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Changes in body temperature during profound hypothermic cardiopulmonary bypass in adult patients undergoing aortic arch reconstruction

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Abstract

Purpose. Our aim was to characterize changes in body temperatures during profound hypothermic cardiopulmonary bypass (CPB) conducted with the sternum opened.

Methods. In ten adult patients who underwent profound hypothermic (<20°C) CPB for aortic arch reconstruction, pulmonary arterial temperature (PAT), nasopharyngeal temperature (NPT), forehead deep-tissue temperature (FHT), and urinary bladder temperature (UBT) were recorded every 1 min throughout the surgery. In addition, the CPB venous line temperature (CPBT), a reasonable indicator of mixed venous blood temperature during CPB and believed to best reflect core temperature during stabilized hypothermia on CPB, was recorded during the period of total CPB.

Results. PAT began to change immediately after the start of cooling or rewarming, closely matching the CPBT ($r = 0.98$). During either situation, the other four temperatures lagged behind PAT ($P < 0.05$); however, NPT followed PAT more closely than the other three temperatures ($P < 0.05$). During stabilized hypothermia, PAT, NPT, and FHT, but not UBT, closely matched the CPBT, with gradients of less than 0.5°C.

Conclusion. During induction of profound hypothermia and its reversal on total CPB with the heart in situ, a PA catheter thermistor, presumably because of its placement immediately behind the superior vena cava, would provide a reliable measure of the mixed venous blood temperature. During stabilized profound hypothermia, PAT, NPT, and FHT, but not UBT, serve as a reliable index of core temperature.

Key words Induced hypothermia · Deep hypothermia · Core temperature · Cardiopulmonary bypass · Thoracic aortic aneurysm

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Introduction

During profound hypothermic cardiopulmonary bypass (CPB) or circulatory arrest, it is essential to monitor body temperatures at several sites, to ensure that the organs vulnerable to decreased O_2 delivery actually receive the benefit of the desired degree of hypothermia, to assess evenness of cooling, and to diagnose hazardous hypothermia [1,2]. It is thus particularly important to use temperature monitoring sites most likely to reflect brain temperature. In this regard, pulmonary arterial, nasopharyngeal, tympanic, and distal esophageal monitoring sites have been used to estimate brain temperature during CPB [1–3]. However, each of these sites has unique problems [3], and none of them may reflect brain temperature reliably throughout the CPB [4]. In addition to core temperature monitoring, temperature monitoring at intermediate or peripheral zones (e.g., rectum, urinary bladder, muscle, skin) has been recommended to assess adequate whole body rewarming during CPB [5–7].

Stone et al. [4] previously demonstrated changes in temperatures measured at various standard monitoring sites (i.e., nasopharynx, esophagus, pulmonary artery, tympanic membrane, urinary bladder, rectum, axilla, sole of the foot) during profound hypothermic CPB conducted for repair of cerebral aneurysms without the sternum opened. They found that, among those monitoring sites, measurements from the nasopharynx, esophagus, and pulmonary artery tended to match brain temperature measured during neurosurgical procedures with the brain exposed to the cool surroundings. If the sternum had been opened, temperatures measured at some of those standard monitoring sites (e.g., pulmonary artery, esophagus) might have changed differently during the profound hypothermic CPB.

Less information is available regarding changes in body temperatures during profound hypothermic CPB conducted for intrathoracic procedures with the

sternum opened. In this study, we therefore investigated changes in pulmonary arterial, nasopharyngeal, forehead deep-tissue, urinary bladder, and fingertip skin-surface temperatures during profound hypothermic CPB in adult patients with thoracic aortic aneurysms who underwent aortic arch reconstruction. The CPB venous line temperature is a reliable indicator of the mixed venous blood temperature during CPB, and is believed to best reflect core temperature (i.e., brain temperature) during CPB when no active core warming or cooling is occurring (i.e., during stabilized hypothermia) [1,2]. Thus, in order to help characterize changes in the above temperatures during hypothermic CPB, we compared them with the CPB venous line temperature.

Patients and methods

With institutional approval and informed consent, we studied ten adult patients with thoracic aortic aneurysm who underwent profound deep hypothermic (<20°C) CPB and circulatory arrest for aortic arch reconstruction. The patient demographics are summarized in Table 1.

The patients were premedicated with oral nitrazepam (2–7.5 mg) and roxatidine acetate hydrochloride (75 mg) 90 min before entering the operating room. Anesthesia was induced with intravenous midazolam (40–150 $\mu g\,kg^{-1}$) and fentanyl (2–10 $\mu g\,kg^{-1}$), and tracheal intubation was facilitated with vecuronium bromide (5–10 mg i.v.). Anesthesia was subsequently maintained with midazolam, fentanyl, and sevoflurane (0.5%–3%) in oxygen. The lungs were ventilated mechanically to maintain Pa_{CO_2} at approximately 35 mmHg.

