Convective warming combined with vasodilator therapy accelerates core rewarming after coronary artery bypass surgery

S. J. HARRISON AND J. PONTE

Summary
In a prospective, randomized, controlled study, we have investigated the effect of forced air warming on the rate of change of nasopharyngeal and rectal temperatures in 20 patients after coronary artery bypass grafting. All patients had nasopharyngeal temperatures less than 36°C on arrival in the intensive care unit and received an infusion of glyceryl trinitrate 15 mg h⁻¹, but none received inotropes. Ten patients were warmed under an aluminized plastic “space” blanket (control group) and 10 were warmed under a “Bair Hugger” blanket connected to its power unit on “high” setting (Bair Hugger group). The rates of increase in nasopharyngeal temperature were 0.4 and 0.95°C h⁻¹, respectively, in the control and Bair Hugger groups (P < 0.01) during the first 2 h after operation. Over the same period of time, rectal temperatures increased at a rate of 0.25 and 0.75°C h⁻¹ in the control and Bair Hugger groups, respectively (P < 0.01). (Br. J. Anaesth. 1996; 76: 511–514)

Key words

A decrease in body core temperature, termed “afterdrop”, occurs frequently during the first 60–90 min of recovery after cardiac surgery; this is often the single factor for delaying return to spontaneous ventilation and tracheal extubation in this group of patients. Postoperative hypothermia may be attenuated by altering the management during cardiac surgery, such as adopting a “normothermic” technique during cardiopulmonary bypass, prolonging the rewarming period or infusing vasodilators while increasing pump flow [1, 2]. Often these measures are not successful and postoperative hypothermia occurs, especially if complications supervene (e.g. difficult haemostasis) which prolong the operative period after restoring normal circulation. Thus there is a clear need for a simple and effective tool to accelerate core rewarming during recovery.

Forced warm air devices are a proven, simple, safe and inexpensive means of preventing hypothermia during operation [3–5]. The available evidence that this type of warming device can be used to accelerate rewarming of core temperature after operation is controversial. In a recent study, Moors and colleagues [6] could not show that forced warm air, after coronary artery bypass surgery, accelerated the rate of increase in core temperature. In contrast, other authors found that in non-cardiac surgery, forced warm air was effective in rewarming the core of patients after operation [7]. We decided to perform this study to ascertain if the concomitant use of forced warm air and cutaneous vasodilatation is effective in accelerating core rewarming after coronary artery bypass surgery. Preliminary results have been published in abstract form [8].

Patients and methods
After obtaining hospital Ethics Committee approval and written informed consent, we studied 20 patients, allocated to two groups of 10 (“control” and “Bair Hugger”) who were admitted for elective coronary artery bypass grafting and who had “good” left ventricular function (ejection fraction > 50%).

The general anaesthetic (based on propofol, fentanyl and vecuronium) and surgical techniques were similar for all patients. In each group, six patients had unilateral stripping of leg veins and subsequent wrapping of the leg with an elastic bandage. In four patients in each group, veins were not harvested for grafting, mammary arteries only were used for coronary bypass conduits. With the exception of one patient who was cooled to 28°C, all other patients were cooled during cardiopulmonary bypass to nasopharyngeal temperatures of 30–32°C and then rewarmed to 37.3–38.3°C; bypass and cooling times were noted. After surgery, patients were transferred to the intensive care unit and included in the study only if their nasopharyngeal temperature was less than 36°C on arrival and inotropic support was not required. Relevant data on the selected patients are shown in table 1; there were no significant differences between the two groups (overall ranges), using the unpaired t test or the Mann–Whitney U test, in bypass time (49–97 min), cooling time (24–61 min) and total operating time (3.5–5 h).

On arrival in the intensive care unit patients were randomized to receive either a “space” blanket (control group) or a Bair Hugger blanket (Actamed Ltd, Wakefield). In the control group an aluminized plastic “space” blanket (Universal Hospital Supplies Ltd, London) was placed above the sheet and beneath the conventional hospital blankets; in the
Bair Hugger group a whole body Bair Hugger blanket was placed on top of the patient next to the skin, beneath conventional hospital sheets and blankets, and connected to a Bair Hugger 275E power unit on “high” setting. In both groups the patient’s head, and the hand and foot from which temperature measurements were being taken were left uncovered.

