Recently, renewed interest has developed in the use of controlled hypothermia for brain protection during neurovascular procedures. Although profound hypothermia with brain surface temperature of 16˚C to 17˚C clearly improves patient outcome from cerebral ischemia, growing evidence suggests that mild hypothermia (33˚C–34˚C) may also result in a better neurological outcome in patients undergoing complex neurovascular procedures.4,8 In addition, the incidence of complications associated with profound hypothermia, such as platelet dysfunction and cardiac arrhythmia, is greatly reduced when mild hypothermia is used.1,5

Although monitoring global brain temperature would be ideal for neurovascular procedures, the difficulty and potential complications of placing brain temperature probes makes the identification of an accurate correlate of brain temperature attractive. Several studies that have used induced hypothermia have demonstrated discrepancies between the common sites of temperature monitoring such as bladder, tympanic membrane, esophagus, and pulmonary artery blood.5,15,17 Furthermore, during induced hypothermia, temperatures at these sites may not correlate closely to those in the brain.17 Finally, the brain temperature appears to be inhomogenous and is dependent on the depth of the probe;7,9 therefore, global brain temperature is difficult to determine without using multiple intraparenchymal probes.

At our institution, patients at risk for cerebral ischemia are routinely cooled to 33˚C to 34˚C. Typically, temperature is monitored at pulmonary artery, esophagus, and bladder sites. In complex neurovascular procedures, we also measure cerebral venous oxygen saturation using a jugular bulb catheter. Two-thirds of the blood from the ipsilateral hemisphere is drained via the jugular bulb and thus this blood sample is considered to reflect accurately the oxygen content and cerebral metabolic state of that hemisphere.14 Ninety-nine percent of the jugular bulb blood has drained from intracerebral vasculature. With these facts in mind, we formed the hypothesis that blood temperature measured at the tip of a jugular bulb catheter

Blood temperature at the jugular bulb was monitored in 10 patients undergoing neurovascular procedures that used induced mild hypothermia, and its correlation with surface brain, core, and peripheral temperatures was determined. The study was motivated by the difficulty encountered in directly measuring global brain temperature and the poor correlations between various core and peripheral sites temperatures and brain temperature, particularly during deep hypothermia. Although not statistically significant, previous studies have suggested a trend toward higher brain temperatures. Temperatures from the jugular bulb (collected using a No. 5 French Swan–Ganz catheter) as well as from subdural, pulmonary artery, esophagus, tympanic membrane, and bladder sites were analyzed during three surgical conditions: prior to incision, with the dura open, and after closure of the dura. No complications related to placement of the jugular bulb catheter, induced hypothermia, or temperature monitoring were seen. The authors found that jugular bulb temperature was similar to pulmonary artery and esophageal temperatures; although prior to incision it tended to be higher than that found at the pulmonary artery, most commonly by 0.2˚C. Surface brain temperature was cooler than all other temperatures (p< 0.05), except that of the tympanic membrane, and was particularly sensitive to environmental variations. Finally, as has been shown by others, bladder temperature lagged substantially behind core temperatures particularly during rapid cooling and rewarming of the patient. In summary, monitoring of jugular bulb temperature is a feasible technique, and temperatures measured in the jugular bulb are similar to core temperatures.

Key Words • hypothermia • jugular bulb • temperature • brain temperature • monitoring
Jugular bulb temperature

