# Influence of Thermoregulatory Vasomotion and Ambient Temperature Variation on the Accuracy of Core-temperature Estimates by Cutaneous Liquid-crystal Thermometers

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*Background:* Recently, liquid crystal skin-surface thermometers have become popular for intraoperative temperature monitoring. Three situations during which cutaneous liquid-crystal thermometry may poorly estimate core temperature were monitored: (1) anesthetic induction with consequent core-to-peripheral redistribution of body heat, (2) thermoregulatory vasomotion associated with sweating (precapillary dilation) and shivering (minimal capillary flow), and (3) ambient temperature variation over the clinical range from 18–26°C.

*Methods:* The core-to-forehead and core-to-neck temperature difference was measured using liquid-crystal thermometers having an ≈2°C offset. Differences exceeding 0.5°C (a 1°C temperature range) were *a priori* deemed potentially clinically important. Seven volunteers participated in each protocol. First, core-to-peripheral redistribution of body heat was

produced by inducing propofol/desflurane anesthesia; anesthesia was then maintained for 1 h with desflurane. Second, vasodilation was produced by warming unanesthetized volunteers sufficiently to produce sweating; intense vasoconstriction was similarly produced by cooling the volunteers sufficiently to produce shivering. Third, a canopy was positioned to enclose the head, neck, and upper chest of unanesthetized volunteers. Air within the canopy was randomly set to 18, 20, 22, 24, and 26°C.

*Results:* Redistribution of body heat accompanying induction of anesthesia had little effect on the core-to-forehead skin temperature difference. However, the core-to-neck skin temperature gradient decreased ≈0.6°C in the hour after induction of anesthesia. Vasomotion associated with shivering and mild sweating altered the core-to-skin temperature difference only a few tenths of a degree centigrade. The absolute value of the core-to-forehead temperature difference exceeded 0.5°C during ≈35% of the measurements, but the difference rarely exceeded 1°C. The core-to-neck temperature difference typically exceeded 0.5°C and frequently exceeded 1°C. Each 1°C increase in ambient temperature decreased the core-to-forehead and core-to-neck skin temperature differences by less than 0.2°C.

*Conclusions:* Forehead skin temperatures were better than neck skin temperature at estimating core temperature. Core-to-neck temperature differences frequently exceeded 1°C (a 2°C range), whereas two thirds of the core-to-forehead differences were within 0.5°C. The core-to-skin temperature differences were, however, only slightly altered by inducing anesthesia, vasomotor action, and typical intraoperative changes in ambient temperature. (Key words: Anesthesia. Hypothermia. Temperature: core; skin; tympanic membrane. Thermometer: liquid-crystal; thermocouple. Thermoregulation: vasoconstriction.)

PHYSIOLOGICALLY, core temperatures are more important than skin temperatures because they provide ≈80% of the thermal input to the hypothalamic regulating system.[1,2] Furthermore, adverse effects of thermal perturbations correlate with small differences in core temperatures.[3-6] Consequently, clinicians usually find core temperature to be the most useful single measure of body temperature.

The standard core-temperature monitoring sites — distal esophagus, tympanic membrane, nasopharynx, and pulmonary artery — are accurate to $\approx 0.2°C$ and precise to $\approx 0.1°C$.[7-9] (Intersite variability is probably greater in children.[10]) The mouth, axilla, bladder, and rectum are considered intermediate sites. These sites reflect core temperature poorly during cardiopulmonary bypass, and often under other circumstances.[11-16] Nonetheless, their accuracy is typically near 0.5°C, and their precision is approximately 0.2°C.[7] Minimum acceptable accuracy and precision for anesthesia thermometers have yet to be established. However, an accuracy near 0.5°C (a 1°C range) seems reasonable because differences of 1°C may have distinct physiologic consequences.[17]

Recently, liquid crystal skin-surface thermometers have become popular for intraoperative temperature monitoring. The correlation between skin and core temperatures has been reported to be poor.[18-22] (In at least one case, data indicate the correlation to be poor, although the authors concluded otherwise.[23]) Others, however, report the method to be reliable.[24,25] A further difficulty with many of these studies is that they were based on simple linear correlation, a frequently inadequate technique.[26]

At least three mechanisms potentially introduce artifact into skin temperature estimates of core temperature. The first is internal redistribution of body heat. Core-to-peripheral redistribution contributes $\approx 80\%$ to the reduction in core temperature during the first hour of general anesthesia and remains the most important cause of hypothermia, even after 3 h of anesthesia.[27] Because core hypothermia results from peripheral tissue warming, redistribution is accompanied by a constant or slightly increased mean skin temperature.[28] Even if skin temperature simply remained constant during redistribution, the typical 1–1.5°C decrease in core temperature would produce a clinically unacceptable artifact.

