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# Do Standard Monitoring Sites Reflect True Brain Temperature When Profound Hypothermia Is Rapidly Induced and Reversed?

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***Background:*** **Brain temperature is closely approximated by most body temperature measurements under normal anesthetic conditions. However, when thermal autoregulation is overridden, large temperature gradients may prevail. This study sought to determine which of the standard temperature monitoring sites best approximates brain temperature when deep hypothermia is rapidly induced and reversed during cardiopulmonary bypass.**

***Methods:*** **Twenty-seven patients underwent cardiopulmonary bypass and deep hypothermic circulatory arrest in order for each to have a giant cerebral aneurysm surgically clipped. Brain temperatures were measured directly with a thermocouple embedded in the cerebral cortex. Eight other body temperatures were monitored simultaneously with less invasive sensors at standard sites.**

***Results:*** **Brain temperature decreased from 32.6 ± 1.4°C (mean ± SD) to 16.7 ± 1.7°C in 28 ± 7 min, for an average cerebral cooling rate of 0.59 ± 0.15°C/min. Circulatory arrest lasted 24 ± 15 min and was followed by 63 ± 17 min of rewarming at 0.31 ± 0.09°C/min. None of the monitored sites tracked cerebral temperature well throughout the entire hypothermic period. During rapid temperature change, nasopharyngeal, esophageal, and pulmonary artery temperatures corresponded to brain temperature with smaller mean differences than did those of the tympanic membrane, bladder, rectum, axilla, and sole of the foot. At circulatory arrest, nasopharyngeal, esophageal, and pulmonary artery mean temperatures were within 1°C of brain temperature, even though individual patients frequently exhibited disparate values at those sites.**

***Conclusions:*** **When profound hypothermia is rapidly induced and reversed, temperature measurements made at standard monitoring sites may not reflect cerebral temperature. Measurements from the nasopharynx, esophagus, and pulmonary artery tend to match brain temperature best but only with an array of data can one feel comfortable disregarding discordant readings. (Key words: Anesthesia: Neurosurgical. Anesthetic technique: deep hypothermic circulatory arrest. Cardiopulmonary bypass. Hypothermia; profound. Temperature: axilla; bladder; brain; esophagus; nasopharynx; pulmonary artery; rectum; skin; tympanic membrane.)**

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WHEN the circulation must be arrested during surgery, deep hypothermia can confer cerebral protection. At 18°C, human brain cells can survive for 30 min without perfusion, but when ischemic time is prolonged or hypothermia is either more or less extreme, the incidence of poor neurologic outcome increases.[1,2] It is not usually feasible to measure intraoperative cerebral temperature directly. This study was undertaken to determine which of the commonly used and easily accessible monitoring sites of the body best approximates the actual temperature of the brain when deep hypothermia is rapidly induced and later reversed using cardiopulmonary bypass.

## Methods

With institutional approval and informed consent, 27 adult patients underwent cardiopulmonary bypass and deep hypothermic circulatory arrest in order for each to have a giant and almost inaccessible cerebral artery aneurysm surgically clipped. Twelve had preoperative neurologic deficits (seven from prior subarachnoid hemorrhage, five were grade III and two grade I), but

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none had significant cardiac dysfunction. There were 18 women and 9 men, ages ranged from 24 to 70 yr, and ASA physical status was either 2 or 3. Anesthesia was induced with intravenous midazolam (50 μg/kg), fentanyl (50 μg/kg), and thiopental (5 mg/kg) and maintained with isoflurane (0.5–1%) in oxygen. Tracheal intubation was accomplished after vecuronium (0.2 mg/kg), lidocaine (100 mg), and esmolol (0–100 mg) were administered intravenously, and the lungs were ventilated to maintain $Pa_{CO_2}$ at approximately 30 mmHg.

