Does Urinary Catheter Temperature Reflect Core Temperature during Cardiac Surgery?

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Values obtained from measurement of body temperature at different sites usually differ from one another. Nasopharyngeal, tympanic membrane, and pulmonary arterial temperatures are generally considered reflective of body “core” temperature. Rectal temperature is not a core temperature.1,2 It lies in an area intermediate between the body core and periphery.1 Urinary catheter temperature (UCT) monitoring is being employed with increasing frequency to measure body temperature.3-5 UCT is equivalent to pulmonary arterial blood temperature.5 Yet, the UCT probe is physically placed in the intermediate zone, not the core zone. UCT may reflect core temperature because urine is a filtrate of core blood.5

Rapid core warming during cardiopulmonary bypass (CPB) would represent a demanding test of the ability of UCT to reflect core temperatures. Large thermal gradients from core to periphery occur while warming during CPB. During such thermal disequilibrium, the more central temperatures measured in the esophagus and pulmonary artery respond more rapidly than the intermediate zone rectal temperature.2

We tested the hypothesis that urinary catheter temperature reflects core temperature during the rapid warming phase of CPB. We discovered that the concordance of UCT to core temperatures depends on urine flow rate.

MATERIALS AND METHODS

With institutional approval, informed consent was obtained from 24 adults undergoing cardiac surgery with CPB. After induction of anesthesia utilizing moderate-to-high-dose opioids (50-150 μg/kg fentanyl or 5-15 μg/kg of sufentanil iv) and non-depolarizing neuromuscular blockade, disposable thermocouple temperature probes (Mon-a-therm Inc., St. Louis, MO) were placed in the distal esophagus, rectum, and urinary bladder. The rectal probe was inserted a minimum of 8 cm and taped in place. The esophageal probe was incorporated into an esophageal stethoscope. Depth of esophageal probe placement was determined by maximum intensity of cardiac sounds by auscultation.6 The urinary catheter thermocouple lies 13 mm proximal to the tip of a 16-Fr silicone Foley catheter. Pulmonary arterial temperature was measured with a thermistor-tipped catheter (American Edwards #93A-131-7F). Esophageal, rectal, urinary catheter, and operating room temperatures were measured with identical thermocouple devices and monitors (Mon-A-Therm model 6500) that were self-calibrated before each use. Day-to-day drift with electronic calibration was less than 0.1°C. Thermocouple accuracy was better than 0.02°C. The cardiac output computer (American Edwards Model 9290) displayed pulmonary arterial thermistor temperature. All temperatures were recorded in Celsius using three significant figures.
A urometer measured urine output. The tubing connecting the Foley catheter to the urometer followed a continuously downhill path, thus preventing pooling of urine in the collecting tube. Beginning with the start of warming on CPB, temperatures and interval urine output were recorded every 10 min for 2 h or until the termination of surgery, whichever occurred first.

CPB employed a Bentley-10 oxygenator and Sarns heater-cooler. Patients were cooled on CPB to esophageal temperatures between 25 and 28° C. A warming blanket (Blanketrol®) under each patient was set to 40° C at the beginning of warming. Warmed blood entered each patient via an aortic cannula. Cold cardioplegia administration occurred prior to the start of warming. A blood warmer was employed for all fluids administered after CPB. For each measurement period, the mean temperatures measured in the pulmonary artery, esophagus, rectum, and urinary catheter were compared to one another using the serial Newman-Kuels test.

Analysis of urine flow data commenced with a histogram of urine flow rates (fig. 1). These data were then retrospectively separated into a lower flow group (<45 ml per 10 min) and a higher flow group (at least 45 ml per 10 min). The temperature data were likewise segregated into lower and higher flow groups according to the urine flow in the 10-min period preceding temperature recording. We calculated for each patient at each measurement time the paired difference between esophageal temperature and UCT. The paired differences for rectal temperature and UCT and for pulmonary artery temperature and UCT were also calculated. Analysis of variance (ANOVA) tested for an effect of urine flow (higher or lower) on temperature difference. This ANOVA was performed for each of the three paired differences (esophageal and UCT, rectal and UCT, pulmonary arterial and UCT). Data during warming and after CPB were analyzed separately. Significant effects were declared at the P < 0.05 level.