A second mechanism potentially confounding cutaneous estimates of core temperature is altered vascular tone. Thermoregulatory changes in vasomotion are common in the perioperative period and can alter skin blood flow enormously. Sufficient core hypothermia triggers intraoperative cutaneous vasoconstriction. The threshold for vasoconstriction (triggering core temperature) is typically 33–35°C[29] but depends on the anesthetic type and dose and ranges from 31–36°C.[30-32] Thermoregulatory vasoconstriction in distal arteriovenous shunts decreases more than ten times.[33] Capillary flow, over the rest of the skin surface, decreases only 25–

50%, but even this amount can reduce skin temperature significantly.[34] Active vasodilation, in contrast, increases capillary flow enormously. Cutaneous vasodilation is mediated by a yet-to-be-identified mediated released by sweat glands[35] and is estimated to reach 7.5 l/min flow to the top millimeter of skin during intense sweating.[36]

Changes in ambient temperature are a third potential source of artifact. (Ambient temperature frequently increases 1°C or more during an operation because air-conditioning systems cannot fully compensate for heat generated by surgical personnel and instruments.) At steady state, mean skin temperature is $\approx 33°C$ when core temperature is 37°C and ambient temperature is 20°C. This suggests that ambient temperature contributes approximately 23% ([37 − 33°C]/[37 − 20°C]) to skin temperature, with the rest coming from core. Sufficient change in ambient temperature might thus produce potentially important alterations in skin-surface temperature.

Thermoregulatory theory thus suggests that several factors may confound estimates of core temperature obtained by liquid-crystal thermography. Accordingly, we evaluated three situations during which cutaneous liquid-crystal thermometry may fail to accurately estimate core temperature: (1) anesthetic induction with consequent core-to-peripheral redistribution of body heat, (2) thermoregulatory vasomotion associated with sweating (complete arteriovenous shunt dilation and some precapillary dilation) and shivering (minimal capillary flow), and (3) ambient temperature variation over the normal clinical range from 18–26°C.

We measured skin temperature at the forehead because it is the most common and convenient cutaneous monitoring site. Furthermore, forehead subcutaneous tissue insulation is usually minimal (and relatively similar among individuals). Finally, the forehead is devoid of thermoregulatory arteriovenous shunts and countercurrent mechanisms that could dramatically alter skin temperature without comparable central temperature changes.[37] Skin over the carotid artery has also been advocated as a cutaneous temperature-monitoring site, on the theory that it will be especially well warmed by the adjacent flow of blood coming directly from the heart. Accordingly, we also recorded neck skin temperature.

## Methods

With approval from the Committee on Human Research at the University of California, San Francisco, and

written consent, we studied 21 volunteers. None was obese, was taking medication, or had a history of thyroid disease, dysautonomia, Raynaud syndrome, or malignant hyperthermia.

Differences between core and skin temperatures defined the accuracy of liquid-crystal thermography under each study circumstance. Changes in the core-to-forehead and core-to-neck differences exceeding 0.5°C were *a priori* deemed potentially clinically important. Smaller changes were considered acceptable because similar variation is typical in other commonly used temperature monitoring sites (*i.e.*, the axilla or mouth) and because reduced resistance to cerebral ischemia is the only perioperative temperature-related complication associated with changes over just a 1°C range (*i.e.*, +0.5 to −0.5°C).[17,38]

We also evaluated the fraction of measurements during which the core-to-forehead and core-to-neck differences exceeded 1°C. This level of accuracy is unlikely to be sufficient for most clinical purposes because core-temperature differences of 2°C (*i.e.*, +1 to −1°C) are associated with many complications.[3-6] All results are reported as means ± SD, and $P < 0.05$ was considered significant.

### Measurements

Core temperature was recorded from the tympanic membrane using Mon-a-Therm thermocouples (Mallinckrodt Anesthesiology Products, St. Louis, MO). The aural probes were inserted by volunteers until they felt the thermocouple touch the tympanic membrane; appropriate placement was confirmed when volunteers easily detected gentle rubbing of the attached wire. The aural canal was then occluded with cotton, the probe was taped securely in place, and a gauze bandage positioned over the external ear.

