

Benefits of intraoperative skin surface warming in cardiac surgical patients

L. HOHN, A. SCHWEIZER, A. KALANGOS, D. R. MOREL, M. BEDNARKIEWICZ AND M. LICKER

Summary

We have investigated patients undergoing cardiac surgery with hypothermic bypass to see if the addition of skin surface warming during systemic rewarming on bypass (heated group, $n = 43$) would improve perioperative thermal balance compared with conventional management without skin warming (control group, $n = 43$) in an open, randomized, controlled study. Intraoperative skin warming with a water mattress and forced warm air over the face, neck and shoulders attenuated the afterdrop in nasopharyngeal temperature after weaning from bypass (2.3 (1.2) °C and 1.3 (0.5) °C in the control and heated groups, respectively) ($P < 0.05$) and resulted in higher rectal temperature 4 h after surgery. Despite similar standard coagulation tests, heated patients had lower blood loss via the chest tubes (600 (264) ml vs 956 (448) ml in control patients) ($P < 0.05$), and less requirements for i.v. colloid infusion (1662 (404) ml vs 1994 (389) ml) ($P < 0.05$). There was a significant inverse correlation between rectal temperature on arrival in the ICU and postoperative blood loss ($r = 0.57$, $P < 0.001$). These data suggest that additional skin surface warming with a water mattress and forced warm air helped to preserve perioperative thermal balance and may contribute to reduced bleeding after cardiac surgery. (*Br. J. Anaesth.* 1998; 80: 318–323)

Keywords: temperature, body; equipment, warming devices; surgery, cardiovascular; blood, coagulation; hypothermia

Hypothermia remains a common problem because of its deleterious haemodynamic, haemostatic, immune and metabolic effects.^{1–4} After non-cardiac surgery, mild hypothermia has also been associated with arterial hypertension and myocardial ischaemia,⁵ in addition to increased blood loss.⁶

Patients undergoing cardiac surgery are often cooled to nasopharyngeal temperatures of 26–28 °C. At the end of cardiopulmonary bypass (CPB), even though nasopharyngeal temperature is restored to pre-bypass levels, a considerable mass of peripheral tissues remains at subnormal temperatures; subsequent redistribution of heat from the core to the periphery causes a decrease in nasopharyngeal temperature ranging from 1 to 3 °C, termed “afterdrop”.^{7,8} During operation, adopting a “normothermic” bypass technique, prolonging the rewarming period or infusing vasodilators while increasing pump flow may attenuate the postopera-

tive central temperature afterdrop.⁹ In the intensive care unit (ICU), external heat, that is convective or radiant, applied over a large body surface area has been shown to accelerate rewarming with the benefits of less shivering, lower oxygen demand and more haemodynamic stability.^{10–13}

Although skin surface warming with a forced warm air device is a simple, safe and efficient means for preventing hypothermia during major surgical procedures,¹⁴ it has not yet been recommended for use during cardiac surgery because the body surface area available for cutaneous warming was thought to be too small.

As the afterdrop in nasopharyngeal temperature is attributed to redistribution of heat from the warm core to the cold peripheral compartment, we hypothesized that the addition of skin warming to heat exchange via the bypass membrane would improve body rewarming and help to preserve body heat content, thereby minimizing the afterdrop in core temperature. Therefore, we investigated if concomitant intraoperative use of a heating water mattress and forced warm air over a limited body surface area (head and neck) would effectively prevent hypothermia and attenuate chest blood loss in the early period after cardiac surgery.

Patients and methods

After obtaining hospital Ethics Committee approval and written informed consent, we studied 91 patients undergoing elective coronary artery bypass graft surgery, aortic valve replacement and mitral valve repair or replacement. Patients with severe left ventricular dysfunction based on clinical history (dyspnoea grade 3 or higher) and ventricular angiograms (left ventricular ejection fraction $< 30\%$) were excluded. In addition, in the early postoperative period, those patients with active bleeding requiring surgical haemostasis were retrospectively excluded. Usual cardiac medications, including nitrates, β -blockers and calcium channel blockers were administered up to the morning of surgery. After an overnight fast, patients were premedicated with morphine 7.5 mg i.m. and diazepam 5–10 mg orally, 1 h before opera-

LAURENT HOHN, MD, ALEXANDRE SCHWEIZER, MD, DENIS R. MOREL, MD, MARC LICKER*, MD (Department of Anaesthesiology, Pharmacology and Surgical Intensive Care); AFKSENDIYOS KALANGOS, MD, MAREK BEDNARKIEWICZ, MD (Clinics of Cardio-Vascular Surgery); University Hospital, Geneva, Switzerland. Accepted for publication: October 31, 1997.