In addition to the standard anesthetic safety monitors, radial, femoral, and pulmonary arterial catheters and transesophageal echocardiography were used to monitor cardiovascular functions. Pulmonary arterial temperature (PAT) was monitored with a thermistor at the tip of a thermodilution catheter placed in the right pulmonary artery (Swan-Ganz CCOmbo CCO/Sv_{O_2}/VIP; Edwards Lifesciences LLC, Irvine, CA, USA), the placement of which was confirmed by preoperative chest X-ray in every patient. Nasopharyngeal temperature (NPT) was monitored by placing a thermistor probe in the posterior nasopharynx (~5 cm from the external naris), and sealing the external naris with cotton gauze. Forehead deep-tissue temperature (FHT) was monitored by placing a 4.5-cm-diameter sensor probe designed for the measurement of deep-tissue temperature (PD-11; Terumo, Tokyo, Japan) on the forehead. Urinary bladder temperature (UBT) was monitored using a thermistor-tipped urinary bladder catheter (Respiratory Support Products, Irvine, CA, USA). Fingertip skin-surface temperature (FSST) was monitored by placing a thermistor probe on the tip of the index finger opposite the nail bed and surrounding the probe with gauze folded in eight (~5 mm in thickness). Mixed venous blood temperature during total CPB was monitored using a thermistor probe (Avecor Cardiovascular, Plymouth, MN, USA) placed in the CPB venous line. All temperature sensors were interfaced with electronic thermometers (AA-900P thermometer [Nihon Kohden, Tokyo, Japan] for pulmonary arterial, nasopharyngeal, forehead deep-tissue, urinary bladder, and fingertip skin-surface temperatures; and a YSI Precision 4000A thermometer [Nikkiso-YSI, Tokyo, Japan] for CPB venous line temperature) whose synchronous digital output was continuously displayed. The data derived from the AA-900P thermometer were electronically sampled and stored at 1-min intervals, while the data displayed on the YSI Precision 4000A thermometer were manually recorded at several (3 to 5)-min intervals. The operating room temperature was thermostatically maintained at 20°C.

Patients were anticoagulated with heparin (300 $U \cdot kg^{-1}$) and subsequent doses were titrated to keep the activated clotting times above 450 s. A 28-Fr cannula (DLP Single Stage Venous Cannula; Medtronics, Minneapolis, MN, USA) and a 32-Fr cannula (Venous Return Catheter; Polystan A/S, Walgerholm, Denmark) were passed into the superior vena cava and inferior vena cava, respectively, via the right atrium. Roller pumps with a membrane oxygenator were used to achieve a 2.5 $l \cdot min^{-1} \cdot m^{-2}$ extracorporeal flow via a 10-mm Microvel vascular graft (Hemashield Gold; Boston Scientific, Natick, MA, USA) sutured to the femoral artery. The bypass circuit was primed using acetated Ringer solution (Veen-F; Nikken Kagaku, Tokyo, Japan) with or without adding packed red blood cells to maintain hematocrit between 20% and 25%.

Profound hypothermia (NPT <20°C) was rapidly (~20 min) induced with a separate water-bath heat exchanger which was initially set at 10°C (Table 2). After NPT became lower than 20°C, the temperature of the water bath was allowed to rise to 15°C–20°C, where it was maintained until rewarming began. After induction

Table 1. Patient demographics

Variable	Values
Age (years)	68 ± 7 (55–78)[a]
Sex (F/M)	4/6[b]
Height (cm)	158.6 ± 9.4 (143.0–171.3)[a]
Weight (kg)	61.3 ± 12.0 (48.8–89.1)[a]
ASA-PS (II/III/IV)	2/4/4[b]

ASA-PS, Physical status defined by the American Society of Anesthesiologists
[a]Mean ± SD (min–max)
[b]Number of patients

Table 2. Data on cardiopulmonary bypass and hypothermia

Variable	
Time from induction of anesthesia (min)	
Beginning of active core cooling	184 ± 59 (80–270)
Time from the beginning of active core cooling (min)	
Pulmonary arterial blood temperature ≤20°C	14 ± 5 (8–25)
Nasopharyngeal temperature ≤20°C	25 ± 9 (16–44)
Ventricular fibrillation	8 ± 4 (3–15)
Retrograde cerebral perfusion	62 ± 42 (32–156)
Active core warming	107 ± 52 (45–175)
Duration (min)	
Retrograde cerebral perfusion (circulatory arrest)	34 ± 8 (22–50)
Cardiopulmonary bypass	226 ± 59 (133–326)
Clamping of the aorta	93 ± 54 (23–170)
Time from the beginning of active core warming (min)	
Pulmonary arterial blood temperature ≥35°C	52 ± 29 (22–105)
Nasopharyngeal temperature ≥35°C	62 ± 24 (41–111)
Temperature when ventricular fibrillation occurred (°C)	
Pulmonary arterial blood temperature	26.8 ± 2.7 (23.7–32.9)
Nasopharyngeal temperature	30.6 ± 2.4 (28.4–35.6)