TABLE 1
Demographics of neurosurgical patients undergoing craniotomy after induced mild hypothermia*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>patients</td>
<td>10</td>
</tr>
<tr>
<td>gender</td>
<td></td>
</tr>
<tr>
<td>male</td>
<td>5</td>
</tr>
<tr>
<td>female</td>
<td>5</td>
</tr>
<tr>
<td>age (yrs)</td>
<td>47.8 ± 3.6</td>
</tr>
<tr>
<td>weight (kg)</td>
<td>73.1 ± 4.7</td>
</tr>
<tr>
<td>height (cm)</td>
<td>170.0 ± 3.6</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.86 ± 0.07</td>
</tr>
<tr>
<td>temperature probe sites</td>
<td></td>
</tr>
<tr>
<td>bladder</td>
<td>9</td>
</tr>
<tr>
<td>brain</td>
<td>9</td>
</tr>
<tr>
<td>esophagus</td>
<td>10</td>
</tr>
<tr>
<td>jugular bulb</td>
<td>10</td>
</tr>
<tr>
<td>pulmonary artery</td>
<td>10</td>
</tr>
<tr>
<td>tympanic membrane</td>
<td>6</td>
</tr>
<tr>
<td>length of study (hrs)</td>
<td>8.4 ± 0.58</td>
</tr>
</tbody>
</table>

* Values given for age, weight, height, and BSA are expressed as means ± standard error of the mean. Abbreviation: BSA = body surface area.

would reflect brain temperature. This report summarizes our findings comparing jugular bulb temperature to core, peripheral, tympanic, and brain surface temperatures measured under three conditions: with the cranium closed, the dura opened, and on rewarthing the patient from mild hypothermia.

Clinical Material and Methods
Patient Population

After obtaining informed consent, 10 patients (five male and five female) undergoing craniotomy for aneurysm (nine patients) and arteriovenous malformation (AVM; one patient) were prospectively entered into this study to undergo temperature monitoring at the sites noted below. The characteristics of the patients are summarized in Table 1.

Induction of Anesthesia

All patients were premedicated with midazolam hydrochloride, which was titrated until the patient was comfortable during arterial line and electroencephalogram needle electrode placement. Induction of anesthesia was performed using pentothal (5 mg/kg) or etomidate (2–4 mg/kg) and fentanyl (5–10 μg/kg). Muscle relaxation was achieved with administration of rocuronium (0.9 mg/kg). After endotracheal tube placement, ventilation was mechanically controlled to maintain a PaCO₂ in the range of 25 to 30 mm Hg. Anesthesia was maintained with 50% oxygen in air, low-dose isoflurane (0.2–0.5 vol%), fentanyl infusion (1–5 μg/kg/hour), and atracurium infusion (1–8 μg/kg/minute). After induction, a No. 7.5 French pulmonary artery catheter was steriley placed in the subclavian vein. Using a retrograde internal jugular vein approach, a No. 5 French Swan–Ganz catheter was inserted in the internal jugular vein via a No. 5 French introducer sheath. The catheter was then advanced to the jugular bulb until a slight resistance was met. All catheter placements were confirmed radiographically. A continuous infusion of normal saline at a rate of 3 cc/hour was administered via the distal port and the central venous pressure port was occluded. The jugular bulb catheter was always placed contralateral to the surgical site to optimize venous drainage and allow surgical exposure of the carotid artery when needed.

Induced Hypothermia

Throughout the induction period, the patients were allowed to cool passively. The ambient room temperature was maintained between 18˚C and 21˚C. After patient positioning, active cooling was begun using either a hyperthermia/hypothermia water mattress (Blanketrol I; Cincinnati Sub Zero, Cincinnati, OH) or a convective air blanket (Polar Bair; Augustine Medical, Minneapolis, MN) set at 10˚C. If the degree of cooling after the 1st hour was less than 0.5˚C, as measured in the pulmonary artery, it was facilitated by nicardipine-induced vasodilation. Once the patient’s temperature reached 33.5˚C, active cooling was stopped and pulmonary artery temperature was allowed to drift passively to a minimum of 33˚C. At this point, active warming was initiated using a convective air blanket (Polar Bair or Bair Hugger, Augustine Medical) to maintain the pulmonary artery temperature between 33˚C and 34˚C throughout the period of aneurysm clipping or AVM resection. Hemodynamic parameters were monitored throughout the intraoperative period.
After angiographically demonstrated aneurysm clipping or AVM resection, active rewarming was instituted by increasing the room temperature to greater than 22°C, administering intravenous fluids heated to 39°C with a blood warmer (Fenwal; Travenol Labs, Deerfield, IL), and by setting the water mattress to 43°C and the convective air blanket to “high.”

