering and poor analgesia) and of trichloroethylene (tachypnoea, slow induction of anaesthesia and delayed recovery) are all but eliminated or substituted when these agents are used together as described above. This may be partly due to the fact that the individual dosage used is so small.

These agents do not form an azeotropic mixture and evidently do not react chemically with each other. I have not seen any ill effects attributable to the method. However, Ozorio and Gabbé (1968, personal communication), who have been working on a technique of giving these two agents via a draw-over system, are going to investigate the effects on liver function of giving these drugs together.

The features I appreciate in this technique are:
1. "standard setting" for the majority of patients being anaesthetized by non-relaxation techniques;
2. rapid induction with excellent analgesia not normally associated with spontaneous respiration techniques;
3. smooth anaesthesia is quickly established following intubation (cords sprayed with 0.5 per cent lignocaine);
4. ideal method to teach beginners;
5. analgesia in the immediate postoperative period;
6. financial considerations.

DAVID L. SCOTT
Manley, Cheshire

REFERENCES


PRE-LUBRICATION BEFORE NASAL INTUBATION

Sir,—Your Symposium on Dental Anaesthesia (Brit. J. Anaesth., 40, March) has prompted me to suggest an adjunct to nasal intubation which is rational and has proved invaluable in practice.

A nasal tube is inevitably a tight fit which must result in the removal of most, if not all, of its surface lubricant during its entry through the nares. This somewhat serious deficiency can be readily overcome by the preliminary passage (during spontaneous respiration) of a well-lubricated rubber urethral rubber catheter into the nasopharynx and withdrawing in a spiral fashion to facilitate the spread of the lubricant evenly through- out the nasal passages.

The main advantages of such a manoeuvre are:
1. A forewarning of obstructions—it is clearly less traumatic to locate these with the blunt end of a catheter than with the open end of a nasal tube. Furthermore, the delay normally occasioned by these during intubation is avoided and the period of anoxia thus shortened.

The economics involved are impressive. At contract rate halothane costs about £5 per 250-ml bottle, while a 500-ml bottle of trichloroethylene costs 6s. 9d.
A single trial will readily confirm the benefits of this simple preliminary preparation.

Michael Kerr
Chelmsford

A SURFACE MARKING FOR CAUDAL BLOCK

Sir,—Although caudal block has not the same necessity today as it had of yore, it still remains unchallenged as the ideal anaesthetic for a few procedures.

Unfortunately, location of the sacral cornua has invalidated its execution in some 20 per cent of cases but it is the purpose of this communication to record a surface marking which obviates this disadvantage.

A needle inserted at the cephalad extremity of the ano-coccygeal raphe will be found to coincide with a bone, (i.e. the posterior surface of the coccyx; with the surface marking which obviates this disadvantage.

A further point in technique is worthy of mention; if using the left lateral position, and the patient is unduly fat, gravity will displace the subcutaneous layer of tissues so that the median plane will, in fact, be higher than is apparent; it is wise, therefore, to locate bone (i.e. the posterior surface of the coccyx) with the first insertion of the needle to avoid an inadvertent patient due to stripping of the periosteum. Latterly, high hazard observation has been adequately confirmed by dissections on cadavers and by consistent clinical approaches.

Another advantage of this approach is a direct visual introduction of the needle in contrast to the method of palpating the cornua and visualizing the position of the palpating fingers. Sufficient clinical application, in addition to anatomical confirmation on cadavers, has confirmed the efficacy of such a surface marking.

Michael Kerr
Chelmsford

CONVULSIONS FOLLOWING PRE-OPE RATIVE MEDICATION

Sir,—Last year Waterhouse (1967) reported epileptic convulsions in children following pre-operative medication with Pamergan. This letter tells a similar yet contrasting story.

The patient, a well-built 14 year-old boy, was to undergo re-exploration of a cyst in the thoracic part of the spinal medulla. Weighing 109 lb. (50 kg), he received intramuscularly the standard adult dose of Omnopon 20 mg and Scopolamine 0.4 mg pre-operatively. Ninety minutes later, in the anaesthetic room, with anaesthesia just about to be induced, he experienced intense itching at the back of the shoulder and then had a single major epileptic convulsion lasting about a minute followed by 30 minutes of post-ictal stupor before he awoke again, a little confused but otherwise normal.

There was no personal or family history of epilepsy, but electroencephalography later the same day, whilst showing "no evidence of a localized or generalized organic change", did reveal "diagnostic signs of a liability to suffer from an 'idiopathic' type of epilepsy".

A few days later the boy received 120 mg of tramprazine orally before his operation without mishap, and did indeed receive Omnopon 10 mg on one occasion postoperatively without ill-effect. I have been unable to discover what drugs he received for his previous operations.

Waterhouse inclined towards promethazine rather than pethidine as the offending agent in his cases, but here one must assume that a generous dose of morphine was responsible.

R. P. Holmes
Bristol

REFERENCE


EVALUATION OF THE EFFECT OF HALOTHANE ON POSTOPERATIVE VOMITING

Sir,—Some patients are sick after the most trivial procedures even when anti-emetic drugs are given, while a small proportion will have neither nausea nor vomiting after any type of operation, premedication or anaesthesia. Between these two extremes lie the patients who are most suitable for studies of anti-emetic drugs such as described by Dr. Ratra and colleagues (Brit. J. Anaesth., 1968), 40, 270 and in whom the beneficial effect of halothane on post-operative vomiting could best be demonstrated. Our own data obtained with the standard gynaecological operation shows an incidence of total emetic effects during the first 6 postoperative hours to be 14-18 per cent with several potent phenothiazine premedicants while when 15 mg morphine was given pre-operatively the incidence was 79 per cent and this latter figure has not been greatly exceeded with large doses of other opiates.

After a moderately potent emetic premedication such as morphine 10 mg, it has been possible to demonstrate conclusively an anti-emetic effect of halothane, but with atropine 0.6 mg and/or pethidine 50 mg the reduction was minimal (Dundee, Kirwan and Clarke, 1965). As with the Lucknow series, we found the reduction to be greatest during the first hour after anaesthesia. Undoubtedly this was due to the relative duration of morphine as compared with halothane. A similar effect has been observed when trimethobenzamid 200 mg is added to pethidine 100 mg premedication. Here the 35 per cent incidence of total emetic effects occurring during the first postoperative hour was reduced to 15 per cent, but the figures during the 1-6 hour period were 43 per cent for pethidine alone and 37 per cent for pethidine-trimethobenzamide (Dundee et al., 1966).
ing attributed to halothane was of no clinical importance, whereas it is most useful when heavy opiate premedication is used.

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CONTRIBUTORS TO THIS ISSUE

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Clinical studies of induction agents. XXVI: The relative potencies of thiopentone, methohexitone and propofol (page 593)

SIR,—We have read with interest the letter of Professor John W. Dundee regarding our paper. Whereas we appreciate the comments, we are not able to support or deny the contention of Professor Dundee that halothane is most useful in patients with heavy opiate premedication, since we did not employ opiates in our group of patients. There is no doubt that halothane reduces the incidence of emergence vomiting. It is, however, conceivable that with opiate premedication, which is characterized by high incidence of emesis, the beneficial effects of halothane may be more clearly observed.

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