*Address for correspondence: Division d'Anesthésiologie, Hôpital Cantonal Universitaire, CH-1211 Genève 4, Switzerland.

tion. In the induction room, peripheral venous, radial arterial and central venous catheters were inserted for fluid and drug administration and for haemodynamic monitoring. A transoesophageal echocardiographic probe was inserted after tracheal intubation. General anaesthesia was conducted according to standard practice by an experienced anaesthetist not involved in the study and was based on moderate doses of fentanyl ($40\text{--}60\ \mu\text{g kg}^{-1}$) and midazolam ($0.03\text{--}0.06\ \text{mg kg}^{-1}$ followed by $0.10\ \text{mg kg}^{-1}\ \text{h}^{-1}$) with pancuronium for neuromuscular block ($0.15\ \text{mg kg}^{-1}$ at induction and $0.05\ \text{mg kg}^{-1}$ during bypass). Mechanical ventilation was adjusted to maintain normocapnia and normoxia. Body temperature was recorded continuously with calibrated thermistors (AS3 Datex Instrum. Corp. Helsinki, Finland) placed in the nasopharynx and in the rectum; nasopharyngeal and rectal temperatures indicated the temperature of the body core and muscular compartments.⁸ All probes were accurate to $\pm 0.1\ ^\circ\text{C}$ over the range $20\text{--}40\ ^\circ\text{C}$.

After aortic and right atrium cannulation, non-pulsatile CPB was commenced with a membrane oxygenator primed with crystalloids 2 litre and body temperature was decreased to achieve moderate hypothermia (lowest nasopharyngeal temperature $28.6\ (2.4)\ ^\circ\text{C}$ and $28.9\ (2.9)\ ^\circ\text{C}$ in the control and heated groups, respectively). After aortic clamping, myocardial protection was provided with St Thomas's solution $600\text{--}900\ \text{ml}$ infused via the aortic root at $25\text{--}30\text{-min}$ intervals. During CPB, pump flow greater than $2\ \text{litre min}^{-1}\ \text{m}^{-2}$ was maintained during moderate hypothermia and increased to $2.4\ \text{litre min}^{-1}\ \text{m}^{-2}$ during rewarming. Vasodilators (nitroprusside) or vasoconstrictors (phenylephrine) were administered to maintain mean arterial pressure (MAP) within an acceptable range ($60\text{--}80\ \text{mm Hg}$). After completion of surgical revascularization or valve repair-replacement, and when rectal temperature was greater than $35.7\ ^\circ\text{C}$, patients were weaned from CPB. Administration of nitroglycerin $1\text{--}3\ \text{mg h}^{-1}$ i.v. was part of our standard management procedure, which was started during rewarming and continued until $6\text{--}8\ \text{h}$ after surgery.

In the ICU, patients underwent mechanical ventilation throughout the 12-h study and, according to routine medical care, repeated bolus doses of morphine $1\ \text{mg}$ were given during emergence from anaesthesia and when increases in heart rate and arterial pressure were interpreted as pain, anxiety or discomfort.

During and after operation, a heat and moisture filter (Pall breathing system, Newquay, UK) was connected to the Y-piece of the non-rebreathing respiratory system. Ambient temperature was maintained at $18\text{--}20\ ^\circ\text{C}$ in the operating theatre and at $22\text{--}24\ ^\circ\text{C}$ in the ICU.

MEASUREMENTS AND CALCULATIONS

During operation, nasopharyngeal and rectal temperatures were recorded at the following times: $10\text{--}15\ \text{min}$ before aortic cannulation; at the end of each cardioplegia; after aortic unclamping; after separation from CPB; and during sternal and skin closures. Total duration of rewarming on bypass was recorded and the afterdrop was calculated as the dif-

ference between nasopharyngeal temperature at the time of separation from CPB and the lowest nasopharyngeal temperature recorded before transfer to the ICU.

After operation, activated clotting time and platelet count were measured on arrival in the ICU; systemic haemodynamic variables (MAP, heart rate, cardiac output) and rectal temperature were recorded hourly for $8\ \text{h}$ and at 10 and $12\ \text{h}$ after the end of surgery.

