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Active Warming during Preanesthetic Period Reduces Hypothermia without Delay of Anesthesia in Cardiac Surgery

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Background: Intra-operative hypothermia adversely affects hemodynamics and post-operative recovery in cardiac surgery patients. This study evaluated the efficacy of active warming during the preanesthetic period on the prevention of intraoperative hypothermia in cardiac surgery patients.

Methods: After gaining the approval of Institutional Review Board and informed consent from the patients, sixty patients undergoing cardiac surgery were divided into control and prewarming group. The control group (n = 30) were managed with warm mattresses and cotton blankets, whereas the prewarming group (n = 30) were actively warmed with a forced-air warming device before anesthesia. Hemodynamic variables and temperature were recorded before anesthesia (Tpre) and at 30 min intervals after anesthesia (T30, T60, and T90).

Results: Before anesthesia, skin temperature was significantly higher in the prewarming group than in the control group. At T90, core temperature was significantly higher in the prewarming group than in the control group. Intraoperative hypothermia (core temperature < 35.5°C) developed by T90 in 78% of patients in the control group and 44% of patients in the prewarming group. Moreover, temperatures below 35°C developed in 58% of the conrol group and 17% of the prearming group.

Conclusions: Active warming just before anesthesia reduced the incidence and degree of hypothermia in patients undergoing cardiac surgery, with no delay of anesthesia. **(Korean J Anesthesiol 2005; 48: S 5～10)**

Key Words: forced-air warming, hypothermia, off-pump coronary artery bypass graft, prewarming, temperature.

INTRODUCTION

In patients undergoing cardiac surgery, unintentional systemic hypothermia causes peripheral vasoconstriction and thus increases myocardial afterload, and coagulopathy and postoperative bleeding can also occur.[1,2] In the field of cardiac surgery, the performance of off-pump coronary artery bypass graft surgery (OPCAB) has become more frequent, and active temperature control management has become one of the important parts of anesthetic management for OPCAB.[3,4] However, elevating room temperature, warming intravenous fluids, and heating ventilator gases have limited effect.[2,5,6]

There have been numerous reports that active warming before anesthesia reduces the core-to-peripheral tissue temperature gradient and the propensity for heat redistribution after the induction of anesthesia and as a result prevents intraoperative hypothermia.[7-9] Previous studies have shown that at least 1-2 hours of prewarming prevented intraoperative hypothermia but was considered as impractical.[8] Recently, it has been reported that a brief period of prewarming, for example 15 minutes, would be easy to accommodate and could be combined with intraoperative warming,[9] which is undoubtedly effective after the redistribution period.

This study was designed to evaluate the efficacy of active preoperative warming with forced air on the development of intraoperative hypothermia in patients undergoing cardiac surgery.

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MATERIALS AND METHODS

After obtaining Research Ethics Committee approval and written, informed consent from patients, 60 patients undergoing cardiac surgery were allocated to two groups. Patients in the control group (n = 30) were scheduled for cardiac surgery with cardiopulmonary bypass, and before and during induction of anesthesia were exposed to room temperature without forced-air warming. The patients in the prewarming group (n = 30) were scheduled for OPCAB were actively warmed with a forced air warming device (Bair Hugger, Augustine Medical Inc., Eden Prairie, MN, USA) before induction of anesthesia. After these patients arrived in the operating room, forced warm air (at 40-42°C) created a high velocity airflow over the legs and trunk under a simple blanket. An airway heater humidifier (34.5-35.5°C) was connected to the breathing circuit after anesthesia in the prewarming group. In both groups, patients were laid on a mattress containing circulating water at 38°C. Patients were excluded who had clinically significant peripheral vascular disease, a history of fever or infection within a week before surgery, or known concomitant life-threatening or debilitating disease of non-cardiac origin requiring vasoconstrictors for blood pressure maintenance.

All recent cardiac medications except digoxin and diuretics were given untill the morning of surgery. As premedication, 0.05 mg/kg of morphine was injected intramuscularly one hour before the surgery. In the operating room, EKG leads II and V5 were monitored and the radial artery was cannulated for continuous monitoring of arterial blood pressure and blood gas analysis.

