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# The Effects of Hydration on Core Temperature in Pediatric Surgical Patients

Tiberiu Ezri, M.D.,* Peter Szmuk, M.D.,† Marian Weisenberg, M.D.,‡ Francis Serour, M.D.,§ Arcadi Gorenstein, M.D.,‖ Daniel I. Sessler, M.D.#

***Background:*** **Reduced vascular volume might influence body temperature by diverting heat flow from peripheral tissues to the central organs. We therefore tested the hypothesis that mild hypovolemia helps to prevent intraoperative hypothermia in pediatric patients.**

***Methods:*** **Twenty-two pediatric patients (aged 1–3 yr) undergoing prolonged minor surgery were randomly assigned to conservative (n = 12) or aggressive (n = 10) perioperative fluid management. The conservative group fasted 8 h before surgery and received a crystalloid at 1 ml · kg$^{-1}$ · h$^{-1}$ during surgery. The aggressive group was allowed to drink liquids until 3 h before surgery and was given a maintenance crystalloid at 8 ml · kg$^{-1}$ · h$^{-1}$. Anesthesia was induced and maintained with halothane in nitrous oxide. Ambient temperature was kept near 25°C, but the patients were not actively warmed. During recovery from anesthesia, additional fluid was given to the conservative group so that perioperative fluid totaled 9.5 ml · kg$^{-1}$ · h$^{-1}$ in both groups.**

***Results:*** **Intraoperative body weight remained unchanged in the aggressive group and decreased only 1% in patients managed conservatively. Heart rate was slightly greater in the conservative group (107 ± 9 *vs.* 95 ± 4 beats/min, *P* = 0.002), but blood pressure was similar. Esophageal temperature in patients whose fluid was managed conservatively increased significantly, by 0.4 ± 0.3°C, to 37.1°C; in contrast, temperature in the aggressive group decreased significantly, by 0.4 ± 0.2°C, to 36.4°C (*P* < 0.001 between groups). Temperatures remained significantly different 1 h after surgery.**

***Conclusions:*** **Conservative fluid management, which decreased body weight by only 1%, prevented reduction in core body temperature, presumably by reducing dissipation of metabolic heat from the core thermal compartment to peripheral tissues.**

PREOPERATIVE requirements have changed recently, and many centers now allow young patients to drink clear fluids up to 2 to 3 h before surgery.[1] Others, however, still require pediatric patients to refrain from eating and drinking from the evening before surgery.[2] Furthermore, some patients end up fasting as long as 18 h because parents are often reluctant to awaken children just to have them drink. Clinicians also use a wide range of fluid replacement strategies; consequently, some pediatric patients are given far more fluid than others.

Different fluid administration strategies appear to have little influence on intraoperative heart rate or blood pressure. Variation in vascular volume may, nonetheless, influence body temperature. The metabolically active central organs are the primary source of body heat; however, this heat must be transferred to peripheral tissues before being dissipated to the environment. Some heat is conducted directly through tissues, but blood-borne convection is by far the most important mechanism for internal heat dissipation in humans.[3,4]

Even slight reductions in vascular volume provoke compensatory peripheral vasoconstriction, which increases systemic vascular resistance and, therefore, maintains normotension without requiring much tachycardia.[5,6] To the extent that peripheral flow is reduced, dissipation of heat will also be reduced. We therefore tested the hypothesis that mild hypovolemia helps to prevent intraoperative hypothermia in pediatric patients.

## Materials and Methods

With approval of the Institutional Review Board at Wolfson Hospital (Holon, Israel) and parental informed consent, we recruited 22 pediatric patients, aged 1–3 yr, scheduled for procedures with an expected duration of at least 2 h. Procedures included hernia, hypospadias repair, diode laser procedures for retinopathy of prematurity, and orthopedic procedures that did not require leg tourniquets. There were no exclusions based on race or sex. Patients with a recent fever or a history of cardiac or renal disease were excluded.

### *Protocol*

Patients were randomly assigned to conservative or aggressive perioperative fluid management. Randomization occurred at the preoperative visit and was based on computer-generated codes kept sealed in opaque, sequentially numbered envelopes until opened. Patients in the conservative group fasted for 8 h before surgery and were given a crystalloid at a rate of 1 ml · kg$^{-1}$ · h$^{-1}$ during surgery. Those assigned to the aggressive group were allowed to drink clear liquids until 3 h before

* Director, Department of Anesthesia, ‡ Attending Anesthesiologist, § Attending Pediatric Surgeon, ‖ Director, Department of Pediatric Surgery, Wolfson Medical Center, Sackler School of Medicine. † Director, Postanesthesia Care Unit, Department of Anesthesiology, Hermann Hospital, The University of Texas Medical School. # Associate Dean of Research, Director of Outcomes Research® Institute, Distinguished University Research Chair, Lolita and Samuel Weakley Professor of Anesthesiology and Pharmacology, Department of Anesthesiology, University of Louisville, and Professor and Vice-Chair, Ludwig Boltzmann Institute, University of Vienna, Vienna, Austria.

