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Temperature Inaccuracies During Cardiopulmonary Bypass

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Abstract: Cerebral hyperthermia caused by perfusate temperature greater than 37°C during the rewarming phase of CPB has been linked to postoperative neurologic deficits. The purpose of this study was to determine the accuracy of the coupled temperature measurement system and the CDI 500 arterial temperature sensor. Seventeen patients undergoing CPB were divided into four groups, each with a different temperature probe coupled to the oxygenator. The coupled temperature measurement system and CDI temperature sensors were compared with an indwelling probe placed in direct contact with the arterial perfusate. Blood, bladder, room and water temperatures, arterial line pressure, blood flow, and hemoglobin were recorded while the patients were supported with CPB. The actual blood temperature was significantly higher than the coupled temperature measurement system for two of the four groups (mean = 1.61°C and 0.91°C, $p < 0.0001$). A significant positive correlation between the actual temperature and the coupled temperature measurement system error was observed for the same two groups ($r = 0.44$, $p < 0.0001$). The actual temperature was significantly higher than the CDI temperature in all patients (mean = 1.2°C, $p < 0.0001$). The coupling mechanism on the oxygenator generates inconsistent temperature readings. The perfusionist should consider these inconsistencies when using coupled temperature measurements and may consider the use of a direct temperature measurement system. The CDI temperature error is probably the result of inadequate flow through the sensor. On the test circuit, the flow of 170 mL/min was inadequate for circuit temperature accuracy. The accuracy of the CDI temperature drastically improved when the flow-through the sensor was increased to approximately 400 mL/min. Thus, the perfusionist must ensure adequate flow through the sensor in order for the temperature mechanism to function properly. Finally, the perfusionist can prevent cerebral hyperthermia by not allowing water temperature to exceed 37°C, when using a coupled temperature measurement system. **Keywords:** temperature probe, cerebral hyperthermia, CPB, cerebral injury. *JECT. 2005;37:38–42*

Cerebral injury is one of the most serious complications faced after cardiopulmonary bypass (CPB) (1). Hypothermia during CPB serves as a neuroprotective mechanism against cerebral injury (2). However, the rewarming phase of CPB frequently leads to cerebral hyperthermia (>37°C), a contributor to postoperative neurologic and neurocognitive dysfunction (3–5). Pervious studies established that elevated temperature after an ischemic insult worsens neurologic outcomes and even a small increase in brain temperature will exacerbate cerebral injury (3,6,7). Wass and associates subjected 21 dogs to cerebral ischemia while undergoing CPB (6). All the dogs that underwent CPB with a hyperthermic perfusion temperature (39°C) fell into coma or died from ischemia-related injury whereas all the dogs that underwent CPB with a normal perfusion temperature (37°C) had normal neurological function postoperatively (6). Thus, cerebral hyperthermia increases cerebral oxygen demand, which reduces the brain's tolerance to ischemia.

The rewarming phase of CPB is associated with decreased cerebral venous oxygen saturation ($cSVO_2$), sometimes falling to less than 50% (3,8–10). Cook et al. (3) conducted a study on cerebral venous temperature and its effects on $cSVO_2$. During the rewarming phase of CPB, cerebral venous temperatures of at least 39°C and a decrease in $cSVO_2$ were measured in all patients. In 50% of these patients, $cSVO_2$ fell to less than 50%, suggesting oxygen supply was not meeting demand. Cook et al. concluded that hyperthermia increases cerebral oxygen demand and may exacerbate ischemia-related cerebral injury, leading to confusion and neurologic dysfunction after CPB.

Yao and associates (11) monitored tympanic membrane, nasopharynx, pulmonary artery, bladder, and oxygenator outlet temperatures of patients undergoing CPB.

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Cerebral hyperthermia occurred in most of these patients during the rewarming phase of CPB, based on tympanic membrane and nasopharyngeal temperature readings. Because cerebral hyperthermia increases neuropsychological complications of cerebral embolic events, Yao et al. recommended keeping the tympanic and nasopharyngeal temperatures at or less than 37°C.

Cerebral hyperthermia should be avoided during CPB and can be achieved through accurate monitoring of brain temperature during the rewarming phase of CPB. However, commonly monitored temperature sites during CPB do not accurately reflect changes in cerebral temperature (3). Marino et al. (12) demonstrated that nasopharyngeal and esophageal measurements were lower than a jugular probe reading during CPB (12). Other studies have demonstrated that nasopharyngeal temperature underestimates cerebral temperature (3,13). Therefore, hyperthermic brain temperatures may occur if only nasopharyngeal or esophageal temperatures are monitored (12). Brain temperature can measure as much as 5°C higher than rectal or bladder temperature (14). The percent of blood flow to these sites is significantly lower than cerebral blood flow. Thus, these sites require longer to rewarm (15) and, consequently, monitoring bladder or rectal temperatures alone can lead to long periods of cerebral hyperthermia (15).