Cardiovascular variables were monitored using electrocardiography, radial and pulmonary artery catheters, and transesophageal echocardiography. EEG signals were obtained from scalp and cortical electrodes. Body temperatures were measured with sterile, disposable, copper-constantan thermocouple sensors (Mon-a-Therm, Mallinckrodt, St. Louis, MO). One was incorporated into the extracorporeal infusion tubing of the bypass circuit. The brain probe was a 21-G flexible Teflon coated wire (Mon-a-Therm, subcutaneous), which was inserted by pushing the tip about 3 cm into the parenchyma of the cerebral cortex at the operative site after the dura had been reflected. Another sensor, which was embedded in a cotton applicator, was placed into the auditory canal and pressed gently against the tympanic membrane. Other thermocouples were put in the posterior nasopharynx, distal esophagus, urinary bladder, rectum, and mid-axilla (arm at patient's side) and against the sole of the foot. Pulmonary artery temperature was monitored with the thermistor at the tip of a Baxter-Edwards VIP pulmonary artery catheter. Pulmonary artery temperature signals were digitized using an algorithm that only responds to temperatures greater than 17°C, and extrapolation was sometimes necessary to obtain the coldest pulmonary artery temperatures. All temperature sensors were interfaced with electronic thermometers whose synchronous digital output was continuously displayed. The data were electronically sampled and stored at 1-min intervals and transcribed onto paper for future analysis. Mallinckrodt specifies an accuracy of ±0.1°C for the thermocouple sensors, and the calibration was verified on several occasions against a standard mercury thermometer. The response time of the equipment to a 37°C temperature change was less than 1 min.

Cardiopulmonary bypass was initiated after intraoperative confirmation that the neurosurgical procedure could be safely accomplished only with circulatory arrest. Patients were anticoagulated with heparin (300 U/kg) and subsequent doses titrated to keep the activated clotting times above 480 s. A 21-Fr cannula was passed into the right atrium *via* a femoral vein, and centrifugal pumps were used with a membrane oxygenator and a 19-Fr femoral arterial cannula to achieve a 2.5 l/min/$m^2$ extracorporeal flow. The bypass circuit was primed with a solution of half Plasma-Lyte 148 (Baxter Healthcare, Deerfield, IL) and half Hespan (Du Pont, Wilmington, DE), and volume was adjusted to maintain hematocrit between 20% and 25%. Hypothermia was induced with a separate water bath heat exchanger which was initially set at 8°C. At circulatory arrest, the temperature of the water bath was allowed to rise to 16°C, where it was maintained until rewarming began. An alpha-stat acid-base strategy was used. If mean arterial pressure increased above 80 mmHg, nitroprusside (200 mg/l) was infused into the bypass circuit. If ventricular fibrillation occurred, conversion to asystole was brought about by injecting KCl (20 mEq boluses, total dose 0–80 mEq) through the right atrial port of a pulmonary artery catheter.

Thiopental was administered by intravenous infusion before bypass and titrated to achieve EEG burst-suppression.[3] The EEG became isoelectric during hypothermia, but thiopental loading continued at 18 ± 5 mg/kg/h until the end of bypass except for the period of circulatory arrest when it was halted. When any three of the patient's monitored temperatures became less than 18°C, the extracorporeal circulation was arrested. Before aneurysm clipping, patients were exsanguinated into the bypass reservoir to further relax the neurovasculature, and the arterial pressure decreased to almost zero. Cardiopulmonary bypass was gradually resumed after the aneurysm was clipped. Rewarming began with the heat exchanger initially set at 40°C and later adjusted downward. A water-filled heating mattress was activated. When any three of the patient's monitored temperatures became higher than 36°C, bypass was terminated and protamine given. At the conclusion of surgery, patients were brought to an intensive care unit, where their lungs were mechanically ventilated until ready for tracheal extubation. Data from 21 of the 27 patients were included in a previously published paper.[3]

The operating room temperature was maintained between 18°C and 21°C, and no attempt was made to keep patients warm before bypass. Temperatures were recorded every minute for the duration of the hypothermic period. Data were tabulated and statistical analyses carried out only at the following six times:

