Anesthesiology
82:870–876, 1995

Thermoregulatory Vasoconstriction Impairs Active Core Cooling

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Background: **Many clinicians now consider hypothermia indicated during neurosurgery. Active cooling often will be required to reach target temperatures < 34°C sufficiently rapidly and nearly always will be required if the target temperature is 32°C. However, the efficacy even of active cooling might be impaired by thermoregulatory vasoconstriction, which reduces cutaneous heat loss and constrains metabolic heat to the core thermal compartment. The authors therefore tested the hypothesis that the efficacy of active cooling is reduced by thermoregulatory vasoconstriction.**

Methods: **Patients undergoing neurosurgical procedures with hypothermia were anesthetized with either isoflurane/nitrous oxide (n = 13) or propofol/fentanyl (n = 13) anesthesia. All were cooled using a prototype forced-air cooling device until core temperature reached 32°C. Core temperature was measured in the distal esophagus. Vasoconstriction was evaluated using forearm minus fingertip skin-temperature gradients. The core temperature triggering a gradient of 0°C identified the vasoconstriction threshold.**

Results: **In 6 of the 13 patients given isoflurane, vasoconstriction (skin-temperature gradient = 0°C) occurred at a core temperature of 34.4 ± 0.9°C, 1.7 ± 0.5 h after induction of anesthesia. Similarly, in 7 of the 13 patients given propofol, vasoconstriction occurred at a core temperature of 34.5 ± 0.9°C, 1.6 ± 0.6 h after induction of anesthesia. In the remaining patients, vasodilation continued even at core temperatures of 32°C. Core cooling rates were comparable in each anesthetic group. However, patients in whom vasodilation was maintained cooled fastest. Patients in whom vasoconstriction occurred required nearly an hour longer to reach core temperatures of 33°C and 32°C than did those in whom vasodilation was maintained ($P < 0.01$).**

Conclusions: **Vasoconstriction did not produce a full core temperature "plateau," because of the extreme microenvironment provided by forced-air cooling. However, it markedly decreased the rate at which hypothermia developed. The ≈1-h delay in reaching core temperatures of 33°C and 32°C could be clinically important, depending on the target temperature and the time required to reach critical portions of the operation. (Key words: Brain protection. Forced-air. Neurosurgery. Temperature, hypothermia. Thermoregulation. Vasoconstriction.)**

MANY clinicians believe hypothermia is indicated during neurosurgery, with target core temperatures between 34°C and 32°C being used in selected cases.[1] Core temperatures ≤ 34°C usually will not develop sufficiently rapidly only from passive exposure of patients to a cool operating room environment.[2] Consequently, a forced-air cooling system was recently developed to facilitate rapid induction of core hypothermia during neurosurgery.[3]

We were concerned, however, that efficacy even of active cooling would be impaired by intraoperative thermoregulatory vasoconstriction, which typically is triggered near 34–35°C.[4–8] Specifically, we thought that the resulting reduction in cutaneous heat loss[9] and constraint of metabolic heat to the core thermal compartment[10] might impair our ability to induce further central hypothermia. We therefore tested the hypothesis that the efficacy of active cooling is reduced by thermoregulatory vasoconstriction.

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Received from the Thermoregulation Research Laboratory, University of California, San Francisco, San Francisco, California, and the Department of Anesthesia and General Intensive Care, University of Vienna, Austria. Submitted for publication July 21, 1994. Accepted for publication December 15, 1994. Supported by National Institutes of Health grant GM49670, the Max Kade and Joseph Drown Foundations, and Augustine Medical, Inc. Dr. Sessler does not consult for, accept honoraria from, or own stock or stock options in any anesthesia-related company.

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Methods

With Institutional Review Board approval and informed consent, we studied 26 ASA Physical Status 1–3 patients undergoing elective neurosurgery. They were sequentially assigned to one of two anesthetic regimens: (1) isoflurane and nitrous oxide (n = 13) or (2) propofol and fentanyl (n = 13).