Values are means ± SD (min–max)

of the profound hypothermia, cold (4°C) crystalloid cardioplegic solution, containing 20 mEq·l^{-1} KCl, was infused into the coronary circulation to arrest the heart (Table 2). During aortic arch reconstruction, the circulation was arrested, except for the cerebral circulation, into which the hypothermic CPB perfusate was infused retrograde at a rate of 150–200 ml·min^{-1} (with central venous pressure raised to 10–15 mmHg) via the 28-Fr cannula placed in the superior vena cava. The CPB was resumed after the aortic arch was reconstructed, and the hypothermia was gradually reversed on CPB (Table 2). During the rewarming period, the heat exchanger was initially set at 39°C and later adjusted downward. In addition, a water-filled heating mattress was activated at 39°C. When UBT became higher than 36°C, the CPB was terminated and protamine given.

Temperatures at the four standard core temperature monitoring sites (i.e., PAT, NPT, FHT, and UBT) were compared with the CPB venous line temperature, using correlation coefficients and Bland and Altman analyses. In these analyses, the data obtained during the first 20 and 60 min of the cooling and rewarming, respectively, were used. Because the data on UBT were relatively variable, we also investigated the relation between urine volume and the changes in UBT during either cooling or rewarming, using simple (either linear or non-linear) regression analyses. The temperature-time relationship during either cooling or rewarming was analyzed using analysis of variance (ANOVA), the Tukey-Kramer test (in case of homogeneous population variances), and the Games-Howell test (in case of heterogeneous population variances). Comparison of the correlation coefficients was made using Fisher's Z-transformation. Any other necessary comparisons between two groups were made by either two-tailed, unpaired Student's *t*-test (in case of homogeneous population variances) or Welch's *t*-test (in case of heterogeneous population variances).

All the above analyses were made on a computer, using GB-Stat v 6.5.6 PPC (Dynamic Microsystems, Silver Spring, MD, USA), PowerStats v 0.9 (Shinko Trading, Tokyo, Japan), or Excel (Microsoft, Redmond, WA, USA). Differences were considered significant at $P < 0.05$. Values were expressed as either means ± SD, means ± 2SD (in the Bland and Altman analyses) or means ± SEM (for clarity in the temperature-time relationship).

Results

Changes in body temperatures during cooling

Temperatures monitored at the four standard core temperature monitoring sites (i.e., PAT, NPT, FHT, and UBT) were closely matched ($P > 0.05$) until the cooling was started (Fig. 1). However, only PAT began to decrease immediately after the start of the cooling (Fig. 1), and NPT, FHT, UBT, and FSST significantly lagged ($P < 0.05$) behind PAT, with lag times of one to several min (Fig. 1). NPT followed PAT more closely ($P < 0.05$) than FHT, UBT, or FSST (Fig. 1). PAT and NPT were significantly lower ($P < 0.05$) than either FHT or UBT at all time points after 3 min and 18 min, respectively, of the cooling (Fig. 1). However, no significant differences ($P > 0.05$) were noted in the temperature-time relationship between FHT and UBT (Fig. 1).

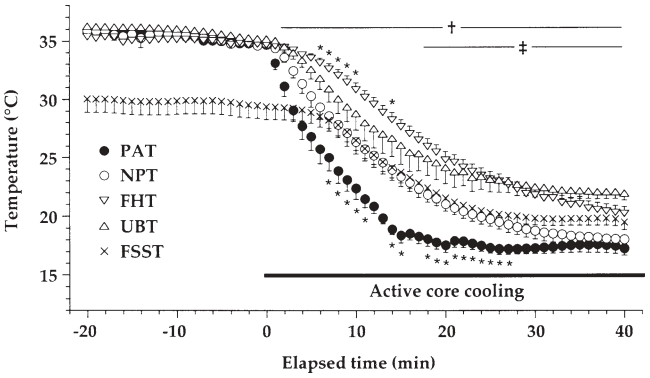

Fig. 1. Changes in body temperatures during active cooling on total cardiopulmonary bypass (CPB). The results of statistical analyses of fingertip skin-surface temperature are omitted for clarity. *PAT*, pulmonary arterial temperature; *NPT*, nasopharyngeal temperature; *FHT*, forehead deep-tissue temperature; *UBT*, urinary bladder temperature; *FSST*, fingertip skin-surface temperature. *$P < 0.05$ vs NPT; †$P < 0.05$, PAT vs FHT, UBT; ††$P < 0.05$, NPT vs FHT, UBT

During the cooling, PAT, NPT, FHT, and UBT were all significantly correlated with the CPB venous line temperature (Fig. 2). Among them, PAT was best correlated with the CPB venous line temperature (Fig. 2). The correlation coefficient for the CPB venous line-pulmonary arterial relation ($r = 0.977$) was significantly larger ($P < 0.05$) than that for the relation of the CPB venous line temperature with either NPT ($r = 0.925$), FHT ($r = 0.883$), or UBT ($r = 0.857$). However, no difference ($P > 0.05$) was observed in the correlation coefficients among the CPB venous line temperature-NPT, CPB venous line temperature-FHT, and CPB venous line temperature-UBT relations. In the Bland-Altman analyses (Fig. 2), the difference from the CPB venous line temperature was significantly lower for PAT (−0.21°C) than for either NPT (−2.6°C), FHT (−5.1°C), or UBT (−4.6°C) ($P < 0.05$). The SD of the difference between CPB venous line temperature and PAT (1.7°C) was smaller than that for the difference between CPB venous line temperature and either NPT (2.5°C), FHT (3.2°C), or UBT (3.4°C).