Forehead and neck skin-surface temperatures were recorded using Mon-a-Therm self-sticking thermocouple probes. A small disk of foam is incorporated into these probes. One skin-surface probe was positioned in the center of the forehead; another was positioned over the carotid artery, as determined by palpation. Ambient temperature was measured from a thermocouple probe positioned between 10 and 30 cm above the forehead. The thermocouples were connected to a calibrated Iso-Thermex 16-channel electronic thermometer having an accuracy of 0.1°C and a precision of 0.01°C (Columbus Instruments International, Columbus, OH).

Forehead and neck skin temperatures were also mea-

sured using Sharn TempAlert Temperature Trend Indicator liquid-crystal thermometers (Tampa, FL). After degreasing the skin with alcohol, self-sticking liquid-crystal strips were positioned adjacent to the thermocouples. Temperatures from the liquid crystal thermometers were evaluated by an investigator blinded to core and ambient temperatures. These thermometers include a 3.5°F (1.95°C) offset correction designed to compensate for skin temperature being less than core temperature. That is, the temperature displayed on the liquid crystal thermometer exceeds actual skin temperature by 1.95°C. This corrected temperature was used for all analyses in this study.

The percentage of body fat was determined using infrared interactance.[39] Air speed near the forehead was evaluated using a calibrated heated-wire anemometer (model FMA-902-V-S; Omega Instruments, Stamford, CT). Forehead capillary flow was estimated using laser Doppler flowmetry with an integrating multiprobe (Periflux 3; Perimed Inc., Piscataway, NJ; on the wide-band setting).[40] This index changes linearly as a function of capillary blood flow. Right index fingertip blood flow was estimated using forearm minus fingertip skin-surface temperature gradients[41] and a pulse-oximeter-based perfusion index. A gradient less than 0°C indicated arteriovenous shunt vasodilation, whereas a gradient ≥4°C identified significant vasoconstriction.

Sweating was measured on the left upper chest using a ventilated capsule as previously described.[42] A sustained sweating rate exceeding $40 \text{ g} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$ was considered significant.[30] Shivering was quantified using electromyographic analysis, as previously described.[43] Sustained, synchronous waxing-and-waning activity identified significant shivering.[44]

### Induction of Anesthesia

We evaluated seven volunteers during induction of general anesthesia. Studies started at approximately 8:30 A.M. and volunteers fasted during the 8 preceding hours. Throughout the study, minimally clothed volunteers reclined on an operating room table set in the chaise lounge position. An intravenous cannula was inserted in the left antecubital fossa, and lactated Ringer's solution warmed to 37°C was infused at a rate of 100 ml/h.

No premedication was given. Volunteers were minimally clothed and maintained in an ambient environment near 22°C during the study. At least 1 h after arteriovenous shunt vasoconstriction was documented, anesthesia was induced by intravenous administration

of propofol (3–5 mg/kg), midazolam (2 mg), and vecuronium bromide (0.1 mg/kg). The volunteers' tracheas were intubated and mechanical ventilation adjusted to maintain end-tidal carbon dioxide pressure near 35 mmHg. Anesthesia was maintained with desflurane (3–5%) in oxygen and air. An infusion of vecuronium was adjusted to maintain one mechanical twitch in response to supramaximal train-of-four electrical stimulation of the ulnar nerve at the wrist. No active warming or cooling was provided during the first hour of anesthesia. Subsequently, the volunteers participated in a separate thermoregulatory protocol.[45]

Arteriovenous shunt and capillary flows were recorded at 10-min intervals for 1 h before anesthesia was induced and for the subsequent hour. Core, skin-surface, and ambient temperatures were similarly recorded. Blood flows and ambient temperatures in each volunteer were averaged over the 1-h control period before anesthesia was induced, and during the first hour of anesthesia. Individual values were then averaged among the volunteers. The effect of anesthetic induction on the core-to-forehead and core-to-neck temperature difference was evaluated using repeated-measures analysis of variance; values were compared with those at elapsed time zero (just before anesthesia was induced) using Dunnett's test.

### Thermoregulatory Vasomotion

Seven unanesthetized volunteers were maintained in an ambient environment at 21.4 ± 0.7°C. In random order, vasoconstriction and vasodilation were induced by surface cooling and warming. Both thermal manipulations were restricted to the legs, leaving the entire upper body exposed to ambient temperature. Vasoconstriction was induced by cooling the volunteers to shivering, and vasodilation was produced by warming them to sweating; each thermal state was maintained for 30 min.