For continuous cardiac output (CO) and mixed venous oxygen saturation (SvO_2) monitoring, pulmonary artery catheter (Swan-Ganz, CCOmbo, Baxter Healthcare Co., Irvine, CA, USA) was inserted through the right internal jugular vein before anesthesia. A skin temperature probe was placed on the right index finger immediately upon arrival of the patient at the operating room and temperature was monitored using an integrated modular device (HP/M1165A, Hewlett Packard Co., Andover, MA, USA). Blood temperature measured by the pulmonary artery catheter was monitored as core temperature. The anesthetic technique was standardized for all patients. Anesthesia was induced with 2.0-3.0 mg of midazolam, 1.0-3.0μg/kg of sufentanil, and 50 mg of rocuronium and maintained with 0.2-0.5 vol% of isoflurane, continuous intravenous infusion of of sufentanil at 0.5-1.5μg/kg/min. Ventilation was controlled with an oxygen-air mixture (FiO_2 0.6) to maintain end-tidal CO_2 at 35-38 mmHg. Patients were also warmed by a mattress through which warm water was circulating and the lower limbs were wrapped with an aseptic forced-air warming blanket after the vein harvest.

Temperature and hemodynamic variables were recorded just after the insertion of pulmonary artery catheter and just before induction of anesthesia (baseline, Tpre), and then at 30 minutes intervals until 90 minutes postinduction (T30, T60, and T90). Hypothermia was defined as a core temperature of below 35.5°C. Hemodynamic variables including heart rate (HR), mean arterial pressure (MAP), central venous pressure (CVP), mean pulmonary artery pressure (MPAP), and CO were recorded and the cardiac index (CI) and systemic vascular resistance index (SVRI) were calculated. The temperature and all hemodynamic variables were expressed as the mean ± standard deviation (SD). Data were analyzed with SPSS for Windows, release 10.0 (SPSS Inc., Chicago, IL, USA). Temperature and hemodynamic changes by time intervals were compared to baseline value using repeated measures of ANOVA. The comparisons of variables between the two groups were performed with unpaired t-tests and Fisher's exact tests. The P values $<$ 0.05 were considered statistically significant.

RESULTS

Demographic data are listed in Table 1. There were no significant differences between the two groups in sex, preoperative left ventricular ejection fraction, body surface area or incidence of preoperative co-morbid risk factors. Patients in the prewarming group were older than in the control group (P = 0.001). The numbers of patients preoperatively medicated with β-blockers and calcium channel blockers was greater in the prewarming group than in the control group (P = 0.017 and 0.001, respectively).

The temperature changes are shown in Table 2. There was no significant difference to these in the periods between the arrival of patients in the operation room and induction of anesthesia (47.0 ± 9.5 and 49.3 ± 8.7 minutes in the control and prewarming groups, respectively, P = 0.333). Core temperature had decreased significantly compared with Tpre by T30, T60 and T90 in both groups. Moreover, core temperature was significantly lower at T90 in the control group than in the

prewarming group. Skin temperature was not significantly changed in either group but it was significantly lower in the control group than in the prewarming group at Tpre, T30 and T60. The difference between core and skin temperature was significantly less in the control group at T90, but was not changed in the prewarming group. The temperature differences at Tpre and T30 were significantly grater in the control group than in the prewarming group.

Hemodynamic data are shown in Table 3. CI was significantly greater at Tpre in the control group than in the prewarming group. CI had decreased significantly at T30 and T60 in the control group but had not changed in the prewarming group. MAP was significantly lower in the control group than in the prewarming group at T60, and had decreased significantly at T30, T60 and T90 compared with Tpre in both groups. CVP was significantly lower in the control group than in the prewarming group at T60 and T90 and had decreased significantly at T30, T60 and T90 compared with Tpre in both groups. MPAP was significantly greater in the control group than in the prewarming group at Tpre, T30 and T60 and had increased significantly at T30, T60 and T90 compared with Tpre in the prewarming group. HR was significantly greater in the control group than in the prewarming group at Tpre and T90. Compared with Tpre, HR was significantly lower at T30, T60 and T90 in the control group, and at T30 and T90 in the prewarming group. SVRI had decreased significantly at T30 and T90 compared with Tpre in the prewarming group but remained unchanged in the control group. However, there was no significant difference in SVRI between the two groups. Although the incidence of intraoperative hypothermia was not significantly different at T90 between control (78%) and prewarming groups (44%), the incidence of patients with core temperature below 35°C was significantly greater at T90 in the control group (58%) than in the prewarming group (17%).

Table 1. Demographic Data

Variables	Control (n = 30)	Prewarming (n = 30)
Patients characteristics		
Age (yr)	53.3 ± 14.8	63.5 ± 7.7*
Sex (M/F)	20/10	19/11
Left ventricular ejection fraction (%)	62.2 ± 8.3	58.4 ± 12.7
Body surface area (m^2)	1.70 ± 0.15	1.70 ± 0.20
Disease (n)		
Coronary	5	30
Valvular	25	
Preoperative medications (n)		
β-blockers	11	18*
Calcium channel blockers	3	13*
Angiotensin converting enzyme inhibitors	12	10
Co-morbid risk factors (n)		
Hypertension	6	7
Diabetes mellitus	3	5
Previous myocardial infarction	0	3

Values are mean ± SD or number of patients. *: P < 0.05 compared with control group.