Comparison of body temperatures during stabilized profound hypothermia

During the period of stabilized hypothermia (i.e., for 20 min before the start of rewarming), PAT (18.5 ± 2.0°C), FHT (18.2 ± 1.6°C), and NPT (18.6 ± 1.8°C) closely matched the CPB venous line temperature (18.1 ± 1.8°C vs PAT, $P = 0.715$; vs FHT, $P = 0.893$; vs NPT, $P = 0.622$). However, UBT (20.0 ± 1.9°C) was significantly higher than the CPB venous line temperature (18.1 ± 1.8°C vs UBT; $P = 0.043$). In addition, as shown in Fig. 3, no significant differences were observed among PAT, FHT, and NPT. However, UBT was slightly, although significantly ($P < 0.05$), higher than these three temperatures (Fig. 3).

Changes in body temperatures during rewarming

Only PAT began to increase immediately after the start of the rewarming, and the other four temperatures significantly lagged ($P < 0.05$) behind PAT (Fig. 3). NPT followed PAT more closely ($P < 0.05$) than the other three temperatures (Fig. 3). As shown in Fig. 3, PAT and NPT were significantly higher ($P < 0.05$) than either FHT, UBT, or FSST at many time points during the rewarming. However, no significant differences ($P > 0.05$) were noted in the temperature-time relationship between FHT and UBT (Fig. 3).

During the rewarming, temperatures at the four standard core temperature monitoring sites were also significantly correlated with the CPB venous line temperature (Fig. 4). Again, among them, PAT was best correlated with the CPB venous line temperature (Fig. 4). The correlation coefficient for the CPB venous line temperature-PAT relation ($r = 0.981$) was significantly larger ($P < 0.05$) than that for the relation of the CPB venous line temperature with either NPT ($r = 0.946$), FHT ($r = 0.939$), or UBT ($r = 0.801$). In addition, the correlation coefficient for either the CPB venous line temperature-NPT relation or the CPB venous line temperature-FHT relation was significantly larger ($P < 0.05$) than that for the CPB venous line temperature-UBT relation. No significant difference ($P > 0.05$) was observed in the correlation coefficients between the CPB venous line temperature-NPT and CPB venous line temperature-FHT relations. In the Bland-Altman analyses, the difference from the CPB venous line temperature was significantly lower for PAT (−0.23°C) than for either NPT (−0.78°C), FHT (2.00°C), or UBT (2.3°C) ($P < 0.05$). The SD of the difference between CPB venous line temperature and PAT (1.6°C) was smaller than that of the difference between CPB venous line temperature and either NPT (2.2°C), FHT (2.3°C), or UBT (3.9°C) (Fig. 4).

Relation between changes in bladder temperature and urine flow rate

A significant linear relation was found between changes in UBT (y) and urine volume (x) during the first 30 min of cooling ($y = 10.7 + 0.0094x$; $r = 0.69$; $P = 0.03$; $n = 10$), but not during the first 60 min of rewarming ($y = 7.2 + 0.013x$; $r = 0.61$; $P = 0.06$; $n = 10$). No significant nonlinear (power, exponential, logarithmic, reciprocal) relation was found between changes in UBT and urine volume during either situation.