**Table 1. Monitored Temperatures**

| | Brain | Tympanum | Nasopharynx | Pulmonary Artery | Esophagus | Urinary Bladder | Rectum | Axilla | Foot Sole | Bypass Infusate |
|---|---|---|---|---|---|---|---|---|---|---|
| Start cooling (°C) | 32.6 ± 1.4 | 33.1 ± 1.2 | 33.0 ± 1.1 | 33.2 ± 1.1 | 33.0 ± 1.5 | 33.4 ± 1.4 | 33.3 ± 1.6 | 32.7 ± 1.8 | 28.4 ± 2.8 | 30.6 ± 1.6 |
| Midway cooling (°C) | 23.4 ± 2.2 | 25.3 ± 2.9 | 24.0 ± 2.5 | 21.3 ± 2.3 | 23.1 ± 2.2 | 25.2 ± 3.2 | 27.8 ± 3.1 | 30.6 ± 2.3 | 27.2 ± 2.2 | 14.9 ± 1.9 |
| Circulatory arrest (°C) | 16.7 ± 1.7 | 18.4 ± 2.8 | 17.5 ± 2.5 | 17.1 ± 2.0 | 17.3 ± 2.2 | 20.2 ± 2.8 | 24.0 ± 3.4 | 26.2 ± 2.6 | 25.4 ± 2.1 | 12.7 ± 1.8 |
| Start rewarming (°C) | 17.5 ± 1.7 | 17.6 ± 1.8 | 17.3 ± 1.9 | 17.6 ± 1.9 | 17.4 ± 1.4 | 20.3 ± 2.4 | 22.6 ± 3.1 | 22.8 ± 2.7 | 24.6 ± 3.0 | 17.7 ± 1.6 |
| Midway rewarming (°C) | 27.4 ± 2.3 | 24.7 ± 1.7 | 23.8 ± 2.6 | 26.8 ± 2.5 | 25.1 ± 2.3 | 21.9 ± 3.0 | 22.7 ± 2.2 | 23.6 ± 1.9 | 24.8 ± 2.8 | 32.1 ± 1.8 |
| End rewarming (°C) | 35.8 ± 1.6 | 35.5 ± 2.1 | 35.8 ± 1.4 | 36.7 ± 1.7 | 35.7 ± 1.9 | 34.5 ± 2.9 | 32.5 ± 3.5 | 30.2 ± 2.4 | 27.0 ± 2.6 | 37.5 ± 0.9 |

Values are means ± SD.

1. just before the initiation of cardiopulmonary bypass,
2. during the rapid cooling phase of cardiopulmonary bypass (when brain temperature was halfway between the beginning of cooling and circulatory arrest),
3. at circulatory arrest,
4. at the beginning of active core rewarming,
5. midway through rewarming (when brain temperature was halfway between the beginning of rewarming and separation from bypass),
6. just before termination of cardiopulmonary bypass.

Average rates of cooling and rewarming were calculated by dividing the temperature change (start cooling to circulatory arrest and start rewarming to end rewarming) by the time taken to accomplish it. Data are expressed as mean ± SD. With brain temperature taken as a target, other temperature monitoring sites within a 95% confidence interval of that target were determined. Pairwise correlations and multiple linear regression analyses were performed to observe how closely the other sites approximated brain temperature. In addition, the individual temperature differences between the brain and each of the other body sites were calculated and plotted against brain temperature. This method of Bland and Altman assesses systematic error (bias) and precision in the comparison of measurement methods.[4,5]

As more patients were studied, we became aware that sometimes a patient's brain temperature measurement differed substantially from that of the other centrally monitored sites when hypothermia was induced. We wondered how much measurement of brain temperature was influenced by ambient temperature. An identical thermal sensor, therefore, was inserted into the cerebral cortex of four other normothermic patients to a depth of 4 cm. Temperature measurements were recorded as the sensor was withdrawn in 1 cm stages. The region used was an epileptic focus which was about to be resected.

## Results

During the 2–3-h before cardiopulmonary bypass, body temperatures drifted down to the levels displayed in table 1 labelled "start cooling." With the exception of the sole of the foot, temperature from none of these sites was significantly different from brain before core cooling began.

The mean perfusate temperature decreased 10 ± 3°C during the first minute of rapid cooling, and another 4 ± 2°C during the second minute. Mean pulmonary artery blood temperature decreased 1 ± 1°C in the first minute and 2 ± 1°C/min for the next 2 min. A lag of 1–2 min occurred before brain temperature began to decrease. It then decreased at 1–2°C/min for a few minutes. At other monitoring sites, lag times of one to several minutes were noted before temperatures began to decrease. In general, all monitored temperatures demonstrated an exponential decline. Figures 1 and 2 display body temperatures from two patients at the ten monitored sites for the duration of the hypothermic period.

