Temperature monitoring during cardiopulmonary bypass—do we undercool or overheat the brain?

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Abstract

Objective: Brain cooling is an essential component of aortic surgery requiring circulatory arrest and inadequate cooling may lead to brain injury. Similarly, brain hyperthermia during the rewarming phase of cardiopulmonary bypass may also lead to neurological injury. Conventional temperature monitoring sites may not reflect the core brain temperature ($T_B$). We compared jugular bulb venous temperatures (JB) during deep hypothermic circulatory arrest and normothermic bypass with Nasopharyngeal (NP), Arterial inflow (AI), Oesophageal (O), Venous return (VR), Bladder (B) and Orbital skin (OS) temperatures.

Methods: 18 patients undergoing deep hypothermia (DH) and 8 patients undergoing normothermic bypass (mean bladder $T_B$—36.29°C) were studied. For DH, cooling was continued to 15°C NP (mean cooling time—66 min). At pre-determined arterial inflow $T_B$; NP, JB and O $T_B$’s were measured. A 6-channel recorder continuously recorded all $T_B$’s using calibrated thermocouples.

Results: During the cooling phase of DH, NP lagged behind AI and JB $T_B$’s. All these equilibrated at 15°C. During rewarming, JB and NP lagged behind AI and JB was higher than NP at any time point. During normothermic bypass, although NP was reflective of the AI and JB $T_B$ trends, it underestimated peak JB $T_B$ ($P = 0.001$). Towards the end of bypass, peak JB was greater than the arterial inflow $T_B$ ($P = 0.023$). Conclusions: If brain venous outflow $T_B$ (JB) accurately reflects brain $T_B$, NP $T_B$ is a safe surrogate indicator of cooling. During rewarming, all peripheral sites underestimate brain temperature and caution is required to avoid hyperthermic arterial inflow, which may inadvertently, result in brain hyperthermia.

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1. Introduction

Temperature management during cardiopulmonary bypass (CPB) continues to be an important issue. Sub-optimal temperature management may contribute to the development of adverse outcomes, in particular, neurological injury. For aortic arch surgery, the increased ischaemic tolerance of deep hypothermia allowing a period of circulatory arrest has become the mainstay of cerebral protection since its introduction by Borst [1] as it facilitates a bloodless operative field without the hindrance of clamps and cannulae. The safe duration of circulatory arrest is dependent upon adequate brain cooling and if this is incomplete, brain injury will result. Hyperthermia during the rewarming phase of hypothermic CPB or normothermic CPB has been shown to be associated with adverse neurological outcomes [2–4].

As direct brain temperature measurement is not possible other than in some neurological procedures, surrogate temperature monitoring sites are used to estimate the brain temperature [5,6]. The most commonly monitored sites include nasopharynx (NP), oesophageal (O), bladder (B) in addition to arterial inflow (AI) and venous return (VR). Other sites include tympanic membrane, rectum and skin. Various studies have shown that these standard sites may not reflect the true brain temperature [3,7].
We sought to compare the routinely monitored temperature sites to the brain temperature. As 99% of jugular bulb blood flow is derived from the brain circulation, jugular bulb (JB) temperature should reliably predict brain temperature. This has been validated in neurosurgical patients undergoing profound hypothermia [7]. We used JB as a reference temperature to compare the accuracy of surrogate sites. In this study, we investigated whether conventional temperature monitoring sites adequately reflect brain temperature during deep hypothermia and normothermic CPB by comparing jugular bulb venous temperature (JB) with routinely monitored peripheral temperature sites including NP, AI, O, VR, B and orbital skin (OS) temperatures.

2. Study design and methodology

After ethical approval and informed consent, 18 patients undergoing hypothermic circulatory arrest for aortic surgery and 8 patients undergoing normothermic CPB for coronary artery bypass or valve surgery were studied. Anaesthetic technique was standardised with etomidate, pancuronium and fentanyl induction and intravenous propofol and alfentanil for maintenance.

