In Vitro Validation of the Affinity NT Oxygenator Arterial Outlet Temperatures

Kieron C. Potger, BSc, CCP; Darryl McMillan, CCP

Department of Anaesthesia and Pain Management, Perfusion and Autotransfusion Unit, Royal North Shore Hospital, Sydney, Australia

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Abstract: During cardiopulmonary bypass, the rates of cooling and rewarming and the maximum temperatures attained are implicated in patient morbidity. Thus, accurate oxygenator arterial outlet temperature measurements are needed. The purpose of this study was to determine the accuracy of the arterial outlet temperature probe on the “Affinity NT” membrane oxygenator in measuring perfusate temperatures. An in vitro circuit was used. Crystalloid solution was recirculated through an Affinity NT membrane oxygenator and, to simulate the patient, a second oxygenator. Water was recirculated through the heat exchanger of the second oxygenator via a reservoir. A myocardial temperature probe was inserted in-line 4 cm distal to the Affinity NT oxygenator arterial outlet temperature probe and was considered to measure the actual temperature of the perfusate. Temperatures were simultaneously recorded from the in-line probe, arterial outlet probe, and reservoir every second. Twenty-seven trials were run using random combinations of three Affinity NT oxygenators and three in-line probes. Each trial entailed cooling an initially normothermic reservoir to 28°C and then rewarming it to normothermia again. The arterial outlet temperature probe on the Affinity NT membrane oxygenator underestimated the perfusate temperatures during early rewarming (bias of 0.72°C; precision of ±1.15°C) and late rewarming (bias of 0.52°C; precision of ±0.97°C). An overestimation of the perfusate temperatures occurred during early cooling (bias of −0.57°C; precision of ±1.37°C). Only during the late cooling phase was the arterial outlet temperature probe accurate (bias of −0.02°C; precision of ±0.3°C). The perfusionist should be aware of the temperature probe monitoring characteristics of the oxygenator to safely perfuse the patient.

Keywords: cardiopulmonary bypass, oxygenator arterial outlet temperature, temperature probe.

Temperature control of patients during cardiopulmonary bypass (CPB) entails cooling or rewarming of the patient’s blood by the oxygenator’s heat exchanger. However, the rates of cooling and rewarming and the maximum temperatures attained are implicated in patient morbidity (1). The brain, with its large blood supply, rapidly equilibrates with the temperature of the perfused blood. Perfusion temperature, as measured in the arterial outlet of the oxygenator, may be a useful and practical indicator of the brain temperature during CPB. Thus, accurate oxygenator arterial outlet temperature measurements are needed.

Previous studies have shown that the standard membrane oxygenator arterial outlet temperature probe may underestimate the temperature of the perfusate (2,3). However, the accuracy of the arterial outlet temperature probe on the Affinity NT membrane oxygenator (Medtronic, Australasia) has not yet been validated.

To determine the accuracy of the arterial outlet temperature probe on the Affinity NT membrane oxygenator, an in vitro circuit was used. Comparisons were made between the temperatures from the arterial temperature probe of this oxygenator with a needle temperature probe inserted within the perfusate.

MATERIALS AND METHODS

A dual in vitro circuit was used that comprised a test extracorporeal circuit and another circuit simulating the patient. The circuit was primed with 840 mL of Hartman’s solution, which was recirculated at 4 L/min through an Affinity NT membrane oxygenator and a second Affinity
NT oxygenator (Medtronic, Australasia). To simulate the heat inertia implicit in a patient, 10 L of water was recirculated at 4 L/min through the heat exchanger of the second oxygenator via a reservoir. A heater-cooler (Jostra HCU-20) was used to adjust the prime temperature and consequently the reservoir temperature. Shown in Figure 1 is a diagram of the circuit. The tubing and oxygenators were recently expired (greater than 48 hours since setup) off-pump standby circuits.

A myocardial temperature probe (18-mm needle, thermister YSI 400, Mallinckrodt) was inserted in-line 4 cm distal to the Affinity NT oxygenator arterial outlet temperature probe site and was considered to measure the actual temperature of the perfusate. Temperatures were simultaneously recorded from the in-line probe, arterial outlet probe (standard Affinity NT oxygenator thermister YSI 400), and reservoir. These temperatures were measured by an anesthetic monitor (Datex AS/3), with the signals being downloaded into a personal computer. LabView software (National Instruments Corporation, Austin, TX) recorded the data every second. The calibration of all temperature probes was verified once against a mercury thermometer (within ± 0.2°C).