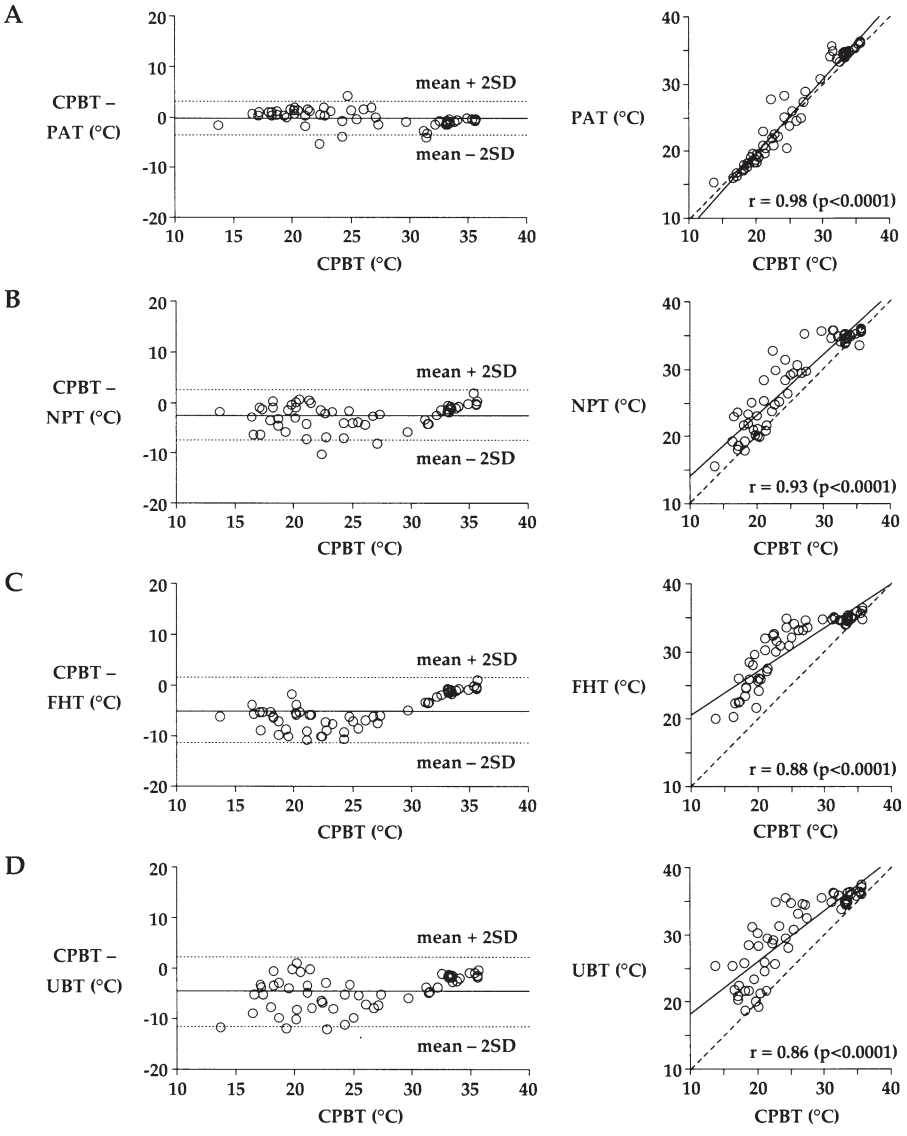

Fig. 2A–D. Bland and Altman comparison (*left*) and regression comparison (*right*) between the CPB venous line perfusate temperature and either pulmonary arterial (**A**), nasopharyngeal (**B**), forehead deep-tissue (**C**), or bladder (**D**) temperature during the first 20 min of active cooling on CPB. *CPBT*, cardiopulmonary bypass venous line temperature; *PAT*, pulmonary arterial temperature; *NPT*, nasopharyngeal temperature; *FHT*, forehead deep-tissue temperature; *UBT*, urinary bladder temperature

Discussion

The immediate response of PAT to the start of active core cooling or rewarming on CPB (i.e., acute changes in blood temperature) implies that PAT closely follows the changes in blood temperature during either the cooling or rewarming. Indeed, during the cooling or rewarming, PAT closely ($r = 0.98$) matched the CPB venous line temperature, a reliable estimate of the perfusing blood temperature during CPB. From another viewpoint, the CPB venous line temperature is a reasonable indicator of mixed venous blood temperature during CPB, and thus would reflect the average temperature within highly perfused organs during CPB. The highly perfused organs during CPB at lower hematocrit (~25%) would include brain, spinal cord, stomach, gut, liver, spleen, kidney, thyroid gland, skeletal muscles, and skin [8]. Because the specific heats of these organs and blood are almost identical (3.56–3.85 $kJ \cdot kg^{-1} \cdot °C^{-1}$) [9], their temperatures would change at similar rates during the cooling or rewarming. In other words, the changes in the CPB venous line temperature would closely reflect those in brain temperature. Indeed, it was previously shown in anesthetized sheep undergoing moderate hypothermia that, during either cooling or rewarming, the temperature of central venous blood in the right atrium closely matched brain temperature, measured with a thermometer inserted deep into the cerebral cortex via a small drill-hole in the skull [10]. Thus, PAT, which excellently correlated with the CPB venous line temperature during the cooling or rewarming, may possibly serve as a reliable index of brain temperature during active cooling or rewarming on CPB.

