Failure of Intraoperative Liquid-Crystal Temperature Monitoring

M. Lou Marsh, MD*, and Daniel I. Sessler, MD†‡

*Frost Street Outpatient Surgical Center, Incorporated, Sun Surgery Corporation, San Diego, California, the †Thermoregulation Research Laboratory, Department of Anesthesia, University of California, San Francisco, San Francisco, California, and the ‡Department of Anesthesia and Intensive Care, University of Vienna, Vienna, Austria

The major reason for monitoring intraoperative temperature is for detection of fever, malignant hyperthermia, and hypothermia. Inadvertent hypothermia—defined by an absolute core temperature <36°C—is by far the most common perioperative thermal disturbance. However, hypothermia may be difficult to evaluate using liquid-crystal thermography, because skin temperatures are 1-4°C less than core temperature (1-3). Furthermore, the difference between skin and core temperatures varies considerably both between and within individuals (4-12).

Liquid-crystal thermography may also fail to detect core hyperthermia. Numerous studies demonstrate that skin-surface thermography often fails to identify clinical fevers, and no published study indicates that liquid-crystal thermography correlates with core temperature during malignant hyperthermia crises in humans. In contrast, the single relevant study showed that changes in forehead and neck skin temperatures failed to identify malignant hyperthermia in swine (13).

Despite the lack of validating data, liquid-crystal thermometers are promoted for “trending” core temperature. The term “trend,” in this context, means that an increase in core temperature will produce an increase in the value displayed on the liquid crystal, without any requirement that the increase be comparable—or even a linear function of core temperature. We now present a case in which liquid-crystal skin-surface thermography failed to “trend” core temperature; monitoring failure prompted otherwise unnecessary laboratory evaluations and cancellation of surgery.

Case Report

An 18-yr-old man was scheduled for anterior cruciate ligament reconstruction. He was otherwise healthy and had a noncontributory personal and family medical history that specifically excluded unexplained perioperative hyperthermia. His preoperative vital signs, including an oral temperature of 36.9°C, were normal. Physical examination was unremarkable. Intravenous metoclopramide, 5 mg, was administered over a 2-min period. General anesthesia was then induced by intravenous administration of 75 µg fentanyl citrate and 100 mg propofol. No muscle relaxants were administered. Anesthesia was maintained with enflurane and nitrous oxide via a laryngeal mask. An infusion of cefazolin sodium was started.

Five minutes after induction of anesthesia, forehead skin-surface temperature, as indicated by a liquid-crystal thermometer (100-Fever Scan®; American Thermometer Company, Glen View, IL), increased from 37.0°C to 38.1°C, and finally to 39.2°C over an 8-min period. The forehead was uncovered throughout, and no active patient warming was used. Cefazolin administration was discontinued (300 mg had been given), and surgery was canceled. Simultaneously, chest skin temperature, measured using a liquid crystal, was 39.2°C, although axillary temperature remained normal at 37.0°C, as measured using a thermistor (Datex, Inc., Tewksbury, MA).

The patient recovered consciousness 5 min after anesthesia was discontinued. At no time was there any evidence of trismus, muscle rigidity, hypercarbia, tachypnea, tachycardia, or hypoxia, nor were flushing, rash, bronchospasm, or other signs of an allergic reaction detected. The patient was monitored for 6 h in the postanesthesia care unit. Serial urine samples failed to reveal myoglobin, and the concentration of creatine kinase in a venous blood sample was normal. Six hours after induction of anesthesia, forehead skin-surface temperature remained 39.2°C (as determined using the liquid crystal). Chest skin temperature (liquid crystal) at that time was 38.1°C, whereas oral and rectal temperatures (thermistor) were 37.2 and 37.4°C, respectively. The patient’s recovery was otherwise uneventful, and he was discharged from the hospital. He has not yet returned for his ligament reconstruction.

Discussion

Physiologically, core temperature is more important than skin temperature because the core provides approximately 80% of the thermal input to control of
autonomic thermoregulatory responses (14). Furthermore, the major complications of mild intraoperative hypoventilation apparently are related to core temperature (15–17). Reliable core temperature monitoring sites include the tympanic membrane, distal esophagus, nasopharynx, and pulmonary artery (1,2). Temperatures measured in any of these sites will be similar, even during rapid thermal perturbations (18). “Intermediate” monitoring sites, such as the mouth, axilla, and bladder, usually correlate well with core temperature except during cardiopulmonary bypass or vigorous exercise (1,2,19).

The standard core-temperature monitoring sites are accurate to approximately 0.2°C and precise to approximately 0.1°C. Under most perioperative circumstances (cardiopulmonary bypass excepted), the intermediate sites are accurate to approximately 0.5°C and probably precise to approximately 0.2°C (1,2). Minimum acceptable accuracy and precision for clinical instruments have yet to be established. However, it is unlikely that accuracy less than 0.5°C would provide sufficient resolution for optimal clinical decision making.

In typical perioperative situations, forehead temperature is 1–4°C less than core temperature (1–3). However, skin-surface temperature is determined by complex interactions among core temperature, ambient temperature, air speed, tissue thermal conductivity, subcutaneous insulation, and tissue perfusion. Core temperature is only one—and not necessarily the most important—of these factors. As a result, the difference between skin and core temperatures varies considerably (4–12). Consistent with this poor correlation, skin-surface temperature monitoring in our patient indicated hyperthermia where none existed.

The most likely explanation for the isolated increase of skin temperature observed in our patient is cutaneous vasodilation, presumably in response to one of the administered drugs. It is striking, however, that vasodilation sufficient to reduce the core-to-skin-temperature gradient more than 2°C was not accompanied by hypotension or other signs of hemodynamic instability and persisted for six hours after anesthesia. It is likely that even larger reductions in the core-to-skin-temperature difference would accompany a severe anaphylactoid reaction. Similarly, skin temperature may fail to reflect clinically important increases in core temperature during malignant hyperthermia if catecholamine release causes intense vasoconstriction (13).

Liquid-crystal thermography indicated that skin-surface temperature in our patient exceeded core temperature, although such reversal of the normal core-to-peripheral gradient occurs only during active cutaneous warming. The explanation is that the 100° Fever Scan® liquid-crystal display is “temperature compensated,” that is, arbitrarily set 2.5°C greater than actual skin-surface temperature. This 2.5°C increase purportedly compensates for the difference between skin and core temperatures. The difficulty with this approach, as illustrated by this case, is that the difference between forehead skin and core temperatures can vary considerably, even in the same patient.

In summary, we present a case in which liquid-crystal skin-surface thermography failed to “trend” core temperature. Specifically, skin-surface monitoring suggested core hyperthermia, although core temperature in fact remained normal. This failure prompted laboratory evaluations and cancellation of surgery; it illustrates the dangers of using skin temperature as a substitute for core-temperature monitoring sites.

This case was referred to the authors by Henry Rosenberg, MD, of the Malignant Hyperthermia Association of the United States (MHAUS). The MHAUS hotline is available 24 hours/day to answer questions and assist with emergency management of malignant hyperthermia crises. It can be reached at 1-800-MH-HYPER in the United States and 1-315-428-7924 internationally.

References

