Temperature During Cardiopulmonary Bypass: The Discrepancies Between Monitored Sites

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We performed studies in patients to determine whether temperature recordings from sites commonly monitored during hypothermic cardiopulmonary bypass adequately reflect cerebral temperature. In Study I ($n = 12$), temperatures monitored in the jugular bulb (JB) were compared with those recorded in the nasopharynx, esophagus, bladder, and rectum. In Study II ($n = 30$), temperature was also monitored in the arterial outlet of the membrane oxygenator. A calibrated recorder continuously and simultaneously recorded all temperatures. Study I found large temperature discrepancies between the JB and all other body sites during cooling and rewarming. There was considerable interindividual variability in the degree of discrepancy between the JB and other sites. Study II produced similar results but also showed that JB temperature reached equilibration with the temperature of blood entering the patient via the arterial outlet of the membrane oxygenator after cooling for 3.3 ± 1.3 min and after rewarming for 16.5 ± 5.5 min. Analysis of variance revealed that this arterial outlet site had the smallest average discrepancy of all temperature sites relative to the JB site ($P < 0.001$). In summary, temperatures measured in body sites over-estimated JB temperature during cooling and under-estimated it during rewarming, whereas arterial outlet blood temperature provided a good approximation.

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Neurologic injury caused by cerebral ischemia remains a devastating complication of cardiac surgery (1). Moderate hypothermia is often used for neuroprotection during routine cardiopulmonary bypass (CPB) and is associated with a reduced stroke rate, but this benefit is offset by a trend towards increased myocardial damage and non-stroke-related perioperative mortality (2). Nevertheless, deep hypothermia unquestionably confers cerebral protection when the circulation must be arrested during cardiac surgery or neurosurgery (3,4).

Whereas hypothermia is neuroprotective, hyperthermia, even if mild (i.e., 2°C–3°C above normal), is deleterious during cerebral ischemia. Hyperthermia delays neuronal metabolic recovery (5) and increases excitotoxic neurotransmitter release (6), oxygen free radical production (7), intracellular acidosis (5), and blood–brain barrier permeability (8). Hyperthermia also affects protein kinase activity (9) and destabilizes the cytoskeleton (10). Even a small increase (as little as 2°C) in cerebral temperature significantly aggravates ischemic neuronal injury and accelerates neuronal death in animal models (11,12). Clinical data are equally convincing: studies have shown that hyperthermia worsens the prognosis of acute stroke patients with respect to infarct size and mortality (13–15).

When deep hypothermia and elective circulatory arrest are used for cerebral protection during cardiac or neurosurgery, concerns about under-cooling or over-heating the brain have led investigators to document the fact that temperature measurements made at standard body monitoring sites provide inaccurate estimates of intracerebral temperature (16,17). Even after routine CPB with moderate hypothermia, nasopharyngeal temperature monitoring is an unreliable indicator of jugular venous bulb (JB) temperature during rewarming (18–20).

The primary aim of the current study was to measure the discrepancies between temperatures measured at several commonly used body sites during the cooling and rewarming phases of routine CPB conducted with moderate hypothermia. We compared temperatures in the nasopharynx, esophagus, bladder, and rectum with temperature in the JB, a reasonable approximation of global brain temperature (21). A

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secondary aim of the study was to determine whether the temperature of the blood entering the patient via the arterial outlet of the membrane oxygenator, an easily accessed, noninvasive monitoring site, accurately reflects JB temperature.

METHODS

Temperature Monitoring Techniques

With the approval of our IRB, we performed two prospective studies. Informed consent was obtained from the patients in each study. Study I examined 12 patients undergoing CPB for elective coronary artery bypass grafting surgery. Throughout CPB, temperatures were monitored simultaneously and continuously from five sites: JB, nasopharynx, esophagus, bladder, and rectum. Temperature was measured at the JB venous site with a 5F catheter with thermistor capability (7.5-cm Swan-Ganz™ Catheter; Baxter Healthcare, Irvine, CA) inserted into the right internal jugular vein after anesthetic induction. The catheter was inserted retrograde until it encountered resistance, and then it was pulled back approximately 0.5 cm until blood could be freely aspirated. This technique, in which no special methods (e.g., fluoroscopy) are used to ensure exact placement of the catheter in the JB, has been described by other investigators (16,19). Before CPB was instituted, two 1-mL samples of jugular venous blood were obtained (one during skin incision and one during cannulation of the great vessels) for measurement of O_2 tension and saturation to verify correct placement of the JB catheter. Temperature was measured at the nasopharyngeal, esophageal, and rectal sites with 12F YSI-400 temperature probes (Mon-a-therm™; Mallinkrodt, St. Louis, MO). Bladder temperature was monitored with a 16F Foley catheter with temperature-sensing capability (Bardex Lubricath™ Temperature-Sensing Foley Catheter; CR Bard, Covington, UK).

All temperatures were collected continuously (10 samples per second) and simultaneously. We used LabVIEW™ VI (National Instruments, Austin, TX) to create a virtual temperature monitor via serial interfaces connected to a laptop computer (Dell Inspiron™ 5000E; Dell Computer Corporation, Round Rock, TX). The external temperatures were multiplexed via a thermistor interface (Precision Smartlink™ KNM-THM31, Keithley Instruments, Cleveland, OH) capable of integrating up to 8 YSI-400 thermistors. A cardiac output computer (Abbott Oximetrix™ Q2a, Abbott Laboratories, Abbott Park, IL) was used to capture invasive temperature via the JB catheter.