DISCUSSION

This study demonstrated that active prewarming with a forced-air warming device elevated skin temperature and decreased the difference between core and skin temperatures. Elevated peripheral temperature with active prewarming seemed to play an important role in maintaining intraoperative core temperature, probably due to a reduction in the redistribution

Table 2. Changes of Skin and Core Temperature

		Tpre	T30	T60	T90
Skin temperature (°C)	Control	26.5 ± 4.6	26.4 ± 5.0*	27.4 ± 4.1*	28.2 ± 3.6*
	Prewarming	30.8 ± 2.8	30.0 ± 3.8	30.6 ± 4.0	30.0 ± 3.7
Core temperature (°C)	Control	36.8 ± 0.4	36.0 ± 0.6*	35.6 ± 0.6*	35.1 ± 0.7*
	Prewarming	36.7 ± 0.3	36.2 ± 0.4*	35.6 ± 0.9*	35.5 ± 0.6*
Difference between core and skin temperature (°C)	Control	10.4 ± 4.8	9.7 ± 5.2	8.2 ± 4.4	6.9 ± 4.0*
	Prewarming	5.9 ± 2.8	6.5 ± 3.8	6.1 ± 4.0	5.8 ± 3.9

Values are mean ± SD. Tpre (baseline): before induction of anesthesia, T30: 30 minutes after induction of anesthesia, T60: 60 minutes after induction of anesthesia, T90: 90 minutes after induction of anesthesia. *: P < 0.05 compared with baseline value.

Table 3. Hemodynamic Changes

		Tpre	T30	T60	T90
Cardiac index (L/min/m^2)	Control	3.6 ± 1.5	2.8 ± 1.1*	2.7 ± 1.0*	2.9 ± 1.0
	Prewarming	2.9 ± 0.8†	2.7 ± 0.6	2.6 ± 0.4	2.7 ± 0.5
Mean arterial pressure (mmHg)	Control	92 ± 17	79 ± 14*	81 ± 11*	79 ± 13*
	Prewarming	94 ± 14	83 ± 12*	85 ± 12*†	86 ± 13*
Central venous pressure (mmHg)	Control	6 ± 3	10 ± 3*	10 ± 3*	9 ± 2*
	Prewarming	7 ± 2	10 ± 3*	12 ± 3*†	12 ± 2*,†
Mean pulmonary arterial pressure (mmHg)	Control	23 ± 10	25 ± 8	25 ± 8	24 ± 7
	Prewarming	18 ± 6†	21 ± 6*†	21 ± 4*†	22 ± 4*
Heart rate (beats/min)	Control	78 ± 16	60 ± 9*	65 ± 11*	68 ± 12*
	Prewarming	68 ± 19†	59 ± 8*	61 ± 10	59 ± 7*
Systemic vascular resistance index (dyne · s/cm^5/m^2)	Control	2259 ± 1137	2234 ± 1025	2305 ± 691	2122 ± 696
	Prewarming	2660 ± 820	2208 ± 590*	2337 ± 573	2216 ± 538*

Values are mean ± SD. Tpre (baseline): before induction of anesthesia, T30: 30 minutes after induction of anesthesia, T60: 60 minutes after induction of anesthesia, T90: 90 minutes after induction of anesthesia. *: $P < 0.05$ compared with baseline value. †: $P < 0.05$ compared with control value.

of heat from the core to the periphery and resultant decrease in core temperature during surgery.[2,6,8-12] Prewarming also decreased systemic vascular resistance and prevented the decrease in cardiac index after anesthesia that was observed in the control group.[13-16]

Hypothermia occurs during general anesthesia because of redistribution of heat from the core to the periphery, decreased metabolic heat production, impaired thermoregulation, and heat loss to the cold operating room environment. Although there are limited data on temperature management during OPCAB, a rapidly expanding surgical field, the adverse effects of hypothermia, are well known. They include impaired coagulation, decreased immune response, increased norepinephrine levels, peripheral vasoconstriction, and compensatory oxygen requirements during warming which increase perioperative morbidity.[1,2] Applying special devices to cardiac patients has been reported to be effective for temperature management.[7,14-17] However, these devices lead to additional cost. In contrast, a convective forced-air warming system is easy to operate and a cost effective means to maintain a patient's intraoperative temperature when used during the preanesthetic period.