TEMPERATURE MEASUREMENTS

Baseline temperature readings of nasopharyngeal, rectal, fingertip and toe temperatures were obtained 10 min after arrival in the intensive care unit and at 15-min intervals thereafter. Nasopharyngeal and rectal temperatures were measured with Yellow Springs 400 series thermistor probes, displayed and stored on a Marquette Tramscope 12C monitor. Fingertip and toe skin temperatures were measured using Mallinkrodt insulated skin thermocouple probes connected to a Mallinkrodt Hi-Lo Temp model 8200 three-channel temperature monitor. The rates of temperature change during the first 2 h after operation were compared between control and Bair Hugger groups for all four measurement sites using the Mann–Whitney U test. Differences in the number of patients achieving core temperatures greater than 37°C at 2 h were compared using the chi-square test with Yates’ correction. Ambient temperature and humidity were recorded for each patient by means of a Vaisala HMI31 monitor. Ambient temperature ranged between 20.8 and 23.3°C and relative humidity between 21% and 38.2% for all patients, with no differences between the two groups.

POSTOPERATIVE MANAGEMENT

All patients received an infusion of glyceryl trinitrate at a minimum rate of 15 mg h⁻¹. Systolic arterial pressure was maintained at 90–130 mm Hg in all patients. This was attained either by an i.v. infusion of gelatin (or blood if haemoglobin was < 8 g dl⁻¹) when systolic arterial pressure approached 90 mm Hg, or by increasing the glyceryl trinitrate infusion when pressure approached 130 mm Hg. Boluses of hydralazine 10 mg i.v. were used occasionally if glyceryl trinitrate 30 mm h⁻¹ was insufficient to maintain systolic arterial pressure at or below 130 mm Hg. All i.v. fluids were warmed to 36.5°C in a water bath. Blood lost through pericardial and mediastinal drains into an autotransfusion reservoir was returned to the patient at room temperature at a maximum rate of 600 ml h⁻¹. Patients were sedated with an infusion of propofol 50–200 mg h⁻¹ and their lungs ventilated mechanically during the period of measurements, maintaining $P_{\text{ACO}_2}$ at 4.5–5.5 kPa. The occurrence of shivering was noted and, if present, was suppressed promptly first by increasing the infusion of propofol and then using morphine boluses (2–5 mg i.v.) supplemented with vecuronium boluses (2–4 mg i.v.) if morphine was ineffective. The incidence of shivering was compared between control and Bair Hugger using the chi-square test with Yates’ correction.

Table 1  Patient data (mean (SD) or median (range)). Comparisons between the two groups showed $P > 0.05$ for all variables. NPT = Nasopharyngeal temperature

<table>
<thead>
<tr>
<th></th>
<th>Control ($n = 10$)</th>
<th>Bair Hugger ($n = 10$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>60.5 (49–73)</td>
<td>64.3 (51–73)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>83.6 (12.1)</td>
<td>74.7 (10.0)</td>
</tr>
<tr>
<td>Body mass index (kg m⁻²)</td>
<td>27.8 (4.0)</td>
<td>26.1 (2.7)</td>
</tr>
<tr>
<td>NPT on arrival at ICU (°C)</td>
<td>35.7 (35.3–35.9)</td>
<td>35.5 (34.7–35.8)</td>
</tr>
<tr>
<td>Ambient temperature (°C)</td>
<td>22.3 (0.9)</td>
<td>22.6 (0.8)</td>
</tr>
</tbody>
</table>

Table 2  Median (range) rate of increase in temperature (°C h⁻¹) during the first 2 h after operation at the four measuring sites in the two groups of patients. The Mann–Whitney U test was used to compared the two groups

<table>
<thead>
<tr>
<th></th>
<th>Control ($n = 10$)</th>
<th>Bair Hugger ($n = 10$)</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasopharyngeal</td>
<td>0.40 (0.75)</td>
<td>0.95 (0.70–1.15)</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Rectal</td>
<td>0.25 (0.65)</td>
<td>0.75 (0.45–1.20)</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Toe</td>
<td>−1.15 (−4.00 to 0.05)</td>
<td>0.75 (−0.55 to 4.90)</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Fingertip</td>
<td>−2.25 (−4.35 to −0.20)</td>
<td>1.30 (−1.45 to 2.75)</td>
<td>&lt; 0.01</td>
</tr>
</tbody>
</table>

Results

The rate of postoperative increase in temperature during the first 2 h was greater in the Bair Hugger group compared to the control group. This was statistically significant ($**P < 0.01$).
group compared with the control group ($P < 0.01$) at all recorded sites (table 2). Patients in the Bair Hugger group often reached a nasopharyngeal temperature in excess of 37°C at 2 h, thus forcing the end of the warming intervention. Although measurements were obtained during the first 4 h after operation in all patients, it was decided to confine the study period for the purpose of comparisons to the first 2 h after operation.