Study II examined a separate group of patients undergoing CPB for elective coronary artery bypass grafting surgery ($n = 30$). Throughout CPB, the temperature was monitored simultaneously and continuously at the same five body sites used in Study I and at the arterial outlet of the membrane oxygenator. The temperature probes used at the arterial outlet were provided by the manufacturers of the individual oxygenators (Cobe Cardiovascular, Arvada, CO; Terumo Cardiovascular Systems Corporation, Ann Arbor, MI; and Bard Cardiopulmonary Division, Tewksbury, MA) and fit into standard connections that are designed for these probes and that are molded into the arterial outlet. In both Studies I and II, a calibrated six-channel recorder continuously and simultaneously recorded all temperatures.

Anesthetic, Surgical, and Perfusion Techniques

The anesthetic protocol in both studies included premedication with a benzodiazepine. Anesthetic induction was achieved with midazolam and fentanyl and supplemented with isoflurane as required. Paralysis was induced with vecuronium, rocuronium, or pancuronium. General anesthesia was maintained with fentanyl (administered to a maximum dose of 2.0 mg). Additional midazolam (to a maximum dose of 20 mg) and muscle relaxant were administered during CPB. No propofol, barbiturates, or inhaled anesthetics were administered during CPB.

CPB was performed with a standard bypass circuit, crystalloid prime, a roller pump, and a membrane oxygenator. The perfusion flow rate was kept between 40 and 60 mL/kg at all times. Mean arterial blood pressure was maintained between 35 and 85 mm Hg; vasopressors and vasodilators were administered as necessary. Hemoglobin levels were maintained between 6.0 and 10.0 g/dL. Cold blood cardioplegia was administered in antegrade fashion in all cases, whereas retrograde cardioplegia was administered at the surgeons' discretion.

The degree of hypothermia induced during CPB (range, 28°C–32°C) was selected by each surgeon, according to the temperature measured in the nasopharynx. Once the target nasopharyngeal temperature was achieved, active cooling ceased and hypothermic temperature was maintained (stable hypothermia) until rewarming commenced. Patients were rewarmed to a target nasopharyngeal temperature of 37.0°C before termination of CPB. When this target normothermic nasopharyngeal temperature was achieved, active rewarming ceased (stable normothermia). In Study II, perfusionists were instructed not to allow the arterial outlet temperature to exceed 37°C. Heating blankets were not used intraoperatively, but crystalloid, colloid, and blood products were warmed with an IV fluid warmer (Hotline™; Sims Level 1, Rockland, MA). Room temperature was maintained at 13°C to 18°C.

Statistical Analysis

Statistical analyses were performed using SAS software (SAS Institute, Cary, NC). For these studies of temperature discrepancies, the periods of cooling and rewarming were the periods of interest. Differences between JB temperature and temperatures at the other monitored sites were analyzed with repeated measures ANOVA at 2-min intervals during cooling and during rewarming and at 3-min intervals during the

periods of stable hypothermia and stable normothermia. When significant differences ($P < 0.05$) were found, *post hoc* comparisons were made using Bonferroni corrections.

In both Studies I and II, demographic and intraoperative factors that might affect the magnitude of temperature discrepancies were recorded. These included age, weight, body surface area, gender, stable hypothermic temperature during CPB, mean flow during CPB, hematocrit during CPB, duration of stable hypothermia, duration of rewarming, rate of rewarming, and duration of CPB. Associations between temperature differences and potential predictors of such differences during cooling or rewarming were assessed with repeated measures ANOVA for each monitored temperature site, with Bonferroni corrections.

Table 1. Study I: Demographic and Perioperative Patient Characteristics ($n = 12$)

Age (yr)	68.2 ± 8.2
Male gender	7 (58%)
Weight (kg)	85.1 ± 15.1
Body mass index (kg/m^2)	28.1 ± 4.6
Body surface area (m^2)	2.0 ± 0.2
Duration of CPB (min)	78.2 ± 40.1
Duration of stable hypothermic CPB (min)	32.4 ± 26.8
Stable hypothermic temperature (°C)	30.2 ± 1.1
Mean hematocrit during CPB (%)	22.7 ± 4.4
Rewarming time (min)	22.6 ± 10.6
Cross-clamp time (min)	47.3 ± 32.3
Duration of rewarming at time of cross-clamp removal (min)	17.2 ± 10.4
Rewarming rate (°C/min)	0.28 ± 0.14
Mean flow during CPB (L/min)	4.6 ± 0.5

Continuous variables are presented as means ± SD. CPB = cardiopulmonary bypass.

RESULTS

Study I

In Study I, patients ($n = 12$) underwent hypothermic CPB with a stable hypothermic temperature of 30.2°C ± 1.1°C (Table 1). Temperatures at the JB site were lower than temperatures at all other body sites, particularly the bladder and rectal sites, during the cooling period (Fig. 1).