After 10 min of equilibration at each thermoregulatory state, flows and core and skin temperatures were recorded at 5-min intervals for 20 min. All values were averaged first within participants and then among them. Changes in arteriovenous shunt and capillary flow and core-to-skin differences induced by surface warming and cooling were evaluated using repeated-measure analysis of variance and Scheffé's F tests.

### Ambient Temperature

Seven unanesthetized volunteers participated in this portion of the study. Their legs were cooled sufficiently

**Table 1. Morphometric Characteristics of the Volunteers Participating in Each Section of the Study**

| | Anesthetic Induction | Thermoregulatory Vasomotion | Ambient Temperature |
|---|---|---|---|
| Age (yr) | 28 ± 4 | 28 ± 6 | 28 ± 5 |
| Weight (kg) | 71 ± 6 | 65 ± 17 | 63 ± 9 |
| Height (cm) | 176 ± 9 | 170 ± 11 | 167 ± 6 |
| Body fat (%) | 19 ± 3 | 20 ± 5 | 19 ± 3 |
| Gender (M/F) | 6/1 | 4/3 | 2/5 |

Values are mean ± SD. There were no statistically significant differences among the groups.

with forced air and circulating water to maintain arteriovenous shunt vasoconstriction (gradient >0°C) during the protocol. The upper chest, neck, and head were covered with a cardboard and plastic canopy, and air circulated at a typical intraoperative flow rate near 5 cm/s). Air temperature within the canopy was randomly set to 18, 20, 22, 24, and 26°C. Each air temperature was maintained for 30 min.

Flows and core and skin temperatures were recorded at 5-min intervals for 30 min at each ambient temperature. All values were averaged first within and then among the volunteers. Changes in core-to-skin temperature differences induced by manipulating ambient temperature were evaluated using linear and second-order regressions.

## Results

Morphometric characteristics of the volunteers participating in each section of the study were similar (table 1). Skin-surface temperatures were similar when evaluated using thermocouples or (uncorrected) liquid crystals. Consequently, we report only liquid-crystal cutaneous temperatures.

### Induction of Anesthesia

Forearm minus fingertip, skin-temperature gradients decreased from +5.9 ± 1.9°C to −4.2 ± 1.3°C (P < 0.001), indicating that induction of anesthesia increased arteriovenous shunt flow more than 20 times.[41] Forehead capillary perfusion, as indicated by laser Doppler flowmetry, was 47 ± 21 before and 31 ± 7 units after induction (P = 0.06). Ambient temperature increased slightly but significantly during the protocol, from 21.1 ± 0.8°C to 21.7 ± 0.4°C (P = 0.02).

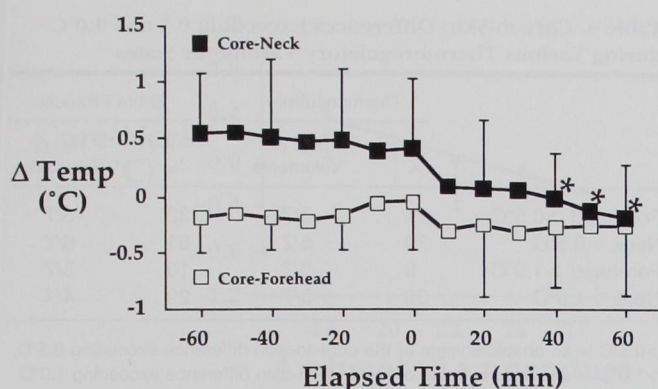Anesthetic-induced vasodilation decreased core tem-

Fig. 1. The difference between tympanic membrane (core) and forehead skin-surface temperature decreased after induction of general anesthesia, but the decrease was not statistically significant. The difference between tympanic membrane and neck skin-surface temperature decreased significantly after induction of general anesthesia. Elapsed time-zero indicates induction of anesthesia. Asterisks (*) indicate values differing significantly from elapsed time-zero; results are presented as means ± SD.

perature ≈2.0°C in the first hour, simultaneously increasing mean skin temperature only ≈0.1°C ($P$ = NS). Nearly constant *mean* skin temperature disguised a large increase in hand and foot skin temperature and a simultaneous smaller decrease in trunk and head skin temperature. For example, neck skin temperature decreased ≈1.4°C. Because neck temperature decreased less than core temperature, the core minus neck difference after 1 h of anesthesia decreased ≈0.6°C. Forehead skin temperature decreased as much as core temperature; consequently, anesthetic induction did not significantly alter the core-to-forehead temperature difference (fig. 1).

