Brachial Arterial Temperature as an Indicator of Core Temperature: Proof of Concept and Potential Applications

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Abstract: There is potential for heat loss and hypothermia during anesthesia and also for hyperthermia if heat conservation and active warming measures are not accurately titrated. Accurate temperature monitoring is particularly important in procedures in which the patient is actively cooled and then rewarmed such as during cardiopulmonary bypass surgery (CPB). We simultaneously measured core, nasopharyngeal, and brachial artery temperatures to investigate the last named as a potential peripheral temperature monitoring site. Ten patients undergoing hypothermic CPB were instrumented for simultaneous monitoring of temperatures in the pulmonary artery (PA), aortic arterial inflow (AI), nasopharynx (NP), and brachial artery (BA). Core temperature was defined as PA temperature before and after CPB and the AI temperature during CPB. Mean deviations of BA and NP temperatures from core temperature were calculated for three steady-state periods (before, during, and after CPB). Mean deviation of BA and NP temperatures from AI temperature was also calculated during active rewarming. A total of 1862 measurements were obtained and logged from eight patients. Mean BA and NP deviations from core temperature across the steady-state periods (before, during, and after CPB) were, respectively: .23 ± .25, –.26 ± .3, and –.09 ± .05°C (BA), and .11 ± .19, –.1 ± .47, and –.04 ± .3°C (NP). During steady-state periods, there was no evidence of a difference between the mean BA and NP deviation. During active rewarming, the mean difference between the BA and AI temperatures was .14 ± .36°C. During this period, NP temperature lagged behind AI and BA temperatures by up to 41 minutes and was up to 5.3°C lower than BA (mean difference between BA and NP temperatures was 1.22 ± .58°C). The BA temperature is an adequate surrogate for core temperature. It also accurately tracks the changing AI temperature during rewarming and is therefore potentially useful in detecting a hyperthermic perfusate, which might cause cerebral hyperthermia. Keywords: core, temperature, brachial, pulmonary, artery, cardiopulmonary bypass (CPB), nasopharynx, aorta, anesthesia. JECT. 2013;45:86–93
forms of surgery. We have used the temperature variation afforded by cardiac surgery with CPB to simultaneously measure several plausible surrogates for core temperature. We discuss possible clinical advantages in the direct measurement of brachial artery temperature in certain clinical situations.

MATERIALS AND METHODS

This work was carried out following review and approval from the New Zealand Ministry of Health, Northern Regional Ethics Committee (approval number NTX/06/04/029).

Data were collected from eight adult patients undergoing open heart surgery at the Green Lane Cardiothoracic Unit, Auckland City Hospital, who required insertion of a Swan-Ganz catheter as part of their standard clinical management were invited to participate. Patient characteristics and surgical procedures are shown in Table 1. All patients underwent CPB with mild hypothermia (average patient temperature on stable bypass was 32.1 ± 1.2°C). Our methods of temperature monitoring and rewarming were standardized. Rewarming was prescribed at a rate of less than .5°C per minute with a maximum arterial blood temperature (as measured just distal to the arterial filter by Capiox thermistor) at 37°C. There was no other requirement for standardization of anesthesia, CPB, or surgical practice, and relevant decisions were left to the discretion of the procedural clinicians.

Temperature was measured at the following sites using commercially available disposable medical sensors:

1. Nasopharynx (NP). A nasopharyngeal temperature probe (Vital Temp Inc.) was inserted into the nasopharynx according to the procedural anesthetist’s standard approach. Temperatures were recorded continuously from this site from the time of insertion to when the patient left the operating room.

2. Pulmonary artery (PA). After induction, a Swann-Ganz catheter (Edwards Lifesciences) was inserted and advanced into the pulmonary artery in the standard fashion. This was used to measure pulmonary artery temperature from insertion and calibration to going on CPB and from separation from CPB to the time the patient left the operating room.

3. Aortic inflow cannula (AI). A three-way tap and Luer thermistor (Medtronic) was placed at the proximal end of the aortic inflow cannula. This is a nonstandard measurement arrangement and so its accuracy was verified as being better than ±1°C using a precision water bath before the clinical monitoring phase. This measured aortic cannula blood inflow temperature during CPB.

4. Brachial artery (BA). A PiCCO thermodilution catheter (Pulsion Medical Systems) containing a temperature thermistor was inserted into the brachial artery at the anteceubital fossa and doubled as the arterial pressure monitoring line and arterial blood sampling portal. This measured brachial artery temperature from the time of insertion to the time the patient left the operating room.

