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Thermoregulatory Thresholds for Vasoconstriction in Pediatric Patients Anesthetized with Halothane or Halothane and Caudal Bupivacaine

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The thermoregulatory threshold for vasoconstriction has been studied in infants and children given isoflurane, but not in those given halothane anesthesia. More importantly, the effect of vasoconstriction on central temperature in pediatric patients remains unknown. Also unknown is the effect of caudal analgesia on vasoconstriction thresholds. Accordingly, in the first portion of this study, we determined the central thermoregulatory threshold in 23 infants and children given ≈0.6% halothane and caudal anesthesia for abdominal surgery. Patients were prospectively assigned to one of four weight groups: 5–10, 10–20, 20–30, and 30–50 kg. The threshold was considered the central temperature triggering peripheral vasoconstriction, and significant vasoconstriction was defined as a forearm − fingertip skin-surface temperature gradient exceeding 4° C. Thresholds were similar (≈35.7° C) in each study group, suggesting that thermoregulatory responses to halothane anesthesia are similar in infants and children of differing weights. However, they were higher than expected based on the previously reported thresholds in pediatric patients given isoflurane anesthesia. After peripheral vasoconstriction, central temperature continued to decrease in patients weighing more than 30 kg but remained constant or increased slightly in the others. These data suggest that thermoregulatory responses are more effective in infants and small children than in bigger children or adults. In the second part of this study we evaluated the effect of caudal analgesia on the thermoregulatory threshold for vasoconstriction. Children undergoing hypospadias repair were anesthetized with halothane (0.9%) and oxygen. Following induction, they were randomly assigned to caudal analgesia (n = 7) or penile nerve block (n = 6). The threshold was 35.9 ± 0.5° C in the caudal group and 35.7 ± 0.5° C in patients given a penile nerve block, indicating that caudal analgesia *per se* has little effect on the thermoregulatory threshold for vasoconstriction during halothane anesthesia. (Key words: Analgesia, caudal: bupivacaine. Anesthesia: pediatric. Anesthetics, volatile: halothane. Hypothermia. Temperature, regulation: setpoint; threshold.)

INTRAOPERATIVE HYPOTHERMIA provokes peripheral thermoregulatory vasoconstriction in adults, children, and infants.[1,2] We have previously demonstrated that vasoconstriction thresholds (central temperatures triggering peripheral vasoconstriction[3]) are similar in pediatric patients of various sizes given isoflurane anesthesia.[2] However, thermoregulatory inhibition is agent- and dose-dependent, and thresholds in adults appear to be better preserved with halothane than with isoflurane.[3,4] The thermoregulatory effects of halothane in patients of differing ages and weights therefore cannot be reliably extrapolated from pediatric patients given isoflurane or from previous results in adults.

Central temperature in anesthetized adults usually decreases until hypothermia triggers peripheral vasoconstriction and then remains nearly constant.[3] Pediatric patients differ from adults in having a larger surface-to-volume ratio and more skin covering central tissues (head and trunk) than covering peripheral tissues (arms and legs). Consequently, infants and children frequently are considered at especially high risk of developing moderate or severe intraoperative hypothermia.‡ The ability of thermoregulatory responses to prevent such hypothermia in anesthetized pediatric patients remains unknown.

We used caudal analgesia to minimize surgical stimulation during our previous evaluation of thermoregulatory responses in infants and children given isoflurane anesthesia.[2] However, the extent to which caudal analgesia influences intraoperative thermoregulatory responses remains unclear. We thus determined vasoconstriction thresholds in children and infants given halothane alone or halothane and caudal analgesia, and the effects of vasoconstriction on central temperature.

Materials and Methods

With approval from the Ethical Committee of the Hospital for Sick Children, we studied 36 ASA physical status 1 or 2 pediatric patients after obtaining written, informed consent from their parents. None was obese, was taking medication, or had a history of thyroid disease, dysautonomia, Raynaud's syndrome, malignant hyperthermia, or recent fever. All patients weighed 5–50 kg, and none was given presurgical medication.

