# Brain temperature exceeds systemic temperature in head-injured patients

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*Objective:* To identify the temperature differences in readings taken from the brain, jugular bulb, and core body in head-injured patients.

*Design:* Prospective, observational study.

*Setting:* Neurosurgical intensive care unit of a university-affiliated county hospital.

*Patients:* Thirty patients with severe head injuries had measurements of brain and core body temperatures. Fourteen patients also had measurements of jugular venous blood at the level of the jugular bulb.

*Interventions:* None.

*Measurements and Main Results:* Brain temperature was increased an average of 2.0°F (1.1°C) over the core body temperature. In individual patients, the average brain temperature increase over the core body temperature ranged from –0.5° to 3.8°F (–0.30° to 2.1°C). Jugular vein and core body temperatures were similar. The difference in the brain and body temperatures increased when cerebral perfusion pressure decreased to between 20 and 50 mm Hg. The difference in the brain and body temperatures decreased in those patients treated with barbiturate coma.

*Conclusions:* Direct measurement of temperature in head-injured patients is a safe procedure. Temperatures in the brain are typically increased over the core body temperature and the jugular bulb temperatures. Jugular vein temperature measurement is not a good measurement of brain temperature since it reflects body, not brain temperature. These findings support the potential importance of monitoring brain temperature and the importance of controlling fever in severely head-injured patients since brain temperature may be higher than expected. (Crit Care Med 1998; 26:562–567)

KEY WORDS: temperature, brain; temperature, jugular bulb; head injury; cerebral blood flow

The injured brain is sensitive to variations in temperature. Hypothermia protects, while hyperthermia exacerbates, ischemic and traumatic brain damage (1–7). In experimental studies, brain temperature is directly monitored and controlled independent of systemic temperature. For clinical purposes, rectal temperature has generally been assumed to be representative of brain temperature (8). Recent studies (9–12), however, have consistently shown that in brain-injured patients, the brain temperature can be significantly higher than the core body temperature.

The purpose of this study was to measure core body temperature, jugular bulb temperature, and brain temperature in patients with severe brain injury, and to evaluate the factors that might be related to the gradient between brain and rectal temperature.

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## MATERIALS AND METHODS

*Patient Characteristics and Management.* Thirty patients with severe head injuries were studied. The protocol was approved by the Institutional Review Board of Baylor College of Medicine, and informed consent was obtained from the nearest relative of each patient. Demographic data for these 30 patients are summarized in Table 1.

Intensive care management at our institution emphasized maintaining cerebral perfusion pressure at ≥60 mm Hg and intracranial pressure at <20 mm Hg. Controlled ventilation (keeping $Pco_2$ at 35 to 40 torr [4.7 to 5.3 kPa]), cerebrospinal fluid drainage, sedation, neuromuscular blocking agents, and mannitol were used to control intracranial pressure. Barbiturate coma was used if these treatments were not adequate.

*Temperature Measurement.* All 30 patients had simultaneous recordings of brain and rectal temperatures. Twenty-one of the patients also had measurement of jugular vein temperature. Global cerebral blood flow of 16 of the patients was measured using the nitrous oxide technique, and cerebral metabolic rates for oxygen ($CMRO_2$) and glucose (CMRglu) were calculated from the product of the cerebral blood flow and the cerebral arterial-venous difference of oxygen and glucose, respectively.

The probe for measuring brain temperature was designed for intraparenchymal placement (Licox, Kiel-Mielkendorf, Germany). The probe consisted of a Type K nickel/nickel-chromium thermocouple that was 0.5 mm in diameter. Insertion was a bedside procedure, generally involving a small twist drill hole placed ~3 cm lateral to the midline and 1 cm anterior to the coronal suture. The dura was penetrated and the probe was inserted intraparenchymally to a depth of 1 to 2 cm. The scalp incision was closed with a single stitch.