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Address reprint requests to Dr. Ezri: Department of Anesthesia, Wolfson Medical Center, P.O. Box 5, Holon 58100, Israel. Address electronic mail to: tezri@wolfson.health.gov.il. Individual article reprints may be purchased through the Journal Web site, www.anesthesiology.org.

surgery and were specifically given 10 ml/kg of clear liquid at that time. During surgery, they were given a crystalloid at a rate of 8 ml · $kg^{-1}$ · $h^{-1}$.

The patients were premedicated with 0.5 mg/kg oral midazolam 20–30 min before induction. Anesthesia was induced and maintained per routine with nitrous oxide in oxygen and halothane with or without fentanyl during maintenance. After induction of anesthesia, an intravenous cannula was inserted, and lactated Ringer's solution at 37°C was given at the randomly designated rates mentioned previously. Fluids were heated with an Astotherm Plus warmer (Stihler Electronic, Stuttgart, Germany). This system includes an actively heated sleeve for the tubing from the warmer to the patient; thus, fluids were administered at 37°C throughout the range of flows used in our patients. Ambient temperature was maintained at 25°C, and the patients were covered with a single blanket; however, they were not actively warmed.

In the PACU, additional fluid was given to the conservative group so that the total perioperative fluid totaled 9.5 ml · $kg^{-1}$ · $h^{-1}$ in both groups. These fluids also were heated to 37°C. Patients spent 1 h in the PACU.

### *Measurements*

End-tidal halothane partial pressures were recorded. Heart rate and blood pressure were measured oscillometrically at 5-min intervals. Core temperature was measured in the distal esophagus at 15-min intervals during surgery. Postoperatively, body temperature was recorded at 15-min intervals for 1 h using a rectal probe inserted 1 to 2 cm past the anus. Temperature measurements were made with M-MNSTPR (YSI-400 standardized) probes with an accuracy of ± 0.1°C over the range of 25–45°C. The probes were connected to a Datex-Engstrom AS/3 Anesthesia Monitor (Datex-Engstrom Division, Instrumentarium Corp., Helsinki, Finland).

The total amounts of intraoperative and preoperative fluids for both groups were recorded. Patients were weighed in the operating room immediately after induction of anesthesia and at the end of surgery by the anesthesiologist in charge of the case, using a scale accurate to ± 5 g (Shekel Electronic Scale T-15-S, Kibbutz Beit Keshet, Israel). The weight of dressings, intravenous cannulae, and so forth was subtracted from the body weight.

### *Statistical Analysis*

Our primary outcome was intraoperative change in core temperature. Heart rate, blood pressure, end-tidal halothane concentration, and ambient temperature were averaged over the intraoperative period for each patient. These values were subsequently averaged among the patients in each treatment group. Normally distributed data were compared with paired or unpaired two-tailed *t* tests, as appropriate. Nonparametric data were analyzed with Mann–Whitney rank sum tests. Results are presented as mean ± SD; $P < 0.05$ was considered statistically significant.

**Table 1. Patient Characteristics and Possible Confounders**

| | Conservative (n = 12) | Aggressive (n = 10) | *P* |
|---|---|---|---|
| Age | 2.1 ± 0.9 | 2.0 ± 0.9 | 0.947* |
| Sex, male/female | 12/0 | 9/1 | — |
| Weight at induction, kg | 13.0 ± 2.6 | 13.3 ± 3.5 | 0.841 |
| Duration of surgery, h | 2.4 ± 0.4 | 2.4 ± 0.5 | 0.444* |
| Mean arterial pressure, mmHg | 76 ± 5 | 78 ± 6 | 0.414 |
| Heart rate, beats/min | 107 ± 9 | 95 ± 4 | 0.002 |
| End-tidal [halothane], % | 0.8 ± 0.4 | 0.8 ± 0.4 | 0.901 |
| Fentanyl, μg | 13 ± 3 | 13 ± 2 | 0.859 |
| OR ambient temperature, °C | 25.0 ± 0.1 | 24.9 ± 0.1 | 0.402* |
| PACU ambient temperature, °C | 22.1 ± 0.1 | 22.1 ± 0.1 | 0.967* |

Data presented as mean ± SD.

* Mann–Whitney-rank-sum tests, other data were compared with unpaired, two-tailed *t* test.

OR = operating room; PACU = postanesthesia care unit.

## Results

Thirty patients were enrolled in the study. The data from eight patients (four in each group) were excluded because their surgeries lasted less than 2 h.