2.1. Jugular bulb temperature monitoring

After induction of anaesthesia, a triple lumen central line was inserted into the jugular bulb by the Seldinger method. Prior to insertion of the triple lumen catheter, a calibrated thermocouple (Columbus Instruments®, Ohio, USA) was inserted through one of the lumens. After the guide wire was placed in the internal jugular vein, the triple lumen catheter was advanced cranially till resistance was encountered. The catheter was then withdrawn while aspirating on the distal lumen till there was an efflux of venous blood into the aspirating syringe. This represented adequate placement of the catheter in the jugular bulb. In patients who underwent hypothermic circulatory arrest, lateral skull radiographs post-surgery were used to confirm catheter position.

2.2. Peripheral temperature monitoring sites

In addition to JB monitoring, calibrated temperature probes were used to measure the NP, AI, VR, B and OS temperatures. The nasopharyngeal probe was placed 5 cm from the external nares and the bladder temperature was measured using a urinary catheter temperature probe. The arterial and venous temperatures were measured directly from the CPB circuit. All the temperature probes were pre-calibrated to an identical reference and data was collected on to a dedicated laptop computer. All patients underwent neurological examination 24–48 h post-procedure.

2.3. Profound hypothermia and circulatory arrest

The cardiopulmonary bypass circuit comprised a single roller pump (Stockert® ‘Shiley caps’) and integrated hard shell venous reservoir, oxygenator and heat exchanger unit (Terumo® Capiox SX-18, Stockert ‘CAPS®’ heater chiller). The circuit was primed with 1500 ml of isotonic electrolyte solution. Oxygenated blood was returned to the patient through an ascending aorta, arch or femoral artery cannula (Sarns® 18–24 Fg) to achieve antegrade flow when possible. Pump flows were maintained at 2.4 l/min/m². A mean blood pressure of 55–70 mmHg was maintained using aliquots of a-adrenergic receptor agonist or nitrate infusion. An alpha stat pH strategy was utilised in all cases. Arterial inflow and water bath temperature gradient was always maintained below 10 °C. Cooling was continued until the NP temperature reached 15 °C. Circulation was arrested at this temperature and following the completion of the arch reconstruction, orthograde cardiopulmonary bypass via a prosthetic graft side-arm, was reinstalled and rewarming commenced. CPB was discontinued when the NP temperature had been maintained at 36 °C for 10 min. All the patients had additional topical head cooling by ice packs and all received dexamethasone 100 mg and mannitol 1 g/kg approximately 20 min prior to circulatory arrest.

2.4. Normothermic bypass

Normothermic bypass patients underwent coronary artery bypass surgery or valve surgery using a similar bypass circuit. Normothermia was defined as maintaining a bladder (B) temperature of 35–37 °C. Anaesthetic induction and maintenance techniques were identical and mannitol 0.5 g/kg was administered pre-bypass. Dexamethasone was not used. The AI temperature was not allowed to exceed 37.5 °C in either group.

2.5. Statistical methods

Collected data were transferred onto a computer spreadsheet and analysed using SPSS (SPSS for windows, version 11.0, Chicago Inc). Data was tested for normality and compared using a t test or Mann Whitney U test as appropriate. Statistical significance was assigned if P ≤ 0.05.

Table 1
Demographic and bypass details

<table>
<thead>
<tr>
<th></th>
<th>HCA</th>
<th>Normothermia</th>
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<tbody>
<tr>
<td>Mean age (SD)</td>
<td>57.6 (14.8)</td>
<td>65.63 (9.08)</td>
</tr>
<tr>
<td>Range</td>
<td>32–81</td>
<td>55–79</td>
</tr>
<tr>
<td>Sex</td>
<td>8M:10F</td>
<td>7M:1F</td>
</tr>
<tr>
<td>CPB time (SD)</td>
<td>221.6 (57)</td>
<td>75 (20.6)</td>
</tr>
<tr>
<td>NP temp on ITU return (SD)</td>
<td>33.1 (0.9)</td>
<td>35.75 (SD 0.44)</td>
</tr>
<tr>
<td>Range</td>
<td>31.8–35</td>
<td>35–36.4</td>
</tr>
</tbody>
</table>
3. Results