Throughout the rewarming period, temperatures at the JB site were higher than temperatures at all other body sites (Fig. 2). After 15 min of rewarming, bladder temperature was 3.2°C ± 1.9°C lower and rectal temperature was 2.2°C ± 1.0°C lower than JB temperature. When rewarming was complete at 37°C (guided by nasopharyngeal temperature, as was our usual practice), bladder and rectal temperatures remained substantially lower (i.e., by 1.5°C–2.5°C). Even at the end of CPB, the bladder-to-JB temperature discrepancy was –1.5°C ± 1.1°C. Although the nasopharyngeal and esophageal temperatures provided closer approximations of JB temperature, these were, respectively, 1.6°C ± 1.2°C and 1.3°C ± 1.2°C lower than JB temperature 15 min after rewarming began, and both were approximately 0.5°C lower when rewarming was complete (Fig. 2). Peak JB temperature during CPB [37.8°C ± 0.4°C] occurred 36.3 ± 14.7 min after rewarming began and was 38.0°C or higher in four of the 12 patients 24 to 41 min after rewarming began.

Study II

The patients in Study II ($n = 30$) underwent hypothermic CPB with a stable hypothermic temperature of 29.6°C ± 0.8°C (Table 2). As in Study I, temperature at the JB site was significantly lower than temperatures at all other body sites throughout the cooling period (Fig. 3). In particular, bladder and rectal temperatures remained significantly higher during the hypothermic period 3.0°C ± 1.1°C for bladder temperature, $P < 0.0001$; and 3.9°C ± 0.9°C for rectal temperature, $P < 0.0001$; at 5 min of stable hypothermia]. However, JB temperature reached equilibration with the temperature of the blood entering the patient via the arterial outlet of the membrane oxygenator after cooling for 3.3 ± 1.3 min.

Throughout the rewarming period, nasopharyngeal, esophageal, bladder, and rectal temperatures were lower than JB temperature (Fig. 4). After 15 min of rewarming, bladder temperature was 2.8°C ± 1.3°C lower and rectal temperature was 3.0°C ± 1.7°C lower than JB temperature. Even during the period of stable normothermia, 2 min after the end of rewarming, the bladder-to-JB temperature discrepancy was −2.3°C ± 1.3°C and the rectal-to-JB discrepancy was −2.9°C ± 1.4°C. However, JB temperature reached equilibration with the temperature of the blood entering the patient via the arterial outlet of the membrane oxygenator after rewarming for 16.5 ± 5.5 min. Analysis of variance revealed that this arterial outlet site had the smallest average discrepancy of all temperature sites relative to the JB site ($P < 0.001$). Peak JB temperature during CPB [37.4°C ± 0.4°C] occurred 29.8 ± 7.4 min after rewarming began but was 38.0°C or higher in only four of 30 patients in Study II.

During rewarming in both Studies I and II, the degree of temperature discrepancy among the various body sites in individual patients was strikingly unpredictable, as suggested by the large standard deviations relative to the mean differences (Figs. 1–4). Because all body site temperatures were significantly different from JB temperature throughout CPB, we examined several demographic and perioperative variables that might have influenced the degree of average temperature discrepancy between these individual temperatures and JB temperature (Tables 1 and 2). In Study I, only the hematocrit during CPB influenced the average temperature discrepancy between the bladder site and the JB site ($P < 0.01$). In Study II, only the duration of the stable hypothermic period of CPB ($P = 0.03$) and the total duration of CPB ($P < 0.01$) influenced the average temperature discrepancy between the JB and all other body sites.