Protocol

On arrival to the operating suite, 10 ml/kg of unwarmed intravenous fluid was administered. Most patients were given 50 mg prednisolone the morning of surgery. Without premedication, anesthesia was induced in the first group of patients with fentanyl (up to 250 μg) and sodium thiopental (4–6 mg/kg). The other patients were given a comparable amount of fentanyl and propofol (2–3 mg/kg). Intubation of the trachea was facilitated by administration of vecuronium bromide (0.1 mg/kg). Mechanical ventilation was maintained with a circle system having a fresh gas flow of 6 l/min, adjusted to maintain end-tidal P_{CO_2} near 35 mmHg. No airway heating or humidification was provided.

In patients assigned to inhalational anesthesia, anesthesia was maintained with 70% N_2O and isoflurane at an end-tidal concentration of 0.5–0.8%. No additional thiopental or opioid was administered. In patients assigned to total intravenous anesthesia, anesthesia was maintained with propofol (6–8 $mg \cdot kg^{-1} \cdot h^{-1}$) and fentanyl (4 $\mu g \cdot kg^{-1} \cdot h^{-1}$) without nitrous oxide.

Supplemental vecuronium was administered as needed to maintain one or two twitches in response to supramaximal stimulation of the ulnar nerve at the wrist. At least 10 $ml \cdot kg^{-1} \cdot h^{-1}$ of fluid was given intravenously, and blood products were replaced to maintain the hematocrit between 25–32%. Fluids were not warmed. Per surgical routine, most patients were given 1 g/kg mannitol shortly after induction of anesthesia.

The patients were covered with a single layer of surgical draping during induction of anesthesia. Starting immediately after induction of anesthesia, active cutaneous cooling was initiated using a prototype forced-air system (Augustine Medical, Eden Prarie, MN).[3] This device provides 1,000 l/min air at 14–15°C into a full-body disposable convective cover. The cover was positioned directly above the patient's anterior skin surface and covered with a single cotton blanket. The arm used to test vasoconstriction was excluded from the forced-air cover. Active cooling continued until core temperature approached 32°C. When surgery was complete, patients were actively rewarmed, again using forced air (Bair Hugger, Augustine Medical).[11,12]

Monitoring

Core temperature, before induction of anesthesia, was measured at the tympanic membrane. The aural probe was inserted until patients felt the thermocouple touch the tympanic membrane; appropriate placement was confirmed when they easily detected a gentle rubbing of the attached wire. The probe was then taped in place, the aural canal occluded with cotton, and the external ear covered with a gauze pad. Tympanic membrane temperatures correlate well with distal esophageal temperatures during anesthesia.[6,13] After induction of anesthesia, core temperature was recorded from the distal esophagus. Mean skin temperature was calculated from four sites: $0.3(T_{chest} + T_{arm}) + 0.2(T_{thigh} + T_{calf})$.[14]

Fingertip blood flow was evaluated using forearm minus fingertip skin-surface temperature gradients; there is an excellent correlation between skin-temperature gradients and volume plethysmography.[15] The gradients were recorded from an arm not having an intravenous cannula or blood pressure cuff. A skin temperature gradient of 0°C coincides with the core temperature plateau[10]; consequently, we considered this gradient to indicate significant vasoconstriction. (A gradient of 4°C, which we have used previously, indicates intense vasoconstriction. However, in this study, we were more interested in the core temperature plateau that starts when the gradient reaches 0°C.) The distal esophageal temperature triggering significant vasoconstriction identified the thermoregulatory threshold. To facilitate comparison with previous studies,[4–6] we also recorded the core temperature triggering a skin-temperature gradient of 4°C (intense constriction). All temperatures were measured using Yellow Springs Instruments thermistors (Yellow Springs, OH).

Heart rate was monitored continuously using three-lead electrocardiography. Blood pressure was determined oscillometrically at 5-min intervals. Respiratory gas concentrations were quantified using a calibrated end-tidal gas analyzer (Dräger, Luebeck, Germany). All other data were recorded at 15-min intervals, starting immediately before induction of anesthesia (control values).