Jugular vein temperature recordings were performed using three different

**Table 1.** Demographic characteristics of the 30 patients studied

| | |
|---|---|
| Age (yr) | 30.7 ± 13.0[a] |
| Gender | |
| Male | 24 (80) |
| Female | 6 (20) |
| Injury | |
| Diffuse axonal injury | 7 (23) |
| Subdural hematoma | 11 (37) |
| Epidural hematoma | 3 (10) |
| Contusion | 3 (10) |
| Gunshot wound | 6 (20) |
| Best Day 1 GCS | |
| 3–5 | 6 (20) |
| 6–8 | 19 (63) |
| >8 | 5 (17) |
| Outcome | |
| Alive | 23 (77) |
| Dead | 7 (23) |

GCS, Glasgow Coma Score.
[a]Mean ± SD.
Values in parentheses indicate percent.

catheters: a) a pediatric 4.5-Fr pulmonary artery flotation catheter (Swan-Ganz™, Abbott Laboratories, North Chicago, IL) in six patients; b) a 4-Fr regional oxygen saturation catheter (Baxter Edwards Critical-Care, Irvine, CA) in four patients; and c) a 5-Fr regional oxygen saturation catheter (Abbott Laboratories) in four patients. The thermistor was 15 mm proximal to the tip of the pulmonary artery flotation catheter and was 5 mm and 8 mm proximal to the tip of the 4-Fr and 5-Fr regional oxygen saturation catheters, respectively. These catheters were inserted through a 5-Fr introducer sheath (Cordis, Miami, FL). The catheter was inserted until the resistance of the catheter tip meeting the roof of the jugular bulb was felt. Then the catheter was withdrawn 0.5 cm. Appropriate placement was confirmed with skull radiographs.

Previous studies (S. P. Gopinath and C. S. Robertson, Unpublished Observations) performed in 14 patients during removal of the jugular bulb catheter measured temperature in the jugular bulb, and in the jugular vein at 1, 2, 3, and 4 cm proximal to the jugular bulb. Although oxygen saturation increases in some patients as the catheter is withdrawn from the jugular bulb, the temperature readings at the different positions in the jugular vein were identical in all 14 patients (S. P. Gopinath and C. S. Robertson, Unpublished Observations). The three catheters used in the present study had temperature sensors that were located at slightly different distances from the tip of the catheter. However, since the previous studies (S. P. Gopinath and C. S. Robertson, Unpublished Observations) had demonstrated no temperature gradient observed between the jugular bulb and the distal 3 cm of the jugular vein, the data collected with the three catheters are summarized together as representing jugular vein temperature.

The temperature probe for measuring rectal temperature was a 10-Fr thermistor probe (400 series, Sheridan Catheter Corporation, Argyle, NY). The rectal probe was connected to the bedside monitor (Hewlett-Packard, Palo Alto, CA).

Brain, jugular vein, and rectal temperature measurements were continuously recorded electronically until the patients had no further need for intracranial pressure monitoring. The duration of the temperature recordings in each patient ranged from 15 to 136 hrs. After removal, the rectal, jugular, and intracranial temperature probes were all tested in a warm water bath by comparing the temperature obtained by each probe with a standard mercury thermometer.

*Data Analysis.* Since temperature did not change rapidly, simultaneous readings from all three sites (rectum, brain, and jugular bulb) at the end of each hour of recording were used for the analyses. To compare temperature findings with demographic characteristics (such as age, gender, severity and type of injury, and outcome), the hourly temperature recordings from the first 5 days after injury were averaged for each patient. Comparisons of mean values for groups were done using repeated-measures analysis of variance, followed by a paired *t*-test with Bonferroni correction for multiple comparisons. Individual measurements of cerebral blood flow and metabolism variables were compared with temperature data obtained at the time of the cerebral blood flow measurement by regression analysis. Summary data in the tables and text are expressed as the mean ± SEM.

