INCIDENTAL HYPOTHERMIA DURING SURGERY FOR PERIPHERAL VASCULAR DISEASE

Case Report

BY

J. F. SEARLE

SUMMARY

A 43-year-old woman undergoing extensive vascular surgery, lasting 8 hours, developed marked hypothermia. Her mean body temperature fell to 31.2°C. This was due to the extensive exposure of the viscera of the chest and abdomen, and peripheral vasodilatation caused by epidural analgesia. The probable consequences of such heat loss during surgery, and methods of its prevention, are discussed. The patient rapidly returned to normal body temperature after operation, without shivering. The management of the cold patient in the immediate postoperative period is reviewed.

Although the effects of low temperature have interested physiologists for many years, it is only in recent years that anaesthetists have begun to pay attention to heat balance during surgery. The following case report is of a woman who developed hypothermia during prolonged peripheral vascular surgery.

CASE REPORT

A 43-year-old housewife, weighing 60 kg, was admitted to hospital in January 1971 complaining of pain in the right thigh and calf on exercise. A lumbar aortogram showed narrowing and irregularity of the abdominal aorta, marked stenosis of the aortic bifurcation, bilateral renal artery stenosis, and complete occlusion of the right common iliac artery, extending to the right femoral artery for 18 cm.

It was decided to perform an aorto-renal-iliac-femoral graft.

Her pre-operative blood pressure was 240/80 mm Hg, and was associated with left ventricular enlargement. Blood picture and electrolytes were normal. Her pre-operative oral temperature ranged from 36.8 to 37.2°C.

Premedication consisted of papaveretum 15 mg and hyoscine 0.3 mg. Anaesthesia was induced at 13.30 hours with thiopentone 250 mg. This was followed by suxamethonium 50 mg and an 8 mm cuffed endotracheal tube was passed. Anaesthesia was maintained with nitrous oxide 65 per cent, oxygen 35 per cent. Phenoperidine 5 mg was given in 1 mg increments. The patient's lungs were ventilated with a Barnet ventilator.

An epidural catheter was inserted via a Tuohy needle at L1-2. Epidural block was performed with 25 ml of 1.5 per cent lignocaine containing 1:200,000 adrenaline, and doses of 7 ml were given 2-hourly during operation.

Blood pressure was monitored using an oscillotonometer. Central venous pressure was measured via a right atrial catheter, inserted percutaneously into the right internal jugular vein. Urinary output was recorded every 30 minutes, an indwelling Foley catheter having been passed immediately before surgery began. Thermistors were placed in the rectum and nasopharynx, and on the skin of the back, abdomen, sole and great toe of the right foot, and the temperature at these sites measured with a Light thermometer. The patient was placed on a water mattress heated to 37°C. The ambient theatre temperature was 21.5°C.

At 14.15 hours, the abdomen was opened through a midline incision from the xiphisternum to the symphysis pubis. This was later extended to a left thoracotomy to improve the surgical exposure. From time to time there was profuse bleeding, requiring rapid blood transfusion to maintain a normal circulating volume. Blood was transfused through an in-line heating coil at 37°C.

Although preoperative hypothermia is not uncommon in elderly patients, with history of smoking, immobilization, and other conditions requiring hospitalization, the degree of postoperative hypothermia encountered in this case was extraordinary. The patient rapidly returned to normal body temperature after operation, without shivering. The management of the cold patient in the immediate postoperative period is reviewed.

The fall in central and peripheral temperature during operation is shown in Table I.

Table I

<table>
<thead>
<tr>
<th>Time</th>
<th>Rectum</th>
<th>Nasopharynx</th>
<th>Abdominal wall</th>
<th>Sole</th>
<th>Great toe</th>
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Surgery lasted 8 hours, during which there was a progressive fall in central and peripheral temperatures. These are shown in table I. Lowest temperatures were recorded at 21.30 hours. Mean body temperature at this time was 31.2°C. Mean body temperature is the average temperature of the body tissues. This was calculated from a nomogram (Vale, personal communication) based on the formula of Burton and Edholm (1955). Back temperature was not used in the calculation. This represents a heat deficit of 169.3 kcal.

While the chest and abdomen were being closed, chlorpromazine 5 mg was given intravenously to enhance peripheral vasodilatation. This was given hourly for 7 hours postoperatively. Surgery was completed at 22.00 hours. The patient was placed on a methyl cellulose mattress at 33°C to avoid further heat loss, and transferred to the intensive care unit. Ventilation was continued with a Cape ventilator.

One hour later the patient's rectal temperature had risen to 36.8°C and toe temperature to 33.8°C. Central and peripheral temperatures during the first 12 hours postoperatively are shown in figure 1. At 09.00 hours the next morning, the patient was extubated and taken off the methyl cellulose mattress. Postoperative analgesia was maintained for 48 hours, via the epidural catheter, using 0.5 per cent bupivacaine, 2-hourly.

**DISCUSSION**

The low temperatures reached during this operation are comparable with those reached in deliberate hypothermia. Newman (1971) reported central temperatures of this order in patients undergoing peripheral vascular surgery. In patients undergoing thoracotomy lasting approximately 2 hours, Dyde and Lunn (1970) found that when no steps were taken to reduce heat loss, average surface temperature fell to 31.8°C and deep body temperature to 35.2°C.

Heat debt, in the cooling subject, may be calculated from the mean body temperature (Burton and Edholm, 1955). The maximum heat debt in this patient was 90 kcal/sq.m of body surface. The accepted tolerance limit for heat debt laid down for the United States and Canadian Armed Forces is 80 kcal/sq.m of body surface (Sipple, 1943; Pugh, 1969).