Core-to-peripheral flow of heat is mediated by blood-borne convection of heat and conduction of heat into adjacent tissues and redistribution of heat is affected by the core-to-peripheral temperature gradient. In association with general anesthesia, the core body temperature usually drops 0.5°C to 1.5°C during the first hour after the induction of general anesthesia.[18] There have been reports that redistribution of heat can be prevented by cutaneous preanesthetic warming, which markedly increases peripheral tissue heat content.[9-19] Prewarming reduces the core-to-peripheral tissue temperature gradient and the propensity for heat redistribution after the induction of anesthesia. In this study, skin temperature was maintained at a significantly higher level and the core-to-peripheral temperature discrepancy was less in the prewarming group than in the control group even before anesthesia, and then throughout the whole study period. As a result, core temperature had been maintained above 35.5°C by T90 in the prewarming group but not in the control group. This is likely to be associated with less internal redistribution of body heat, which decreases core temperature, and a proportionally increased temperature of peripheral tissues under general anesthesia in the prewarming group.[20,21] When compared to baseline values, skin temperature significantly increased after anesthesia in the control group, in contrast to no significant change in the prewarming group. Although there was no difference in the number of patients whose core temperature decreased below 35.5°C through the whole study period, the number of patients whose core temperature had decreased below 35.0°C by T90 was significantly less in the prewarming group than in the control group ($P = 0.032$).

The convective forced-air warming system used in this study is easy to operate, and its efficacy has been reported even when it covers only one third of body surface.[6,7] Interestingly,

heat transfer has been shown to be effective as in this study when forced air is delivered beneath a simple bed sheet instead of using a commercial blanket whose rigid nature prevents a close approximation to non-planar anatomical structures and whose pinhole gridwork pattern causes low flow velocity. Heat loss to the cold operating room environment is also an important factor in the development of hypothermia during general anesthesia,[22] and the forced-air warming device played an important role in maintaining preanesthetic skin temperature in the prewarming group. Although an airway heater humidifier was used in the prewarming group during the operation, airway heating and humidification was reported to have little influence on core temperature[2] and therefore, this intra-operative warming did not seem to significantly influence the result. Heating with a circulating-water mattress was also reported as unlikely to be effective in maintaining core temperature during general anesthesia.[2] Therefore, prevention of intra -operative hypothermia in the prewarming group can be attributed to both active forced-air warming and avoiding cold environment before anesthesia rather than the airway heater humidifier and heating with a circulating-water mattress.

Previous studies have suggested that 1-2 hours, or as little as 30 minutes, of prewarming is required for clinical benefit.[3,8,9] However, an hour or more of prewarming dose not suit the clinical routine of most hospitals, so prewarming has not been commonly used. Recently, there was a report that a brief period of prewarming, (for example 15 minutes), would be easy to accommodate and could be combined with intra-operative warming.[9] The recent study also showed that in patients undergoing cardiac surgery, if active prewarming is started immediately after the patient's arrival at the operating room and continued untill the induction of anesthesia, it is helpful in the prevention of intraoperative hypothermia.

This study was not a prospective randomized, blinded study and the resultant difference in patients' characteristics was a limitation of this study. In the preliminary study, prewarming was associated with the patient's intraoperative temperature and patients who underwent OPCAB without active prewarming frequently developed intraoperative hypothermia. Since most coronary bypass graft surgery was performed using OPCAB and temperature management was critical during OPCAB, patients with coronary disease were assigned to the prewarming group. The body surface area, extreme age of the patients,[23,24] the size of the surgical incision, and the presence of neuropathy are known as risk factors for intraoperative hypothermia.[25] There was no difference in body size between the two groups and patients with neuropathy were not included in this study. The higher core temperature. despite old age and a higher incidence of preoperative medication with (β-blockers and calcium channel blockers[7]) in the prewarming group, supported the fact that prewarming is effective.

In this study, the distinguishing feature of prewarming was a higher skin temperature before anesthesia and that the surgical procedure was unlikely to affect core temperature significantly. SVRI decreased significantly at T30 and T90 only in the prewarming group. The decrease in SVRI contributed to the increase in CI through vasodilation, which was known as the beneficial effect of prewarming.

In conclusion, active warming with a forced-air warming system during the preanesthetic period reduced the differrebce between skin and core temperature and the development of hypothermia in patients undergoing cardiac surgery with, no delay of anesthesia. This method might be especially useful for maintaining temperature in patients undergoing OPCAB.

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