

Perioperative Temperature Management

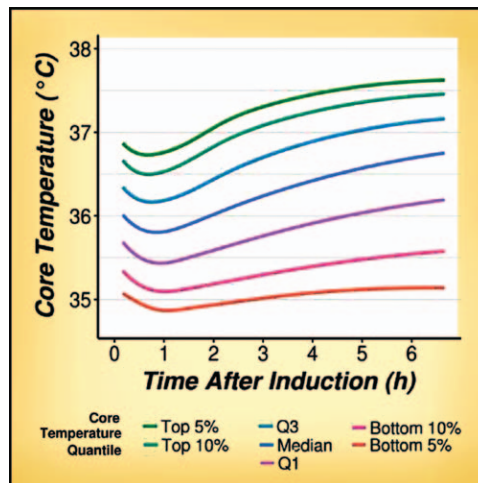
Time for a New Standard of Care?

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This article has been selected for the ANESTHESIOLOGY CME Program. Learning objectives and disclosure and ordering information can be found in the CME section at the front of this issue.

WHEN I started anesthesia residency in 1988, patients undergoing major surgery routinely arrived in the postanesthesia care unit (PACU) with a core temperature of 34.5° to 35°C. We did not fully understand how anesthesia causes hypothermia; we did not have practical, effective means of warming patients; and we did not have evidence of harm—although the shivering patients in the PACU probably had a different perspective. All this changed during my first decade in practice; by 1999, maintenance of perioperative normothermia had been incorporated into practice guidelines.¹ The most common definition of perioperative normothermia is core temperature at least 36.0°C on arrival in the PACU. This number was extrapolated from studies that compared outcomes between patients with relatively large differences in core temperature (1° to 2°C) on arrival in the PACU. Sun *et al.*² in this issue, using innovative analyses of a large patient dataset, demonstrate that, although most patients meet criteria for normothermia on arrival in the PACU, *intraoperative* hypothermia (35° to 36°C) is common. Moreover, longer duration of hypothermia is associated with a significant increase in transfusion requirement and a small but statistically significant increase in hospital length of stay. These results suggest the need for a more comprehensive definition of perioperative normothermia and more aggressive efforts to prevent intraoperative hypothermia.



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strated that central hypothermia does not require a change in total body heat content when there is redistribution of heat from the core to the periphery. Glosten *et al.*⁴ demonstrated that, even with active warming, redistribution (and not heat loss) leads to an early decrease in core temperature under general and regional anesthesia. In awake patients, cold exposure leads to vasoconstriction and redistribution of heat to the core; anesthetic agents cause vasodilation and redistribution of heat to the periphery. Forced-air warming and

In the 1980s and 1990s, Daniel Sessler and colleagues systematically defined the physiology of anesthesia-induced hypothermia. Perioperative hypothermia was demonstrated to have detrimental effects on patient comfort and recovery time, coagulation, and drug metabolism. Scott Augustine developed the forced air warmer (Bair Hugger, 3M, St. Paul, MN) and it became commercially available in 1988. The ability to warm patients effectively led to randomized controlled trials that demonstrated reduced surgical site infections, blood loss and transfusion, and cardiac complications in patients with a normal core temperature compared with patients with a core temperature 1° to 2°C lower on arrival in the PACU.

To understand the results of those clinical trials and the implications of the current large database study, it is important to understand the underlying physiology. Holdcroft *et al.*³ demon-

Image: Core temperature trajectories in 58,814 patients undergoing noncardiac surgery. From Sun *et al.*,² figure 3 (this issue).

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other active warming methods transfer heat to the patient and, over time, return core temperatures to normal.

In the current study, Sun *et al.*² evaluated esophageal core temperature throughout surgery in more than 50,000 adults having surgery lasting over an hour who were actively warmed intraoperatively. The core temperature 45 min after induction was less than 36°C in 64% of patients and less than 35.5°C in 29% of patients. Hypothermia lasting more than an hour was common although 91% of patients were normothermic by the end of surgery. Accounting for variables including type and duration of surgery, preoperative hemoglobin, and comorbidities, there was a significant association between degree-hours of hypothermia and transfusion.

The study has some limitations. The database did not include all outcomes of interest, so we do not know whether these effects are pertinent for surgical site infection or cardiac complications. The study is retrospective, so the identified associations cannot be considered evidence of causality. However, clinical trials have already established causal relations.

One major potential confounder in this study is the complex relation between duration of surgery, blood loss, fluid and blood product administration, and core temperature. Patients with more blood loss might be more likely to become hypothermic, rather than *vice versa*, because of administration of cold fluid and blood products. Inclusion of these confounders in the multivariable analysis strengthens the argument for hypothermia-driving blood loss. Moreover, exclusion of massively transfused patients gave the same association between hypothermia exposure and transfusion. However, patients in the highest quartile for hypothermia exposure had longer duration of surgery (289 [238 to 355] min) compared with the lowest quartile (137 [104 to 191] min). Given that patients with a longer duration of surgery are more likely to be normothermic at the end of surgery (because of the longer exposure to active warming), there is likely a more complicated interaction between blood loss and hypothermia.

What are the implications of this study for anesthetic practice? First, it is time to reevaluate our definition of normothermia. A first step would be to assess not only core temperature on arrival in the PACU but also the lowest core temperature and the duration of hypothermia intraoperatively. Electronic medical records could easily calculate such a variable. We also need more reliable measures of core temperature. Esophageal temperature is considered the definitive standard, but esophageal measurements are available only for anesthetized patients, the probe must be inserted to adequate depth to be accurate, and the esophagus is not always accessible.

Although better metrics for hypothermia are important, a critical implication of this study is that current standards

and practice routinely lead to intraoperative hypothermia, which is associated with a higher transfusion requirement. These results should be an impetus for changes in practice that lead to lower rates of intraoperative hypothermia. The practice at most centers is to apply the warming device after induction of anesthesia and application of surgical drapes. As demonstrated in the current study, this predictably leads to hypothermia in the first hour in the majority of patients. Application of an active warming device preoperatively (*i.e.*, in the preoperative holding area) reduces the decrease in core temperature in the first hour after induction.^{4,5} We implemented routine prewarming for most patients in our hospitals several years ago. Our experience suggests that routine prewarming is both feasible and effective.

The study by Sun *et al.*² starts a new conversation on perioperative temperature management. Future studies should evaluate the effectiveness of interventions to reduce the degree and duration of intraoperative hypothermia and the effect of these interventions on the broad range of outcomes known to be temperature sensitive. These studies will require development of better methods of assessing core temperature throughout the perioperative period.

Competing Interests

The author is not supported by, nor maintains any financial interest in, any commercial activity that may be associated with the topic of this article.

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