After use and before disposal, the sensors were tested in a temperature-controlled water bath against a reference thermometer (Hart Scientific 5612). In every case, the accuracy was well within the manufacturer’s specification (Table 2).

We divided the operation into five periods: pre-CPB, CPB—active cooling, stable CPB, CPB—active rewarming, and post-CPB. We compared peripheral site temperatures (i.e., BA and NP) with a surrogate for core temperature during periods of relative temperature stability (“steady-state periods”); that is, during the pre-CPB, stable CPB, and post-CPB periods when no active cooling or warming was taking place and when any lag in NP temperature after active cooling and active rewarming during CPB had resolved (Figure 1 shows these periods for a representative patient). Core temperature during these periods was assumed to be represented by the PA temperature in the pre- and post-CPB periods and by the AI during the stable CPB period.

Table 1. Patient characteristics and surgical procedures.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Gender</th>
<th>Surgery</th>
<th>CPB Minimum Temperature</th>
<th>Steady State Time</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Pre-CPB</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>65</td>
<td>M</td>
<td>CABG</td>
<td>31.7</td>
<td>9:26–9:52</td>
</tr>
<tr>
<td>2</td>
<td>81</td>
<td>M</td>
<td>CABG</td>
<td>32.1</td>
<td>11:59–12:52</td>
</tr>
<tr>
<td>3</td>
<td>52</td>
<td>M</td>
<td>CABG + MVR</td>
<td>32.1</td>
<td>14:04–14:23</td>
</tr>
<tr>
<td>4</td>
<td>60</td>
<td>F</td>
<td>CABG</td>
<td>30.7</td>
<td>8:35–10:16</td>
</tr>
<tr>
<td>5</td>
<td>49</td>
<td>M</td>
<td>Mediastinal re-exploration</td>
<td>31.1</td>
<td>12:03–12:23</td>
</tr>
<tr>
<td>6</td>
<td>87</td>
<td>F</td>
<td>AVR</td>
<td>28.7</td>
<td>13:50–13:54</td>
</tr>
<tr>
<td>7</td>
<td>59</td>
<td>M</td>
<td>CABG</td>
<td>30</td>
<td>9:45–9:55</td>
</tr>
<tr>
<td>8</td>
<td>70</td>
<td>M</td>
<td>CABG</td>
<td>32.1</td>
<td>10:12–10:27</td>
</tr>
</tbody>
</table>

AVR, aortic valve replacement; CPB, cardiopulmonary bypass; CABG, coronary artery bypass graft; MVR, mitral valve replacement.
As a secondary comparison, we compared the aortic inflow temperature with peripheral site temperatures during rewarming. The aim was to evaluate how accurately the brachial thermistor tracked the arterial inflow temperature at a time when the nasopharyngeal temperature is known to lag behind the latter. BA and NP responsiveness was estimated by measuring the time taken from the onset of rewarming until the respective site temperature started to increase (the “initial lag”) and the time between the core temperature maximum until the peripheral site maximum (the “equilibrium lag”).

All measurements made during the respective periods were entered into the analysis providing the appropriate core site and both of the peripheral sites were concomitantly monitored. Data from all four sites were continuously fed to a personal computer and standardized to 30-second intervals. The BA epoch was 12 seconds, so the measurement closest in time to the 30-second interval point was used when compiling the data. Because operation lengths varied, the total number of measurements varied between patients (and operating periods).

### Statistical Analyses

For each patient, we examined the deviation between the core temperature and the peripheral sites and calculated the mean square error (MSE) for each steady-state operative period. MSE can be decomposed into the (sum of) average deviation and the variance of the estimator (NP or BA) (see equation 1)

$$MSE_i = E(\hat{\theta} - \theta)^2 + Var(\hat{\theta} - \theta)$$  \hspace{1cm} (1)

where $\hat{\theta}$ is the peripheral temperature (i.e., BA or NP), and $\theta$ is the core temperature measured for the $i$th patient.

The square root of the first component in equation 1 is a measure of “bias”—the difference between the peripheral temperature and the core temperature for each patient. The square root of the second component in equation 1 quantifies peripheral temperature “stability” relative to the core, i.e., it is a measure of how the deviation between the core and peripheral site varies over time. Both of these measures were then averaged across patients to give interpatient estimates of bias and stability for BA and NP. Confidence intervals for the average interpatient bias and stability were typically calculated using the t-distribution; however, when significant evidence of nonnormality data was found (using a Shapiro-Wilk test), a confidence interval for the median was approximated using linear interpolation on an inverted sign test.