Data Analysis

Morphometric data and core temperatures were compared using two-tailed, unpaired *t* tests. Isoflurane

concentration, propofol dose, vasoconstriction threshold, time of constriction, time required for patients to reach target core temperatures, mean skin temperature, heart rate, and arterial blood pressures also were compared using two-tailed, unpaired t tests. Tumor locations were compared using Fisher's exact test. All values are expressed as mean ± SD; differences were considered significant when $P < 0.01$.

Results

No complications specifically related to deliberate hypothermia (*e.g.,* cardiac arrhythmias, hemodynamic instabilities) were detected. Age, gender, weight, and height did not differ significantly in the two anesthesia groups (table 1). We administered isoflurane at an end-tidal concentration of 0.6 ± 0.2%; propofol was administered at a rate of 7.5 ± 1.2 $mg \cdot kg^{-1} \cdot h^{-1}$. Specific neurosurgical procedures (*e.g.,* location) and indications (tumor *vs.* aneurysm) were comparable in the patients given each type of anesthesia.

Vasoconstriction was present in most patients in the immediate preoperative period, but vasodilation was observed in every case shortly after induction of anesthesia. In 6 of 13 patients given isoflurane, vasoconstriction (skin-temperature gradient = 0°C) was observed at a core temperature of 34.4 ± 0.9°C and a mean skin temperature of 28.9 ± 1.2°C, 1.7 ± 0.5 h after induction of anesthesia. Similarly, in 7 of the 13 patients given propofol, vasoconstriction occurred at a core temperature of 34.5 ± 0.9°C and a mean skin temperature of 28.7 ± 1.4°C, 1.6 ± 0.6 h after induction of anesthesia. Patients reaching a gradient of 0°C proceeded to a gradient of 4°C in 44 ± 12 min, when the core temperature decreased 0.4 ± 0.2°C. Core cooling rates were comparable in each anesthetic group (fig. 1).

Patients remaining vasodilated cooled faster than those in whom vasoconstriction was observed. Consequently, patients given both types of anesthesia were subdivided on the basis of vasomotor responses. Specifically, we compared patients who at some time reached a gradient ≥ 0°C (which we considered evidence of vasoconstriction) with those in whom vasoconstriction was never detected. Similar amounts of fluid and blood were given to the patients in each group, and ambient temperatures did not differ significantly. Morphometric characteristics also were similar (table 2). Preoperative and immediate postoperative blood pressures and heart rates did not differ significantly in the two groups.

Table 1. Morphometric Characteristics and Vasoconstriction Thresholds by Anesthetic Type

	Isoflurane/ Nitrous Oxide	Propofol/ Fentanyl
Age (yr)	38 ± 11	39 ± 10
Male/n	6/13	7/13
Weight (kg)	56 ± 12	68 ± 11
Height (cm)	168 ± 10	171 ± 7
Threshold (°C)	34.4 ± 0.9	34.5 ± 0.9
Time to threshold (h)	1.7 ± 0.5	1.6 ± 0.6
Skin temperature at threshold (°C)	28.9 ± 1.2	28.7 ± 1.4

Values are mean ± SD.

Morphometric characteristics, vasoconstriction threshold (skin temperature gradient = 0°C), time to vasoconstriction, and mean skin temperature at vasoconstriction did not differ significantly in the patients given each type of anesthesia. Patients who vasoconstricted and those remaining vasodilated are both included

Core cooling rates in the patients in whom vasoconstriction was observed and those in whom it was not (fig. 2), and times required to reach core temperatures of 33°C and 32°C differed significantly in the two groups (table 3). Intraoperative vasoconstriction was not correlated with the indication for surgery (tumor *vs.* aneurysm) or the location within the brain (table 4).