Cerebral temperature was midway through its decline after 8 ± 2 min of rapid cooling and measured 23.4 ± 2.2°C. At that time, only esophageal and nasopharyngeal temperatures were not significantly different from brain. Pulmonary artery temperature was less than and tympanic membrane, urinary bladder, rectal, axillary,

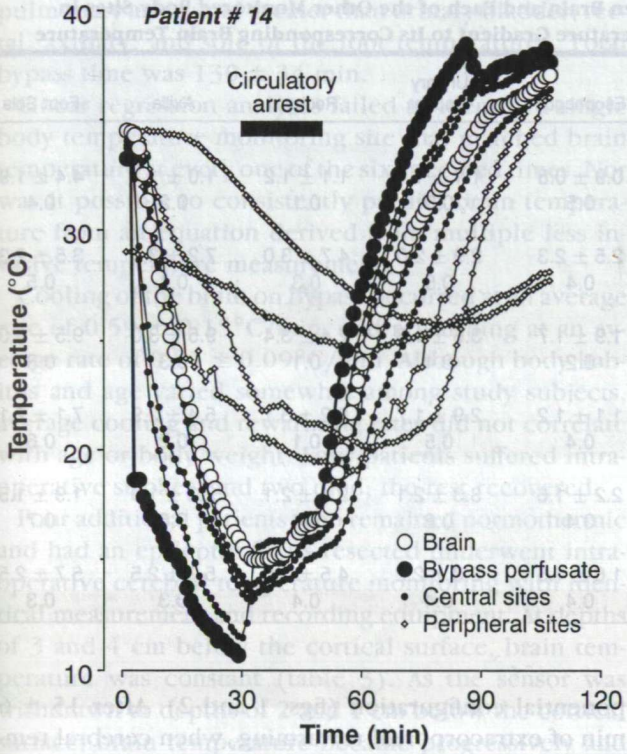


**Fig. 1. Graphic depiction of temperature:time relationship during cardiopulmonary bypass and deep hypothermic circulatory arrest for patient 14. Temperatures from each monitoring site are plotted every minute. Central sites are nasopharynx, tympanic membrane, esophagus, and pulmonary artery. Peripheral sites are bladder, rectum, axilla, and sole of the foot. The two fastest cooling sites were pulmonary artery and esophagus.**

and sole of the foot temperatures were greater than brain. Temperature gradients between the brain and the other sites were very large during rapid cooling (table 2), and large systematic errors were seen.

The rapid core cooling phase lasted 28 ± 7 min and ceased when brain temperature was 16.7 ± 1.7°C. At that time, the circulation was arrested for 24 ± 15 min while the aneurysms were clipped. Mean brain temperature was not significantly different from mean nasopharyngeal, esophageal, or pulmonary artery temperature at circulatory arrest, but it was less than that recorded from tympanic membrane, urinary bladder, rectum, axilla, and the sole of the foot (table 1). Temperatures varied considerably at circulatory arrest; brain temperature ranged from 14.5°C to 18.6°C, nasopharyngeal from 12.9°C to 22.7°C, esophageal from 13.5°C to 22.6°C, pulmonary artery from 15.9°C to 20.3°C, and tympanic membrane from 13.3°C to 22.6°C. Sometimes there were large temperature gradients between the brain and the other central sites, e.g. nasopharyngeal temperature varied from 4.7°C less than to 4.9°C greater than brain at circulatory arrest, esophageal temperature varied from 4.4°C less than to 6.7°C greater than brain, pulmonary artery varied from 4.6°C less than to 3.8°C greater than, and tympanic membrane varied from 3.9°C less than to 6.7°C greater than. Two examples of large gradients at circulatory arrest are illustrated in figures 1 and 2. Note that temperatures at centrally monitored sites differed from brain by several degrees, sometimes overestimating and other times underestimating it. When individual differences between the brain and nasopharyngeal temperature at circulatory arrest were plotted against brain temperature, differences frequently exceeded 2°C (fig. 3). When differences between the brain and tympanic membrane temperature at circulatory arrest were plot-

