Delayed forced air warming prevents hypothermia during abdominal aortic surgery

J. KARAYAN, D. THOMAS, L. LACOSTE, K. DHOSTE, J. B. RICCO AND J. FUSCIARDI

Summary

We have evaluated the efficacy of the delayed forced air warming during abdominal aortic surgery in 18 patients. Patients were allocated randomly to one of two groups: the control group (n = 9) received no intraoperative warming device; the Bair-Hugger group (n = 9) had active skin surface warming with an upper body cover. The device was activated when core temperature decreased to less than 36 °C. The reduction in core temperature was 0.6 °C during the first hour after induction and 0.4 °C during the second hour in both groups. In the control group, core temperature continued to decrease until the end of surgery, whereas in the Bair-Hugger group, the reduction in core temperature stopped after 1 h of warming, and then rewarming began. At the end of surgery, core temperature in the Bair-Hugger group was similar to core temperature before induction, and was higher than core temperature in the control group (P < 0.003). (Br. J. Anaesth. 1996; 76: 459–460)

Key words


Intraoperative hypothermia is common during abdominal aortic surgery [1]. Forced air warming has been shown to be an efficient preventive method against hypothermia in a wide variety of surgical procedures [2–5]. In these studies, skin warming was started at the beginning of anaesthesia. After induction of anaesthesia for abdominal aortic surgery, insertion of i.v. cannulae, haemodynamic monitoring and subsequent surgical preparation delay warming for 1 h, while initial hypothermia induced by internal heat redistribution continues. The aims of this study were to evaluate the efficacy of forced air warming during abdominal aortic surgery when initial hypothermia had begun.

Methods and results

In a prospective design, we studied 18 adult ASA II or III patients undergoing elective abdominal aortic surgery (aortic aneurysm repair or aortobifemoral bypass). None was obese, febrile or had a history of endocrine disease. Two hours before surgery, patients received preoperative cardiovascular treatment orally and morphine 0.1 mg kg⁻¹ i.m. Anaesthesia was induced with flunitrazepam 0.03 mg kg⁻¹ and fentanyl 5 µg kg⁻¹ i.v.; pancuronium 0.1 mg kg⁻¹ was then administered and the trachea intubated. The lungs were ventilated via a semi-closed circle system, at a fresh gas flow of 1.5 litre min⁻¹ to maintain end-tidal PCO₂ at 4.8–5.3 kPa. Anaesthesia was maintained with isoflurane and 50% nitrous oxide in oxygen, fentanyl 5 µg kg⁻¹ h⁻¹ and pancuronium. Core temperature (Tc) and haemodynamic state were monitored using a pulmonary artery catheter inserted before induction of anaesthesia. The operating room temperature was kept at 20–21 °C throughout surgery. All infused i.v. fluids were warmed (Fenwal). Patients were then allocated randomly to one of two groups. In nine patients a warm cotton sheet was placed over the upper chest and arms (control group (CG)). In the other nine patients the upper chest and arms (i.e. 24% of body surface area) were covered by an upper body forced air blower cover (model 520) attached to a Bair-Hugger model 500 (Augustine Medical, Eden Prairie, MN, USA) which was set on high (Bair-Hugger group (BHG)). The forced air blanket was covered by two cotton sheets. The warming system was activated as soon as Tc decreased to less than 36 °C. At the end of surgery, patients were transferred to the intensive care unit for continuous ventilation until complete recovery. Data were analysed with ANOVA and the Mann–Whitney U test. Differences were considered statistically significant when P < 0.05.

The two groups did not differ in age (mean 59 (range 42–79) yr in group CG vs 59 (43–79) yr in group BHG), mean duration of anaesthesia (366 (50) min in group CG vs 400 (50) min in group BHG), duration of surgery (278 (54) min in group CG vs 312 (47) min in group BHG), end-tidal isoflurane concentration (0.6 (0.3)% in group CG vs 0.8 (0.2)% in group BHG), blood loss (0.4 (0.2) litre in group CG vs 0.8 (0.4) litre in group BHG), infused volume of solutions (3.1 (0.6) litre in group CG vs 3.8 (0.8) litre in group BHG) and operating room temperature (21 (0.5) °C in group CG vs 21 (0.7) °C in group BHG).

J. KARAYAN*, MD, D. THOMAS, MD, L. LACOSTE, MD, K. DHOSTE, MD, J. FUSCIARDI, MD (Department of Anaesthesiology and Surgical Intensive Care); J. B. RICCO, MD (Department of Vascular Surgery); University Hospital, Poitiers, France. Accepted for publication: November 10, 1995.

*Address for correspondence: Département d’Anesthésie-Réanimation, CHU Poitiers, 86021 Poitiers Cedex, France.
Variations in $T_c$ for the patients are shown in figure 1. Before induction of anaesthesia there was no difference in mean $T_c$ between the groups (36.5 (sd 0.2) °C in group CG vs 36.7 (0.2) °C in group BHG). During the first hour after induction of anaesthesia, $T_c$ in both groups decreased (0.6 °C), and by the second hour a subsequent reduction of 0.4 °C had occurred. In group CG, $T_c$ continued to decrease until the end of the intervention (34.7 (1.0) °C). In group BHG, $T_c$ decreased to less than 36 °C (35.8 (0.2) °C) 2 h after induction. At this time the warming system was activated. One hour after the onset of active warming the reduction in temperature was stopped, and then the patients began to rewarm. During the subsequent course of the procedure, $T_c$ remained significantly higher in group BHG than in group CG ($P < 0.003$). $T_c$ in group BHG at the end of surgery was 36.5 (0.3) °C, that is similar to $T_c$ before induction.

Comment

All unwarmed patients were hypothermic at the end of surgery. In contrast, delayed forced air warming prevented intraoperative hypothermia.

Several authors have shown that the use of a limited skin surface is sufficient to prevent post-operative hypothermia [2–5], a finding confirmed by our study. However, in these studies skin warming was started just after induction of anaesthesia. The main purpose of our study was to show that delayed skin warming may be efficient during abdominal aortic surgery lasting more than 5 h.

A decrease in $T_c$ was observed during the first hour of anaesthesia in all patients. This initial hypothermia cannot be prevented, even if warming is started as soon as the patient is placed on the operating table [2]. In our study warming was started later, when $T_c$ decreased to less than 36 °C. There were several reasons for this delay in activating the warming system: first, the use of a warming system before induction would have required a cover on the lower limbs. In our opinion this seemed hazardous in patients with aortoiliac occlusive disease because of the risk of burning. Moreover, Beebe and colleagues showed that in pigs, warming of the hind legs during abdominal aortic surgery worsened the haemodynamic consequences of aortic declamping [6]. Second, putting the cover over the upper limbs would have avoided this risk, but insertion of invasive monitoring in the upper part of the body would have been precluded. Third, because of these two imperatives and in order to standardize the BHG group, we arbitrarily activated cutaneous warming at 36 °C.

Our study demonstrated that limited warming of the upper chest and arms was effective after the initial hypothermia by redistribution had started; it stopped the cooling and provided a normal $T_c$ at the end of the intervention. However, a minimum duration of 2 h is mandatory to start core warming, and 4 h of warming are needed to normalize $T_c$.

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References