In the first portion of the study, patients undergoing elective intraabdominal surgery were prospectively as-

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‡ Flacke JW, Flacke WE: Inadvertent hypothermia: Frequent, insidious, and often serious. Seminars in Anesthesia 2:183–196, 1983.

signed to one of four weight groups: 5–10, 10–20, 20–30, and 30–50 kg. Anesthesia was induced with halothane and 70% nitrous oxide in oxygen, and the trachea of each patient was intubated following administration of vecuronium, 0.1 mg/kg. Patients' lungs were mechanically ventilated with a tidal volume near 12 ml/kg, and the respiratory rate was adjusted, as needed, to maintain an end-tidal P_{CO_2} near 35 mmHg. Bupivacaine (1.0–1.25 ml/kg, 0.125%) was injected into the epidural space *via* a caudal approach immediately after induction of anesthesia. Our clinical experience with similar pediatric patients has shown that this dose of bupivacaine produces a sympathetic blockade extending no higher than the twelfth thoracic dermatome.

Intraoperatively, anesthesia was maintained with halothane in oxygen, at end-tidal concentrations near 0.6%. Muscle relaxation was maintained with intravenous administration of vecuronium, 0.05 mg/kg, as needed, to maintain a one- or two-twitch mechanical response to stimulation of the ulnar nerve by a peripheral nerve stimulator. No barbiturates or opioids were given during surgery.

In the second part of the study, patients undergoing hypospadias repair were anesthetized with halothane (0.9% end-tidal) and oxygen. They were then randomly assigned to caudal analgesia (bupivacaine 0.125%, 1.0–1.25 ml/kg) (n = 7) or dorsal penile nerve block using 0.1 ml/kg of 0.5% bupivacaine (n = 6). The nerve blocks were performed shortly after tracheal intubation.

In all cases, operating rooms were maintained at a normal temperature (≈20° C) to provide similar cutaneous input to the thermoregulatory system. Intravenous fluids were not warmed and were administered in similar weight-adjusted quantities in each group. Respiratory gases were not humidified, and circulating-water warming blankets were not used.

End-tidal gases were sampled from a piece of polyethylene tubing inserted through the endotracheal tube to a position estimated to be 2 cm above the carina.[5] End-tidal halothane concentrations were quantified using a Capnomac® (Datex Medical Instrumentation, Inc., Tewksbury, MA) end-tidal gas analyzer. Heart rate was continuously monitored during each study using lead-2 electrocardiography. Blood pressure was evaluated oscillometrically (Dinamap® 1846 SX, Critikon Inc., Tampa, FL) at 5-min intervals throughout surgery.

Temperatures were monitored using disposable thermocouples and model 6500 digital thermometers (Mona-Therm,® Inc., St. Louis) that require no user calibration and have an accuracy near 0.1° C. Skin-surface temperatures were monitored using disposable, 1-cm-diameter, self-sticking thermocouples, and central temperatures using a flexible, cotton-covered thermocouple placed in contact with the tympanic membrane. Average skin-surface temperatures were calculated using a standard formula: $0.3 \cdot [t_{chest} + t_{upper\ arm}] + 0.2 \cdot [t_{thigh} + t_{calf}]$.[6]

Peripheral vasoconstriction was evaluated using forearm − fingertip, skin-surface temperature gradients. We have previously demonstrated an excellent correlation between these gradients and absolute fingertip blood flow in adults.[7] Furthermore, skin-temperature gradients correlate well with laser Doppler flowmetry in pediatric patients of various sizes.[2] The forearm thermocouple was placed on the radial side of the arm, midway between the wrist and the elbow; the fingertip probe was positioned on the tip of the index finger. The monitored arm did not have an intravenous catheter or blood pressure cuff, and all thermocouple sites were fully exposed to room air.

As in previous studies, we prospectively defined significant thermoregulatory vasoconstriction as a skin-temperature gradient ≥4° C. The tympanic membrane temperature at which the skin-temperature gradient first exceeded 4° C was considered the central thermoregulatory threshold for vasoconstriction.