A trial commenced by initially establishing “normothermia” (reservoir 37 ± 0.5°C), and then cooling began by setting the heater-cooler unit to 20°C. Once the reservoir had reached 28°C, rewarming commenced by setting the heater-cooler unit to 41°C. A trial was completed when the reservoir reached 36.9°C.

To minimize any sequencing effects or the possibility of using unrepresentative examples of temperature probes or oxygenators, 27 trials were run using random combinations of three Affinity NT oxygenators and three in-line probes. Each trial lasted approximately 20 minutes.

The data were analyzed by identifying early and late cooling and warming phases. Early cooling was defined as the period from initial cooling until the temperature of the reservoir decreased to 34°C, decreasing the temperature further until the reservoir was at least 28°C was classified as late cooling. Similarly, early warming was defined as the period from initial rewarming until the temperature of the reservoir reached 31°C with the subsequent heating until the reservoir was 36.9°C being classified as the late warming phase.

Unpaired t tests were performed to determine the statistical significance between the temperatures measured by the in-line probe and the arterial outlet probe during each of the cooling and rewarming phases. A p value of less than 0.05 was considered significant. To assess how these data can be interpreted clinically, the agreement between the two variables was analyzed using Bland and Altman’s method (4). Here, the relationship between the two variables is presented in terms of accuracy or bias (mean difference between the two variables) and precision or 95% limits of agreement (1.96 times the standard deviation of the difference between the two variables). Bland and Altman graphs plot the differences between the two probes’ temperatures against the averages of the two measurements. Data are summarized as the mean ± one standard deviation (SD).

RESULTS

More than 36,000 seconds of data were collected. Figure 2 shows a typical temperature profile taken from trial 12. There was a lag in temperatures attained by the reservoir compared with the arterial outlet and in-line sites. The in-line probe was statistically cooler than the arterial outlet probe during early cooling, with no difference occurring during the late cooling phase. Conversely, the in-line temperature becomes significantly warmer than that of the arterial outlet probe during both the early and later phases of rewarming (Table 1).

During early cooling, the arterial outlet probe over-read the perfusate temperature by an average of 0.57°C,
Cerebral hyperthermia is implicated in cerebral dysfunction. Elevating the brain by 1 to 2°C significantly worsened postischemia neurological outcome in dogs (7), whereas studies of focal ischemia in pigs showed a direct relationship between high intracerebral temperatures and poor outcomes (8). Relatively high perfusate temperatures are used to promote rewarming of the patient. However, this occurs at the same time as the brain is recovering from multifocal ischemia secondary to microemboli or hypoperfusion, thereby contributing to post CPB neurological dysfunction (1). Likewise, exposing the brain to hyperthermic blood during rapid rewarming was shown in a randomized clinical study to diminish the neuroprotective effect of hypothermia during CPB (9). A strict adherence to normothermic CPB also may cause cerebral overheating and subsequent neurological morbidity as the brain becomes bathed in hyperthermic perfusate (10).

The rate of rewarming after hypothermic CPB also is implicated in patient morbidity. Faster rewarming rates are associated with jugular venous hemoglobin desaturation indicating an imbalance between cerebral oxygen supply and demand (11,12). A relationship between the rewarming associated jugular venous desaturation and the severity of postoperative neuropsychological dysfunction was established by Croughwell and colleagues (13). Additionally, a recent prospective study examining the consequences of the rewarming rate during hypothermic CPB showed that slower rewarming rates were associated with an improved neurocognitive outcome (14). Clinically, the rate of rewarming may have greater significance in patients with impaired cerebral autoregulation such as the elderly, diabetic and those with pre-existing cerebrovascular disease.

The nasopharangeal temperature is notorious for underestimating cerebral temperatures on rewarming with a potential for cerebral hyperthermia (15–17). Because the brain is unlikely to be hotter than the perfusate temperature when warming or colder than the perfusate when cooling, the perfusate temperature as measured in the arterial outlet of the oxygenator may be a useful and practical measure of the brain temperature during CPB. With a typical adult pump flow of 4 L/min or more, and with one fifth of this blood perfusing the brain with negligible cooling of the blood between the oxygenator and the aorta, the brain should rapidly equilibrate with the perfusate temperature as measured in the outlet of the oxygenator (17).

The bias or difference observed between the arterial outlet and in-line probes may be explained by the lower mass and complete immersion of the myocardial needle probe within the perfusate. The proprietary temperature probe is larger, more complex in structure, and has only its tip inserted into the prime with the rest of its structure exposed to air. During periods when the arterial blood

Table 1. Mean temperatures between arterial outlet and in-line probes during different phases of cooling and warming °C (±1 SD).