Urinary volume, blood loss through the chest tubes, i.v. fluid requirements (crystalloids and colloids), transfusion of blood products and the need for additional vasodilators (nitroprusside) and inotropic support (dobutamine or dopamine $> 5\ \mu\text{g kg}^{-1}\ \text{min}^{-1}$, norepinephrine or epinephrine $> 0.05\ \mu\text{g kg}^{-1}\ \text{min}^{-1}$) were also recorded.

EXPERIMENTAL DESIGN

On the day of surgery, patients were allocated randomly to routine thermal management (control group) or to additional skin warming (heated group). In both groups, core body rewarming (systemic rewarming) was accomplished by heat exchange through the oxygenator (maximum gradient of $10\ ^\circ\text{C}$ between the patient's blood temperature and water temperature in the oxygenator). In the heated group, extra warming of the skin included transfer of convective heat with a Bair Hugger system (Bair-Hugger Model 500, Augustine SA, Berne, Switzerland) and conductive heat with a thermostatic water mattress (Thermostat T1000, JMW Medical Systems Ltd, Midlothian, UK). Heated patients lay on a mattress containing circulating water at $38\ ^\circ\text{C}$ ($5\ \text{litre}$ with a mean flow of $3\text{--}4\ \text{litre min}^{-1}$) in contact with approximately $0.3\text{--}0.4\ \text{m}^2$ of body surface area (from the buttocks up to the shoulders) while forced warm air (at $38\ ^\circ\text{C}$) created a high velocity "wind" over the head, neck and shoulders ($10\text{--}15\%$ of body surface area) under a simple blanket. Systemic and skin rewarming were started during completion of the last distal graft anastomosis or at the end of valvular repair. Systemic rewarming ended when rectal temperature had reached $35.7\ ^\circ\text{C}$ in the two groups, while skin rewarming was continued until skin closure (in the heated group).

In the ICU, nurses and physicians administering fluids and treating pain were blinded to the patient's group.

STATISTICAL ANALYSIS

Parametric data were expressed as mean (SD) and analysed using two-way analysis of variance with Dunnett's test for within-group changes with respect to baseline. The relationships between chest tube fluid losses (dependent variable) and other independent variables, such as rectal temperature, afterdrop, length of CPB and aortic clamping were studied by uni- and multiple stepwise regression analysis. Pearson product-moment or Spearman rank correlation coefficients were calculated depending on the parametric or non-parametric nature of the data. Drug treatment, need for transfusion and type of surgery were compared using chi-square with Yates' correction. Differences were considered significant at $P < 0.05$.

Results

We studied 91 patients; two patients in each group were subsequently excluded because of bleeding which required surgical haemostasis within the first 3 h after operation. Another patient in the control group died within 2 h after surgery in cardiac failure as a result of a massive myocardial infarct.

Preoperative clinical, surgical and anaesthetic data are summarized in table 1. The two groups were similar in patient characteristics and surgical data. The risk of bleeding, as indicated by the type of surgery (combined valvular surgery and myocardial revascularization) and the use of aprotinin or antiaggregant and anticoagulant medications were also comparable in the two groups. Activated clotting time and platelet count were similar on arrival in the ICU, in addition to packed cell volume at the end of the study. Postoperative shivering requiring administration of pethidine or a muscle relaxant was observed in five patients in the heated group and in nine patients in the control group.

PERIOPERATIVE BODY TEMPERATURE

Duration of systemic rewarming and the gradients between nasopharyngeal and rectal temperatures after weaning from CPB did not differ between the two groups (table 2). However, body temperature was better maintained in the heated group, as: (1) the afterdrop in nasopharyngeal temperature was smaller; (2) fewer patients presented with nasopharyngeal temperatures less than 35.5 °C at the end of surgery (15 and 28 patients in the heated and control groups, respectively) ($P < 0.05$); and (3) rectal temperature was significantly higher from the time of sternal closure up to 4 h after operation (fig. 1).

In the control patients, rectal temperature on arrival in the ICU was 35.3 (0.4) °C and increased at a rate of approximately 0.3 °C h⁻¹, whereas in the heated group, initial postoperative rectal temperature was 36.0 (0.4) °C and increased more slowly (approximately 0.2 °C h⁻¹) over the subsequent 6 h. At the end of the 12-h study, rectal temperatures did not differ between the two groups (37.5 (0.5) °C in the heated and 37.4 (0.5) °C in the control group).