Discussion

Core body temperature normally is precisely regulated by effective thermoregulatory responses, which are initiated by small thermal perturbations. Heat stress provokes sweating[16] and active precapillary vasodilation[17]; cold stress initiates, in turn, arteriovenous shunt vasoconstriction,[18] nonshivering thermogenesis (in infants),[19,20] and shivering.[21] The interthreshold range is defined by core temperatures between the sweating and vasoconstriction thresholds *not* triggering autonomic thermoregulatory responses.[22] This range usually is only ≈0.2°C,[23] but typical doses of all general anesthetics so far tested increase the range 10–20-fold. Volatile anesthetics increase the sweating threshold,[24,25] but the interthreshold range is augmented mostly by a reduction in the vasoconstriction threshold.[4–8] In contrast, propofol increases the interthreshold range by reducing the vasoconstriction threshold without much increases the sweating threshold.[8]

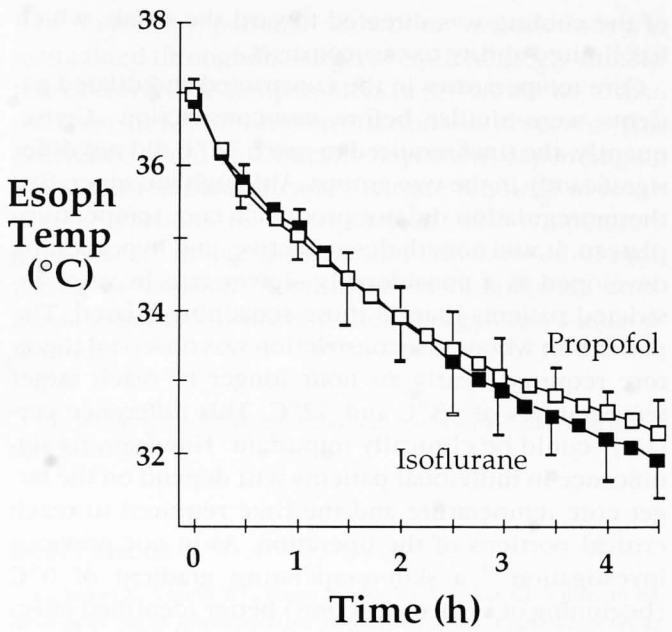

Fig. 1. Core cooling rates were comparable in patients given isoflurane/nitrous oxide and propofol/fentanyl anesthesia. (Patients who were vasoconstricted and those who remained vasodilated are included.) Results are presented as mean ± SD.

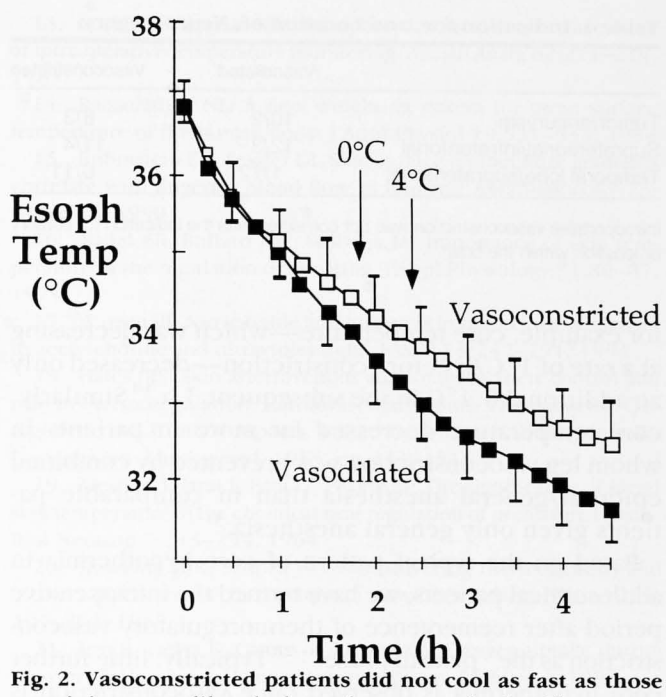

Fig. 2. Vasoconstricted patients did not cool as fast as those remaining vasodilated throughout anesthesia. The arrow marked "0°C" identifies the mean time at which significant thermoregulatory vasoconstriction was first detected (skin-temperature gradient = 0°C). For comparison, the second arrow indicates the time at which gradients reached 4°C (intense vasoconstriction). Temperatures in the two groups differed significantly at all times after 2 h of anesthesia ($P < 0.01$). After 4.5 h, the temperatures differed by 0.8°C. (Patients given both types of anesthesia are included, divided based on their vasomotor responses.) Results are presented as mean ± SD.