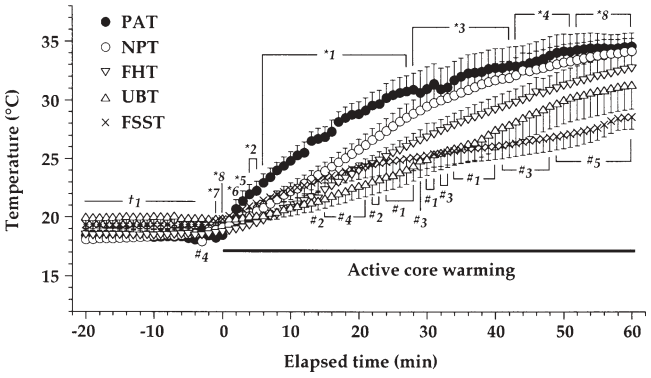

Fig. 3. Changes in body temperatures during reversal of profound hypothermia on CPB. *PAT*, pulmonary arterial temperature; *NPT*, nasopharyngeal temperature; *FHT*, forehead deep-tissue temperature; *UBT*, urinary bladder temperature; *FSST*, fingertip skin-surface temperature. $^{*1}P < 0.05$, PAT vs NPT, FHT, UBT, FSST; $^{*2}P < 0.05$, PAT vs NPT, FHT, UBT; $^{*3}P < 0.05$, PAT vs FHT, UBT, FSST; $^{*4}P < 0.05$, PAT vs UBT, FSST; $^{*5}P < 0.05$, PAT vs NPT, FHT; $^{*6}P < 0.05$, PAT vs FHT; $^{*7}P < 0.05$, PAT vs UBT; $^{*8}P < 0.05$, PAT vs FSST; $^{\#1}P < 0.05$, NPT vs FHT, UBT, FSST; $^{\#2}P < 0.05$, NPT vs FHT, UBT; $^{\#3}P < 0.05$, NPT vs UBT, FSST; $^{\#4}P < 0.05$, NPT vs UBT; $^{\#5}P < 0.05$, NPT vs FSST; $^{\dagger 1}P < 0.05$, UBT vs PAT, NPT, FHT

During total CPB, in spite of cessation of pulmonary blood flow and the intrathoracic procedure with the sternum opened, PAT closely matched the CPB venous line temperature (i.e., mixed venous blood temperature during CPB). We speculate that the temperature measured with the pulmonary arterial catheter thermistor closely reflected the temperature of venous blood flowing at a high rate in the superior vena cava (SVC), because the thermistor was placed in the right pulmonary artery immediately behind the SVC (Fig. 5). The SVC blood temperature would represent the average temperature within highly perfused (i.e., vessel-rich) regions of the upper body (i.e., brain, thyroid gland, skeletal muscles, and skin). Because of the aforementioned identity of specific heat among the highly perfused organs [9], the SVC blood temperature would be identical to the mixed venous blood temperature during CPB. Thus, it is conceivable that, in spite of cessation of pulmonary blood flow, PAT closely matched the CPB venous temperature in this study. During our measurements, the right pulmonary arterial segment where the thermistor was placed had not been exposed to the air with the heart in situ (i.e., without being overturned), and the pleural cavity or the pericardium was not filled with cold irrigating solution. Thus, PAT was, presumably, little influenced by the cool surroundings, closely matching the CPB venous line temperature.

Based on the assumption that PAT closely reflected changes in brain temperature (as discussed above), none of NPT, FHT, and UBT could be considered as a reliable index of brain temperature during rapid induction of profound hypothermia and its reversal on CPB. However, during stabilized profound hypothermia on CPB, both NPT and FHT, but not UBT, could be considered as a reliable index of brain temperature.

Posterior NPT has long been used to estimate core or brain temperature during hypothermic CPB in the clinical setting [5,11–13]. However, in earlier animal studies [10,14], as well as in a recent human study [4], NPT has been shown to modestly, although significantly, either overestimate or underestimate brain temperature (measured with a thermometer placed in the cerebral cortex) during rapid (20–40 min) cooling or rewarming. However, in those studies [4,10], during stabilized profound hypothermia, the NPT closely matched the brain temperature with gradients of less than 1.0°C. All these findings are not inconsistent with our findings.

Deep tissue temperature can be estimated using an insulated thermistor probe placed on the skin surface that creates an area of zero thermal flux between the skin surface and subcutaneous deep tissue [15–18]. In practice, the probe is insulated by electrically heating the upper surface of the probe and thereby eliminating thermal gradients between its upper and lower surfaces. This insulation would eventually lead to the creation of an area of zero thermal flux between the skin surface and subcutaneous deep tissue. Thus, utilizing this deep-tissue thermometry, brain temperature could be estimated noninvasively by placing the insulated thermistor probe at the forehead skin surface. However, the principle of deep-tissue thermometry suggests its slow responsiveness, and this thermometry may not be useful in detecting rapid changes in core temperature [19]. Indeed, this thermometry has been shown to be useful in estimating relatively slow changes in core temperature during general surgery [20], but not in estimating rapid changes in core temperature during the induction of moderate hypothermia (25°C–28°C) on CPB [19]. However, previous studies have yielded conflicting results regarding its usefulness in estimating changes in core temperature during reversal of moderate hypothermia on CPB [19,21,22].