## RESULTS

The temperature measurements were completed without difficulty in all cases. There were no complications related to catheter insertion or removal. During the first 5 days after injury, the average brain temperature in the 30 patients was 102.0 ± 1.8°F (38.9 ± 1.0°C), compared with the average rectal temperature of 100.0 ± 0.7°F (37.8 ± 0.4°C) ($p < .001$, paired *t*-test). In the 14 patients who had jugular vein temperature measurements, the average jugular vein temperature was 99.9 ± 0.9°F (37.7 ± 0.5°C) compared with 99.9 ± 0.7°F (37.7 ± 0.4°C) for rectal temperature ($p = .63$, paired *t*-test). As illustrated in Figure 1, all three temperatures tended to change over time together, but there was a gradient between the brain temperature and the rectal/jugular temperatures.

The difference between the average brain and rectal temperatures was 2.0 ± 1.1°F (1.1 ± 0.6°C), although, in the individual patients, the difference ranged from –0.5 to 3.8°F (–0.3 to 2.1°C). Eighteen (60%) of the patients had an average difference between the brain and rectal temperatures of >1.8°F (>1°C). Three (10%) patients had an average difference between the brain and rectal temperatures of >3.6°F (>2°C). Only two patients had an average brain temperature that was less than the rectal temperature. As shown in Figures 1 and 2, the difference between brain and rectal temperatures was fairly constant over time for each patient, increasing somewhat when patients became febrile and decreasing with brain death.

The difference between the average jugular vein and rectal temperatures was –0.05 ± 0.18°F (–0.03 ± 0.1°C), and ranged from –0.4 to 0.2°F (–0.2 to 0.1°C). As illustrated in Figures 1 and 2, the jugular vein temperature tended to follow the rectal temperature over time in each patient.

The only demographic characteristic that was significantly related to either the brain temperature or the difference between the brain and rectal temperatures was age. The age of the patient was inversely related to the difference between the brain and rectal temperatures ($r^2 = .14$, $p = .038$). Neither average brain temperature nor difference between the brain and rectal temperature were significantly related to injury severity, gender, type of injury, or outcome (Table 2).

In 21 patients who had measurement of global cerebral blood flow and metabolism, the cerebral blood flow

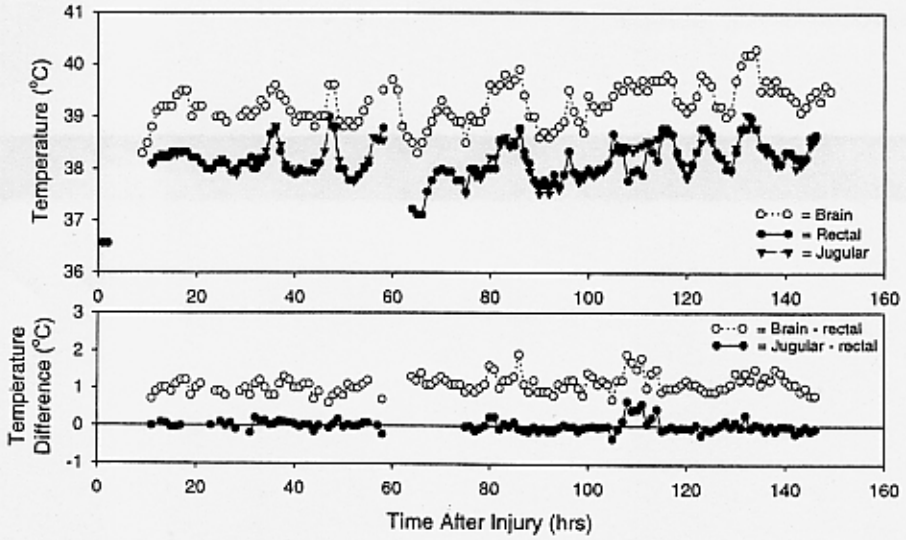


**Figure 1.** Example of changes in brain, rectal, and jugular bulb temperatures over time. Brain temperature is ~1.8°F (~1°C) higher than both rectal and jugular temperatures during the first 5 days after injury.