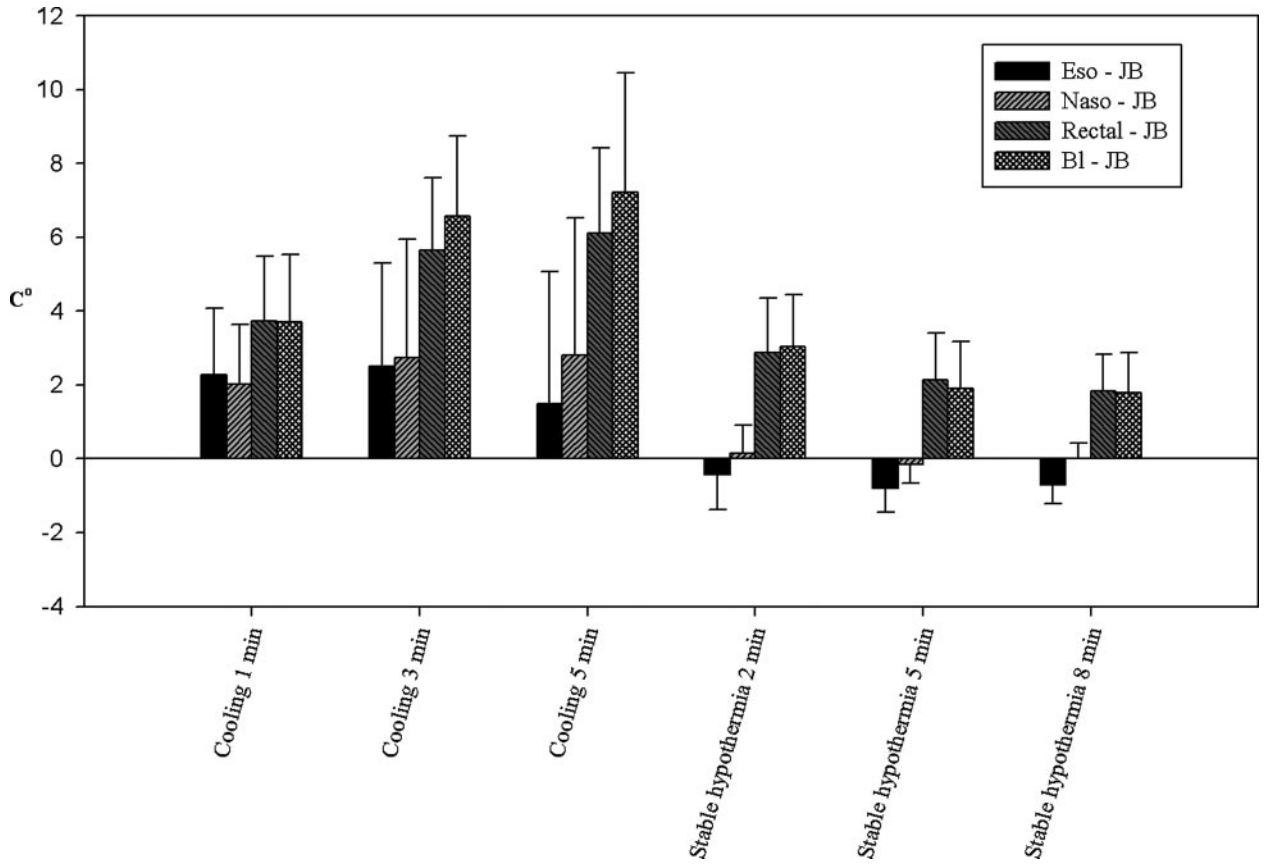

Figure 1. Study I: temperature difference between jugular bulb (JB) and other sites during cooling (mean ± SD; $n = 12$). Bl, bladder; Eso, esophageal; Naso, nasopharyngeal.

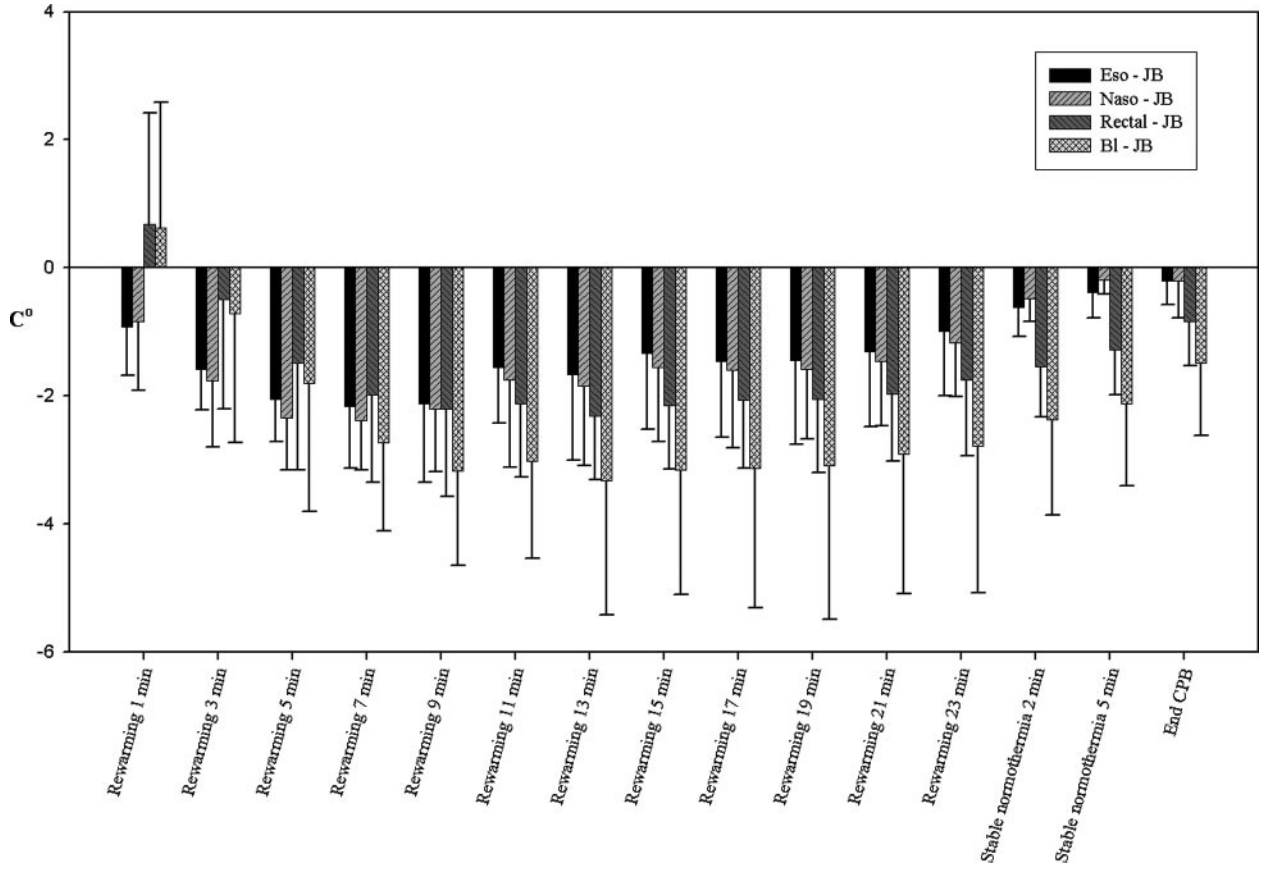

Figure 2. Study I: temperature difference between jugular bulb (JB) and other sites during rewarming (mean ± SD; $n = 12$). Bl, bladder; CPB, cardiopulmonary bypass; Eso, esophageal; Naso, nasopharyngeal.