This study, for the first time, investigated the possible usefulness of forehead deep-tissue thermometry in estimating core blood (or brain) temperature during profound hypothermic (≤20°C) CPB, i.e., during cooling, stabilization, and rewarming. In our patients, FHT significantly lagged behind PAT and NPT during either the cooling or rewarming, consistent with the previously reported discrepancy between NPT and FHT during the induction of moderate hypothermia (25°C–28°C) and its reversal on CPB [19]. As inferred from its principle, deep-tissue thermometry would fail to exteriorize the deep-tissue temperature if the ambient temperature is

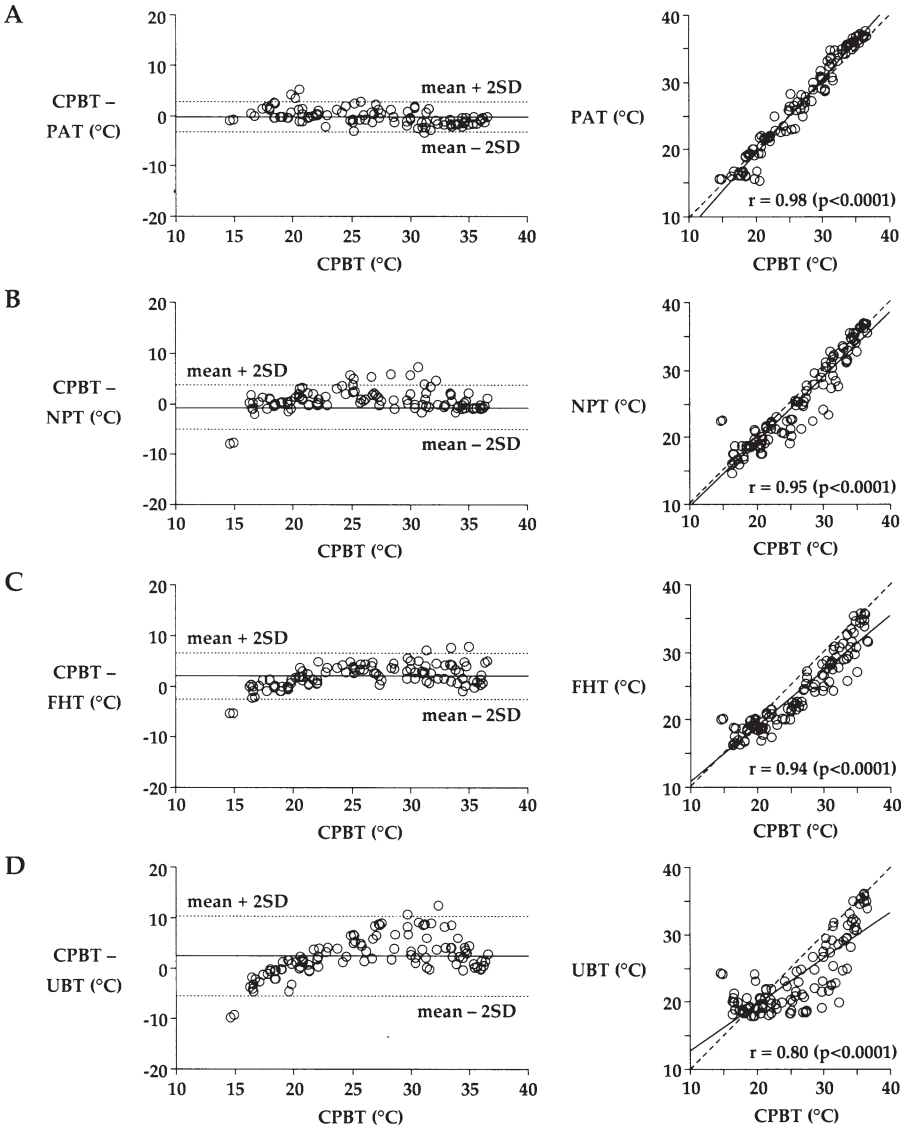

Fig. 4A–D. Bland and Altman comparison (*left*) and regression comparison (*right*) between the CPB venous line perfusate temperature and either pulmonary arterial (**A**), nasopharyngeal (**B**), forehead deep-tissue (**C**), or bladder (**D**) temperatures during the first 60 min of active rewarming on CPB. *CPBT*, cardiopulmonary bypass venous line temperature; *PAT*, pulmonary arterial temperature; *NPT*, nasopharyngeal temperature; *FHT*, forehead deep-tissue temperature; *UBT*, urinary bladder temperature

higher than the deep-tissue temperature (because of incomplete thermal insulation). Nevertheless, in our measurements made with the ambient temperature thermostatically controlled at 20°C, FHT closely matched both PAT and NPT during the profound hypothermia stabilized at ~18°C. Because the air movement is controlled using vertical flow in our operating rooms, the vertical airflow may have decreased the temperature in the vicinity of the patient's forehead to lower than 18°C and thereby enabled FHT to reflect the deep-tissue temperature. Forehead deep-tissue thermometry may be useful in estimating brain temperature during a period of retrograde cerebral circulation. However, we did not investigate this issue because of the lack of information on the reference temperature, i.e., directly measured brain temperature or average temperature of the blood returning from brain.

