MESENTERIC INFARCTION DURING HYPOTHERMIA

A Case Report

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SUMMARY

A case is reported of the development of “shock” during an operation performed under hypothermia, which was apparently caused by a mesenteric infarct. Resection of the infarcted segment of gut was followed by an improvement in the patient’s condition.

Most of the hazards and complications of induced hypothermia are well known and fully documented. However, the English and American literature on the subject contains no reference to the occurrence of mesenteric infarction during hypothermia. A case is reported in which such an incident did occur, due to either mesenteric thrombosis or embolus.

CASE REPORT

A man aged 46 years, with malignant hypertension (blood pressure 240/180 mm Hg), was found to have a stenosed left renal artery. In order to improve the blood flow to the left kidney a lienorenal arterial anastomosis was planned. It was decided to perform the operation under hypothermia to protect the kidney while its blood supply was cut off.

The patient was dyspnoeic on moderate exertion and had been in left ventricular failure on several occasions, treated with hypotensive agents, digitalis and diuretics. The e.c.g. showed left ventricular hypertrophy and a possible previous infarct. Haemoglobin and white cell count were normal. Cold haemaglutinins precipitated heavily at 4°C and faintly at 11°C, but as this temperature range was far outside that to which it was intended to cool the patient, no significance was attached to this observation.

Anaesthetic management.

On the night before operation, the patient was given chlorpromazine 50 mg orally with no effect on his pulse rate or blood pressure. At 7 a.m. on the morning of operation he was given pethidine 50 mg, chlorpromazine 50 mg and atropine 0.6 mg and at 8 a.m. arrived at the anaesthetic room. His pulse rate was 70 b.p.m. and his blood pressure was 180/90 mm Hg. After attaching the cardio-oscilloscope leads, anaesthesia was induced with thiopentone 250 mg (2.5 per cent), and d-tubocurarine 40 mg, and after inflating the patient's lungs with oxygen and spraying the pharynx, larynx, and trachea with 4 per cent lignocaine, a No. 10 cuffed armoured latex endotracheal tube was passed. A thermocouple thermometer was then inserted in his oesophagus at heart level. The patient's ventilation was controlled, with carbon dioxide absorption, using nitrous oxide and oxygen (3:2) throughout the period of hypothermia, operation and rewarming. Increments of tubocurarine were given up to a total of 85 mg. The patient's temperature, pulse rate and blood pressure were recorded every 15 minutes and the e.c.g. tracing on the oscilloscope was constantly watched.

The patient was put in a bath of water at an initial temperature of 12°C and this was then lowered by the addition of fresh-water ice to 8-10°C. The head, hands and feet were supported above the water level.

After 75 minutes in the bath, the oesophageal temperature had fallen to 32.5°C. The pulse rate was 48 b.p.m. and the blood pressure was 140/80 mm Hg. His e.c.g. tracing had undergone the changes associated with cooling, namely lengthening of the QRS complex and prolongation of the PR interval. The patient was removed from the bath, dried and placed on the operating table. After-cooling continued for 40 minutes, the temperature stabilizing at 29.5°C.

The operation was performed with the patient supine, via a transverse upper abdominal incision. As the peritoneum was being opened, the patient’s condition suddenly deteriorated. His pulse rate rose to 76 b.p.m. and his systolic blood-pressure fell to 90-100 mm Hg. The e.c.g. tracing changed only in amplitude. No cause for the deterioration was apparent until the gut was exposed, when a 2-inch length of ileum was found to be oedematous and discoloured. A warm pack was placed over this while the renal pedicle was being exposed, but it was felt that the segment of gut was infarcted and that resection would be necessary. Pressure recordings were taken from within the aorta and renal artery proximal and distal to the stenosis. The systolic pressure at all three sites was 70 mm Hg. This procedure took 15 minutes, and as the patient's condition continued to deteriorate it was decided to do only a nephrectomy. An additional inducement to haste was the now obviously infarcted segment of gut. The nephrectomy took about 5 minutes but there was no improvement in the patient's condition until the resection and anastomosis of the infarcted gut was performed. Immediately this was completed, the patient's condition began to improve, and within 5 minutes was back to the pre-incisional state.

For the remainder of the operation and during the rewarming period there was no further cause for anxiety. When the patient's temperature had reached
34.5°C active rewarming was stopped and he was allowed to regain consciousness, which took less than 5 minutes. His temperature overshot on the evening after the operation to 37.8°C but returned to normal in 4 hours. Bowel sounds were heard within 12 hours of the operation, his urine output was satisfactory, and he was allowed up for bedmaking on the 3rd post-operative day. He was discharged home on the 13th day.

DISCUSSION

It is well known that some of the factors associated with blood clotting undergo alteration during operations performed under hypothermia. There is a reduction in the number of circulating platelets (Villalobos, Adelson and Riley, 1956), and in the serum prothrombin (Bunker and Goldstein, 1958). Although the reduction in platelets is rapidly made up during rewarming, it has been suggested by Dundee and King (1959) that intravascular clotting is responsible for these findings.

There is also a reduction in the rate of blood flow throughout the body during hypothermia, due to the fall in cardiac output, and this is particularly marked in the hepatic and splanchnic vascular beds (Cooper and Sellick, 1960). These factors, plus the increased viscosity of the blood at low temperature would contribute to the formation of thrombi or emboli. Gray (1955) has suggested that pulmonary emboli are commoner after operations performed under hypothermia than after those performed under normothermic conditions.

The hazards of hypothermia must be balanced against the indications for its use. In the case reported, the patient's expectation of life untreated was very short, and the pre-operative investigations, which included intravenous pyelography, differential renal function studies and arteriography, suggested that improvement in the blood supply to the kidney might reasonably be expected to lower the blood pressure. Hypothermia was used to safeguard the kidney for the period during which its blood supply was to be cut off. As it turned out, the operation planned was not possible.

The actual mechanism of shock production in this case is obscure. There was no overt blood loss or loss of intravascular fluid into the gut from the infarcted segment. There is no way of knowing whether there was functional hypovolaemia due to pooling or regional vasodilatation. It would be expected that neurogenic shocking stimuli would be obtunded by the combined effects of anaesthesia and hypothermia.

It is unsatisfactory to postulate that the cause was the absorption of a hypothetical toxin produced either by the infarcted gut itself or by gut contents. Consequently, the immediate cause of the collapse remains a matter for conjecture.

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REFERENCES