Data Analysis

All temperatures were recorded every 15 minutes. For each probe site, temperatures were analyzed for four consecutive readings during three specific conditions as follows: “preincision,” the first 45 minutes after all probes were placed during patient positioning and preparation; “dura open,” the first 45 minutes after the dura was opened and brain temperature recording was initiated; and “closure,” the last 45 minutes of the surgical procedure. In four patients intraoperative tympanic membrane temperature could not be monitored due to early dislodgment of the probe; in one patient the brain surface temperature probe failed because of a connection problem; and in one patient bladder temperature was not monitored. Given the small sample size, we did not fill in missing data with sample derived estimates. The data were analyzed by repeated measures analysis of variance followed by pairwise comparisons between probe sites using Scheffé’s test with a significance level of 0.05. This method is known to provide conservative results. The time points chosen for pairwise comparison were: “preincision T1,” the first time point after all probes were placed; “dura open T4,” the last time point recorded with the dura open; and “closure T4,” the last set of recordings when all probes were still in place.

Results

The patients’ demographic data are shown in Table 1. No complications related to placement of the jugular bulb catheter, induced hypothermia, or temperature monitoring occurred.

Temperature data obtained at each of the four time points during the three different conditions are shown in Table 2. All patients cooled significantly and reached the target pulmonary artery temperature of 33.5°C ± 0.5°C; subsequently all rewarmed to an average temperature of 35.4°C by the end of the procedure. Statistical comparisons between temperatures at the six probe sites are shown in Table 2. At no time during the three conditions analyzed was any significant difference found between jugular bulb temperature and the temperature recorded from the pulmonary artery or the esophagus. During “preincision,” even though statistical significance was not reached, we observed a trend toward a higher temperature at the jugular bulb than at the pulmonary artery (Fig. 1). The temperature difference between the jugular bulb and the pulmonary artery was greater than 0 in 74% of the observations and the mean difference between temperatures was 0.2°C. Temperatures recorded from the tympanic membrane were significantly lower than those recorded from the jugular bulb, esophagus, and bladder, but not from those obtained at pulmonary artery sites. At “dura open,” surface brain temperatures were significantly
Jugular bulb temperature

![Graph](image)

**Fig. 1.** Scatterplot displaying the temperature differences between jugular bulb (JB) and pulmonary artery (PA) sites ($\Delta T$JB–PA (°C)) during the “preincision” period at each of the four time points (T1–4). Each symbol represents one patient. At each time point, the data points are separated on the horizontal axis for visualization purposes only.

lower than all other temperatures recorded except those from the tympanic membrane. During the third condition, “closure,” no significant differences in temperature could be found between any of the sites.

**Discussion**

Profound hypothermic circulatory arrest requiring cardiopulmonary bypass has gained favor in neurosurgery by providing a bloodless surgical field and possible protection of ischemic brain. Because of significant complications including cardiac arrhythmia, bleeding diathesis, and myocardial ischemia, hypothermia induced to the level of 16°C to 18°C is reserved for cerebral protection from circulatory arrest used for facilitating exposure of large vascular lesions (such as giant intracranial aneurysm). An alternative to profound hypothermia is mild hypothermia (33°C–34°C), which has been shown by histopathology to limit brain tissue damage in animals following forebrain ischemia. In humans, Marion, et al., demonstrated a trend toward a better neurological outcome in head-trauma patients treated with mild hypothermia (33°C). Brain temperature was monitored via a ventriculostomy with thermistors placed in the frontal horn of the lateral ventricle. Also in humans, BAKER and colleagues have shown that mild hypothermia (33°C–34°C) monitored at pulmonary artery, esophageal, or tympanic locations was not associated with the aforementioned cardiac or hemostatic complications.