UBT and rectal temperature, commonly used to estimate core temperature during general surgery, have both been shown to significantly lag behind PAT, NPT, or esophageal temperature during induction of hypothermia and its reversal [23–26], consistent with our results. Because urine is a filtrate of blood, a thermistor-tipped urinary catheter would provide a reliable measure of core temperature if the urine flow rate were high. Thus, during a period of hypothermic CPB when the urine flow rate normally decreases, UBT would fail to accurately reflect changes in core temperature. Indeed, it was reported that, during rewarming on CPB, the difference between UBT and NPT increased with lower urine flow rates [25]. In this study, we confirmed the dependence of UBT on the urine flow rate.

Distal esophageal temperature has been suggested to serve as a reliable index of brain or central blood

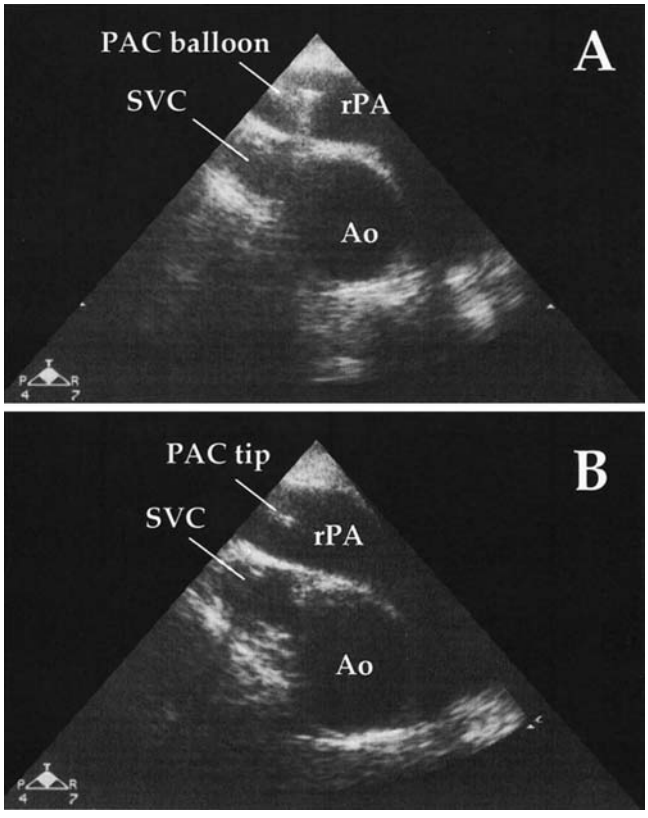

Fig. 5A,B. Transesophageal echocardiogram indicating anatomical relation between the superior vena cava and the tip of a thermodilution catheter placed in the right pulmonary artery to measure pulmonary capillary wedge pressure. These images were obtained before the start of CPB, using a multiplane transducer positioned at 0° (**A**) or 17° (**B**). In image **A**, the pulmonary arterial catheter balloon was inflated, as indicated by the presence of the acoustic shadow, while the balloon was deflated in image **B**. Please note that the pulmonary arterial catheter tip was placed in the immediate posterior vicinity of the superior vena cava. Results identical to these images were obtained during CPB, although the pulmonary arterial diameter was decreased by ~30%. *Ao*, aorta; *PAC*, pulmonary artery catheter; *rPA*, right pulmonary artery; *SVC*, superior vena cava

temperature during the induction of hypothermia or hyperthermia, and its reversal [10,27–29]. However, in our recent clinical practice, it is becoming rare to insert a thermometer into the esophagus during cardiac surgery because of the routine intraoperative use of transesophageal echocardiography.

Earlier studies [30,31] had proposed that tympanic membrane temperature could be considered as a gold reference for brain or hypothalamic temperature. However, more recent studies have suggested that the tympanic temperature can be significantly influenced by changes in ambient temperature (i.e., face or head skin temperature), and may not accurately reflect brain temperature [32,33]. Particularly in operating rooms in which air movement is controlled using vertical flow, the tympanic temperature would not reflect brain temperature precisely because cold air is blowing on the patient's head and face. In addition, perforation of tympanic membrane was previously reported as a complication of tympanic thermometry during anesthesia [34]. Thus, tympanic thermometry is rarely used in our clinical practice.

In conclusion, during a period of total CPB when pulmonary blood flow has nearly ceased and distal pulmonary arteries are not exposed to the air, a pulmonary arterial catheter thermistor located immediately behind the superior vena cava appears to provide a reliable estimate of the temperature of the blood returning from the upper body and, thus, possibly, the brain temperature. During the induction of profound hypothermia and its reversal on total CPB, the pulmonary arterial temperature, but neither the nasopharyngeal, forehead deep-tissue, nor urinary bladder temperature, would closely reflect changes in the mixed venous blood temperature, indicative of the efficiency of active core cooling or rewarming. On the other hand, during stabilized profound hypothermia on CPB, the pulmonary arterial, nasopharyngeal and forehead deep-tissue temperatures, but not urinary bladder temperature, appear to provide a reliable measure of core temperature (i.e., brain temperature).

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