There were no complications due to the JB temperature probe insertion and no patients developed a neurological deficit. Lateral skull X-ray confirmed adequate positioning of the JB catheter in all patients in the circulatory arrest group. Patient demographics are summarised in Table 1 and the procedures in Table 2.

3.1. Deep hypothermia

For the profound hypothermia—circulatory arrest group, the mean cooling time was 66 min (SD 18.5) (Range 40–116). During the cooling phase, NP lagged behind the AI and JB temperatures (Fig. 1) but all temperatures equilibrated when the NP reached the nadir of 15 °C. The mean circulatory arrest time was 29.1 min (SD 12.5). Mean rewarming time was 65.1 min (SD 17.6) (Range 45–106). During the rewarming phase, JB and NP temperatures lagged behind AI temperatures. JB temperature was noted to be higher than the NP temperature at all time points during rewarming (mean difference 1.2 °C $P < 0.001$). Despite prolonged rewarming, there was a significant temperature drop following return to intensive care unit.

3.2. Normothermia

In the normothermic CPB patients, the mean NP, JB and B temperatures were 35.2, 35.2 and 35.7 °C, respectively, at the start of CPB. AI temperature was maintained below 37.5 °C. The temperature trends for one patient are depicted in Fig. 2. The bladder (B) and orbital skin (OS) temperatures were consistently lower than NP and JB temperatures (Fig. 3). Towards the end of bypass, all the temperatures including NP (mean 37.06 °C SD 0.42) ($P = 0.001$), AI (mean 37.02 °C SD 0.43) ($P = 0.023$) and B (mean 36.74 °C SD 0.45) ($P < 0.001$) underestimated the peak JB temperature (mean 37.52 °C SD 0.29). JB temperatures $>37$ °C were observed if rewarming continued (despite control of AI temperature) after nasopharyngeal temperature exceeded 36.8 °C. In all the patients,

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**Table 2**

Operative details

<table>
<thead>
<tr>
<th></th>
<th>Deep hypothermia group ($n = 18$) (Extent of aortic replacement)</th>
<th>Normothermia group ($n = 8$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ascending aorta</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Arch</td>
<td>1</td>
<td>Valve replacement</td>
</tr>
<tr>
<td>Arch and descending aorta</td>
<td>6</td>
<td>Combined CABG and valve surgery</td>
</tr>
<tr>
<td>Descending and thoraco-abdominal</td>
<td>6</td>
<td></td>
</tr>
</tbody>
</table>

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Fig. 1. Mean temperature trends during the cooling and rewarming phase of deep hypothermic circulatory arrest. JB, jugular bulb; NP, nasopharyngeal; Art Inflow, arterial inflow temperature.

Fig. 2. Jugular Bulb (JB), Nasopharyngeal (NP) and Arterial (AI) temperature trends during CPB in one patient undergoing normothermic cardiopulmonary bypass. Note the x axis is measured in seconds.

Fig. 3. Bladder (B), and Skin (OS) temperature in relation to Nasopharyngeal (NP) and Jugular Bulb (JB) during CPB in one patient undergoing normothermic cardiopulmonary bypass. Note the x axis is measured in seconds.
when the peak JB temperature was reached, all other peripheral temperatures including the NP temperature under-estimated this peak temperature ($P = 0.001$) (Fig. 4). This underestimation of peak JB temperature was not observed in the patients undergoing profound hypothermia.