RESULTS

Twenty men and four women participated in the study. Age was 60 ± 10 (SD) yr, height 68 ± 4 inches, weight 82 ± 16 kg, and body surface area 1.9 ± 0.24 m². At the start of warming, operating room temperature was 25.0 ± 1.9° C and esophageal temperature was 26.3 ± 1.5° C. Warming lasted for 37 ± 10 min. Figure 2 shows the mean ± SEM temperatures for all patients. At 10, 20, 30, and 40 min after the start of warming, esophageal and pulmonary arterial temperatures were not statistically different, nor were UCT and rectal temperature. But pulmonary arterial or esophageal temperatures were higher than rectal or urinary catheter temperatures. Following CPB, a period of relatively greater thermostability, temperatures from all four sites were not different statistically.

During warming, urine flow rate was 26 ± 21 (range 0–80) ml per 10 min. Of the 82 observations during warming, the high urine flow rate group contained 14 observations; urine flow rate was 64 ± 11 ml in this group. Urine flow rate in the low flow group (N = 68) was 19 ± 12 ml. During warming, urine flow rate category (low or high group) affected the pulmonary arterial-UCT difference (P < .001) and the esophageal-UCT difference (P < .0001), but not the rectal-UCT difference (table 1). With lower urine flow, UCT was about 5° C cooler than esophageal or pulmonary arterial temperature, whereas with higher urine flow, UCT was only about 2° C cooler.

After CPB, urine flow rate was 52 ± 44 (range 0–200) ml per 10 min. Urine flow rate category affected the pulmonary arterial-UCT difference and the esophageal-UCT difference, but not the rectal-UCT difference.
TABLE 1. Mean ± SD Temperature Differences (°C) during Warming and After CPB

<table>
<thead>
<tr>
<th></th>
<th>Higher Flow</th>
<th>Lower Flow</th>
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<tbody>
<tr>
<td>Warming</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ET-UCT</td>
<td>2.1 ± 1.3 (14)</td>
<td>4.8 ± 1.9 (68)*</td>
</tr>
<tr>
<td>RT-UCT</td>
<td>0.0 ± 1.1 (14)</td>
<td>0.8 ± 1.9 (66)</td>
</tr>
<tr>
<td>PAT-UCT</td>
<td>2.2 ± 1.9 (13)</td>
<td>4.7 ± 2.4 (66)*</td>
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<tr>
<td>After CPB</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ET-UCT</td>
<td>0.0 ± 0.6 (71)</td>
<td>0.3 ± 1.0 (122)*</td>
</tr>
<tr>
<td>RT-UCT</td>
<td>0.3 ± 0.4 (71)</td>
<td>0.3 ± 0.5 (122)</td>
</tr>
<tr>
<td>PAT-UCT</td>
<td>0.0 ± 0.5 (71)</td>
<td>0.3 ± 0.9 (122)*</td>
</tr>
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</table>

CPB = cardiopulmonary bypass; ET = esophageal temperature; UCT = urinary catheter temperature; RT = rectal temperature; PAT = pulmonary arterial temperature. Numbers in parentheses indicate the number of observations.

* F < 0.05 higher vs. lower flow rate group by ANOVA and Scheffe’s F test.

after CPB. The mean core-UCT temperature differences were less than 0.1° C at higher urine flows. At lower urine flows, these temperature differences (0.3° C) were statistically greater than those at higher flows.

DISCUSSION

During the marked thermal disequilibrium with cooling and warming on CPB, intermediate zone (rectum) and peripheral (skin or muscle) temperatures lag behind those measured from the central compartment (esophagus, pulmonary artery, nasopharynx, or tympanic membrane). Our data show that during warming on CPB, UCT is not different from rectal temperature (fig. 2). UCT is not a “core” temperature. In contrast, Lilly et al. found that the rate of increase of UCT was “very close” to the rate of increase of pulmonary artery temperature during warming on CPB. Of note is that the esophageal temperatures measured by Lilly et al. were cooler than the urinary catheter temperatures.