Skin-surface and tympanic temperatures and end-tidal isoflurane concentrations were recorded every 10 min from induction of anesthesia until closure of the surgical incision. Subsequent intraoperative and postoperative management were determined by the attending anesthesiologist.

Continuous variables were analyzed using unpaired, two-tailed *t* tests, one-way analysis of variance, or repeated-measures analysis of variance, as appropriate. Intragroup differences were identified using Student-Newman-Keuls tests. All values are expressed as the mean ± the standard deviation. Differences were considered significant when $P < 0.05$.

Results

Patients in the first portion of the study differed significantly only in weight and age (table 1). Gender, ambient temperature, thermoregulatory thresholds, time of vasoconstriction, and end-tidal halothane concentrations were not different; skin-surface temperatures rarely changed more than 1° C throughout study.

All patients developed significant peripheral vasoconstriction. The mean threshold temperatures in each group ranged from 35.5° C to 35.9° C, and did not differ significantly among groups (fig. 1).

Central temperature decreased in each group until the time of peripheral vasoconstriction and then continued to decrease in patients weighing more than 30 kg while remaining constant or increasing slightly in the other groups (fig. 2). An increase in minute ventilation frequently was required near the time of vasoconstriction (to maintain a normal end-tidal P_{CO_2}) in the 5–10-kg in-

TABLE 1. Morphometric Data and Summary of Results in Patients Divided by Weight

Group	Weight (kg)	Age (yr)	Gender (male/n)	Ambient (° C)	TM (° C)	Time (min)	Skin (° C)	[Halothane]
5–10	6.7 ± 1.5	0.8 ± 0.3	1/5	20.0 ± 0.5	35.7 ± 0.4	48 ± 11	33.3 ± 0.5	0.6 ± 0.1
10–20	16.9 ± 1.9	2.6 ± 1.0	5/8	20.0 ± 1.1	35.7 ± 0.6	81 ± 45	33.4 ± 1.0	0.6 ± 0.1
20–30	26.6 ± 2.5	6.5 ± 2.5	2/5	20.5 ± 0.9	35.5 ± 0.2	88 ± 23	33.7 ± 0.7	0.7 ± 0.2
30–50	42.8 ± 5.8	12.1 ± 2.9	1/5	20.0 ± 0.3	35.9 ± 0.3	40 ± 17	33.0 ± 0.8	0.5 ± 0.2

The central temperature thermoregulatory threshold for vasoconstriction is indicated in the column marked TM (° C). End-tidal halothane concentrations ([Halothane]) at the time of vasoconstriction are not age-corrected. Mean skin-surface temperatures at the time of vasoconstriction are labeled Skin (° C). Values are expressed as means ± standard deviations. There were no statistically significant differences between the groups in gender, ambient temperature, threshold temperature, time of vasoconstriction, or end-tidal halothane concentration.

fants, but the magnitude of this increase was not quantified.

Intravenous fluids were administered at a rate of 10–15 $ml \cdot kg^{-1} \cdot h^{-1}$; vasoconstrictor medications were never administered. No blood transfusions were required during surgery, and no patient developed hypertension or bradycardia at the time of thermoregulatory vasoconstriction.

Mean skin-surface temperature, ambient temperature, halothane concentration, age, and weight in patients given caudal analgesia and penile nerve blocks were similar. The central-temperature thresholds for vasoconstriction also were similar: 35.9 ± 0.5° C *versus* 35.7 ± 0.5° C (*P* not significant) (table 2).