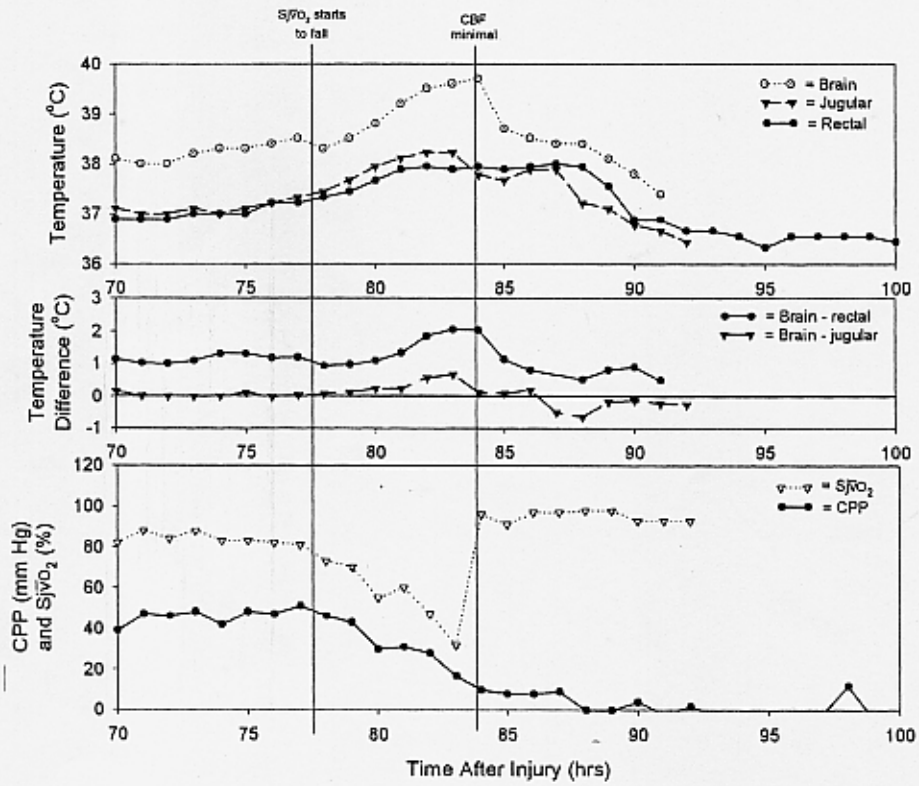


**Figure 2.** Example of changes in brain, rectal, and jugular bulb temperatures as cerebral blood flow (*CBF*) decreases due to severe intracranial hypertension. As CBF decreases, brain temperature and the brain-rectal temperature gradient increase. As CBF ceases, brain temperature and the brain-rectal temperature gradient decrease, and the brain-jugular temperature gradient reverses. $Sj\bar{v}o_2$, jugular venous oxygen saturation; *CPP*, cerebral perfusion pressure.

averaged 70.6 ± 3.4 mL/100 g/min and $CMRO_2$ averaged 1.00 ± 0.05 μmol/g/min. CMRglu and cerebral metabolic rate of lactate averaged 0.22 ± 0.02 and –0.030 ± 0.010 μmol/g/min, respectively. Cerebral blood flow, $CMRO_2$, and CMRglu were not significantly related to either brain temperature or to the difference between brain and rectal temperatures. However, these global measurements were obtained at times when the patients were relatively stable, and therefore did not include any ischemic levels of cerebral blood flow. To examine the consequences of more severe reductions in cerebral blood flow, the six patients who died of refractory intracranial hypertension were examined using jugular venous oxygen saturation and cerebral perfusion pressure as surrogate measures of cerebral blood flow. An example of the biphasic pattern of changes in brain temperature that was observed is shown in Figure 2, and the average data from all six patients are shown in Table 3. In each of these six patients, as the cerebral perfusion pressure decreased from 50 to ~20 mm Hg, the jugular venous oxygen saturation decreased, indicating a decrease in cerebral blood flow. During this phase, brain temperature increased, and the gradient between brain and rectal temperatures, as well as between jugular vein and rectal temperatures, increased. As cerebral perfusion pressure decreased to <20 mm Hg, jugular venous oxygen saturation increased as blood flow to the brain ceased and the blood in the jugular vein became primarily extracerebral. During this phase, the following temperature changes were observed: a) brain temperature decreased at an average rate of 0.9°F/hr (0.5°C/hr) (~0.2°F/min [~0.01°C/min]); b) the gradient between the brain and rectal temperatures decreased; and c) the gradient between the jugular vein and rectal temperatures reversed.