Table 2. Study II: Demographic and Perioperative Patient Characteristics ($n = 30$)

Age (yr)	65.1 ± 10.5
Male gender	23 (77%)
Weight (kg)	85.5 ± 17.1
Body mass index (kg/m^2)	27.9 ± 4.6
Body surface area (m^2)	2.0 ± 0.2
Duration of CPB (min)	70.2 ± 21.0
Duration of stable hypothermic CPB (min)	16.8 ± 15.3
Stable hypothermic temperature (°C)	29.6 ± 0.8
Mean hematocrit during CPB (%)	22.0 ± 2.9
Rewarming time (min)	27.5 ± 4.6
Cross-clamp time (min)	37.6 ± 13.8
Duration of rewarming at the time of cross-clamp removal (min)	18.1 ± 7.5
Rewarming rate (°C/min)	0.24 ± 0.06
Mean flow during CPB (L/min)	4.5 ± 0.8

Continuous variables are presented as means ± SD. CPB = cardiopulmonary bypass.

DISCUSSION

To our knowledge, this is the first study of the relationship between JB temperature and several commonly used body sites during routine CPB conducted with moderate hypothermia. We found significant differences between JB venous temperature and temperatures in the nasopharynx and esophagus, and there were even larger differences between JB temperature and temperatures in the bladder and rectum. Monitoring bladder or rectal temperature to estimate "core" temperature is standard practice in many institutions. Many clinicians aggressively rewarm patients in an attempt to "normalize" temperature in these sites before termination of CPB. According to our data, if bladder or rectal temperatures are brought to 37°C, brain temperature is likely to be 2°C to 4°C higher.

A disconcerting finding was the striking unpredictability of the degree of temperature discrepancy among standard monitored sites in individual patients. In fact, we could not identify a single "best" body site at which to monitor temperature relative to temperature in the JB, although the nasopharyngeal and esophageal sites were consistently better than the rectal or bladder sites. Discrepancies between temperatures in the JB and temperatures at all other sites during CPB probably result from rapid cerebral blood-flow during rewarming and the proximity of the carotid origins to the aortic cannula (22). Therefore, it is not surprising that blood temperature in the arterial line leading from the oxygenator to the aortic cannula provides a better indicator of JB temperature (18).

Cerebral temperature may influence the extent or severity of neurologic injury during cardiac surgery involving CPB (22–24). Our data suggest that the temperature in the arterial line that exits the oxygenator should be monitored to determine when the desired temperature has been reached, because equilibration of the JB temperature occurs within 5 min (3.3 ± 1.3 min) after cooling begins. Most importantly, the rewarming phase must be carefully monitored and managed to avoid cerebral hyperthermia (22). More than half of the overt strokes that are observed after cardiac surgery presumably occur during the surgery itself (25). When this is the case, the presence of cerebral hyperthermia can only aggravate the tissue injury that ensues (26). Cerebral embolization, the likely cause of most overt strokes, usually occurs during periods when the brain is warm or warming, particularly during removal of the aortic cross-clamp (27). Therefore, overly aggressive rewarming in an attempt to avoid the "afterdrop" in temperature that usually occurs after CPB is discontinued may cause cerebral hyperthermia during or shortly after a period when cerebral embolization is likely. Furthermore, intraoperative hyperthermia has been associated with postoperative neuropsychological impairment (24,28).

Clinicians are often concerned about the risks posed by postoperative hypothermia (29). These include coagulopathy, shivering (which increases myocardial oxygen consumption), arrhythmias, a greater risk of wound infections, and longer hospital stays (30–32). On the other hand, the potential neuroprotective benefits of perioperative hypothermia may have been overlooked in patients undergoing cardiac surgery, and the practice in some institutions is to wean patients, particularly those at increased risk of stroke, from CPB at temperatures that are below normothermia (33,34). Based on our observations in Study I and the data of Grigore et al. (35), changes were made to our practice for Study II that are now permanent: earlier and slower rewarming was used, the temperature of the CPB perfusate was kept at or below 37.0°C (water bath settings exceeding 38°C were avoided), and CPB was discontinued at a nasopharyngeal temperature of 36.5°C.