Discussion

During halothane anesthesia, the central-temperature thermoregulatory threshold for vasoconstriction was similar in pediatric patients ranging from 5 to 50 kg in weight. These data are consistent with our previous report that the thresholds during isoflurane anesthesia were similar in children and infants of different sizes.[2]

MAC is higher in infants and small children than in older children and adults.[8] We did not increase end-tidal halothane concentrations in the smallest (and youngest) patients because there is no *a priori* reason to expect that age-related alteration in MAC and thermoregulatory inhibition are similar. Had we provided similar anesthetic

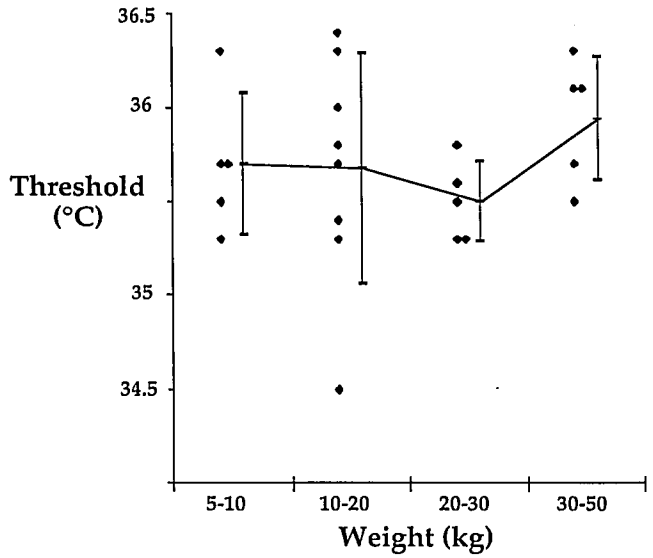

FIG. 1. The central thermoregulatory threshold in 23 healthy children and infants undergoing abdominal surgery with halothane anesthesia. The threshold was defined as the tympanic membrane temperature when the forearm − fingertip skin-temperature gradient first exceeded 4° C. Differences between the groups were not statistically significant.

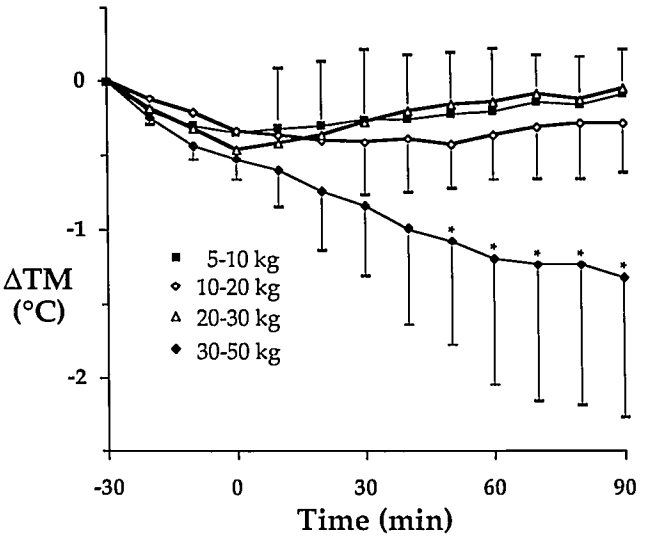

FIG. 2. All patients became progressively hypothermic following induction of general anesthesia. After significant peripheral vasoconstriction was observed (defined as time = zero), central temperature continued to decrease in patients weighing more than 30 kg but remained constant or increased slightly in the others. Temperatures in the 30–50-kg group were significantly different from each of the others after 40 elapsed minutes.

TABLE 2. Morphometric Data and Summary of Results in Patients Anesthetized with Halothane and Given Caudal Analgesia or Penile Nerve Block

	Weight (kg)	Age (yr)	Ambient (° C)	TM (° C)	Time (min)	Skin (° C)
Caudal (n = 7)	18.1 ± 3.6	4.0 ± 0.9	20.1 ± 0.2	35.9 ± 0.5	46 ± 17	33.2 ± 0.4
Penile block (n = 6)	20.5 ± 4.4	4.4 ± 0.8	20.2 ± 0.9	35.7 ± 0.5	68 ± 34	33.9 ± 0.2*

The central temperature thermoregulatory threshold for vasoconstriction is indicated in the column marked TM (° C). Mean skin-surface temperatures at the time of vasoconstriction are labeled Skin (° C). Values are expressed as means ± standard deviations. There were no statistically significant differences between the groups in ambient temperature, threshold temperature, time of vasoconstriction, or end-tidal halothane concentration.