There was no significant association between the afterdrop in nasopharyngeal temperature and aortic clamping time ($r = 0.12$), CPB duration ($r = 0.09$), lowest temperature during CPB ($r = 0.08$) or body surface area ($r = 0.25$) in the two groups. In the control group only, the afterdrop in nasopharyngeal temperature was significantly correlated with the ratio of rewarming time to duration of aortic clamping ($r = 0.86$); in these patients, duration of systemic rewarming, representing >80% or <80% of clamping time, was associated with an afterdrop in nasopharyngeal temperature of -1.7 °C or -2.5 °C, respectively.

HAEMODYNAMIC STATE

Haemodynamic data (heart rate, MAP and cardiac output) were comparable in the two groups in addition to requirements for inotropic or vasopressor support (15 patients in the heated group and 20 patients in the control group) and for additional

Table 1 Clinical, surgical and anaesthetic data (mean (range) or number of patients). No significant differences between groups

	Heated group (n=43)	Control group (n=43)
Patient characteristics		
Age (yr)	60 (42-76)	66 (50-85)
Sex (M/F)	34/9	35/8
Body surface (m ²)	1.9 (1.6-2.1)	1.9 (1.6-2.2)
Left ventricular ejection fraction (%)	61 (31-88)	63 (33-84)
Preoperative antiaggregant and anticoagulant treatment		
Salicylates (n)	23	18
Heparin (n)	5	1
Dicoumarol (n)	1	1
Surgical procedures		
CABG (n)	30	29
Valvular surgery (n)	9	8
Combined (n)	4	6
Aortic clamping (min)	99 (18-181)	101 (29-250)
CPB duration (min)	138 (49-217)	142 (48-297)
Anaesthesia, analgesia and sedation		
Fentanyl (µg kg ⁻¹)	58 (39-92)	61 (31-96)
Midazolam (mg kg ⁻¹)	0.7 (0.4-1.1)	0.8 (0.5-1.2)
Morphine (mg kg ⁻¹)	0.12 (0.04-0.19)	0.10 (0.03-0.17)

Table 2 Rewarming time, gradients of nasopharyngeal to rectal temperature and afterdrop in nasopharyngeal temperature (mean (SD) or number). * $P < 0.05$ between groups

	Heated group (n=43)	Control group (n=43)
Rewarming on bypass (min)	71 (21)	77 (22)
Afterdrop in nasopharyngeal temp. (°C)	-1.3 (0.5)	-2.3 (1.2)*
Gradients of nasopharyngeal to rectal temp. (°C)		
Aortic unclamping	2.1 (1.9)	2.5 (2.0)
Weaning from bypass	0.7 (0.9)	0.6 (0.8)
Sternal closure	-0.6 (0.4)	-0.5 (0.8)
End of surgery	-0.6 (0.5)	-0.7 (0.4)

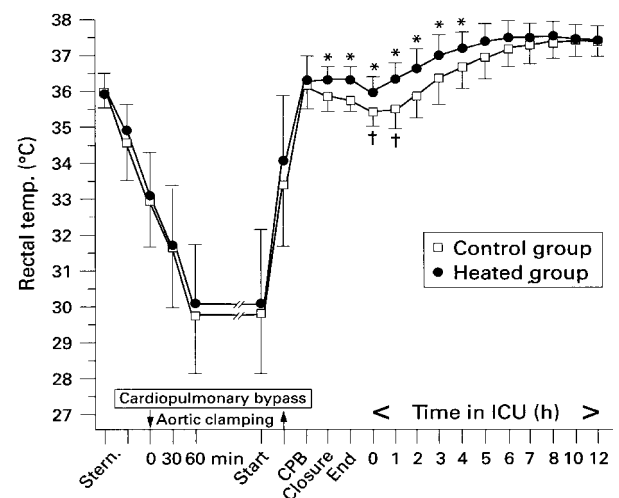


Figure 1 Perioperative changes in rectal temperature in patients undergoing cardiac surgery with standard intraoperative thermal management (control group) and in those with additional skin warming (heated group). Significant differences ($P < 0.05$): *between the two groups; †compared with values at weaning from CPB in the same group.

vasodilators (five patients in the heated group and seven in the control group). Nitroglycerin was administered at a median rate of 2 mg h⁻¹ in the two groups and over a median period of 8 h in the control group and 7 h in the heated group.