To examine the effect of a pharmacologic reduction in $CMRO_2$ on brain temperature, the brain temperature was compared before and after induction of barbiturate coma (Table 4). Eight of the patients monitored required barbiturate coma for treatment of refractory intracranial hypertension. Seven of these eight patients had measurements of brain temperature before and after receiving the loading dose of barbiturates. The eighth patient was already receiving barbiturates at the time that the brain temperature monitoring was initiated. There was a trend for brain and rectal temperatures to decrease slightly by 5 hrs after receiving the loading dose of pentobarbital, but these values were not significantly different. There was not a change in the difference between brain and rectal temperatures after pentobarbital was administered.

**Table 2.** The relationship of brain and rectal temperatures and demographic characteristics in the 30 patients was examined by repeated-measures analysis of variance (mean ± SEM)

| | Rectal Temperature | Brain Temperature | Temperature Difference (Brain – Rectal) |
|---|---|---|---|
| Injury Severity[a] | | | |
| GCS 3–6 | 37.7 ± 0.3 | 38.6 ± 0.3 | 1.1 ± 0.7 |
| GCS 6–8 | 37.9 ± 0.2 | 39.1 ± 0.2 | 1.3 ± 0.55 |
| GCS >8 | 37.8 ± 0.3 | 38.5 ± 0.3 | 0.6 ± 0.7 |
| Gender[b] | | | |
| Male | 37.9 ± 0.1 | 39.0 ± 0.1 | 1.2 ± 0.7 |
| Female | 37.6 ± 0.3 | 38.4 ± 0.3 | 0.8 ± 0.4 |
| Type of Injury[c] | | | |
| Diffuse | 38.0 ± 0.2 | 39.1 ± 0.2 | 1.1 ± 0.6 |
| Mass lesion | 37.7 ± 0.1 | 38.6 ± 0.1 | 1.0 ± 0.7 |
| Gunshot wound | 37.9 ± 0.2 | 39.2 ± 0.2 | 1.3 ± 0.5 |
| Outcome[d] | | | |
| Survived | 37.9 ± 0.1 | 39.0 ± 0.1 | 1.1 ± 0.6 |
| Died | 37.6 ± 0.3 | 38.5 ± 0.4 | 1.0 ± 0.7 |

GCS, Glasgow Coma Score.

[a]GCS effect ($p = .41$), location effect ($p < .001$), GCS × location interaction ($p = .22$); [b]gender effect ($p = .13$), location effect ($p < .001$), gender × location interaction ($p = .46$); [c]injury effect ($p = .33$), location effect ($p < .001$), injury × location interaction ($p = .52$); [d]outcome effect ($p = .17$), location effect ($p < .001$), outcome × location interaction ($p = .43$).

For each demographic factor, the main effect of temperature location (brain or rectal) was significant ($p < .001$) but the main effect of the demographic factor and the interaction of the temperature location and demographic factor were not.

**Table 3.** Changes in brain temperature as refractory intracranial hypertension impaired cerebral blood flow in six patients were analyzed by repeated-measures analysis of variance (mean ± SEM)

| | Time Period | | | |
|---|---|---|---|---|
| | CPP >50 mm Hg | CPP <50 >20 mm Hg | CPP <20 mm Hg | *p* Value |
| Brain temperature (°C) | 38.6 ± 0.3 | 39.6 ± 0.4 | 37.2 ± 0.7[a] | .007 |
| Temperature difference (°C, brain – rectal) | 1.1 ± 0.2 | 1.7 ± 0.3 | 0.2 ± 0.6[a] | .003 |
| Temperature difference (°C, jugular – rectal) | –0.05 ± 0.05 | 0.45 ± 0.25 | –0.50 ± 0.20 | .184 |

CPP, cerebral perfusion pressure.