Core-to-forehead temperature differences exceeded 0.5°C in 18% of the control measurements and in 35% of the measurements when the entire protocol was considered. However, this difference only once exceeded 1°C. Core-to-neck differences frequently exceeded 0.5°C and sometimes also exceeded 1°C (table 2).

*Thermoregulatory Vasomotion*

According to the protocol, evaporative water loss exceeded 40 g · m$^{-2}$ · h$^{-1}$ through sweating and averaged 72 ± 26 g · m$^{-2}$ · h$^{-1}$. Induction of sweating and shivering significantly altered arteriovenous shunt and capillary blood flow (table 3). The core-to-forehead difference (including the offset correction) was −0.1 ± 0.3°C during the thermoneutral control period. The difference decreased slightly, to −0.4 ± 0.5°C during sweat-

**Table 2. Core-to-Skin Differences Exceeding 0.5 and 1.0°C before and after Anesthetic Induction**

| | Control Period | | Entire Protocol | |
|---|---|---|---|---|
| | % Time | No. of Volunteers | % Time | No. of Volunteers |
| Forehead >0.5°C | 18 | 2/7 | 35 | 7/7 |
| Neck >0.5°C | 51 | 4/7 | 47 | 6/7 |
| Forehead >1.0°C | 0 | 0/7 | 3 | 1/7 |
| Neck >1.0°C | 22 | 3/7 | 13 | 3/7 |

>0.5°C = an absolute value of the core-to-skin difference exceeding 0.5°C; >1.0°C = an absolute value of the core-to-skin difference exceeding 1.0°C.

Core and skin-surface temperatures were recorded at 10-min intervals. The "Control Period" extended from −60 to 0 elapsed minutes. The "Entire Protocol" included the Control Period and the first 60 min after induction of anesthesia.

ing, and increased slightly to +0.3 ± 0.4°C during shivering (fig. 2). However, these changes were neither statistically significant nor clinically important. The pattern was similar but the mean changes were smaller when the core and neck temperatures were compared. The core-to-neck difference was 0.2 ± 1.1°C during the control period, decreased minimally to 0.1 ± 1.0°C during sweating, and then increased to 0.4 ± 0.8°C during shivering (fig. 3).

The core-to-skin temperature difference frequently exceeded 0.5°C both during the control period, and when the entire protocol was considered. Core-to-neck skin temperatures sometimes also exceeded 1°C, although the core-to-forehead difference did so rarely (table 4).

*Ambient Temperature*

According to the protocol, fingertip vasoconstriction (skin-temperature gradient >0°C) was maintained

**Table 3. Thermoregulatory Vasomotion**

| | Thermoneutral | Sweating | Shivering |
|---|---|---|---|
| Perfusion index (units) | 2.0 ± 0.7 | 2.6 ± 1.1 | 0.2 ± 0.2* |
| Forearm minus fingertip, temperature gradient (°C) | −2.0 ± 1.2 | −1.3 ± 0.7 | 9.2 ± 3.5* |
| Laser Doppler (units) | 40 ± 7 | 93 ± 31* | 32 ± 9 |
| Ambient temperature (°C) | 21.3 ± 0.7 | 21.3 ± 0.7 | 21.4 ± 0.8 |
| Core − forehead (°C) | −0.1 ± 0.3 | −0.4 ± 0.5 | 0.3 ± 0.4 |
| Core − neck (°C) | 0.2 ± 1.1 | 0.1 ± 1.0 | 0.4 ± 0.8 |

Values are mean ± SD.

* Statistically significant differences from thermoneutral.

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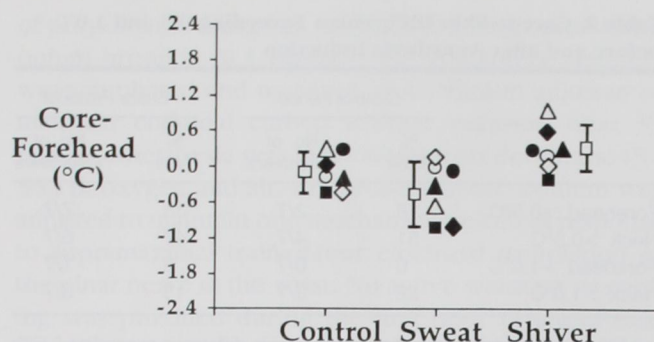


Fig. 2. The tympanic membrane (core) minus forehead skin-surface temperature difference during a thermoneutral control period was 0.1 ± 0.3°C. This difference did not change significantly during vasodilation associated with sweating or vasoconstriction associated with shivering. Results are presented as means ± SD.

throughout the study. Laser Doppler flow averaged 28.2 ± 70 units at 22°C and did not change significantly at any tested ambient temperature. Air speed averaged 3.4 cm/s and never exceed 8.4 cm/s.