One limitation of these studies is the possibility that minor variations in the positioning of the nasopharyngeal and esophageal probes may have influenced temperature readings (36). Similarly, variations in urinary flow may affect the bladder temperature sensors (37), and variations in the position of rectal probes in fecal matter (which produce variations in the probes' insulation from surrounding tissues) may affect accuracy (38). However, these sources of temperature variation would be present in any clinical setting, and therefore they would not affect the validity of the present study. Other limitations include the possibility of inaccurate positioning of the JB catheter and the uncertain extent to which temperature at the JB site reflects global brain temperature. Although JB temperature does not precisely measure cerebral cortical temperature, it cannot be more than brain temperature, as shown by direct measurements of cerebral temperature in patients with head injuries (19). Finally, this investigation is purely observational, without any intervention and with no attempt to correlate temperature data with any short- or long-term neurological or other patient outcomes. A much larger study will be required to determine whether preventing

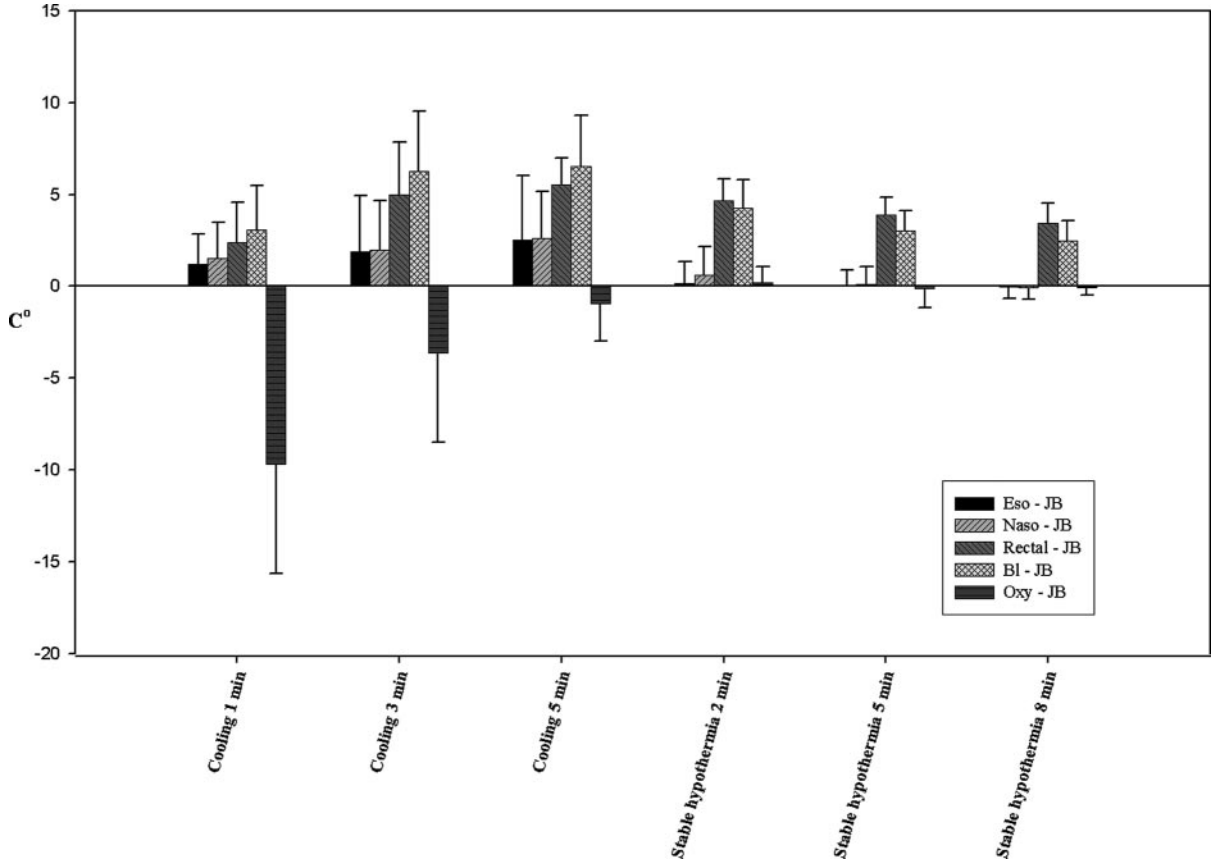

Figure 3. Study II: temperature difference between jugular bulb (JB) and other sites during cooling (mean ± SD; $n = 30$). Bl, bladder; Eso, esophageal; Naso, nasopharyngeal; Oxy, oxygenator exit.