Table 3 Intra- and postoperative fluid balance and blood coagulation tests on arrival in the ICU (mean (range) or number). * $P < 0.05$ compared with control group

	Heated group ($n = 43$)	Control group ($n = 43$)
Intraoperative fluid balance		
I.v. crystalloids (ml)	2149 (700–4500)	2158 (1000–4100)
I.v. synthetic colloids (ml)	345 (0–1900)	414 (0–2100)
Cardioplegic solution (ml)	1929 (300–5900)	1673 (300–4500)
Autologous blood (ml)	548 (190–1500)	566 (100–2050)
Red blood cell (n)	17	18
Fresh frozen plasma (n)	8	5
Urine output (ml)	1843 (300–550)	1940 (450–4000)
Postoperative fluid balance		
Crystalloids (ml)	1891 (600–4350)	2227 (750–4950)
Colloids (ml)	1662 (500–3500)*	1994 (1000–4450)
Red blood cells (n)	6	13
Fresh frozen plasma (n)	7	13
Chest tube (ml/12 h)	660 (230–1870)*	956 (340–5480)
Urine output (ml/12 h)	1877 (195–3450)	1862 (250–3850)
Blood coagulation tests in ICU		
Activated coagulation time (s)	121 (84–175)	118 (78–164)
Platelets (number/ml)	109 (81–161)	116 (82–158)

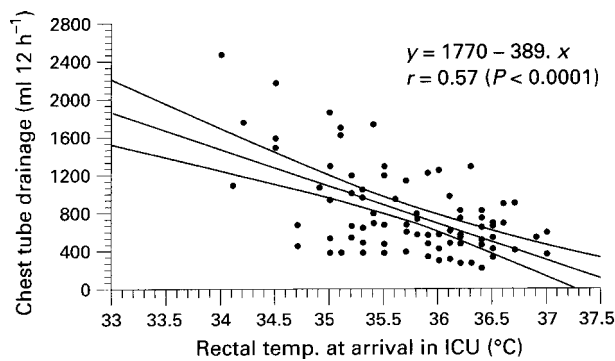


Figure 2 Relationship between fluid loss via the chest tubes over the first 12 h after cardiac surgery and rectal temperature on arrival in the ICU (regression line and 95% confidence intervals).

FLUID BALANCE AND HAEMOSTATIC VARIABLES

During operation, i.v. clear fluids, transfusion of homologous blood products and urine output did not differ between groups. During the first 12 h in the ICU, the amount of blood loss through the chest tubes was smaller in the heated group (-31% , $P < 0.05$) and was accompanied by reduced i.v. infusion of fluids (-23% , $P < 0.05$) compared with the control group (table 3). Transfusion of homologous blood components tended to be reduced in the heated group, although this was not significant (seven patients compared with 13 patients in the control group). There was a significant inverse relationship between rectal temperature on arrival in the ICU and postoperative fluid loss via the chest tubes in the pooled data for the two groups ($r = -0.57$, $P < 0.0001$). Regression analysis between these two variables is illustrated in figure 2.

Discussion

As expected, many patients became hypothermic at the end of cardiac surgery, despite systemic rewarming until rectal temperature reached at least 35.7°C , and when no precautions were taken to avoid "redistribution hypothermia". In contrast, intraoperative combination of two simple skin warming methods (Bair Hugger and water mattress) in addition to core rewarming attenuated the core temperature afterdrop and resulted in higher rectal temperatures in the

early hours after CPB. Importantly, better thermal homeostasis was associated with reduced postoperative fluid losses and i.v. fluid requirements.

Application of skin warming did not adversely affect the working conditions in the operating theatre compared with the routine management procedure. Additional noise was barely noticeable, i.v. cannulae were secured on the operating table, the patient's face and eyes were checked regularly and the transoesophageal echocardiographic probe was manipulated easily.

THERMAL BALANCE

In unwarmed patients, a core temperature afterdrop of -2°C has been associated with a heat deficit of more than 300 kJ which is equivalent to basal heat production over 1–2 h and requires 50–100% increase in energy expenditure in the early postoperative period.^{7,15} In this study, we hypothesized that heat storage in peripheral tissues would reduce the core-to-periphery gradient and attenuate heat flow redistribution. In the heated group, skin warming with forced warm air and a water mattress resulted in a mean decrease in nasopharyngeal temperature of 39% less and a higher rectal temperature during the first 4 h in the ICU compared with control patients. Presumably, higher mean body temperature ($+0.8^\circ\text{C}$ compared with the control group) was associated with better restoration of body heat content after weaning from bypass (approximately +195 kJ in a 70-kg adult) and with diminished thermoregulatory responses to cold, as indicated by a slower postoperative increase in rectal temperature in heated patients (0.2°C h^{-1} compared with 0.3°C h^{-1} in control patients).