Inspection of the raw data indicated that manipulation of ambient temperature altered skin-surface temperature within minutes, and that skin temperature subsequently remained constant for the duration of each 30-min trial. The average difference between core and corrected forehead skin-surface temperatures ($\Delta T$) at ambient temperatures ($T_{ambient}$) between 18 and 26°C fit a second-order regression: $\Delta T = -0.58 + 0.29(T_{ambient}) - 0.01(T_{ambient})^2$, $r^2 = 0.999$. Each 1°C change in ambient temperature, starting near 22°C, thus altered core-to-forehead bias $\approx 0.16°C$ (fig. 4). The core and neck temperatures fit a

**Table 4. Core-to-Skin Differences Exceeding 0.5 and 1.0°C during Various Thermoregulatory Vasomotor States**

| | Thermoneutral | | Entire Protocol | |
|---|---|---|---|---|
| | % | No. of Volunteers | % | No. of Volunteers |
| Forehead >0.5°C | 21 | 3/7 | 32 | 6/7 |
| Neck >0.5°C | 79 | 6/7 | 61 | 6/7 |
| Forehead >1.0°C | 0 | 0/7 | 10 | 3/7 |
| Neck >1.0°C | 39 | 3/7 | 29 | 4/7 |

>0.5°C = an absolute value of the core-to-skin difference exceeding 0.5°C; >1.0°C = an absolute value of the core-to-skin difference exceeding 1.0°C.

Core and skin-surface temperatures were recorded four times each at 5-min intervals during each thermoregulatory vasomotor state.

linear regression: $\Delta T = 1.8 - 0.08(T_{ambient})$, $r^2 = 0.95$ (fig. 5).

## Discussion

Major reasons for monitoring intraoperative core temperature include detection of (1) fever (*e.g.*, from mismatched blood transfusions, blood in the fourth cerebral ventricle, allergic reactions, or infection), (2) malignant hyperthermia and hyperthermia from other causes (*i.e.*, excessive patient heating), and (3) inadvertent hypothermia. Hypothermia is by far the most common among these thermal disturbances, and reductions in core temperature of only 2°C are associated with adverse outcomes including prolonged postanesthetic re-
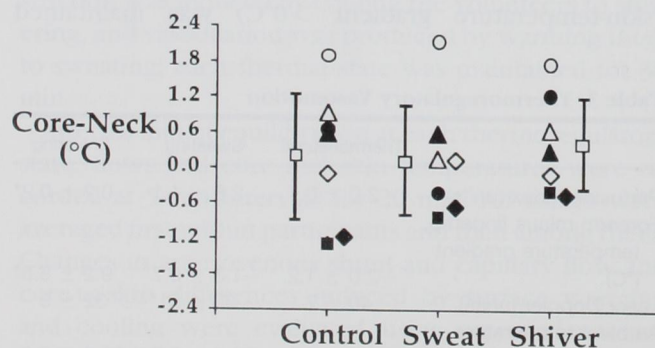


Fig. 3. The tympanic membrane (core) minus neck skin-surface temperature difference during a thermoneutral control period was 0.2 ± 1.1°C. This difference did not change significantly during vasodilation associated with sweating or vasoconstriction associated with shivering. Results are presented as means ± SD.