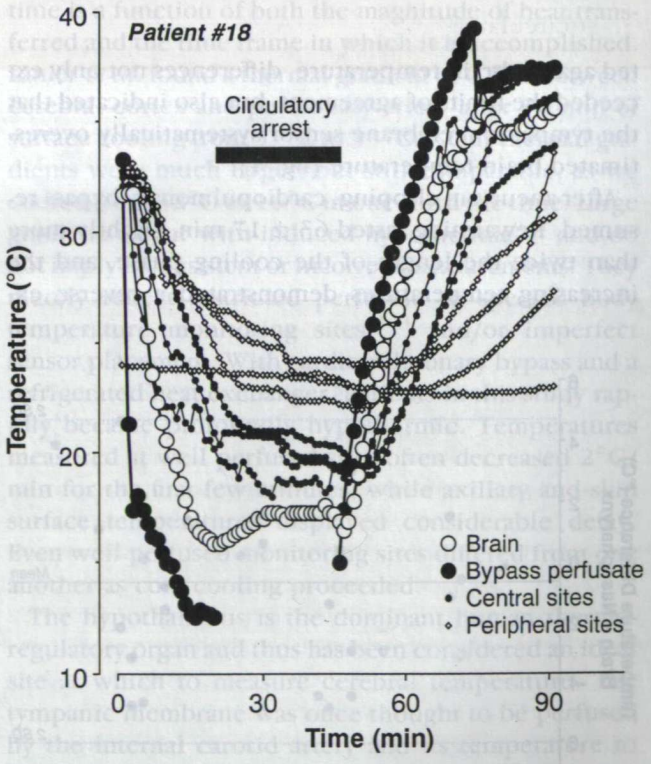


**Fig. 2. Graphic depiction of temperature:time relationship during cardiopulmonary bypass and deep hypothermic circulatory arrest for patient 18. Monitoring site temperatures are plotted every minute. Central sites are nasopharynx, tympanic membrane, esophagus, and pulmonary artery. Peripheral sites are bladder, rectum, axilla, and sole of the foot. The two slowest cooling central sites were tympanic membrane and esophagus.**

**Table 2. Temperature Gradients (Temperature Differences between Brain and Each of the Other Monitored Body Sites in Absolute Numbers) and $r^2$ Regression Values Relating Each Temperature Gradient to Its Corresponding Brain Temperature**

| | Tympanum | Nasopharynx | Pulmonary Artery | Esophagus | Urinary Bladder | Rectum | Axilla | Foot Sole |
|---|---|---|---|---|---|---|---|---|
| Start cooling | | | | | | | | |
| (°C) | 0.7 ± 1.0 | 0.7 ± .09 | 0.7 ± 1.0 | 0.9 ± 0.8 | 1.1 ± 1.1 | 1.1 ± 1.2 | 1.0 ± 1.7 | 4.4 ± 1.9 |
| $r^2$ | 0.3 | 0.4 | 0.4 | 0.5 | 0.6 | 0.1 | 0.2 | 0.4 |
| Midway cooling | | | | | | | | |
| (°C) | 2.7 ± 1.9 | 2.4 ± 1.8 | 2.9 ± 2.1 | 2.5 ± 2.3 | 2.9 ± 2.0 | 4.7 ± 3.0 | 7.2 ± 3.0 | 3.5 ± 2.3 |
| $r^2$ | 0.3 | 0.1 | 0.5 | 0.4 | 0.5 | 0.3 | 0.4 | 0.5 |
| Circulatory arrest | | | | | | | | |
| (°C) | 2.6 ± 1.5 | 2.1 ± 1.6 | 1.5 ± 1.1 | 1.9 ± 1.7 | 3.6 ± 1.8 | 7.4 ± 3.4 | 9.5 ± 3.0 | 9.5 ± 3.0 |
| $r^2$ | 0.1 | 0.1 | 0.6 | 0.2 | 0.4 | 0.1 | 0.3 | 0.3 |
| Start rewarming | | | | | | | | |
| (°C) | 1.2 ± 1.0 | 1.0 ± 1.0 | 0.8 ± 0.6 | 1.1 ± 1.2 | 2.9 ± 1.7 | 5.2 ± 3.1 | 5.4 ± 2.9 | 7.1 ± 2.1 |
| $r^2$ | 0.2 | 0.1 | 0.4 | 0.4 | 0.5 | 0.1 | 0.2 | 0.6 |
| Midway rewarming | | | | | | | | |
| (°C) | 2.6 ± 1.5 | 2.4 ± 2.2 | 1.8 ± 1.6 | 2.2 ± 1.6 | 3.3 ± 2.1 | 2.4 ± 2.1 | 2.3 ± 1.9 | 1.9 ± 1.5 |
| $r^2$ | 0.4 | 0.1 | 0.1 | 0.4 | 0.6 | 0.4 | 0.6 | 0.7 |
| End rewarming | | | | | | | | |
| (°C) | 1.5 ± 1.6 | 1.2 ± 1.3 | 1.2 ± 1.3 | 1.6 ± 1.5 | 2.3 ± 2.2 | 4.5 ± 3.0 | 5.7 ± 2.5 | 5.7 ± 2.5 |
| $r^2$ | 0.2 | 0.1 | 0.6 | 0.4 | 0.4 | 0.4 | 0.3 | 0.3 |

ted against brain temperature, differences not only exceeded the limits of agreement, but also indicated that the tympanic membrane sensor systematically overestimated brain temperature (fig. 4).