Comparisons of the current report with that of Lilly et al. are obscured by several factors. First, Lilly et al. report neither the number of patients studied during CPB nor any measure of the variance of their observations. They provide no information regarding what, if any, statistical tests were used to interpret the data. Second, their patients were cooled only to 30° C, thus establishing a smaller thermal gradient from core to periphery than in our patients. Third, the position of their esophageal probe was unspecified. If placed too proximally in the esophagus, the esophageal probe would read several degrees C cooler than if placed more distally. Distal esophageal temperature is considered more reflective of central compartment temperature. Placement of the probe in the proximal or middle esophagus may explain why the mean esophageal temperatures reported by Lilly et al. during warming on CPB are 1–2° C cooler than their mean pulmonary arterial temperatures. Finally, because our data indicate that high urinary flow rates can increase UCT, it is possible that high urinary flow rates increased the UCT values obtained by Lilly et al.; urinary flow rate was not quantified in their study.

Moorthy et al. report in a study of 12 patients during warming on CPB that esophageal and nasopharyngeal temperatures were not different, but rectal and urinary catheter temperatures were significantly cooler than nasopharyngeal temperature. Mean UCT values were between those of the rectum and esophagus. The study by Moorthy et al. agrees more closely with ours both in methods and results: their patients were cooled to 27° C esophageal, ours to 26.3° C esophageal; the probes in each investigation were placed in the distal esophagus. Moorthy et al.’s determination that UCT falls between esophageal and rectal temperatures differs from our conclusion that UCT and rectal temperature do not differ. Several explanations are possible. First, variances may be smaller in that study, reflecting better control of unidentified variables. Second, urinary flow rate may have been larger than in our study, raising UCT closer to pulmonary arterial temperature. Third, this statistical difference may be a reflection of the different statistical methods employed (Dunnett’s test versus the Newman-Kuels test).

UCT should reflect the temperature of the urinary bladder, because urine is drained from the bladder by the catheter. Since the bladder wall is in close proximity to the rectum, UCT and rectal temperature should be similar. This is the case when urine flow rate is low or moderate. Modest rates of urine production provide sufficient time for the urine to exchange heat with tissues surrounding the ureters and bladder.

High urine flow rates might transfer calories to the bladder from the core via blood filtration. Thus, UCT would better reflect the “core” temperatures measured in the esophagus or pulmonary artery during high rates of urine flow. Our data support this theory. With lower urine flows, UCT was 4.7° C cooler than pulmonary arterial temperature, compared with 2.2° C cooler at higher urine flows. After CPB, differences of UCT from other temperatures, although statistically significant, were small enough (0.3° C or less) to be of little clinical significance.

This study employed an esophageal site to measure central compartment temperature, rather than a nasopharyngeal site. Nasopharyngeal temperature is considered more reflective of brain temperature than esophageal temperature, because the latter can be affected by the temperature of inspired gases. However, esophageal temperature is more suitable than nasopharyngeal temperature for this study, because manipulation of the nasal mucosa is undesirable in patients to be totally anticoagulated. Furthermore, retrocardiac esophageal probes are not affected by airway temperature.
Transcatheter Thoracic Epidural Neurolysis Using Ethyl Alcohol

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Sporadic reports of pain relief following the injection of ethyl alcohol into the epidural space have appeared in the literature since 1930. Early reports described the injection of large volumes of local anesthetic and alcohol into the caudal epidural space for relief of intractable pelvic and perineal pain due to rectal or prostatic cancers.1–5

Despite mention in scattered case reports, no study of the efficacy and safety of epidural neurolysis using ethyl alcohol has been reported.6–7 Of concern are the inaccuracies of the technique, the possibility of injection pain, the chance that “alcohol neuritis” might develop, and the danger that accidental unrecognized dural puncture might lead to disastrous consequences in the face of the relatively large volume of alcohol that is used. After an initial success using transcatheter thoracic epidural neurolysis with ethyl alcohol to treat a patient with pancreatic cancer pain who did not respond to celiac plexus block, this study was undertaken to document the safety and efficacy of this technique in the management of intractable pain of malignant and benign origins.

MATERIALS AND METHODS

Following approval of the institutional review board, informed consent was obtained from 36 consecutive in-