All statistical analyses were done using R (Version 2.15.1) with associated packages (10).

### RESULTS

#### Steady-State Measurements

During steady-state periods, both peripheral sites gave very stable estimates of the measured core temperature (Table 3), and the average temperature deviation for these sites (from the core) for any patient was less than 1°C (Figure 2).

There was some evidence that the BA temperature was biased (relative to core temperature) in the pre-CPB, CPB, and post-CPB steady-state periods ($p = .04, .04$, and $.002$, respectively). However, the size of BA bias was small in all periods; the largest BA bias occurred during

<table>
<thead>
<tr>
<th>Location</th>
<th>Sensor</th>
<th>Accuracy (°C)</th>
<th>Epoch</th>
<th>Manufacturer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brachial artery</td>
<td>3-Fr thermodilution catheter</td>
<td>.1</td>
<td>12 seconds</td>
<td>Pulsion Medical Systems</td>
</tr>
<tr>
<td>Nasopharynx</td>
<td>Vital temperature probe</td>
<td>.1</td>
<td>30 seconds</td>
<td>Vital Signs Inc.</td>
</tr>
<tr>
<td>Arterial cannula</td>
<td>Luer thermistor</td>
<td>.1</td>
<td>30 seconds</td>
<td>Terumo</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>1.5-mL CAP 131HF7</td>
<td>.2</td>
<td>30 seconds</td>
<td>Edwards Life Sciences</td>
</tr>
</tbody>
</table>

*All sensors were disposable, precalibrated, single-use devices.
Table 3. The brachial artery (BA) and nasopharyngeal (NP) sites average bias (from core temperature) and stability (average standard deviation).

<table>
<thead>
<tr>
<th>Period</th>
<th>Bias</th>
<th>Stability</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BA</td>
<td>NP</td>
</tr>
<tr>
<td>Pre-CPB</td>
<td>.23 ± .25</td>
<td>.11 ± .19*</td>
</tr>
<tr>
<td>CPB</td>
<td>−.26 ± .30</td>
<td>−.10 ± .47</td>
</tr>
<tr>
<td>Post-CPB</td>
<td>−.09 ± .05</td>
<td>−.04 ± .30*</td>
</tr>
</tbody>
</table>

*An outlier in the distribution, so medians were used for inferential tests. CPB, cardiopulmonary bypass.

CPB; the 95% confidence interval (CI) suggests the bias was between .01 and .51°C lower than the core (AI) temperature in this period (Table 3). There was also no evidence that NP temperature had any bias that was of practical significance during the steady-state periods. The largest 95% CI for NP suggested that the NP temperature was somewhere between .3°C above and .49°C below the core temperature (during CPB).

The interpatient distributions of pre- and post-CPB NP deviations from the core temperature both contained outliers. The median NP deviation from the core temperature (and corresponding 95% CI) for these periods were: .11°C (−.05 to .29) and −.08°C (−.28 to .21), respectively.

Rewarming Measurements

During rewarming, the BA site accurately tracked the AI temperature; both the initial temperature lag and the equilibrium temperature lag were less than the 30-second sampling resolution. The mean difference between the BA and AI temperatures over this period was .14 ± .36°C. In contrast, there was a clear lag in equilibration of NP temperatures for all patients (Figure 3 shows rewarming values for a representative patient.) leading to differences up to 5.3°C between the NP and AI temperatures (Table 4). The mean difference between the NP and AI temperature was 1.2 ± .58°C.

Table 4. The responsiveness of the nasopharyngeal probe (NP) and the maximum temperature between the aortic arterial inflow (AI) and NP during cardiopulmonary bypass rewarming.*

<table>
<thead>
<tr>
<th>Patient</th>
<th>Initial Lag (minutes)</th>
<th>Equilibrium Lag (minutes)</th>
<th>Maximum Difference (°C) (AI – NP)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>2</td>
<td>.8</td>
</tr>
<tr>
<td>2</td>
<td>1.5</td>
<td>6</td>
<td>2.6</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>23</td>
<td>3.2</td>
</tr>
<tr>
<td>4</td>
<td>3</td>
<td>9</td>
<td>1.9</td>
</tr>
<tr>
<td>5</td>
<td>3</td>
<td>41</td>
<td>5.3</td>
</tr>
<tr>
<td>6</td>
<td>2.5</td>
<td>2.5</td>
<td>3.6</td>
</tr>
<tr>
<td>7</td>
<td>2</td>
<td>9</td>
<td>1.6</td>
</tr>
<tr>
<td>8</td>
<td>2</td>
<td>4.5</td>
<td></td>
</tr>
</tbody>
</table>