After aneurysm clipping, cardiopulmonary bypass resumed. Rewarming lasted 63 ± 17 min, slightly more than twice the length of the cooling phase, and the increasing temperatures demonstrated a reverse exponential configuration (figs. 1 and 2). After 15 ± 6 min of extracorporeal rewarming, when cerebral temperature had increased halfway, all temperatures except pulmonary artery were significantly different from brain. When cardiopulmonary bypass was terminated and active rewarming ceased, brain temperature was indistinguishable from tympanic membrane, nasopharyngeal, and esophageal temperatures but was less than

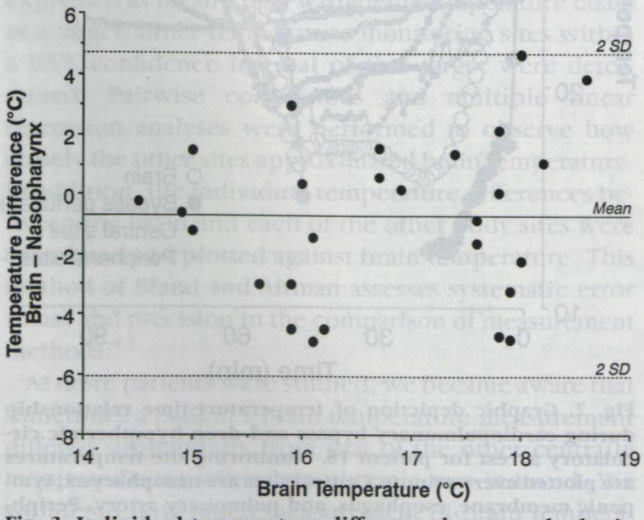


**Fig. 3. Individual temperature difference between the brain and nasopharynx as a function of brain temperature at circulatory arrest.**

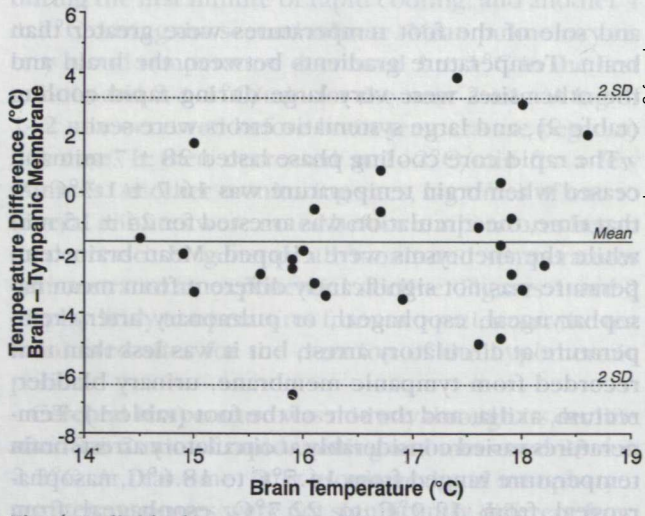


**Fig. 4. Individual temperature difference between the brain and tympanic membrane as a function of brain temperature at circulatory arrest.**

pulmonary artery and greater than urinary bladder, rectal, axillary, and sole of the foot temperatures. Total bypass time was 130 ± 34 min.

Linear regression analyses failed to identify a single body temperature monitoring site that matched brain temperature at every one of the six specified times. Nor was it possible to consistently predict brain temperature from an equation derived from multiple less invasive temperature measurements.

Cooling of the brain on bypass occurred at an average rate of 0.59 ± 0.15°C/min, and rewarming at an average rate of 0.31 ± 0.09°C/min. Although body habitus and age varied somewhat among study subjects, average cooling and rewarming rates did not correlate with age or body weight. Four patients suffered intraoperative strokes, and two died; the rest recovered.

Four additional patients who remained normothermic and had an epileptic focus resected underwent intraoperative cerebral temperature monitoring with identical measurement and recording equipment. At depths of 3 and 4 cm below the cortical surface, brain temperature was constant (table 3). As the sensor was withdrawn to depths of 2 and 1 cm below the cortical surface, brain temperature became progressively and significantly less than that at greater depth. Throughout these experiments, esophageal, tympanic membrane, and operating room temperatures all remained constant.