Morphometric and demographic characteristics of the patients were similar in the two groups, as was surgery time. Heart rate was slightly greater in the conservative group (107 ± 9 *vs.* 95 ± 4 beats/min, $P = 0.002$), but mean arterial blood pressure in the groups was similar. Ambient room temperatures in the operating room and PACU were comparable for each group (table 1).

Weight decreased 0.15 ± 0.13 kg during surgery in the conservative group (approximately 1%) and 0.05 ± 0.06 kg in the aggressive group (table 2; $P = 0.07$). Intraoperative core temperatures are shown in figure 1. Core temperature in the conservative group increased by 0.4 ± 0.3°C to 37.1°C ($P < 0.001$). In contrast, core temperature in the aggressive group decreased by 0.4 ± 0.2°C to 36.4°C ($P < 0.001$). Consequently, temperatures in the two groups differed significantly at the end of surgery ($P < 0.001$; table 2 and fig. 2). Even after one postoperative hour, core temperatures continued to differ significantly: 37.1 *versus* 36.4°C.

## Discussion

Even mild reductions in vascular volume provoke a compensatory vasoconstriction that reduces peripheral perfusion. The patients assigned to conservative fluid management became slightly dehydrated and lost about 1% of their body weight during surgery. This small re-

**Table 2. Major Outcomes**

| | Conservative | Aggressive | *P* |
|---|---|---|---|
| Intraoperative fluid, ml | 34 ± 8 | 263 ± 77 | <0.001 |
| Postoperative fluid, ml | 260 ± 53 | 40 ± 11 | <0.001 |
| Total fluid, ml | 293 ± 58 | 303 ± 88 | 0.763 |
| Intraoperative weight change, kg | −0.15 ± 0.13 | −0.05 ± 0.06 | 0.069* |
| Initial core temperature, °C | 36.7 ± 0.4 | 36.8 ± 0.3 | 0.639 |
| Intraoperative core temperature change, °C | 0.4 ± 0.3 | −0.4 ± 0.2 | <0.001 |
| Esophageal–rectal temperature at end of surgery, °C | 0.06 ± 0.12 | 0.03 ± 0.15 | 0.971 |
| Postoperative temperature, °C | 37.1 ± 0.4 | 36.4 ± 0.4 | <0.001 |

Data presented as mean ± SD.

* Mann–Whitney-rank-sum tests, other data were compared using unpaired, two-tailed *t* test.

duction is far less than that used in physiologic studies[7] or the reduction of 5% or more that occurs commonly during bouts of pediatric gastroenteritis.[8] Furthermore, it was insufficient to reduce intraoperative mean arterial pressure and increased heart rate by only 10%.

Redistribution hypothermia reduced core temperature in both groups during the first 30 min of anesthesia. However, over the duration of surgery, conservative fluid management increased core temperature by 0.4°C, whereas those patients who were given aggressive amounts of fluid became hypothermic by the same amount. Consequently, core temperature in the two groups differed by 0.7°C at the end of surgery. Although only 0.5°C of hypothermia significantly increases blood loss,[9] it seems unlikely that the observed difference was clinically important in our population. The effect of conservative fluid management is nonetheless of considerable mechanistic interest and is consistent with the thermal effects of other conditions in which peripheral flow is restricted.

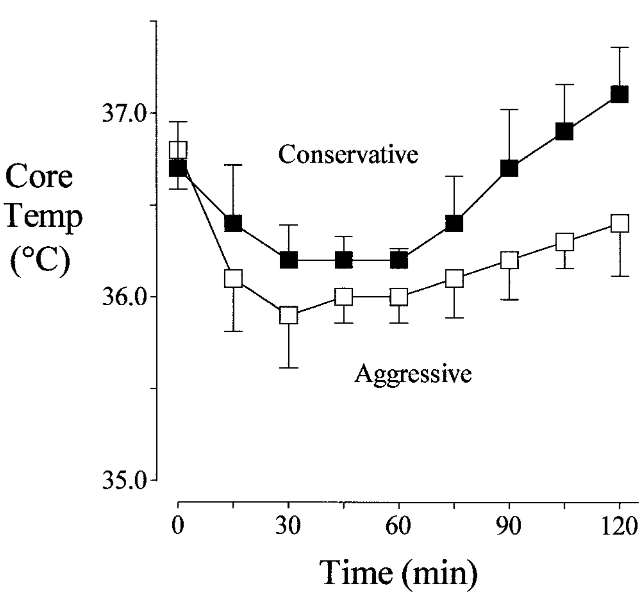


**Fig. 1. Core temperature in the conservative (solid squares) and aggressive (open squares) groups. Elapsed time zero is induction of anesthesia. Error bars show 95% confidence intervals.**

Arteriovenous shunt constriction is by far the most commonly used thermoregulatory response, typically being activated numerous times daily to prevent excessive heat loss. General anesthetics, however, centrally impair thermoregulatory control to prevent intraoperative activation of this defense at near-normal temperatures.[10,11] The result is an initial rapid core-to-peripheral redistribution of body heat[12] followed by a slower, linear decrease in core temperature that results from heat loss exceeding heat production.[13]