*“Initial lag” measured the time between the start of rewarming and when NP started to warm. “Equilibrium lag” was the time between the core temperature (as measured by AI) reaching its maximum to when NP temperature reached its maximum.
Relevant NP, BA, PA, and AI temperature measurements for all patients recorded in this study are shown in Figure 4.

**DISCUSSION**

There is a well-known propensity for core temperature to fall during the administration of a general anesthetic. This occurs quickly at first, resulting mainly from the redistribution of heat from core to periphery as a result of vasodilation induced by anesthetic agents (11). Subsequently there may be heat loss, mainly by radiation and convection, the rate of which is dependent on multiple factors such as patient exposure, the temperature of the operating room, and the nature of the surgery. Heat conservation or active warming measures including warmed fluids, warming mattresses, and forced-air warmers are consequently used in an attempt to maintain normothermia. Thus, anesthetized patients may easily become hypothermic because of heat loss or indeed hyperthermic if warming strategies are used to excess. Either extreme is potentially harmful. Hypothermia may have adverse effects on the myocardium (1,12), impair hemostasis (13–15), and promote surgical wound infection (16). Hyperthermia may be harmful to the brain, particularly in patients with cerebral ischemia or head injury (2,17–19). It follows that there is good reason for wanting accurate, continuous temperature monitoring in patients undergoing prolonged general anesthesia.

“Core temperature” is a poorly defined term. In practice, one is often interested in the temperature of specific organs, notably the heart or the brain, and these may differ from each other and from other organs depending on the clinical setting. There are multiple sites that offer different perspectives on the body’s thermodynamic state. Such sites include the tympanic membrane, esophagus, nasopharynx, and bladder, each with its own normal
temperature range (20). It can be argued that the tympanic membrane should be viewed as the gold standard because it accurately reflects both the arterial inflow temperature to the brain (21) (often a critically important clinical consideration) and the temperature at the body’s thermoregulatory center (within the hypothalamus) (20). However, with respect to the latter point, normal thermoregulation is substantially deranged during anesthesia (22,23) and the relevance of the hypothalamic temperature is therefore less obvious in this context. Moreover, others have argued that multiple extrahypothalamic sites have a role in thermoregulatory control and that there is little physiological justification from this perspective in focusing on the hypothalamic (or tympanic membrane) temperature for monitoring purposes (24). We have chosen to use the temperature of circulating blood in the thorax as an operational definition of core temperature because this reflects both the temperature of arterial blood leaving the central circulation (en route to the brain and other destinations) and the reservoir of body heat that is most isolated from temperature gradients at the skin.

We have shown that the BA temperature is an adequate surrogate for core temperature. Indeed, both the NP and BA temperatures reflected core temperature (defined as previously) with acceptable accuracy when the patient was in a steady state (no active warming or cooling). The accuracy of the BA temperature is not surprising because of the short distance between the central circulation and the antecubital fossa (approximately 50 cm) and the high velocity of blood flow over this distance. Blood velocities of 9 cm/sec and approximately 30 cm/sec (25,26) have been measured in the brachial artery (11) and aorta, respectively. So with a transit time between 2 and 6 seconds, there is little opportunity for heat loss from deep vessels that are not arranged for this purpose.