Fig. 4. The difference between core and forehead skin-surface temperatures ($\Delta T$) at ambient temperatures ($T_{ambient}$) between 18 and 26°C. The data were fit to a second-order regression: $\Delta T = -0.58 + 0.29(T_{ambient}) - 0.01(T_{ambient})^2$, $r^2 = 0.999$. Each 1°C change in ambient temperature, starting near 22°C, thus altered skin temperature $\approx 0.16°C$. Results are presented as means ± SD. Horizontal error bars (variation in ambient temperatures) are not displayed because they were smaller than the size of the markers.
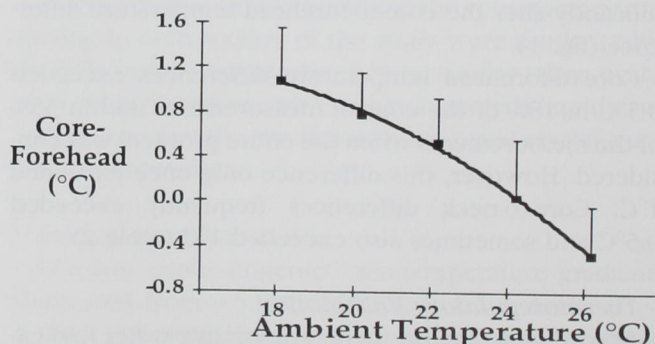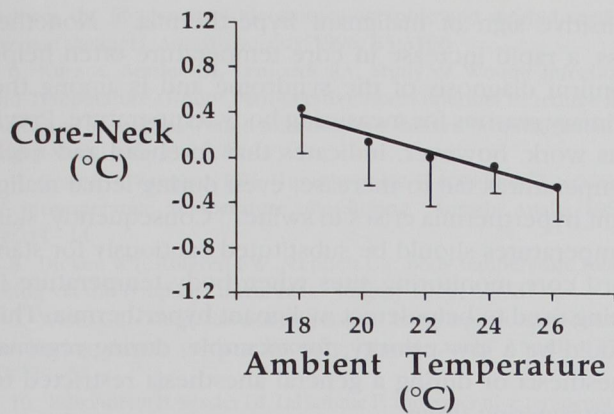
**Fig. 5. The difference between core and neck skin-surface temperatures ($\Delta T$) at ambient temperatures ($T_{ambient}$) between 18 and 26°C. The data were fit to a linear regression: $\Delta T = 1.8 - 0.08(T_{ambient})$, $r^2 = 0.95$. Results are presented as means ± SD. Horizontal error bars (variation in ambient temperatures) are not displayed because they were smaller than the size of the markers.**

covery,[3] increased bleeding and transfusion requirement,[4] ventricular tachycardia and morbid cardiac events,[5] and reduced resistance to surgical wound infections and prolonged hospitalization.[6] Conversely, mild hypothermia may be induced therapeutically because in animals it may protect against cerebral ischemia[17,38] and malignant hyperthermia.[46,47]

When the entire anesthetic induction and vasomotion protocols were considered, core-to-forehead temperature difference exceeded 0.5°C during ≈35% of the measurements but rarely exceeded 1°C. In contrast, the core-to-neck difference exceeded 0.5°C in more than half of the measurements and frequently exceeded 1°C. These data indicate that estimates of core temperature obtained from the forehead are superior to those from the neck. Why accuracy at the neck should be so much worse than at the forehead remains unclear, but forehead temperature is clearly better linked to the thermal core than the neck is. Most of the unacceptable core-to-skin differences resulted from random variation (lack of precision) rather than a consistent bias from tympanic membrane temperature. Consequently, simply changing the offset correction incorporated into liquid crystal thermometers is unlikely to much improve overall accuracy.

The apparent accuracy of cutaneous monitoring would, of course, have been better had greater deviations between core and (corrected) skin temperatures been considered clinically acceptable. Conversely, accuracy would have been worse had smaller deviations

been required. An additional caveat is that our results apply only to liquid crystals and to other simple skin-surface thermometers. More sophisticated systems, such as "deep temperature" thermometers that actively null cutaneous heat flux, are known to be reliable.[48-51]

In addition to being influenced by core temperature, skin-surface temperature is determined by multiple factors, including metabolic heat production, skin blood flow, counter-current mechanisms, subcutaneous insulation, ambient temperature (radiation and conduction), wind speed (convection), and surface insulation (conduction). We tested three circumstances that potentially confound estimates of core temperature obtained by liquid-crystal thermography.

The first potential confounding factor we tested was induction of general anesthesia and consequent redistribution hypothermia. As in previous similar studies, an ≈2°C decrease in core temperature during the first hour was accompanied by only an ≈0.1°C increase in mean skin temperature.[28] Skin temperature increased most in the hands and feet, which is consistent with the observed 20-fold increase in arteriovenous shunt flow and established patterns of heat transfer.[27] In contrast, capillary flow decreased slightly. Consequently, forehead and neck skin temperatures (which depend on both cutaneous flow and core temperature) decreased during redistribution. This indicates that these skin-surface sites are relatively closely linked to core temperature, making them far more suitable for core temperature estimates than distal areas.