Even though temperature-monitoring sites fail to accurately predict the exact temperature of the brain during CPB, monitoring the temperature of the arterial blood leaving the oxygenator may prevent cerebral hyperthermia. Dexter and Hindman (16) demonstrated that arterial blood temperature closely predicts cerebral temperature. However, hyperthermic nasopharyngeal blood temperatures greater than 38°C are still common during the rewarming phase of CPB (17). These hyperthermic temperatures may occur because of temperature-measurement errors in the extracorporeal circuit and can lead to inaccurate temperature measurement of the arterial blood. This study examined the temperature reading accuracy in the extracorporeal circuit at Rush-Presbyterian St-Luke's Medical Center, Chicago, Illinois (RPSLMC).

At RPSLMC, arterial blood temperature is monitored at two different sites: the temperature port built into the arterial outlet of the membrane oxygenator and the arterial blood gas shunt sensor (CDI). The temperature readings were recorded in a prospective, in vivo study using 17 patients undergoing CPB and compared with an indwelling temperature probe in direct contact with the arterial blood.

METHODS

The accuracy of the arterial temperature port on the Spiral Gold™ membrane oxygenator (Jostra Bentley, Irvine, CA; oxygenator temperature) and the CDI-500 arterial blood gas shunt sensor (Terumo Cardiovascular Systems, Ann Arbor, MI; CDI temperature) were compared with an indwelling temperature probe (Mallinckrodt, St. Louis, MO) in 17 patients undergoing routine coronary or valvular procedures using CPB. The temperature of the indwelling probe was considered the actual temperature of the arterial blood. This study was approved by The Internal Review Board of RPSLMC. All patients underwent moderate hypothermic bypass (28–32°C) and were rewarmed to a bladder temperature of 36°C before weaning from CPB.

The Extracorporeal Circuit

The extracorporeal circuit at RPSLMC is comprised of a roller pump (Terumo Cardiovascular System), a heater-cooler (TCM II; Terumo Cardiovascular Systems), a hard-shell venous reservoir with the Spiral Gold™ membrane oxygenator, an arterial filter (Jostra Bentley, Irvine, CA), and CDI 500 blood gas analyzer (Figure 1). The temperature measurement system consists of a port, which is coupled to a temperature probe The Spiral Gold oxygenator has a built-in coupled temperature measurement system in the arterial outlet (Oxygenator Temperature). The CDI 500 arterial blood gas shunt sensor is connected to a stopcock at the end of the arterial filter purge line. Thus, the blood is purged from the arterial filter through the purge line, the sensor, and returned to the venous reservoir through a 24-inch female–male pressure line. The indwelling temperature probe was inserted into the luer of a 3/8-inch connector cut into the tubing between the oxygenator outlet and the arterial filter.

Data Collection

The patients were randomized into four groups. Each of these groups was assigned a temperature probe coupled to the oxygenator. Readings of the four temperature probes used in this study were identical to a mercury thermometer and to an indwelling temperature probe during water immersion. Oxygenator, CDI, indwelling temperature probe, bladder, room and heater-cooler water temperatures, as well as arterial line pressure, mean arterial pressure, blood flow, and hemoglobin were recorded throughout CPB and every minute after the start of rewarming.

Statistical Analysis

Mean temperature differences (actual temperature minus oxygenator temperature, and actual temperature minus CDI temperature) within each group were compared using the Student's paired *t* test. Mean hemoglobin, blood flow, room temperature, line pressure, bladder temperature, water temperature, and mean arterial pressure were compared between groups using the Student's *t* test with equal variance. All correlations were measured using