At the end of the second hour after operation, one patient in the control group and nine patients in the Bair Hugger group had reached a nasopharyngeal temperature of 37°C or higher ($P < 0.01$) (fig. 1). Shivering was noted in five patients in the control group and in three patients in the Bair Hugger group but this difference was not significant. In the control group five patients were given morphine boluses, three were given hydralazine and another three vecuronium to suppress shivering. In the Bair Hugger group three patients were given morphine, one was given hydralazine and none needed vecuronium to suppress shivering.

**Discussion**

In contrast with a recent study in a similar group of patients [6], our results demonstrated that forced warm air, using a Bair Hugger system, accelerated the rate of warming not only of skin but also of nasopharyngeal and rectal temperatures in patients after coronary artery surgery with hypothermic bypass. This discrepancy may be explained by differences in study design; for example, the routine use of high doses of glyceryl trinitrate after operation in our patients may have caused cutaneous vasodilation, modifying the exchange of heat between the warming device and the patient. It is also possible that the doses of glyceryl trinitrate used in our study accelerated cutaneous heat loss in the patients in the control group. This, however, seems unlikely as patients in this group did eventually warm up, despite the absence of shivering, at a rate close to that usually observed in the absence of glyceryl trinitrate.

Studies of heat transfer through the skin into the body are scarce. There are reports in awake normal subjects managed with surface warming devices [9] but none in unconscious patients undergoing ventilation. Our results should be regarded as preliminary in this respect; they indirectly suggest that heat transfer from the warming device into the patient is possible, compared with merely preventing heat loss. The rate of increase of temperature in the four measured sites is not easily accounted for by the physiological non-shivering thermogenesis expected in postoperative, sedated patients. In awake, normal, non-shivering subjects, metabolism generates heat close to that usually observed in the absence of glyceryl trinitrate.

Heat flow through the skin, if not actual heat gain from the warming device.

The discrepancy between our results and those of Moors and colleagues [6] in which it was suggested that forced air warming devices can only expand the volume of the body “core” rather than increase its temperature, may be explained by differences in study design other than the postoperative use of vasodilators not used routinely by these authors. The use of inotropes was not stated and, more importantly, the management of shivering was not addressed. In our view, studies failing to accurately report the use of inotropes during recovery should be interpreted with caution because of the well known thermogenic effects of catecholamines [10, 11]. We were meticulous in our efforts to promptly abolish shivering in our patients because of its contribution to total body heat generation. For this purpose morphine was used [12] and, if necessary, vecuronium, in addition to a routine background sedative dose of propofol. Such manoeuvres attenuated the physiological responses to hypothermia other than shivering [13] allowing for a clearer comparison of the effects of the warming strategy between the two groups of patients. Although the difference was not significant, five patients shivered in the control group compared with three in the Bair Hugger group. Had we not abolished shivering, we would have expected acceleration of the rate of warming in the shivering patients which might have blurred the difference between the two groups. To enhance further the expected difference between the two groups, the “high” setting (43°C) on the Bair Hugger power unit was used routinely, as reported previously by others [3, 4].

Perioperative hypothermia remains a common problem in cardiac surgery and is associated with increased plasma catecholamine concentrations and an increase in metabolic rate (shivering), causing increased cardiovascular morbidity [14]. Our findings appear to illustrate the stressful effects of postoperative hypothermia: for example, more patients needed hydralazine, morphine and vecuronium in the control than in the Bair Hugger group. Although these differences did not reach significance...
we suspect that this was because of the small number of patients studied.

The recent trend towards a shorter postoperative period of mechanical ventilation and early extubation has highlighted the need for prevention and treatment of hypothermia. Despite careful temperature management during bypass, a proportion of patients is hypothermic at the end of surgery. The management of this condition has been based traditionally on preventing further heat losses by nursing the patient in a warm environment and covering the skin with thermal insulation devices such as aluminium foil. Despite these measures, core temperature often decreases further during the first hour after operation, as indeed we observed in several of our patients in the control group. Such “after-drop” in core temperature is possibly related to the redistribution of heat from the core to other body compartments, especially skeletal muscle [15]. Other types of heating devices have been tried in this setting; the oesophageal heat exchanger, for example, has been shown to be ineffective and radiant heaters to be effective only in warming the skin but at the expense of convenience [16].

References