Based on previous studies[5,6,8,26] and the age of the patients,[27] we expected the relatively low anesthetic doses given our patients to reduce the vasoconstriction threshold 2–3°C. Consistent with this experience, patients who vasoconstricted did so at core temperatures near 34.5°C. But surprisingly, vasoconstriction was not observed in half the patients in each anesthetic group, even at core temperatures of 32°C. Although thermoregulatory responses may be especially impaired in patients undergoing neurosurgery, the absence of concurrent non-neurosurgical control patients does not permit us to make any firm conclusion in this regard.

Table 2. Fluid Administration and Morphometric Characteristics by Vasomotor Response

	Vasoconstriction	Vasodilation
Fluid administration rate (L/h)	0.8 ± 0.2	0.9 ± 0.1
Age (yr)	36 ± 12	38 ± 11
Male/n	5/13	8/13
Weight (kg)	57 ± 11	65 ± 13
Height (cm)	170 ± 9	169 ± 11

Values mean ± SD.

Fluid administration rate and morphometric characteristics did not differ significantly in the patients in whom vasoconstriction was observed and in those remaining vasodilated throughout surgery.

Nonshivering thermogenesis probably contributes little in unanesthetized adults[28,29] and does not occur during anesthesia.[30] However, thermoregulatory vasoconstriction reduces cutaneous heat loss[9] and constrains metabolic heat to the core thermal compartment.[10] Consequently, vasoconstriction is effective in minimizing further core hypothermia. In one study,

Table 3. Time Required to Reach Core Temperature Targets

	Vasodilated	Vasoconstricted
Time to 34°C (h)	1.7 ± 0.4	2.3 ± 0.6
Time to 33°C (h)	2.6 ± 0.6	3.4 ± 0.6*
Time to 32°C (h)	3.6 ± 0.7	4.8 ± 0.8*

Values are mean ± SD.

The times required for vasodilated and vasoconstricted patients to reach core temperatures of 33 and 32°C differed significantly (*, $P < 0.01$). Patients given both types of anesthesia are included, divided based on their vasomotor responses.

Table 4. Indication for, and Location of, Neurosurgery

	Vasodilated	Vasoconstricted
Tumor/aneurysm	10/2	8/3
Supratentorial/infratentorial	12/3	11/4
Temporal lobe/supratentorial	1/12	0/11

Intraoperative vasoconstriction was not correlated with the indication for surgery or location within the brain.

for example, core temperature—which was decreasing at a rate of 1°C/h before constriction—decreased only an additional 0.4°C in the subsequent 3 h.[10] Similarly, core temperature decreased far more in patients in whom leg vasoconstriction was prevented by combined epidural/general anesthesia than in comparable patients given only general anesthesia.[31]

Based on the typical pattern of core hypothermia in adult surgical patients, we have termed the intraoperative period after reemergence of thermoregulatory vasoconstriction as the "plateau phase." [22] Typically, little further core hypothermia is observed once vasoconstriction is triggered in adults.[10,31] However, observed core temperature changes following vasoconstriction depend on a number of factors, including environmental temperature[2,32] and the patient's age and morphometric characteristics.[33] Core temperature, for example, may increase in hypothermic infants once vasoconstriction (and perhaps nonshivering thermogenesis) is triggered.# Similarly, infants in a warm environment become hyperthermic when orthopedic tourniquets constrain metabolic heat to the core thermal compartment.[34]