* Skin temperatures were statistically, but not clinically, significantly different ($P < 0.05$).

potency in each group, inhibition of thermoregulatory vasoconstriction would have been greater in infants and small children than in older patients.

Central temperature thresholds in these pediatric patients (≈35.7° C) were higher than those we reported previously in adults given halothane anesthesia (34.4 ± 0.2° C).[3] However, the adults received 0.86% halothane, whereas infants and children in this study required only ≈0.6% because surgical pain was prevented by caudal anesthesia. During isoflurane anesthesia in humans, the vasoconstriction threshold is linearly decreased by increasing the end-tidal concentration of isoflurane: Threshold (°C) = 37.1 − 3.1 · [isoflurane].[4] (Inhibition of sweating also is a linear function of isoflurane concentration.[9]) It is thus likely that inhibition of thermoregulatory responses is proportional to halothane concentration. Supporting this possibility is our finding that central thermoregulatory thresholds in these infants and children were only slightly higher than would be predicted from a simple linear extrapolation from the threshold reported in adults and a "normal" unanesthetized temperature near 37° C. More importantly, thresholds in our current study were ≈1° C higher than in our previous study,[2] despite administration of a higher MAC-corrected anesthetic concentration (0.6% halothane *vs.* 0.8% isoflurane). A similar difference in vasoconstriction threshold during halothane and isoflurane anesthesia was previously observed in adult patients.[3,4]

After vasoconstriction, central temperature continued to decrease in children weighing more than 30 kg but remained constant or increased slightly in the others. A central temperature "plateau" was observed in these patients, despite large surgical incisions, relatively cool operating rooms, and the absence of active warming. This plateau might have resulted from decreased heat loss to the environment, increased heat production, or altered distribution of heat within the body.

We have previously demonstrated that thermoregulatory vasoconstriction in anesthetized[10] and unanesthetized[11] adults minimally decreases cutaneous heat loss. The ability of vasoconstriction to decrease heat loss to the environment in infants and children remains to be tested, but, *per se,* is unlikely to provide a sufficient decrement in heat loss to explain the observed central temperature plateau in patients weighing less than 30 kg.

Nonshivering thermogenesis can double heat production in unanesthetized infants[12] but is probably of little consequence in children and adults.[13,14] (Nonshivering thermogenesis does not increase heat production in anesthetized adults.[15]) The increase in minute ventilation required to maintain constant end-tidal carbon dioxide in the infants weighing less than 10 kg indicates that nonshivering thermogenesis may have increased metabolic heat production at that time. However, a similar increase was not required in the 20–30-kg patients, suggesting that nonshivering thermogenesis is not the sole etiology of the observed central temperature plateau.

The remaining potential etiology for the central temperature plateau in patients weighing less than 30 kg is altered distribution of heat within the body. We have previously demonstrated in adults that the initial hypothermia following induction of epidural[16] and general[17] anesthesia results from redistribution of heat within the body. Vasoconstriction cannot "push" heat from the periphery to the core (heat flowing *up* a temperature gradient would violate the Second Law of Thermodynamics). However, peripheral vasoconstriction during anesthesia may reestablish the normal central-to-peripheral thermal gradient by limiting heat transfer within the body. With vasoconstriction, metabolic heat (which is produced mostly centrally) might be constrained to the relatively small central compartment, allowing central temperature to plateau or even increase.§ Similar constraint of metabolic heat likely causes the relative hyperthermia observed in children requiring limb tourniquets for orthopedic surgery.[18]

Although we did not quantify the specific contributions of peripheral vasoconstriction, metabolic heat production, and altered distribution of body heat, our data suggest

§ Unpublished data in adults indicate that thermoregulatory vasoconstriction prevents further central hypothermia even when cutaneous heat loss far exceeds metabolic heat production.

that intraoperative thermoregulatory defenses in infants and small children are more effective than in adult patients.