4. Discussion

Utilisation of hypothermia has been an important neuroprotective strategy throughout the history of cardiac surgery. Bigelow et al. demonstrated that oxygen consumption falls in a linear fashion with body temperature and that ischaemic tolerance is extended by cooling [8,9]. Cerebral oxygen consumption decreases to approximately 20% of normothermic levels when the temperature is reduced to 20 °C [9]. In addition, hypothermia may afford neuroprotection by a variety of complex mechanisms including decreased excitatory transmitter release, reduced ion influx and reduced vascular permeability [10]. Even a 2 °C reduction from normothermia may have beneficial effects [11]. During core-cooling on CPB, brain cooling may not be homogenous. Thus, conventional temperature monitoring sites such as NP temperature may not reflect true brain temperature [12,13].

While deep hypothermia has an established role in aortic surgery, there has been a renewed interest in normothermia during CPB for cardiac surgery including coronary and valve surgery [14]. Normothermic bypass has been shown to have various systemic advantages [15–17] but there have also been reports of increased adverse neurological events [18,19]. One possible mechanism for increased brain injury is the potential development of brain hyperthermia during CPB [4]. Brain hyperthermia may not be apparent if conventional temperature monitoring sites are used as these may be inaccurate and may therefore underestimate true brain temperature.

In cardiac surgery, there is a necessary reliance on indirect peripheral temperature measurements as guide to brain temperature and the most commonly used site is the nasopharynx. Crowder et al. [7] have shown that the core brain temperature is reflected by the JB temperature. Little is known about intra-cerebral shunts and how they change during deep hypothermia and normothermic CPB. Stone et al. [3] demonstrated that there is a significant gradient between the near surface temperature (depth of 1 cm) and that measured at an intra-parenchymal depth of 4 cm, the latter being close to 3 °C warmer than the NP temperature. But this study was done in patients whose cranium was open which could influence the temperature changes.

Our study demonstrates that during the cooling phase of profound hypothermic CPB, nasopharyngeal temperature monitoring is a satisfactory surrogate of brain temperature. Thus if a target NP temperature is chosen for initiation of circulatory arrest, the operator can be confident that true brain temperature is at or below the NP measurement. The findings in our study provide a degree of confidence in the nasopharyngeal temperature site as an index of brain temperature during cooling.

We are not aware of any previous reports of jugular bulb temperature measurement during profound hypothermia and normothermic CPB. The thermocouple we used was chosen due to its large calibrated range of temperature measurement, thus allowing accurate measurement of temperatures as low as 15 °C. Studies on cooling prior to HCA have reported that a cooling duration of 50 min is required to achieve electrocerebral silence [20]. Though we did not use EEG monitoring, which we recognise as a limitation, our mean cooling duration was in excess of 50 min. By taking JB temperature as a marker of brain temperature, we are confident that by achieving a NP temperature of 15 °C with the cooling protocol we have described, we have achieved target brain cooling prior to HCA. A faster rate of cooling might not be able to achieve this [3].

During normothermic CPB, brain temperature may be higher than the temperature observed at any surrogate site particularly towards the end of bypass and brain hyperthermia may occur inadvertently. Unless monitored, arterial inflow temperatures may exceed the corporeal temperature and lead to brain heating. Further, increasing brain and corporeal metabolism during rewarming may lead to additional heat generation. The findings of our study are consistent with previous reports of temperature measurement in moderately hypothermic bypass. Grocott et al. showed that during rewarming from moderate hypothermia, NP underestimates the JB temperature [21] while Bissonnette reported a 1–2 °C higher JB temperatures when compared to tympanic, rectal or oesophageal temperatures in children and infants having cardiac operations [22]. In these studies, excessive arterial inflow temperature was regarded as the main culprit for increased brain temperatures.
Our study demonstrated that JB temperature can exceed AI temperature. This finding is important as it indicates a clear potential to generate brain hyperthermia despite cautious rewarming strategies. The mechanism for this effect is unclear but possibly reflects active brain metabolism and heat generation during normothermic CPB. This finding was not observed in the deep hypothermic group possibly due to continued suppressed cerebral metabolism [23].