As might be expected, core temperatures were comparable in all patients before vasoconstriction. In contrast to the typical adult pattern, patients in whom vasoconstriction was observed continued to become hypothermic during neurosurgery. Cutaneous blood traverses arteriovenous shunts perfusing distal tissues[18] and capillaries that supply the remaining skin.[35] During the initial phase of anesthesia, shunt flow is near maximal, whereas capillary flow remains low.[36] During cold stress, vasoconstriction decreases shunt flow more than tenfold with a smaller decrease in capillary flow.[37] (In contrast, heat stress increases capillary flow to as much as 7.5 l/min.[38,39]) Core cooling in our patients continued despite vasoconstriction, in part because temperature in the microenvironment under the forced-air cooling cover was quite cold. Equally important, much of the cooling was directed toward the trunk, which has limited ability to vasoconstrict.

Core temperatures in the constricted and dilated patients were similar before vasoconstriction. Consequently, the time required to reach 34°C did not differ significantly in the two groups. Although intraoperative thermoregulation did not produce a core temperature plateau, it was nonetheless effective, and hypothermia developed at a considerably slower rate in vasoconstricted patients than in those remaining dilated. The patients in whom vasoconstriction was observed therefore required nearly an hour longer to reach target temperatures of 33°C and 32°C. This difference certainly could be clinically important. However, its significance in individual patients will depend on the target core temperature and the time required to reach critical portions of the operation. As in our previous investigation,[10] a skin-temperature gradient of 0°C (beginning of vasoconstriction) better identified effective thermoregulation than a gradient of 4°C (intense constriction).

Thermoregulation may be the most common trigger for intraoperative peripheral vasoconstriction, but it is not the only one. Inadequate anesthesia, α-adrenergic drugs, and vascular volume depletion also cause constriction. Furthermore, thermoregulation and volume depletion synergistically increase vasoconstriction.[40] Patients undergoing neurosurgery—in whom fluid administration is often restricted—are thus at particular risk for hypovolemia-induced vasoconstriction. Intraoperative vasoconstriction, unresponsive to additional anesthetic, may be relieved by administration of fluid.

Because vasoconstriction significantly increases the time required to reach therapeutic target core temperatures, clinicians may find it helpful to monitor cutaneous vasomotion. The decrease in arteriovenous shunt flow is far more dramatic than the reduction in capillary flow. Consequently, fingers and toes are optimal monitoring sites. Thermoregulatory vasoconstriction can be monitored using a variety of methods, including volume plethysmography,[41] laser Doppler flowmetry,[42,43] and skin-temperature gradients.[4–6] Forearm minus fingertip skin-temperature gradients have the advantage of being inexpensive and easy to implement. There is an excellent correlation between skin-temperature gradients and volume plethysmography (which generally is considered the "gold standard").[15] Calf minus toe gradients also are reliable.[6] Gradients will be accurate, however, only if measured on an extremity adequately shielded from active cooling.

Bissonnette B: Unpublished data. 1991.

In summary, patients in whom vasodilation was maintained throughout surgery required nearly an hour less to reach core temperatures of 33°C and 32°C than did those in whom vasoconstriction was observed. This difference could be clinically important, depending on the target temperature and the time required to reach critical portions of the operation. Skin-temperature gradients are an inexpensive and easy method of evaluating vasomotion. Administration of sufficient anesthesia usually will prevent thermoregulatory vasoconstriction.

The authors thank Gerhard Pavecic and Gepa-Med, Austria, for their assistance.

References

1. Baker K, Young WL, Stone JG, Kader A, Baker CJ, Solomon RA: Deliberate mild intraoperative hypothermia for craniotomy. ANESTHESIOLOGY 81:361–367, 1994

2. Morris RH: Operating room temperature and the anesthetized, paralyzed patient. Surgery 102:95–97, 1971

3. Lanier WL, Iaizzo PA, Murray MJ: The effects of convective cooling and rewarming on systemic and central nervous system physiology in isoflurane-anesthetized dogs. Resuscitation 23:121–136, 1992