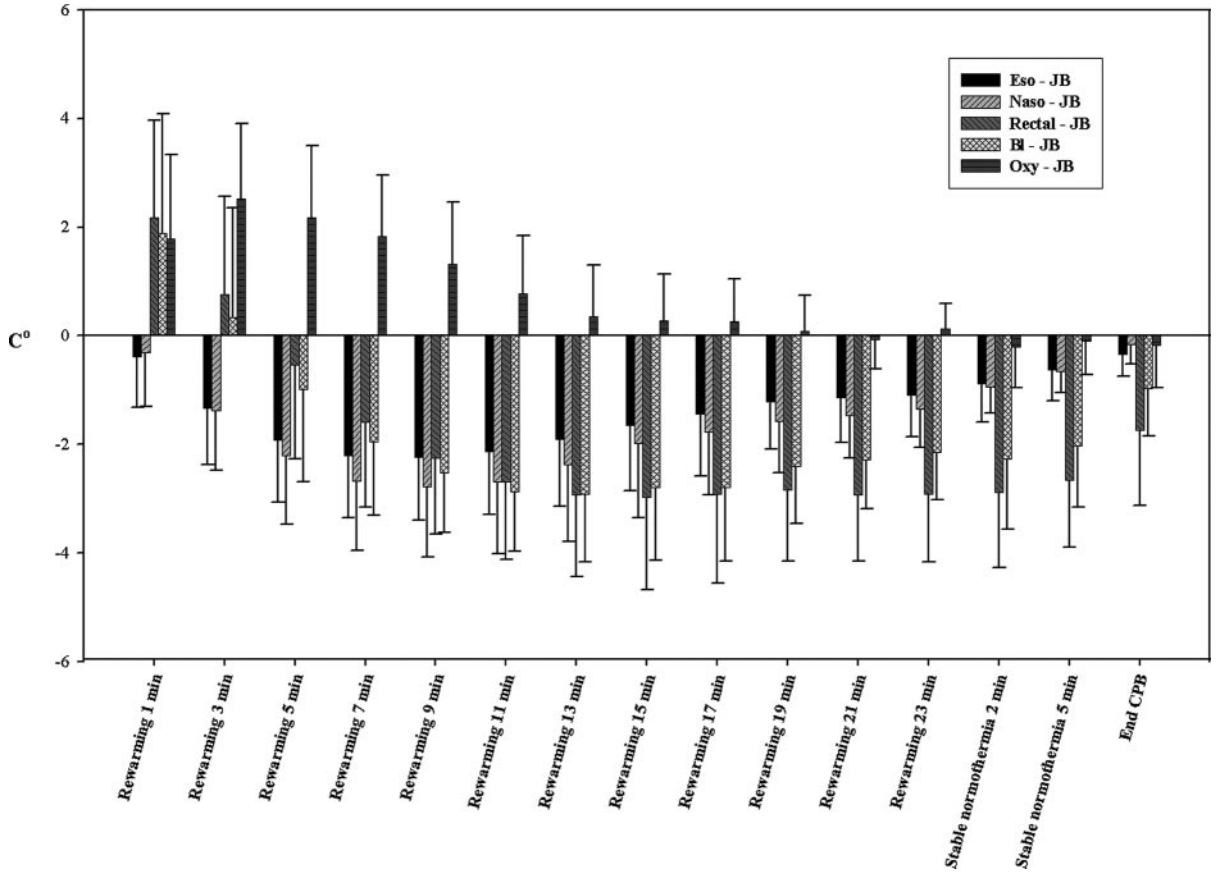

Figure 4. Study II: temperature difference between jugular bulb (JB) and other sites during rewarming (mean ± SD; $n = 30$). Bl, bladder; CPB, cardiopulmonary bypass; Eso, esophageal; Naso, nasopharyngeal; Oxy, oxygenator exit.

hyperthermia improves cerebral or other outcomes after CPB.

CONCLUSIONS

We found substantial temperature differences between the JB and all other commonly monitored sites during the cooling and rewarming phases of hypothermic CPB. The discrepancies between rectal or bladder temperatures and JB temperature were particularly large. Striking interpatient variability in temperature gradients prevented identification of a "best site." Only the temperature of blood entering the patient via the arterial outlet of the membrane oxygenator provided a good approximation of JB temperature.