Clearly, the potential benefits and problems associated with hypothermia require an accurate way of monitoring the temperature of the end organ in question, the brain. Few studies have addressed the difficulty of accurately monitoring brain temperature in humans. MELLERGÅRD and NORDSTRÖM monitored brain temperature at the anterior horn through a ventriculostomy catheter equipped with thermocouples, but this technique is applicable only for patients requiring ventriculostomies and the equipment is unavailable at the present time. More recently, Stone, et al., suggested that due to the lack of uniformity between temperatures at different sites, monitoring at multiple sites may provide a more accurate indication of brain temperature during profound hypothermia. Moreover, they demonstrated a significant gradient between near surface (depth of 1 cm) temperature and that measured at a intraparenchymal depth of 4 cm, the latter being close to 3°C warmer. Similarly, MELLERGÅRD and NORDSTRÖM measured a temperature gradient of 0.4°C to 1°C between the epidual space and the lateral ventricle, the epidural space being colder. It is interesting to note that the gradient was less pronounced in the latter study, perhaps because measurements were performed through a burr hole instead of through a craniotomy as in the former study.

Given the difficulties of directly measuring global cerebral temperature, we set out to determine the feasibility of jugular bulb temperature monitoring and its correlation with temperatures measured at other sites. Our rationale for monitoring jugular bulb temperature was based on the fact that 99% of the blood at this level has circulated throughout the brain and that venous blood sampled at the jugular bulb is an acceptable means of monitoring cerebral metabolic states. Thus, we believe that temperature measured at the jugular bulb may reflect the temperature of the brain.

Our results with the cranium closed failed to show any significant difference between jugular bulb temperature and that measured at any other site except the tympanic membrane. However, as shown in Fig. 1, jugular bulb temperature tended to be higher than pulmonary artery temperature. These data are in agreement with the results of LANIER, et al., who demonstrated in dogs prior to cooling a temperature gradient of 0.2°C between pulmonary artery and intraparenchymal temperatures, a difference that was not statistically significant. Similarly in humans, MELLERGÅRD and NORDSTRÖM demonstrated a median 0.3°C difference between rectal and intraventricular temperature, the latter being higher. However, the correlation between rectal and “core” temperatures during induced hypothermia is controversial. Furthermore, MELLERGÅRD

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**Table 3**

<table>
<thead>
<tr>
<th>Probe Site</th>
<th>Preincision at T1</th>
<th>Dura Open at T4</th>
<th>Closure at T4</th>
</tr>
</thead>
<tbody>
<tr>
<td>bladder</td>
<td>35.7 ± 0.11</td>
<td>33.5 ± 0.25†</td>
<td>34.9 ± 0.29</td>
</tr>
<tr>
<td>brain</td>
<td>NA</td>
<td>32.2 ± 0.51‡</td>
<td>NA</td>
</tr>
<tr>
<td>esophagus</td>
<td>35.5 ± 0.23</td>
<td>33.2 ± 0.24‡</td>
<td>35.2 ± 0.27</td>
</tr>
<tr>
<td>jugular bulb</td>
<td>35.6 ± 0.18</td>
<td>33.4 ± 0.24‡</td>
<td>35.3 ± 0.30</td>
</tr>
<tr>
<td>pulmonary artery</td>
<td>35.4 ± 0.16</td>
<td>33.3 ± 0.24‡</td>
<td>35.4 ± 0.26</td>
</tr>
<tr>
<td>tympanic membrane</td>
<td>35.0 ± 0.21§</td>
<td>32.7 ± 0.20</td>
<td>34.5 ± 0.52</td>
</tr>
</tbody>
</table>

* Values are expressed as means ± standard error of the mean. See Data Analysis for explanation of time points (T1 and T4).

† $p < 0.05$ compared to brain probe site.
‡ $p < 0.05$ compared to bladder, esophagus, jugular bulb, and pulmonary artery probe sites.
§ $p < 0.05$ compared to bladder, esophagus, and jugular bulb probe sites.
and Nordström’s data were not subjected to statistical analysis, making a direct comparison with our results difficult.