<table>
<thead>
<tr>
<th>Phase</th>
<th>Arterial Outlet</th>
<th>In-Line</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early cooling</td>
<td>29.7 (3.1)</td>
<td>29.2 (2.9)*</td>
</tr>
<tr>
<td>Late cooling</td>
<td>25.4 (1.4)</td>
<td>25.4 (1.4)</td>
</tr>
<tr>
<td>Early warming</td>
<td>31.8 (3.6)</td>
<td>32.5 (3.6)*</td>
</tr>
<tr>
<td>Late warming</td>
<td>37.4 (1.0)</td>
<td>37.9 (0.9)*</td>
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*p < 0.0001.

whereas the precision revealed that 95% of samples were as much as 1.37°C greater or less than this bias (Table 2, Figure 3). The agreement between the two probes improved during late cooling, with hardly any bias observed and very tight precision; clinically the arterial outlet probe was accurate during this phase. However, there was a reduction in the agreement upon early cooling, with the arterial outlet probe underestimating the perfusate temperature by an average of 0.72°C (with an upper limit of agreement that was 1.15°C greater than this bias). During late warming, the accuracy and precision slightly improved, but clinically, the perfusate temperature could still be as much as 1.5°C greater than that measured by the arterial outlet probe (Table 2, Figures 4 to 6).

**DISCUSSION**

This in vitro study determined that the arterial outlet temperature probe on the Affinity NT membrane oxygenator can underestimate the perfusate temperatures by as much as nearly two degrees on rewarming and can overestimate the perfusate temperatures by as much as nearly two degrees on early cooling. Only during stable hypothermia is the arterial outlet probe accurate.

Overheating of the blood by the extracorporeal heat exchanger can cause blood trauma, resulting in hemolysis and plasma protein denaturation (5). Excessively high temperature gradients might precipitate microbubble generation as gas solubility decreases (6). Furthermore, the brain, with its high blood flow, warms rapidly and could be hyperthermic for a significant period during rewarming on CPB.

Table 2. Accuracy and precision between arterial outlet and in-line probes during different phases of cooling and warming.

<table>
<thead>
<tr>
<th>Phase</th>
<th>Accuracy*</th>
<th>Precision†</th>
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<tbody>
<tr>
<td>Early cooling</td>
<td>−0.57</td>
<td>±1.37</td>
</tr>
<tr>
<td>Late cooling</td>
<td>−0.02</td>
<td>±0.3</td>
</tr>
<tr>
<td>Early warming</td>
<td>0.72</td>
<td>±1.15</td>
</tr>
<tr>
<td>Late warming</td>
<td>0.52</td>
<td>±0.97</td>
</tr>
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*Bias: mean difference between arterial outlet and in-line probe (°C).
†95% limits of agreement: 1.96 times the standard deviation of the difference between arterial outlet and in-line probes.
temperature approximates the venous blood, eg, during stable hypothermia or normothermia, sufficient time may occur for the equilibration of the arterial outlet probe with the perfusate temperature resulting in an improvement in the arterial outlet probe’s accuracy.

When examining the various trials, it became apparent that not all the temperature probes were performing the same as a bimodal distribution of biases was observed; one of the three proprietary arterial outlet probes performed more aberrantly. The difference in performance observed among the arterial outlet temperature probes may be the result of a variability of individual probe design. The arterial outlet probe is an elaborate device: the thermistor wires are embedded in Silastic then surrounded by a plastic coating before being encased in stainless steel. This probe is then inserted into another stainless-steel sheath.
before finally coming into contact with the blood. It may be prudent to test and discard probes yielding too high a discrepancy before using them clinically. Caution must be used when applying these findings to clinical practice. Crystalloid, with its higher specific heat capacity than blood, was used as the perfusate in this in vitro study. Also, the relatively aggressive cooling and rewarming regimen that was used to simulate a worse case scenario may not be indicative of that seen clinically. Furthermore, although it may be anticipated that other brands of oxygenators using a similar temperature probe design would follow the trends shown in this study, it would be advisable to confirm this.

In summary, the Affinity NT oxygenator arterial outlet temperature probe may overestimate the arterial perfusate temperature during early active cooling and underes-

Figure 5. Agreement between in-line and arterial outlet temperature probes during the early warming phase.

Figure 6. Agreement between in-line and arterial outlet temperature probes during the late warming phase.
timate the arterial perfusate temperature during all phases of active warming. Also, the proprietary temperature probes may vary in their measurement performance. The perfusionist should be aware of the temperature probe monitoring characteristics of the oxygenator being used to safely perfuse the patient.

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REFERENCES