**Table 3. Cerebral Temperature as Sensor Withdrawn**

| Sensor depth from cortical surface (cm) | 4 | 3 | 2 | 1 |
|---|---|---|---|---|
| Cerebral temperature (°C) | 36.9 ± 0.2 | 36.8 ± 0.2 | 35.9 ± 0.1* | 33.3 ± 0.3* |

* Significantly different from previous temperature, $P < 0.05$.

## Discussion

When the circulation is arrested, the brain is the organ that first sustains injury. Cerebral protection can be conferred by hypothermia. For every 1°C that brain temperature is decreased, cerebral oxygen consumption decreases by about 8%. Hypothermia also protects the brain by other less well understood but probably more important means.[6,7] Nevertheless, human neurons remain viable without blood supply at 18°C for at least 30 min and often much longer,[1-3] and induced hypothermia has long been an adjuvant to neurosurgery when adequate tissue perfusion could not be assured.[8]

Accurate knowledge of cerebral temperature is critical during deep hypothermic circulatory arrest, because neurologic outcome worsens when ischemic time is prolonged or hypothermia becomes either more or less extreme.[1,2] Brain temperature cannot always be measured directly, however, and alternative, less invasive, monitoring sites frequently are substituted.[9] Under normothermic steady-state conditions, these alternate sites usually reflect true cerebral temperature.[10] In this study as well, all monitored temperatures, with the exception of the skin surface over the sole of the foot, exhibited considerable thermal homogeneity during the hours of anesthesia before cardiopulmonary bypass.

In contrast, temperatures measured at standard monitoring sites often lack uniformity during profound and rapid thermal change: some respond almost immediately whereas others display significant inertia.[11] Lag time is a function of both the magnitude of heat transferred and the time frame in which it is accomplished. Lanier *et al.* found a thermal gradient of 0.5°C between cerebral cortex and pulmonary artery after 90 min of surface cooling from 37°C to 33°C.[12] Our thermal gradients were much larger, but still comparable, as we cooled from 32°C to 16°C in one-third the time. Large gradients occur with induced hypothermia[9,11] and do not imply inconsistent or incorrect measurements. They merely reflect restricted perfusion to specific body temperature monitoring sites[13,14] and/or imperfect sensor placement. With cardiopulmonary bypass and a refrigerated heat exchanger, patients in this study rapidly became profoundly hypothermic. Temperatures measured at well perfused sites often decreased 2°C/min for the first few minutes, while axillary and skin surface temperatures displayed considerable delay. Even well perfused monitoring sites differed from one another as core cooling proceeded.

The hypothalamus is the dominant human thermoregulatory organ and thus has been considered an ideal site at which to measure cerebral temperature. The tympanic membrane was once thought to be perfused by the internal carotid artery and its temperature to correlate well with thalamic temperature.[15] Both theories have been questioned,[16,17] as studies demonstrate that fanning the face decreases the temperature of the tympanic membrane but not the brain.[17,18] Moreover, sensor probes do not always abut the tympanum, and cerumen or dried blood in the auditory canal can result

in a delayed response time. In this study, we did not obtain otoscopic assurance that the tympanic membrane was free of wax before sensor placement; therefore, erroneous measurements may have occurred.

Nasopharyngeal temperatures are readily monitored during anesthesia and usually correlate well with other centrally measured temperatures.[19] However, nasopharyngeal bleeding can occur, and temperatures vary between different probe positions.[20,21] Likewise, thermal sensors in the proximal esophagus are influenced by endotracheal ventilation, and some recommend placing probes where the loudest heart sounds are heard,[19,22] whereas others opt for a deeper, more distal position.[23,24] Neither esophageal nor pulmonary artery thermal sensors remain as "centrally located" when the circulation becomes distorted during cardiopulmonary bypass or circulatory arrest. Cold cardioplegia and flooding the thorax with iced saline also alter esophageal and pulmonary artery temperatures disproportionately.[20]