After opening of the dura and during active cooling, jugular bulb temperature remained similar to all other temperatures except that measured by the subdural brain probe. However, the temperatures measured by the brain probe fluctuated greatly, particularly during irrigation, despite taking precautions to isolate the probe by placing it deeply under the dura. The same phenomenon was described by Stone, et al.,17 who suggested that these large fluctuations in brain temperature are the result of contamination from the surrounding environment and may not accurately reflect global brain temperature. Similarly, the colder temperature that we measured at the brain surface may also result from contamination by the environment (that is, room air and irrigation). Thus, at best, surface brain temperature may only measure nearby brain tissue and is unlikely to be a reliable indicator of global cerebral temperature. During the active hypothermic phase, jugular bulb temperature was not different from pulmonary artery temperature. These results appear to contradict those of Lanier and colleagues7 who found significantly higher temperatures at an intraparenchymal probe site than at the pulmonary artery site. But in their study, brain temperatures were recorded in a closed cranium with no cooling effect from the environment. In our study, any initial temperature gradient between the pulmonary artery and jugular bulb may be abolished by cooling the blood draining the exposed brain surface to a temperature approaching that of the core. A larger series of patients will be required to verify the initial gradient between jugular bulb and pulmonary artery temperatures and their subsequent equalization on brain exposure.

Whether tympanic temperature is representative of brain temperature in humans remains a controversy.2-3 Nicholson and Iserson12 demonstrated in dogs a close correlation between tympanic and brain temperature. Human studies have shown agreement between temperatures measured from tympanic and brain probes.3 In contrast, Shiraki, et al.,16 found that fanning the face reduced the temperature of the tympanic membrane but not that of the brain in a nonanesthetized subject. Also, tympanic temperatures are influenced by the position of the head, the lower side of which is warmer.13 An additional difficulty with tympanic temperature monitoring is its sensitivity to proper placement, best accomplished with otoscopic confirmation of contact between the probe and the tympanic membrane. In the study by Stone, et al.,17 the tympanic temperatures were warmer than those of the brain except during the rewarming phase when they were cooler; the authors attributed this discrepancy to possible probe malplacement. In our observations, temperatures measured at the tympanic membrane did not correlate with those measured at the jugular bulb with a closed cranium and, in fact, were significantly colder, despite monitoring from the dependent ear. This poor correlation might be explained by a malpositioning of the tympanic probe and by contamination of tympanic temperature with cooler room air; otoscopic confirmation of probe position was not performed in our patient series. It was obvious that complete dislodgment of the sensor in the tympanic membrane occurred in four patients.

Esophageal, pulmonary artery, and jugular bulb temperatures remained extremely close during cooling and rewarming and did not exhibit any lag time. This similarity suggests that all three approximate true core temperature. In contrast, as others have shown for peripheral sites during induced hypothermia,6 bladder temperature, which varies with urinary flow, lagged behind the core temperatures by up to 0.7°C.

Summary

In summary, temperatures monitored at the jugular bulb were similar to those recorded at pulmonary artery and esophageal sites. Surface brain temperature did not appear to reflect global brain temperature accurately, because it was overly sensitive to the environment; this demonstrates again the difficulty in assessing the temperature of the brain as a whole organ. Although prior to incision the jugular bulb did exhibit a trend toward a higher temperature than the pulmonary artery, albeit without reaching significance, our data did not demonstrate any significant gradient between temperatures recorded at core and jugular bulb sites. Similar to our results in determining jugular bulb and core temperatures, the observed differences between brain and core temperatures in normothermic subjects have not been found to be significant in any of the series reviewed, despite a trend in each study toward a warmer brain.7,9,10 Whether jugular bulb blood is in thermal equilibrium with brain, suggesting that global brain and core temperatures are not significantly different in this clinical situation, remains to be determined. Nevertheless, our study demonstrates that jugular bulb temperature monitoring is a feasible technique and that jugular bulb temperature is similar to core temperature.

References

Jugular bulb temperature


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