Neck skin temperature decreased somewhat less than core temperature during redistribution hypothermia. The core-to-neck difference thus decreased ≈0.6°C during the first hour after induction of general anesthesia. Although this decrease improved absolute accuracy, it would nonetheless appear to clinicians as a 0.6°C (artifactual) increase in core temperature. In contrast, forehead and core temperatures decreased comparably during redistribution, and the core-to-forehead difference thus remained constant near −0.2°C. These data indicate that induction of general anesthesia produces a potentially important artifact when core temperature is estimated from neck skin temperature. However, redistribution does not significantly alter forehead skin temperature.

The second potential confounding factor we tested was thermoregulatory vasomotion associated with sweating and shivering. The core-to-skin differences decreased slightly during sweating, as would be expected from vasodilation, and the core-to-skin differences in-

IKEDA *ET AL.*

creased slightly during shivering, as might be expected during vasoconstriction. The direction of these changes thus was as expected from sweating-induced vasodilation and shivering-induced vasoconstriction. However, the magnitude of the changes was small and unlikely to be clinically important.

The third potential confounding factor we tested was alteration in ambient temperature. Our data indicate that ambient temperature changes do alter the core-to-skin temperature difference, with the forehead being more susceptible to this artifact than the neck. Nonetheless, the magnitude of the artifact was relatively small, suggesting that usual intraoperative alterations in ambient temperature are unlikely to produce clinically important bias. Transferring a patient from a typical 20°C operating room to a typical 25°C postanesthesia care area, however, would reduce the core-to-forehead difference approximately 1°C. Depending on clinical needs, liquid-crystal estimates of core temperature during substantial changes in ambient temperature might thus be accepted, abandoned, or arithmetically corrected.

The effects of thermoregulatory vasomotion on skin temperature were evaluated in unanesthetized volunteers rather than anesthetized patients. Although general anesthesia alters the thresholds for thermoregulatory responses,[29-32] the maximum intensity of vasoconstriction[34] and vasodilation[52] remains essentially normal. (Volatile anesthetics reduce the gain of vasoconstriction,[53] but the reduction is small unless high local skin temperature is maintained.) It is thus unlikely that the observed (small) core-to-skin temperature changes will differ much during general anesthesia. Because most general anesthetics produce vasodilation, ambient temperature alterations are likely to change core-to-skin differences even less in surgical patients than in our volunteers.

Aside from estimating core temperature, there are several other reasons anesthesiologists may wish to measure skin-surface temperatures: (1) average skin temperature is an important thermal input to the central thermoregulatory system[1,2,54]; (2) local skin temperature can indicate the extent of sympathetic blockade during regional anesthesia[55,56]; (3) skin-temperature gradients are a simple method to quantify peripheral thermoregulatory vasoconstriction[41]; and (4) skin temperature monitoring can prevent burns during active external rewarming. Monitoring for each purpose has a place in clinical practice.

Core hyperthermia is neither the first nor the most sensitive sign of malignant hyperthermia.[57] Nonetheless, a rapid increase in core temperature often helps confirm diagnosis of the syndrome and is among the primary reasons for measuring body temperature. Previous work, however, indicates that forehead and neck temperatures fail to increase, even during lethal malignant hyperthermia crises in swine.[16] Consequently, skin temperatures should be substituted cautiously for standard core monitoring sites when body temperature is being used to help detect malignant hyperthermia. This would be a low priority, for example, during regional anesthesia or during a general anesthesia restricted to nontriggering drugs.

In summary, we evaluated skin-temperature thermometry under three conditions in which alterations in the core-to-skin temperature difference were likely. The core-to-peripheral redistribution of body heat that accompanies induction of general anesthesia had little effect on the core-to-forehead skin temperature difference. However, the core-to-neck skin temperature gradient decreased ≈0.6°C in the hour after anesthesia was induced. Thermoregulatory vasoconstriction associated with shivering and vasodilation associated with mild sweating did not produce clinically important alterations in the core-to-skin temperature difference. Core-to-neck temperature differences frequently exceeded 1°C (a 2°C range), whereas two thirds of the core-to-forehead differences were within 0.5°C. Both forehead and neck skin temperatures were sensitive to changes in ambient temperature. However, typical intraoperative increases in ambient temperature did not produce clinically important changes in either measure.

## References

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