[a]Significant difference ($p < .05$) compared with values at the "CPP >50 mm Hg" time period by paired *t*-test with Bonferroni correction.

**Table 4.** Effect of pentobarbital coma on brain and rectal temperatures in seven patients (mean ± SEM)

| | Before Pentobarbital | 1 Hour After Loading Dose of Pentobarbital | 5 Hours After Loading Dose of Pentobarbital |
|---|---|---|---|
| Temperatures[a] | | | |
| Rectal (°C) | 38.1 ± 0.3 | 38.2 ± 0.3 | 37.8 ± 0.3 |
| Brain (°C) | 38.9 ± 0.3 | 39.1 ± 0.3 | 38.6 ± 0.3 |
| Temperature Difference[b] (°C, brain – rectal) | 0.8 ± 0.4 | 0.9 ± 0.3 | 0.8 ± 0.4 |

[a]Location effect ($p = .025$), time period effect ($p = .452$), location × time interaction ($p = .97$); [b]time effect ($p = .654$).

Effect was examined by repeated-measures analysis of variance. Only the main effect of temperature location (rectal vs. brain) was significant. The main effect of time, and the interaction between time period and temperature location, were not significant.

When the temperature probes were removed and compared, the following data were recorded: a) the temperature difference between the brain temperature probe and the standard thermometer averaged 0.05 ± 0.05°F (0.03 ± 0.03°C); b) the temperature difference between the jugular vein probe and the standard thermometer averaged –0.05 ± 0.23°F (–0.03 ± 0.13°C); and c) the temperature difference between the rectal probe and the standard thermometer averaged 0.00 ± 0.04°F (0.00 ± 0.02°C).

## DISCUSSION

*Clinical Importance of Brain Temperature.* Evidence has begun to accumulate that traumatic brain injury is sensitive to temperature changes. Animal studies (5, 7, 13, 14) have shown mild hypothermia to decrease mortality after a traumatic brain injury, decrease histopathologic changes, and improve behavioral outcomes. Hypothermia as a treatment of head-injured patients decreases $CMRO_2$, cerebral blood flow, and intracranial pressure (6). Two clinical studies (6, 15) have observed a trend toward an improved clinical outcome in patients with head injuries treated with hypothermia as a part of their overall care.

The beneficial effects of hypothermia in cases of ischemia are well known. Research in animal models has shown decreased neuronal damage when hypothermia is employed after ischemic insult, while increased injury is seen with hyperthermia (1). Hypothermia is commonly used in cardiovascular anesthesia, and often in neurosurgical anesthesia, when cerebral blood flow is expected to be decreased or interrupted (8, 16). Multiple cases exist of people surviving submersion in cold water for prolonged periods (17).

Abnormalities of local cerebral blood flow and local ischemia are likely present after head injury. Mechanical stresses on blood vessels from head trauma can cause injury to those vessels, with contusion formation and disruption of local blood circulation (7). N-methyl-D-aspartate receptor-mediated calcium entry into the cell is a step in the process of free-radical production and cellular injury in both ischemia and head injury. Excitatory amino acids, such as glutamate and aspartate, are potent agonists of N-

methyl-D-aspartate receptors, and excessive concentrations of these substances have been found in the extracellular space after experimental brain injury (18, 19). Research has indicated that a major benefit of hypothermia is the prevention of excessive levels of these amino acids from developing (20).