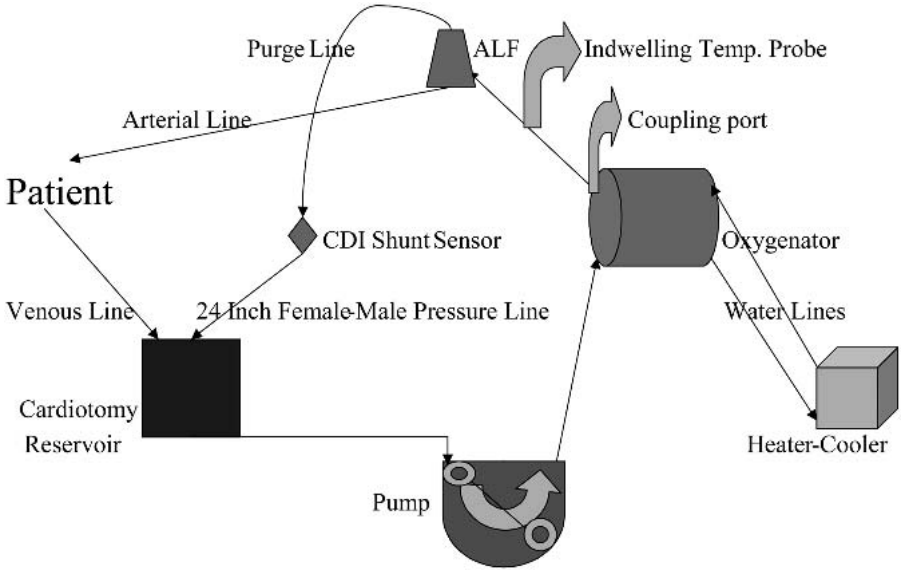

Figure 1. The extracorporeal circuit.

Pearson's correlation. Significance was considered when $p < 0.01$.

RESULTS

The actual blood temperature was significantly greater than the oxygenator outlet temperature in two of the four groups (groups 1 and 2). In addition, a significant positive correlation between the actual temperature and the oxygenator temperature-error was observed for the same two groups, as indicated by Figure 2 ($r = 0.44$, $p < 0.0001$). Furthermore, the actual temperature was significantly greater than the CDI temperature for all four groups. However, CDI temperature-error did not significantly correlate with the actual temperature in any of the four groups ($p > 0.01$). Tables 1 and 2 compare the temperature readings of the oxygenator and the CDI to the actual temperature for each group.

Water, room, and bladder temperatures; line pressure and mean arterial pressure; and hemoglobin and blood flow were not significantly different in any of the four groups ($p > 0.01$). Hyperthermic blood temperatures greater than 37°C were observed in 13 of the 17 patients (maximum temperature 38.9°C).

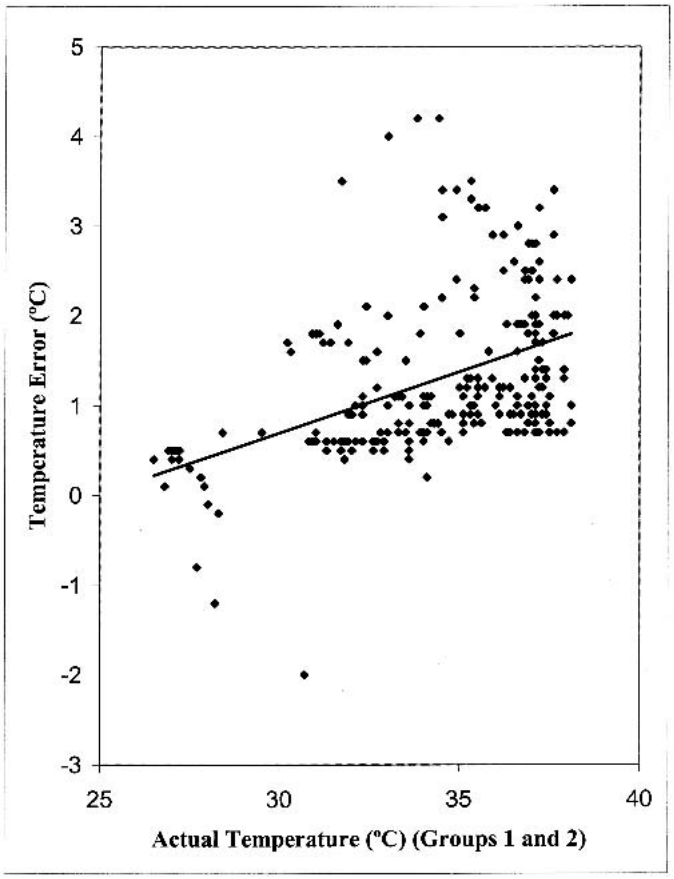

Figure 2. Actual temperature (groups 1 and 2) vs. oxygenator temperature error ($r = 0.44$, $p < 0.0001$).

DISCUSSION

The actual temperature of the arterial blood was significantly greater than the oxygenator temperature for groups 1 and 2. The four temperature probes used in each group were identical to a mercury thermometer during water immersion and were identical to each other; thus, observed temperature error in group 1 and group 2 could not be attributed to mechanical error in the probes. This temperature error may, however, have been caused by a mechanical error in the coupling mechanism of the Spiral Gold oxygenator. The temperature probe may not couple with the port perfectly, which would leave an "empty space" between some areas of the temperature well and

Table 1. Actual temperature vs. oxygenator temperature (°C).