4. Sessler DI, Olofsson CI, Rubinstein EH, Beebe JJ: The thermoregulatory threshold in humans during halothane anesthesia. ANESTHESIOLOGY 68:836–842, 1988

5. Sessler DI, Olofsson CI, Rubinstein EH: The thermoregulatory threshold in humans during nitrous oxide-fentanyl anesthesia. ANESTHESIOLOGY 69:357–364, 1988

6. Støen R, Sessler DI: The thermoregulatory threshold is inversely proportional to isoflurane concentration. ANESTHESIOLOGY 72:822–827, 1990

7. Washington DE, Sessler DI, McGuire J, Hynson J, Schroeder M, Moayeri A: Painful stimulation minimally increases the thermoregulatory threshold for vasoconstriction during enflurane anesthesia in humans. ANESTHESIOLOGY 77:286–290, 1992

8. Leslie K, Sessler DI, Bjorksten A, Ozaki M, Matsukawa T, Schroeder M, Lin S: Propofol causes a dose-dependent decrease in the thermoregulatory threshold for vasoconstriction, but has little effect on sweating. ANESTHESIOLOGY 81:363–360, 1994

9. Sessler DI, Hynson J, McGuire J, Moayeri A, Heier T: Thermoregulatory vasoconstriction during isoflurane anesthesia minimally decreases heat loss. ANESTHESIOLOGY 76:670–675, 1992

10. Belani K, Sessler DI, Sessler AM, Schroeder M, McGuire J, Washington D, Moayeri A: Leg heat content continues to decrease during the core temperature plateau in humans. ANESTHESIOLOGY 78: 856–863, 1993

11. Kurz A, Kurz M, Poeschl G, Faryniak B, Redl G, Hackl W: Forced-air warming maintains intraoperative normothermia better than circulating-water mattresses. Anesth Analg 77:89–95, 1993

12. Giesbrecht GG, Ducharme MB, McGuire JP: Comparison of forced-air patient warming systems for perioperative use. ANESTHESIOLOGY 80:671–679, 1994

13. Cork RC, Vaughan RW, Humphrey LS: Precision and accuracy of intraoperative temperature monitoring. Anesth Analg 62:211–214, 1983

14. Ramanathan NL: A new weighting system for mean surface temperature of the human body. J Appl Physiol 19:531–533, 1964

15. Rubinstein EH, Sessler DI: Skin-surface temperature gradients correlate with fingertip blood flow in humans. ANESTHESIOLOGY 73: 541–545, 1990

16. Nadel ER, Bullard RW, Stolwijk JA: Importance of skin temperature in the regulation of sweating. J Appl Physiology 31:80–87, 1971

17. Warren JB: Nitric oxide and human skin blood flow responses to acetylcholine and ultraviolet light. FASEB J 8:247–251, 1994

18. Hales JRS: Skin arteriovenous anastomoses, their control and role in thermoregulation, Cardiovascular Shunts: Phylogenetic, Ontogenetic and Clinical Aspects. Edited by Johansen K, Burggren W. Copenhagen, Munksgaard, 1985, pp 433–451

19. Mestyan J, Jarai I, Bata G, Fekete M: The significance of facial skin temperature in the chemical heat regulation of premature infants. Biol Neonate 7:243–254, 1964

20. Dawkins MJR, Scopes JW: Non-shivering thermogenesis and brown adipose tissue in the human new-born infant. Nature 206: 201–202, 1965

21. Just B, Delva E, Camus Y, Lienhart A: Oxygen uptake during recovery following naloxone. ANESTHESIOLOGY 76:60–64, 1992

22. Sessler DI: Perianesthetic thermoregulation and heat balance in humans. FASEB J 7:638–644, 1993

23. Lopez M, Sessler DI, Walter K, Emerick T, Ozaki M: Rate and gender dependence of the sweating, vasoconstriction, and shivering thresholds in humans. ANESTHESIOLOGY 80:780–788, 1994

24. Lopez M, Ozaki M, Sessler DI, Valdes M: Physiological responses to hyperthermia during epidural anesthesia and combined epidural/enflurane anesthesia in women. ANESTHESIOLOGY 78:1046–1054, 1993