This enormous heat loss in this patient occurred despite the use of a heating mattress. This was due to the prolonged exposure of the viscera of the left chest and abdomen and to the vasodilatation in the lower half of the body caused by the epidural block. Heat loss would almost certainly have been less in the absence of an epidural block, because peripheral vasoconstriction is part of the response to the cooling.

Vale and Lunn (1969) have shown that if the peripheral vasoconstriction caused by incidental cooling is allowed to persist into the postoperative period, central temperature rises, peripheral temperature continues to fall and a condition of hyperpyrexia develops. They suggest, therefore, that if in the hypothermic patient central and peripheral temperatures begin to diverge, chlorpromazine should be used to cause peripheral vasodilatation. This allows heat distribution to the periphery and prevents hyperpyrexia occurring. In this patient, the rapid rise in peripheral temperature and the achievement of a normal central temperature within 1 hour of the completion of surgery, without overt shivering, was almost certainly due to the persistent vasodilatation caused by the epidural block, augmented by chlorpromazine.

Chlorpromazine is also a useful sedative in these patients because hypothermic patients usually shiver, are restless and anxious.

Hypothermia causes a number of other changes. Metabolic rate rises (Cannon and Keatinge, 1960). Benzingher (1967) found that metabolic rate rises when deep body temperature is below 35°C and surface temperature below 33°C. This response is not entirely abolished by anaesthesia (Mellinger, 1965).

It is also known that a falling deep body temper-
ature is accompanied by a rising oxygen consumption (Roe et al., 1966). Vale and Lunn (1969) suggest that this rise in oxygen consumption may contribute to postoperative hypoxaemia in patients who develop hypothermia during anaesthesia.

The return to consciousness may be delayed in patients whose body temperature falls during anaesthesia. The reported levels at which consciousness is lost are variable. Some patients may be conscious when rectal temperatures are as low as 27°C and others are unconscious at 31°C (Cooper, Hunter and Keatinge, 1964).

Various methods have been advocated to prevent heat loss during anaesthesia and surgery. Newman (1971) recommends the use of an electric blanket heated to between 40-42°C, and the warming of intravenous fluids. Morris (1971) has shown that if the operating room temperature is between 21-24°C, oesophageal temperature does not fall below 26°C in patients undergoing intra-abdominal surgery. However, when the ambient temperature is 24°C, working conditions are uncomfortable, and patients tend to gain heat (Lunn, 1969). Water-heated mattresses may also be used and Vale and Lunn (1969) have described a mattress of methyl cellulose gel which is pre-heated. Patients may be insulated by wrapping the lower limbs in a heat-reflecting blanket (“space blanket”). This has been shown to prevent the falls in deep and surface temperatures in patients undergoing thoracotomy (Dyde and Lunn, 1970). It is the usual practice in this department for patients undergoing major surgery to be placed either on a water-heated or methyl cellulose mattress, and for the lower limbs to be wrapped in a space blanket. This effectively maintains peripheral temperature above 33°C and deep body temperature above 35°C. The use of such measures, however, requires the monitoring of peripheral and central temperatures because overheating may occur. In this patient a space blanket was not used, because exposure of the lower limbs was necessary for surgery.

The rapid transfusion of cold stored blood also lowers body temperature (Boyan and Howland, 1961). All the blood given to this patient was warmed to 37°C by passing it through an in-line heating coil.

REFERENCES


HYPOTHERMIE ACCESSOIRE DURANT LA CHIRURGIE POUR MALADIE VASCULAIRE PERIPHERIQUE: DESCRIPTION D'UN CAS

SOMMAIRE

Une femme de 43 ans manifesta au cours d'une intervention chirurgicale vasculaire etendue, qui dura 8 heures, une hypothermie marquée. Sa température corporelle moyenne tomba à 31,2°C. Cette chute fut due à l'exposition massive des viscères du thorax et abdomen et à la vasodilatation périphérique causée par l'analgésie épidermale. L'auteur discute des conséquences probables de pareille perte de chaleur durant la chirurgie, et des méthodes de prévention. La patiente récupéra rapidement, sans frissonner, sa température corporelle normale après l'opération. La conduite à suivre chez un malade refroidi au cours de la période postopératoire immédiate est revue.
ZUSAMMENFASSUNG


CORRESPONDENCE

THE ALBERT-SANDERS ADAPTOR FOR VENTILATING ANAESTHETIZED PATIENTS FOR MICRO-LARYNGEAL SURGERY

Sir,—I read with interest, in the Correspondence section, the letter of Drs Sellars and Gordon (1971) concerning a modification of the Kleinsasser laryngoscope for endolaryngeal surgery. It certainly is a practical way of administering gases to the non-paralyzed patient breathing spontaneously. The difficulty arises when one tries to ventilate the anaesthetized patient rendered apnoeic with relaxants. We adapted the Sanders oxygen injector utilized in bronchoscopy to the Jako laryngoscope (figs. 1 and 2). Ventilation can be maintained adequately for a long period of time (Barr et al., 1971).

Oxygen under pressure is released intermittently by a valve incorporated in the oxygen line. It is advisable to utilize an oxygen tank adapted with a pressure gauge and a reducing valve that can be regulated and adjusted to deliver oxygen between 40–80 p.s.i. We find this system to be preferable to the piped oxygen which is fixed at 45–50 p.s.i. In infants and young adults a lower pressure would be necessary to ventilate the patient while in the elderly patient a higher pressure is required to achieve adequate ventilation.

S. N. ALBERT
Washington, D.C.

REFERENCES