Bladder temperature sensors are convenient,[25] but they are influenced by urinary flow and consequently do not provide a reliable measure of core temperature during hypothermic cardiopulmonary bypass.[22] Rectal probes can become lodged in fecal matter, which insulates them from the surrounding tissues.[11] Thermoregulatory vasoconstriction shunts blood from axillary and other skin surface temperature sensors during moderately hypothermic anesthesia and prolongs their thermal response times.[13,14] During profound hypothermia, intense vasoconstriction redirects flow primarily to a very diminished central compartment. Thermal response time is altered by the volume of connective tissue and fat separating peripheral compartments from perfused vasculature.[26]

Even direct cerebral temperature measurement is influenced by the surrounding environment and may not provide an accurate representation of the entire brain.[21,27] Cerebral metabolic activity generates heat and causes a small gradient (less than 1°C) between deep brain temperature and that of the superficial parenchyma through which the proximal cerebral arteries course.[27] Our supplementary data in normothermic patients provide evidence that ambient operating room temperature can create a much larger gradient between the exposed surface of the brain and the tissue beneath it. An example of this effect in a study patient can be seen in figure 5. Note that, before cardiopulmonary bypass began, brain temperature measured less than the others and, during hypothermia, never reached the nadir achieved by the centrally monitored sites. During rewarming, irrigation of the neurosurgical field also caused local brain temperature fluctuation.

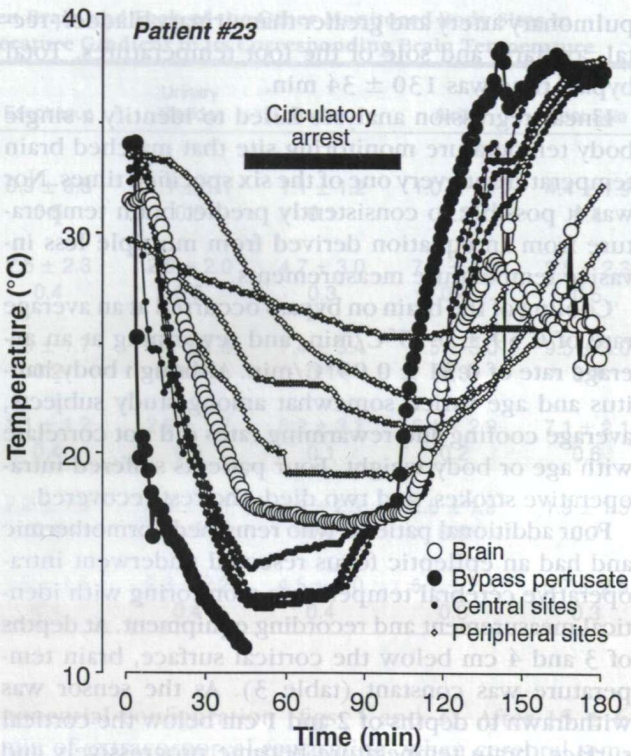


**Fig. 5. Graphic depiction of temperature:time relationship during cardiopulmonary bypass and deep hypothermic circulatory arrest for patient 23. Temperatures from each monitoring site are plotted every minute. Central sites are nasopharynx, tympanic membrane, esophagus, and pulmonary artery. Peripheral sites are bladder, rectum, axilla, and sole of the foot.**

As a technology, thermometry is reproducible, precise, and accurate. Yet the contradictory nature of body temperature measurement during thermal flux stands as the most important finding of this study. Nine body sites were monitored during rapidly induced profound hypothermia and, although most of the central sites were in agreement much of the time, in almost every patient, one usually displayed conflicting data. Moreover, variability was so large that none of the standard sites consistently corresponded to brain temperature, and no equation could be derived to always predict it. Thus, when cerebral temperature must be known, but cannot be measured directly, multiple less invasive substitutes are clinically helpful. Only then can one feel comfortable disregarding a discordant reading. Two central sites will not even suffice; for if they differ, how

will the correct one be identified? Redundancy is expensive, but when surgery requires profound hypothermia, our data indicate multiple central temperature monitoring sites must be used and be in near-agreement before one can have reasonable confidence that brain temperature is represented accurately.

In conclusion, when deep hypothermia is rapidly induced and reversed during cardiopulmonary bypass, cerebral temperature is poorly tracked by less invasive measurements made at standard body monitoring sites. Nevertheless, at circulatory arrest, nasopharyngeal, esophageal, and pulmonary artery temperatures usually closely approximate the temperature of the brain. Not infrequently, however, one measurement is inconsistent, and it is difficult to disregard discordant clinical values unless multiple others exist. We recommend monitoring all three of the above sites, when accurate knowledge of true brain temperature is critical.

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