The duration of rewarming and control of thermal gradients may also be important. Slow rewarming to prevent rapid heterogeneous changes in brain temperature may be beneficial.

During warm CPB techniques, arterial inflow temperature should be carefully monitored and a nasopharyngeal temperature ceiling of somewhat less than 37 °C should be a prompt to discontinue rewarming prior to CPB separation if brain hyperthermia is to be avoided.

References


Appendix A. Conference discussion

Dr D. Chambers (London, UK): I wonder whether you could develop from your data some sort of factor whereby you could calculate the right temperature from your nasopharyngeal temperature, if you like. Did you always see the same sort of difference between your jugular bulb temperature and your nasopharyngeal temperature, or was it variable?

Dr Kaukuntla: Well, because in the normothermic group, there was hardly any rewarming involved. The patients would have probably cooled to about 34.5° during the time the mammary artery was being harvested. So there was only a very short period of rewarming. Once the required temperature was attained, there was no definite relationship between the temperature just after the oxygenator and the bulb temperatures were a good reflection of the arterial inflow; but they hardly any rewarming involved. The patients would have probably cooled to about 34.5° during the time the mammary artery was being harvested. So there was only a very short period of rewarming. Once the required temperature was attained, there was no definite relationship between the temperature just after the oxygenator. Did you always see the same sort of difference between your jugular bulb temperature and your nasopharyngeal temperature, or was it variable?
always either lagged behind or lagged in front, if you see, depending upon where in the hypothermic circulatory curve the time point was.

What we found was that in the hypothermic group they tend to even out. But in the normothermic group, towards the end of bypass, the jugular bulb temperature was actually even more than the arterial inflow. This was a very surprising result for us. We did ponder on the reasons of why the jugular bulb temperature is more than the arterial inflow temperature, and we can only speculate the reasons. We think that one of the reasons is - in normothermia there is no heat debt, if I could use that term. So the brain venous outflow temperature is temperature of the blood going into the brain plus the brain metabolic heat which is generated in normothermia. So we think that probably is the reason.

A. Philipp (Regensburg, Germany): Why didn’t you correlate the temperature of the jugular venous blood and the temperature detected with a probe in the typanum? Tympanic temperature is just easy to monitor, and, as you certainly know, it correlates very exactly with the brain temperature.

Dr Kaukuntla: Well, there is conflicting evidence. Some authors have, yes, as you’ve rightly said, say that it reflects the core brain temperature quite well. But at the same time, there are some authors who have said that tympanic membrane probably doesn’t represent the true temperature at different parts of the brain. Besides this another reason was that when we set up this study, we didn’t have the kit to measure tympanic membrane temperature in the theaters.

Dr J. Wistbacka (Vaasa, Finland): Doesn’t this study and the data from the literature point out that we should use rewarming water temperature at the maximum of 37° all the time? That will leave our patients a little bit hypothermic, but I think that’s the price we have to pay in order to avoid the risks of neurologic sequelae.

Dr Kaukuntla: I entirely agree with you. Because we do not have a definite relationship between the temperature monitoring sites and the core brain temperature, we cannot say it is safe to heat up to this nasopharyngeal temperature or this bladder temperature or this arterial inflow. The safest way is, as you rightly said is to never allow any of the temperatures to go beyond 37, so the likelihood of the brain going much higher than 37.5 is rather negligible.

Dr A. Hassouna (Cairo, Egypt): I wanted to know when you have made your correlation between the arterial flow and jugular venous bulb temperatures?

In the graph, we see they cross each other at some points. Did you make a correlation over time or did you make a correlation at a certain time after bypass?

Dr Kaukuntla: No, the correlation was done at the point when the jugular bulb temperature reached its peak for each patient.

Dr Hassouna: Just peak correlation?

Dr Kaukuntla: Yes, peak correlation.