	Actual Temperature*	Oxygenator Temperature*	Mean Difference*	Range in Differences	*p* Value
Group 1	33.9 ± 3.4	32.3 ± 2.9	1.6 ± 1.1	–2 to 4.2	<0.0001
Group 2	34.7 ± 2.2	33.8 ± 2.1	0.9 ± 0.3	0.4 to 1.5	<0.0001
Group 3	34.7 ± 2.7	34.6 ± 2.6	0.05 ± 0.3	–0.4 to 1.8	0.08
Group 4	35.1 ± 2.4	35 ± 2.4	0.09 ± 0.5	–2.9 to 1.2	0.26

*Mean ± standard deviation.

Table 2. Actual temperature vs. CDI temperature (°C).

	Actual Temperature*	CDI Temperature*	Mean Difference*	Range in Differences	*p* Value
Group 1	33.9 ± 3.4	32.7 ± 3.3	1.2 ± 0.5	0.3 to 3.7	<0.0001
Group 2	34.7 ± 2.2	33.5 ± 2.2	1.2 ± 0.4	0.6 to 2.2	<0.0001
Group 3	34.7 ± 2.7	33.6 ± 2.7	1.1 ± 0.5	0.4 to 4.9	<0.0001
Group 4	35.1 ± 2.4	33.9 ± 2.3	1.2 ± 0.5	0.6 to 2.3	<0.0001

*Mean ± standard deviation.

probe; thus, thermal conduction to the probe is not 100% complete. Furthermore, the temperature errors could not be attributed to any of the other variables measured because they were not significantly different between the four groups.

The actual temperature was significantly greater than the CDI temperature in all four groups. This temperature error did not significantly correlate with the actual temperature, which suggests that the temperature error was constant throughout the procedures. The CDI temperature error did not significantly correlate with any of the measured parameters in each group; thus, CDI temperature error could not be attributed to any of these parameters. After careful inspection of the extracorporeal circuit at RPSLMC, we observed that the CDI sensor might not have had sufficient flow going through it. This insufficiency may have been caused by the presence of the 24-inch female–male pressure line on the outlet of the sensor, which probably increased the resistance to flow through the sensor. After the pressure line was removed from the sensor and the sensor was directly connected to the venous reservoir, flow through the sensor increased from 170 mL/min to 400 mL/min. Also, the temperature readings from the CDI sensor were more consistent with the indwelling temperature probe with the increased flow. From this, we can conclude that the CDI temperature error is probably caused by insufficient flow through the sensor, and the suggested minimal flow of 35 mL/min recommended by the manufacturer of the CDI 500 in probably not high enough for the temperature sensor to read the temperatures accurately.

In summary, the rewarming phase of CPB can lead to cerebral hyperthermia, which may exacerbate cerebral injury. Hyperthermia increases cerebral oxygen demand, which reduces its tolerance to ischemic-related injury. Hyperthermia also leads to cerebral blood desaturation to levels less than 50%. Saturation this low indicates inadequate oxygen supply to the brain, which would expose cerebral tissue to ischemia. Commonly monitored temperature sites, such as bladder or nasopharyngeal temperature, do not accurately reflect cerebral temperature and often have a lower temperature reading than the brain. Thus, monitoring temperature at these sites alone may lead to cerebral hyperthermia. Monitoring the temperature of the arterial blood, however, can prevent cerebral hyperthermia if blood temperatures are not allowed to reach hyperthermic levels. The perfusionist usually monitors temperature of the arterial blood, and it is usually monitored at the outlet of the oxygenator used for CPB.

This study has provided some evidence showing that this site may provide inaccurate temperature measurements when compared with an indwelling temperature probe that is in direct contact with the blood. We speculated that this inaccuracy might be caused by the coupling mechanism on the oxygenator used at our institution. The temperature probe may not couple to the temperature port perfectly, leading to temperature readings below the actual temperature of the blood. Therefore, the perfusionist is warned when using a coupling mechanism to monitor arterial blood temperature. Coupling mechanisms may not function properly, which would lead to underestimation of the true temperature of the blood; this may produce excessive warming of the blood and perhaps causing cerebral hyperthermia. Therefore, the perfusionist is advised to use a direct temperature measurement system to ensure a blood temperature greater than 37°C does not reach the patient. Also, the perfusionist can prevent cerebral hyperthermia by keeping the water temperature under 38°C, to prevent the blood temperature from reaching hyperther-

mic levels. Through continued efforts to improve temperature control in the ECC, the perfusionists can play an important role in eliminating the incidence of cerebral hyperthermia during cardiopulmonary bypass.

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