During non-cardiac surgery, when 80–90% of the body surface is exposed to forced warm air (38°C), a large amount of heat (approximately 200 kJ h^{-1}) has been shown to be transferred across the skin.¹⁶ During cardiac surgery, despite a smaller surface available (only 10–15% of the total body surface), transfer of convective heat was likely facilitated by several factors, namely the underlying highly perfused anatomic structures of the upper body, large air-skin temperature difference, prolonged duration of rewarming (2–2.5 h) and a high velocity convective "wind". Interestingly, heat transfer has

been shown to be twice as effective when forced air is delivered beneath a simple bed sheet instead of using a commercial blanket whose rigid nature prevents the close approximation to non-planar anatomical structures and whose pinhole gridwork pattern causes low flow velocity.¹⁷

Conductive heat flow was also transferred across the larger skin surface in contact with the warm water mattress (approximately 0.3–0.4 m²). Its modest rewarming efficiency in non-cardiac surgery has been attributed to the relatively small surface available and to poor perfusion in dependent areas.¹⁸ Although many centres currently use a circulating water mattress in adult cardiac surgery, there is no clear evidence concerning its beneficial effects in terms of heat balance when used as a sole adjuvant to core rewarming.

Nitroglycerin and high perfusion flow rates were used in all patients during systemic rewarming in order to increase heat delivery to the “cold” muscles and thereby expand the “warm” body core. Hence in the two groups pharmacological vasodilatation contributed to the attenuation in subsequent redistribution hypothermia (as heat can only flow down a temperature gradient). As reported previously by Noback and Tinker,⁹ the afterdrop in nasopharyngeal temperature in control patients was related to the ratio between duration of systemic rewarming and hypothermic clamping: if systemic rewarming was prolonged >80% of clamping time, the afterdrop in nasopharyngeal temperature was attenuated. In this regard, additional skin surface warming could help to reduce the rewarming time on bypass.

TEMPERATURE AND HAEMOSTASIS

Chest fluid drainage consists of blood oozing from the surgical wound that is mixed with residual ice-slush solution and transudated fluids from the visceral pleura. An important observation was a significant correlation ($r = -0.57$) between postoperative blood loss via the chest tubes and rectal temperature on arrival in the ICU. This could explain approximately 33% of the relationship between these two variables, and the slope of the linear regression equation predicts an average additional fluid loss of 389 ml °C⁻¹ over a range of rectal temperatures from 37 to 34 °C. Except for the difference in rectal temperature, all other factors that would affect postoperative bleeding (i.e. activated clotting time, platelet concentration and administration of antiaggregant, nitrates and aprotinin) were similar in the two groups.

Unless considered specifically, the contribution of hypothermia to haemorrhagic diathesis is overlooked in clinical practice as standardized procedures for coagulation testing are performed at 37 °C and the number of platelets is unaffected by cold.¹⁹ The disparity between near-normal *in vitro* clotting studies and clinically non-surgical bleeding can be attributed to both coagulation and cold-induced platelet dysfunction. First, the process of coagulation involves a series of enzymatic reactions and the speed at which these reactions occur is temperature-dependent; prothrombin and activated partial thromboplastin times are increased by approximately 10–15% when temperature decreases from 37 to

34 °C.^{20,21} Second, cold-induced activation of the fibrinolytic system can further impair the haemostatic capacity, but to a much lesser extent.²² Third, prolongation of bleeding time during mild hypothermia is caused by a defect in the ability of platelets to produce thromboxane A₂, a proaggregatory factor, at the bleeding site.²³ In heated patients, the beneficial effects of skin warming in terms of reduced bleeding were more likely attributed to better preservation of platelet function as local rewarming has been demonstrated to normalize both bleeding time and platelet aggregation.^{23,24}

LIMITATIONS OF THE STUDY

In this study, extra skin warming resulted in higher rectal and nasopharyngeal temperatures in heated patients undergoing surgery of long duration with hypothermic bypass. However, we were unable to measure heat fluxes because surgical incision precluded positioning of skin temperature probes over the thorax, legs and arms. Furthermore, it is not known if extra skin warming is (more or less) beneficial during shorter surgical procedures, under normothermic conditions or in selected patients with co-morbidities (i.e. obesity, heart failure).