*Brain Temperature Regulation.* The temperature of the brain has been observed to vary locally, and can be up to 2.5°F (1.4°C) higher in the center of the brain than at the surface (21, 22). Studies (23–25) have suggested that brain temperature is determined by three major factors: a) the production of local heat by metabolic processes in the brain; b) the rate of the local cerebral blood flow; and c) the level of the arterial temperature. The temperature of the perfusing arterial blood is normally lower than that of the brain, and blood flow through the brain is an important route of heat removal by the brain. This characteristic of the brain has been exploited as a method for measuring cerebral blood flow using the thermal diffusion technique (26).

Age was also a significant factor in the present study. The mechanism of this association is not clear but perhaps involves alterations in skull density and/or scalp thickness with aging that might increase diffusion of heat generated by the brain.

*Cerebral Metabolic Rate.* The metabolic rate of the brain is normally high, accounting for 20% of systemic resting oxygen consumption and 25% of systemic glucose consumption. After brain injury, when cerebral metabolic rate is reduced from normal, the brain's metabolism still accounts for ~10% of the total body energy expenditure. In the present study, global $CMRO_2$ averaged ~58% and CMRglu averaged 55% of the normal value. Because of this reduced metabolic rate, the temperature of the brain might be expected to be lower than normal after severe head injury. Mellergard and Nordstrom (11, 22) noted a trend for a decreased difference in brain and rectal temperatures in unconscious patients compared with awake patients. However, a significant relationship between measures of cerebral metabolism and brain temperature could not be demonstrated in the present study, and brain temperature was consistently higher than rectal temperature. Only in circumstances where brain metabolism was markedly altered did brain temperature seem to be related to $CMRO_2$. When barbiturate coma, which typically reduces $CMRO_2$ by 50%, was induced as a treatment of intracranial hypertension, there was a trend for brain temperature to decrease. With brain death, there was a reduction in brain temperature of 0.02°F/min (0.01°C/min). This finding is similar to data in an experimental study (24) in which circulatory arrest resulted in an abrupt decrease in the temperature of the brain by 0.07°F/min (0.04°C/min) and to data in recent case reports (22, 27) in which brain temperature decreased below rectal temperature with brain death. The brain's metabolism generates heat, but an increase in global cerebral metabolism does not explain the large gradient between brain and rectal temperature after head injury.

*Cerebral Blood Flow.* Cerebral blood flow is normally closely coupled to the brain's metabolic rate. However, after injury, cerebral blood flow can be uncoupled and be increased or decreased relative to metabolic rate (28, 29). Since some of the heat generated by the brain is removed by the circulation, it would be expected that a decrease in cerebral blood flow could increase brain temperature. In the present study, there was not a significant relationship between global cerebral blood flow and brain temperature. Only when cerebral blood flow was markedly decreased by severe intracranial hypertension was an increase in brain temperature observed.

*Jugular Vein Temperature.* Direct measurement of intracranial temperature requires an invasive procedure that many intensivists are not capable of performing or would prefer to avoid. Brain temperature has been compared with temperature obtained at other sites in an attempt to find a site that reliably reproduces the brain temperature (8, 9, 22).

The comparison of jugular bulb temperatures with brain temperatures has shown differing results. One abstract (30) concluded that brain and jugular bulb temperatures were essentially equivalent, but other work (12, 31) has found that the temperatures do not correspond well. The present study demonstrates that jugular bulb temperature is similar to core body temperature and poorly correlated to the temperature of the brain. Similarly, variations in the magnitude of the difference between the brain and body temperature seen in individual patients with continued recording makes estimation of brain temperatures difficult to accurately predict.

*This study shows brain temperature to be significantly higher than the body temperature in patients with head injuries.*

*Conclusions.* This study shows brain temperature to be significantly higher than the body temperature in patients with head injuries, and there is considerable individual variation in the brain-to-rectal temperature gradient that cannot be predicted based on any clinical findings, such as severity or type of injury. Jugular vein temperature will not substitute for brain temperature, since jugular vein temperature reflects body, not brain temperature. These findings support the potential importance of brain temperature monitoring and also the importance of controlling fever in severely injured patients, since brain temperature may be higher than expected.

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