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REFERENCES

1. Roach GW, Kanchuger M, Mangano CM, et al. Adverse cerebral outcomes after coronary bypass surgery. Multicenter Study of Perioperative Ischemia Research Group and the Ischemia Research and Education Foundation Investigators. N Engl J Med 1996;335:1857–63.
2. Rees K, Beranek-Stanley M, Burke M, et al. Hypothermia to reduce neurological damage following coronary artery bypass surgery. Cochrane Database Syst Rev 2001;1:CD002138.
3. Kirklin JW, Barratt-Boyes BG. Hypothermia, circulatory arrest and cardiopulmonary bypass. In: Cardiac surgery. 2nd ed. New York: Churchill Livingstone, 1993:61–127.
4. Williams MD, Rainer WG, Fieger HG Jr, et al. Cardiopulmonary bypass, profound hypothermia, and circulatory arrest for neurosurgery. Ann Thorac Surg 1991;52:1069–74.
5. Chopp M, Welch KM, Tidwell CD, et al. Effect of mild hyperthermia on recovery of metabolic function after global cerebral ischemia in cats. Stroke 1988;19:1521–5.
6. Castillo J, Davalos A, Noya M. Aggravation of acute ischemic stroke by hyperthermia is related to an excitotoxic mechanism. Cerebrovasc Dis 1999;9:22–7.
7. Globus MY, Busto R, Lin B, et al. Detection of free radical activity during transient global ischemia and recirculation: effects of intraischemic brain temperature modulation. J Neurochem 1995;65:1250–6.
8. Dietrich WD, Busto R, Halley M, Valdes I. The importance of brain temperature in alterations of the blood-brain barrier following cerebral ischemia. J Neuropathol Exp Neurol 1990; 49:486–97.
9. Busto R, Globus MY, Neary JT, Ginsberg MD. Regional alterations of protein kinase C activity following transient cerebral ischemia: effects of intraischemic brain temperature modulation. J Neurochem 1994;63:1095–103.
10. Morimoto T, Ginsberg MD, Dietrich WD, Zhao W. Hyperthermia enhances spectrin breakdown in transient focal cerebral ischemia. Brain Res 1997;746:43–51.
11. Dietrich WD, Busto R, Valdes I, Loor Y. Effects of normothermic versus mild hyperthermic forebrain ischemia in rats. Stroke 1990;21:1318–25.
12. Minamisawa H, Smith ML, Siesjo BK. The effect of mild hyperthermia and hypothermia on brain damage following 5, 10, and 15 minutes of forebrain ischemia. Ann Neurol 1990;28: 26–33.
13. Azzimondi G, Bassein L, Nonino F, et al. Fever in acute stroke worsens prognosis: a prospective study. Stroke 1995;26:2040–3.
14. Reith J, Jorgensen HS, Pedersen PM, et al. Body temperature in acute stroke: relation to stroke severity, infarct size, mortality, and outcome. Lancet 1996;347:422–5.
15. Kammersgaard LP, Jorgensen HS, Rungby JA, et al. Admission body temperature predicts long-term mortality after acute stroke: the Copenhagen Stroke Study. Stroke 2002;33:1759–62.
16. Kaukuntla H, Harrington D, Bilkoo I, et al. Temperature monitoring during cardiopulmonary bypass—do we undercool or overheat the brain? Eur J Cardiothorac Surg 2004;26:580–5.
17. Stone JG, Young WL, Smith CR, et al. Do standard monitoring sites reflect true brain temperature when profound hypothermia is rapidly induced and reversed? Anesthesiology 1995;82: 344–51.
18. Johnson RI, Fox MA, Grayson A, et al. Should we rely on nasopharyngeal temperature during cardiopulmonary bypass? Perfusion 2002;17:145–51.
19. Grocott HP, Newman MF, Croughwell ND, et al. Continuous jugular venous versus nasopharyngeal temperature monitoring during hypothermic cardiopulmonary bypass for cardiac surgery. J Clin Anesth 1997;9:312–6.
20. Cook DJ, Orszulak TA, Daly RC, Buda DA. Cerebral hyperthermia during cardiopulmonary bypass in adults. J Thorac Cardiovasc Surg 1996;111:268–9.
21. Rumana CS, Gopinath SP, Uzura M, et al. Brain temperature exceeds systemic temperature in head-injured patients. Crit Care Med 1998;26:562–7.
22. Cook DJ. Cerebral hyperthermia and cardiac surgery: consequences and prevention. Semin Thorac Cardiovasc Surg 2001; 13:176–83.
23. Gaudino M, Martinelli L, Di Lella G, et al. Superior extension of intraoperative brain damage in case of normothermic systemic perfusion during coronary artery bypass operations. J Thorac Cardiovasc Surg 1999;118:432–7.
24. Mora CT, Henson MB, Weintraub WS, et al. The effect of temperature management during cardiopulmonary bypass on neurologic and neuropsychologic outcomes in patients undergoing coronary revascularization. J Thorac Cardiovasc Surg 1996;112:514–22.
25. Salazar JD, Wityk RJ, Grega MA, et al. Stroke after cardiac surgery: short- and long-term outcomes. Ann Thorac Surg 2001;72:1195–201.
26. Ginsberg MD, Busto R. Combating hyperthermia in acute stroke: a significant clinical concern. Stroke 1998;29:529–34.
27. Barbut D, Hinton RB, Szatrowski TP, et al. Cerebral emboli detected during bypass surgery are associated with clamp removal. Stroke 1994;25:2398–402.
28. Buss MI, McLean RF, Wong BI, et al. Cardiopulmonary bypass, rewarming, and central nervous system dysfunction. Ann Thorac Surg 1996;61:1423–7.
29. Sessler DI. Complications and treatment of mild hypothermia. Anesthesiology 2001;95:531–43.
30. Frank SM, Fleisher LA, Breslow MJ, et al. Perioperative maintenance of normothermia reduces the incidence of morbid cardiac events: a randomized clinical trial. JAMA 1997;277:1127–34.
31. Insler SR, O'Connor MS, Leventhal MJ, et al. Association between postoperative hypothermia and adverse outcome after coronary artery bypass surgery. Ann Thorac Surg 2000;70:175–81.
32. 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. N Engl J Med 1996;334:1209–15.
33. Bar-Yosef S, Mathew JP, Newman MF, et al. Prevention of cerebral hyperthermia during cardiac surgery by limiting on-bypass rewarming in combination with post-bypass body surface warming: a feasibility study. Anesth Analg 2004;99: 641–6.
34. Nathan HJ, Wells GA, Munson JL, Wozny D. Neuroprotective effect of mild hypothermia in patients undergoing coronary artery surgery with cardiopulmonary bypass: a randomized trial. Circulation 2001;104:I85–91.
35. Grigore AM, Grocott HP, Mathew JP, et al. The rewarming rate and increased peak temperature alter neurocognitive outcome after cardiac surgery. Anesth Analg 2002;94:4–10.
36. Whitby JD, Dunkin LJ. Cerebral, oesophageal and nasopharyngeal temperatures. Br J Anaesth 1971;43:673–6.
37. Horrow JC, Rosenberg H. Does urinary catheter temperature reflect core temperature during cardiac surgery? Anesthesiology 1988;69:986–9.
38. Severinghaus JW. Temperature gradients during hypothermia. Ann N Y Acad Sci 1959;80:515–21.