The amount of postoperative blood loss varies largely among different centres and reproduction of the study results depends on the adoption of similar medical management procedures. All patients were given nitroglycerin and nitroprusside which may cause impairment of lung oxygenation and exert an antiplatelet effect via increase in cyclic guanine monophosphate and reduction of proaggregatory endoperoxide and thromboxane A₂.²⁵ However, nitrate-induced postoperative bleeding is questionable in the context of major surgery as high plasma concentrations of epinephrine have been shown to reverse nitrate-induced platelet inhibition.²⁶

Although we observed a similar haemodynamic pattern in heated and control patients, we did not evaluate myocardial ischaemia or plasma concentrations of catecholamines which could provide a better indication of hypothermia-induced stress. Nevertheless, in the context of cardiac surgery, it is likely that activation of the sympatho-adrenal and renin-angiotensin systems, in addition to the release of vasopressin in response to surgical trauma, largely overwhelmed the mild physiological response that could be attributed to a small difference in nasopharyngeal temperature (0.8 °C).^{27,28}

In summary, mild postoperative hypothermia is known to be associated with bleeding diathesis and increased oxygen demand. Our data suggest that intraoperative skin warming with a water mattress and forced warm air over the patient's face, neck and shoulders is a simple and efficient means of reducing the temperature afterdrop after hypothermic CPB and reducing postoperative blood loss. Complete rewarming of the body contributes to improved quality of perioperative care, allowing fast recovery while potentially reducing perioperative respiratory morbidity.²⁹ In our institution, the standard perioperative management for most cardiac surgical patients has evolved with the use of additional skin warming and short-acting anaesthetics. Such strategies are aimed at achieving earlier tracheal extubation and shorter duration of ICU stay, thereby reducing medical costs.