Thermoregulatory responses in anesthetized patients resemble those of individuals in a thermoneutral environment. Specifically, there is an absence of tonic active thermoregulatory vasoconstriction (in arteriovenous shunts located in the fingers, toes, nose, *etc.*) and an absence of active vasodilation (in capillaries covering the remainder of the body).[19] In a thermoneutral environment, only ≈10% of cardiac output traverses the skin surface. (In contrast, as much as 7.5 l/min flows through the top millimeter of skin during heat stress.[20]) It is thus not surprising that vasoconstriction in arteriovenous shunts produced little hemodynamic change because only a small fraction of total cardiac output is affected. Although small differences may have been obscured by autonomic responses to endotracheal intubation, these data suggest that thermoregulatory vasoconstriction does not produce clinically important hemodynamic consequences. Our current results are consistent with those observed previously in anesthetized adults.[3]

Epidural analgesia decreases efferent sympathetic tone[21,22] (which promotes redistribution hypothermia[16]) and inhibits afferent input to the brain (which increases thermal comfort[23] and prevents pain). Nonetheless, central thermoregulatory responses remain intact during conduction analgesia as indicated by vasoconstriction (above the level of the block) and shivering.[24] Combined with general anesthesia however, the thermoregulatory consequences of caudal or epidural analgesia are probably less important. Volatile anesthetics are direct vasodilators,[25] and additionally decrease efferent sympathetic tone *via* central thermoregulatory inhibition.[1-4] It is thus unlikely that regional anesthesia significantly enhances cutaneous heat loss or the redistribution hypothermia produced by general anesthesia.[17] Consistent with this reasoning, the time required to become sufficiently hypothermic to trigger vasoconstriction was only slightly less in patients given caudal analgesia than penile nerve blocks.

More importantly, regional analgesia may alter thermoregulatory responses by preventing cutaneous thermal input from the anesthetized region.[16,23] However, cutaneous input contributes far less than central temperature to overall thermoregulatory responses when skin temperature is moderate and relatively constant.[26] It is thus not surprising that vasoconstriction thresholds during halothane anesthesia were unaffected by caudal analgesia.

Painful surgical stimulation may increase vasoconstriction thresholds by reducing the effective anesthetic level.[27] The extent to which stimulation contributes to thermoregulatory responses remains unclear because pain was prevented both in patients receiving caudal analgesia and in those given penile nerve blocks. Consequently, the thresholds defined in these pediatric patients should be cautiously compared with those previously reported in adults because regional analgesia was not used to block surgical pain in the adult studies. However, vasoconstriction thresholds are similar in adult surgical patients[4] and in volunteers given isoflurane anesthesia without surgery,[10] suggesting that surgical stimulation *per se* may not increase the vasoconstriction threshold in patients with clinically adequate anesthesia (*e.g.* blood pressure and heart rate within 20% of control values).

In summary, we first determined the central-temperature thermoregulatory threshold for vasoconstriction in 23 infants and children given ≈0.6% halothane and caudal anesthesia for abdominal surgery. Patients were prospectively assigned to one of four weight groups: 5–10, 10–20, 20–30, and 30–50 kg. Thresholds were similar (≈35.7° C) in each study group, suggesting that thermoregulatory responses to halothane anesthesia are similar in infants and children of differing weights. However, they were higher than expected based on the previously reported thresholds in pediatric patients given isoflurane anesthesia. After peripheral vasoconstriction, central temperature continued to decrease in patients weighing more than 30 kg but remained constant or increased slightly in the others. These data suggest that thermoregulatory responses are more effective in infants and small children than in older children or adults. We then determined vasoconstriction thresholds in 13 children undergoing hypospadias repairs who were randomly assigned to receive caudal analgesia or penile nerve block after induction of halothane anesthesia. The thresholds were 35.9 ± 0.5° C in the caudal group and 35.7 ± 0.5° C in patients given a penile nerve block (*P* not significant), indicating that caudal anesthesia *per se* has little effect on the thermoregulatory threshold for vasoconstriction during halothane anesthesia.

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