During active rewarming, the BA temperature accurately tracked the AI temperature from the CPB machine, whereas the NP temperature lagged behind (Table 2; Figure 1). The responsiveness of the NP probe varied considerably between patients. The time it took to reach the BA temperature varied between 2 and 41 minutes. We suspect that this variation may be introduced by the position of the temperature sensor; the probe may not be in good contact with the surrounding tissue in those patients exhibiting low NP responsiveness. These observations are not new but deserve to be reinforced. The potential for NP temperature to lag behind arterial inflow temperature and, consequently, to lag behind the temperature of well-perfused tissues such as the brain during active rewarming has previously been demonstrated (27,28). Not surprisingly, cerebral hyperthermia can occur during rewarming even when NP temperatures are normal if the AI temperature from the CPB machine is allowed to exceed 37°C (29). Because even mild cerebral hyperthermia may be harmful, this finding has formed the basis for advocating avoidance of arterial inflow temperatures greater than 37°C during rewarming (30). This recommendation is supported by human data suggesting better neurocognitive outcomes when rewarming is slower and peak cerebral rewarming temperature is reduced (31). Not surprisingly, it has been enthusiastically embraced by clinical perfusionists. However, it is recognized that numerous factors can influence the accuracy of the arterial perfusate temperature measurement (32). Indeed, one study using relevant equipment configurations warns that the temperature of the perfusate can be significantly (more than 1°C) higher than what is measured by the CPB machine stating: “The coupling mechanism on the oxygenator generates inconsistent temperatures readings. The perfusionist should consider these inconsistencies when using coupled temperature measurements and may consider the use of a direct temperature measurement system” (33).

Although there are various ways in which this hazard can be managed, none are likely to be completely failsafe. Thus, one potential advantage for measuring temperature in the BA during cardiac surgery (in which patients require an invasive arterial catheter for monitoring of blood pressure irrespective of the temperature monitoring strategy) is that it serves as a “final check” on the arterial inflow temperature during rewarming. A BA temperature less than 37°C would confirm that inadvertent cerebral hyperthermia is being avoided. However, because the BA temperature is so responsive to AI temperature, it is not a suitable measure for completeness of tissue rewarming, and the nasopharyngeal temperature should still be measured for this purpose.

When else might BA temperature monitoring be used? At the present time, the most common means of intraoperative temperature monitoring in our experience is the insertion of a flexible probe with a distal thermistor into the NP position. This is a common and safe intervention, but it is not without problems. First, although complications are rare, they do occur and are probably underreported. Nasal probe insertions can result in epistaxis, necessitating transfusion, nasal packing or surgery, and occasionally intensive care admission (34). Such a case occurred (but was not reported in the literature) in our own institution in late 2008 (Dr. N. Forbes, personal communication). A NP temperature probe might be avoided in a patient with such a history or a history of recurrent epistaxis from any cause. Second, there are occasional cases when a NP temperature probe either cannot be used or is relatively contraindicated such as in some complicated nasofacial procedures, in patients who have undergone recent transphenoidal pituitary surgery, or in patients with a base of skull fracture. We
do accept that there are simpler temperature monitoring alternatives to a NP probe than a BA catheter and we wish to emphasize that our commentary should not be interpreted as advocacy for routine or widespread use of BA temperature monitoring during anesthesia. However, in a case in which a NP probe is contraindicated and the patient is to have an arterial cannula placed as a matter of routine, a thermistor-equipped cannula is unlikely to increase the risk and is a plausible temperature-monitoring solution. It is notable that in our series, the thermistor catheters functioned adequately as arterial blood sampling portals in all cases. No complications arose as a result of cannulation of the BA in this small group.

**Study Limitations**

There are two potential limitations that deserve mention. First, the study was limited by its small sample size. However, although there were only eight patients in the study, the intrapatient BA measurements (relative to the core temperature) were very stable over time, and there was relatively little interpatient bias or variability. Second, the different recording times and epochs could also cause discrepancies when the data were compiled into 30-second intervals. Because BA temperature measurements were made every 12 seconds and a measurement was chosen every 30 seconds to match the NP and core temperatures, a discrepancy of up to 6 seconds could have occurred. However, this would have been irrelevant during periods of temperature stability, and the possible temporal discrepancies were small relative to the slow responsiveness at the NP site during times of temperature change. The 30-second resolution of the data also meant that we were unable to accurately estimate the responsiveness of the BA temperature; all BA temperatures responded to the core temperature by the next measurement.

This study identifies the BA as a plausible site for accurate monitoring of core temperature. At present (to our knowledge), there is no thermistor-equipped cannula suitable for insertion in the radial artery (where invasive arterial pressure monitoring is most commonly established), but if a suitable device could be produced, it would be interesting to test the possibility that this site would be as suitable as the BA. However, our real interest in demonstrating the value of the BA as a site for measuring temperature lies in planned work to develop a percutaneous BA temperature probe. Our results support the conceptual basis for this project and also suggest that the use of invasive BA temperature monitoring may be valuable as a check on arterial inflow temperatures during rewarming from CPB, a matter to which attention has been drawn in a recent evidence-based review of perfusion best practice (23).

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