Mild hypovolemia presumably similarly prevented hypothermia by restricting convective transfer of heat from metabolically active central organs to peripheral tissues, thus constraining heat to the core rather than facilitating dissipation of heat to the environment.[14] Mild hypovolemia increases the threshold for thermoregulatory vaso-

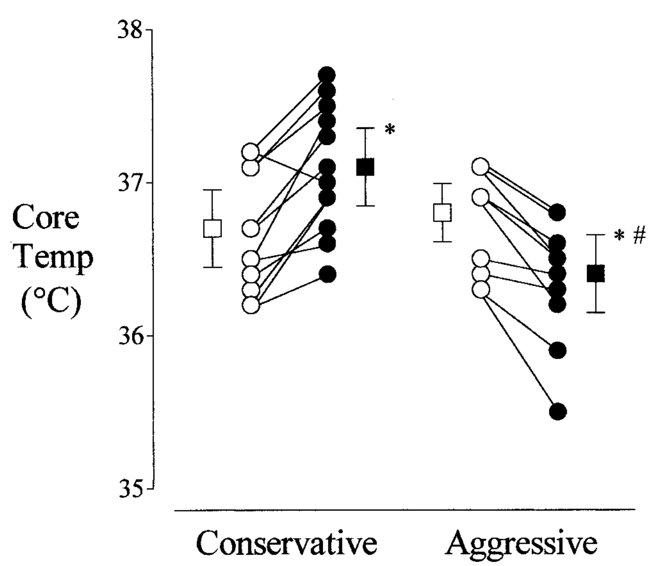


**Fig. 2. Preoperative (open circles) and postoperative (solid circles) core body temperature in individual pediatric patients who were given conservative or aggressive perioperative fluid management. Squares show the mean (± 95% confidence intervals) for each group. Asterisks (*) indicate that the average postoperative temperature was significantly different from the average preoperative temperature in the same group (paired *t* tests, *P* < 0.001). The pound sign (#) indicates that the average postoperative temperatures in the conservative and aggressive groups were significantly different as well (unpaired *t* test, *P* < 0.001).**

constriction by 0.4°C without increasing the gain.[15] Hyperhydration has no effect on the vasoconstriction[15] or sweating[16] thresholds. We did not measure arteriovenous shunt perfusion; however, it is highly unlikely that our patients were cold enough to trigger thermoregulatory vasoconstriction since during anesthesia the threshold is usually decreased by 2 to 3°C.[10,11] Decreased peripheral blood flow in our anesthetized patients was therefore most likely a cardiovascular reflex rather than a thermoregulatory one.

A surgical tourniquet obliterates extremity blood flow and can thus be considered the ultimate vasoconstrictor. It is therefore unsurprising that a single leg tourniquet makes pediatric patients hyperthermic, and that hyperthermia is even worse when two tourniquets are used.[17] Leg tourniquets also slow reduction of core temperature during cold-water immersion.[18] As might be expected, subsequent release of the tourniquets is associated with a core-to-peripheral redistribution of body heat[19,20] similar to that normally accompanying induction of general[12] or regional[21] anesthesia. An even more extreme example is cardiogenic shock, which can be associated with enormous core-to-peripheral tissue temperature gradients.[22]

Perioperative core temperatures are modulated by changes in the internal distribution of body heat and by systemic heat balance. Heat balance is, in turn, largely determined by ambient exposure, which includes the effect of passive insulation[23] and active heating.[24] Core temperature in our hypovolemic patients increased, whereas temperature decreased in those who were given larger amounts of fluid. The magnitude or even direction of the changes would presumably vary in other environments. However, the relative effect of mild fluid deprivation is likely to be similar under other conditions.

In addition to its obvious effects on blood pressure and heart rate, fluid management affects numerous perioperative responses. For example, aggressive hydration reduces the risk of postoperative nausea and vomiting.[25] It also improves subcutaneous tissue oxygenation,[26] which may reduce the risk of surgical wound infection.[27] Our observation that slight dehydration facilitates maintenance of normothermia therefore should not be considered a recommendation to restrict perioperative fluids. Instead, it is but one of many factors that might be considered when planning an individual vascular volume management strategy.

In summary, conservative fluid management decreased body weight by only 1%, did not reduce mean arterial pressure, and produced only a slight tachycardia; however, esophageal temperature increased significantly, by 0.4 ± 0.3°C, in these patients and decreased significantly, by 0.4 ± 0.2°C, in patients who were aggressively hydrated. Very mild dehydration thus helped to prevent intraoperative hypothermia, presumably by reducing dissipation of metabolic heat from the core thermal compartment to peripheral tissues.

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