References

1. Editorial. Perioperative hypothermia. *Lancet* 1991; **338**: 547–549.
2. Spaniol SE, Bond EF, Brengelmann GL, Savage M, Pozos RS. Shivering following cardiac surgery: predictive factors, consequences and characteristics. *American Journal of Critical Care* 1994; **3**: 356–367.
3. Kurz A, Sessler DI, Lenhardt R. Perioperative normothermia to reduce the incidence of surgical-wound infection and shorten hospitalization. Study of Wound Infection and Temperature Group. *New England Journal of Medicine* 1996; **334**: 1209–1215.
4. Kurz A, Sessler DI, Narzt E, Bekar A, Lenhardt R, Huemer G, Lackner F. Postoperative hemodynamic and thermoregulatory consequences of intraoperative core hypothermia. *Journal of Clinical Anesthesia* 1995; **7**: 359–366.
5. Frank SM, Beattie C, Christopherson R, Norris EJ, Perler BA, Williams GM, Gottlieb SO. The Perioperative Ischemia Randomized Anesthesia Trial Study Group. Unintentional hypothermia is associated with postoperative myocardial ischemia. *Anesthesiology* 1993; **78**: 468–476.
6. Schmied H, Kurz A, Sessler DI, Kozek S, Reiter A. Mild hypothermia increases blood loss and transfusion requirements during total hip arthroplasty. *Lancet* 1996; **347**: 289–292.
7. Davis FM, Parimelazhagan KN, Harris EA. Thermal balance during cardiopulmonary bypass with moderate hypothermia in man. *British Journal of Anaesthesia* 1977; **49**: 1127–1132.
8. Ramsay JG, Ralley FE, Whalley DG, Delli Colli P, Wynands JE. Site of temperature monitoring and prediction of afterdrop after open heart surgery. *Canadian Journal of Anaesthesia* 1985; **32**: 607–612.
9. Noback CR, Tinker JH. Hypothermia after cardiopulmonary bypass in man: amelioration by nitroprusside-induced vasodilation during rewarming. *Anesthesiology* 1980; **53**: 277–280.
10. Joachimsson P-O, Nyström S-O, Tyden H. Postoperative ventilatory and circulatory effects of extended rewarming during cardiopulmonary bypass. *Canadian Journal of Anesthesia* 1989; **36**: 9–19.
11. Joachimsson P-O, Nyström S-O, Tyden H. Heating efficacy of external heat supply during and after open-heart surgery with hypothermia. *Acta Anaesthesiologica Scandinavica* 1987; **31**: 73–80.
12. Joachimsson P-O, Nyström S-O, Tyden H. Postoperative ventilatory and circulatory effects of heating after aortocoronary bypass surgery: extended rewarming during cardiopulmonary bypass and postoperative radiant heat supply. *Acta Anaesthesiologica Scandinavica* 1987; **31**: 543–549.
13. Harrison SJ, Ponte J. Convective warming combined with vasodilator therapy accelerates core rewarming after coronary artery bypass surgery. *British Journal of Anaesthesia* 1996; **76**: 511–514.
14. Sessler DI. Perianesthetic thermoregulation and heat balance in humans. *FASEB Journal* 1993; **7**: 638–644.
15. Chiara O, Giomarelli PP, Biagioli B, Rosi R, Gattinoni L. Hypermetabolic response after hypothermic cardiopulmonary bypass. *Critical Care Medicine* 1987; **15**: 995–1000.
16. Sessler DI, Moayeri A. Skin-surface warming: heat flux and central temperature. *Anesthesiology* 1990; **73**: 218–224.
17. Kempen PM. Full body forced air warming: commercial blanket vs air delivery beneath bed sheets. *Canadian Journal of Anaesthesia* 1996; **43**: 1168–1174.
18. Kurz A, Kurz M, Poeschl G, Faryniak B, Redl G, Hackl W. Forced-air warming maintains intraoperative normothermia better than circulating-water mattresses. *Anesthesia and Analgesia* 1993; **77**: 89–95.
19. Reed RL, Johnson TD, Hudson JD, Fisher RP. The disparity between hypothermic coagulopathy and clotting studies. *Journal of Trauma* 1992; **33**: 465–470.
20. Rohrer MJ, Natale AM. Effect of hypothermia on the coagulation cascade. *Critical Care Medicine* 1992; **20**: 1402–1405.
21. Staab DB, Sorensen VJ, Fath JJ, Raman SB, Horst HM, Obeid FN. Coagulation defects resulting from ambient temperature-induced hypothermia. *Journal of Trauma* 1994; **36**: 634–638.
22. Yoshihara H, Yamamoto T, Mihara H. Changes in coagulation and fibrinolysis occurring in dogs during hypothermia. *Thrombosis Research* 1985; **37**: 503–512.
23. Valeri CR, Feingold H, Cassidy G, Ragno G, Khuri S, Altschule MD. Hypothermia-induced reversible platelet dysfunction. *Annals of Surgery* 1987; **205**: 175–181.
24. Valeri CR, Khabbaz K, Khuri SF, Marquardt C, Ragno G, Feingold H, Gray AD, Axford T. Effect of skin temperature on platelet function in patients undergoing extracorporeal bypass. *Journal of Thoracic and Cardiovascular Surgery* 1992; **104**: 108–116.
25. Knight CJ, Panesar M, Wilson DJ, Chronos NA, Patel D, Fox K, Goodall AH. Different effects of calcium antagonists, nitrates, and beta-blockers on platelet function: possible importance for the treatment of unstable angina. *Circulation* 1997; **95**: 125–132.
26. Harris SN, Rinder CS, Rinder HM, Tracey JB, Smith BR, Hines R. Nitroprusside inhibition of platelet function is transient and reversible by catecholamine priming. *Anesthesiology* 1995; **83**: 1145–1152.
27. Frank SM, Higgins MS, Breslow MJ, Fleisher LA, Gorman RB, Sitzmann JV, Raff H, Beattie C. The catecholamine, cortisol, and hemodynamic responses to mild perioperative hypothermia. A randomized clinical trial. *Anesthesiology* 1995; **82**: 83–92.
28. Taggart DP, Fraser W, Gray CE, Beastall G, Shenkin A, Wheatley DJ. The effects of systemic intraoperative hypothermia on the acute-phase and endocrine response to cardiac surgery. *Thoracic and Cardiovascular Surgery* 1992; **40**: 74–78.
29. Johnson D, Thomson D, Mycyk T, Burbridge B, Mayers I. Respiratory outcomes with early extubation after coronary artery bypass surgery. *Journal of Cardiothoracic and Vascular Anesthesia* 1997; **11**: 474–480.