25. Washington D, Sessler DI, Moayeri A, Merrifield B, Prager M, McGuire J, Belani K, Hudson S, Schroeder M: Thermoregulatory responses to hyperthermia during isoflurane anesthesia in humans. J Appl Physiol 74:82–87, 1993

26. Ozaki M, Sessler DI, Suzuki H, Ozaki K, Tsunoda C, Starashi K: Nitrous oxide decreases the threshold for vasoconstriction less than sevoflurane or isoflurane. Anesth Analg (in press)

27. Kurz A, Plattner O, Sessler DI, Huemer G, Redl G, Lackner F: The threshold for thermoregulatory vasoconstriction during nitrous oxide/isoflurane anesthesia is lower in elderly than young patients. ANESTHESIOLOGY 79:465–469, 1993

28. Jessen K: An assessment of human regulatory nonshivering thermogenesis. Acta Anaesthesiol Scand 24:138–143, 1980

29. Jessen K, Rabøl A, Winkler K: Total body and splanchnic thermogenesis in curarized man during a short exposure to cold. Acta Anaesthesiol Scand 24:339–344, 1980

30. Hynson JM, Sessler DI, Moayeri A, McGuire J: Absence of nonshivering thermogenesis in anesthetized humans. ANESTHESIOLOGY 79: 695–703, 1993

31. Joris H, Ozaki M, Sessler DI, Hardy AF, Lamay M, McGuire J, Blanchard D, Schroeder M, Moayeri A: Epidural anesthesia impairs both central and peripheral thermoregulatory control during general anesthesia. ANESTHESIOLOGY 80:268–277, 1994

32. Morris RH: Influence of ambient temperature on patient temperature during intraabdominal surgery. Ann Surg 173:230–233, 1971

33. Bissonnette B, Sessler DI: Thermoregulatory thresholds for vasoconstriction in pediatric patients anesthetized with halothane or halothane and caudal bupivacaine. ANESTHESIOLOGY 76:387–392, 1992

34. Bloch EC, Ginsberg B, Binner RA, Sessler DI: Limb tourniquets and central temperature in anesthetized children. Anesth Analg 74: 486–489, 1992

35. Rowell LB: Active neurogenic vasodilation in man, Vasodilatation. Edited by Vanhoutte PM, Leusen I. New York, Raven, 1981, pp 1–17

36. Rowell LB: Cardiovascular aspects of human thermoregulation. Circ Res 52:367–379, 1983

37. Sessler DI, Moayeri A, Støen R, Glosten B, Hynson J, McGuire J: Thermoregulatory vasoconstriction decreases cutaneous heat loss. ANESTHESIOLOGY 73:656–660, 1990

38. Detry JR, Brengelmann GL, Rowell LB, Wyss C: Skin and muscle components of forearm blood flow in directly heated resting man. J Appl Physiol 32:506–511, 1972

39. Rowell LB, Brengelmann GL, Murray JA: Cardiovascular responses to sustained high skin temperature in resting man. J Appl Physiol 27:673–680, 1969

40. Nadel ER, Fortney SM, Wenger CB: Effect of hydration state on circulatory and thermal regulations. J Appl Physiol 49:715–721, 1980

41. Burch GE, Cohn AE, Neumann C: A study by quantitative methods of the spontaneous variations in volume of the finger tip, toe tip, and postero-superior portion of the pinna of resting normal white adults. Am J Physiol 136:433–447, 1942

42. Carpenter RL, Kopacz DJ, Mackey DC: Accuracy of laser Doppler capillary flow measurements for predicting blood loss from skin incisions in pigs. Anesth Analg 68:308–311, 1989

43. Hales JRS, Stephens FRN, Fawcett AA, Daniel K, Sheahan J, Westerman RA, James SB: Observations on a new non-invasive monitor of skin blood flow. Clin Exp Pharmacol Physiol 16:403–415, 1989

Anesthesiology
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