

Core Temperature Changes during N₂O Fentanyl and Halothane/O₂ Anesthesia

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Hypothermia during surgery results from the interaction of three factors that depend on the depth and type of anesthesia: 1) decreased metabolic heat production, 2) increased heat loss to the environment from cutaneous vasodilation, surgical exposure, dry respiratory gases, and unwarmed intravenous fluids, and 3) reduced compensatory response (vasoconstriction, non-shivering thermogenesis, and shivering) due to hypothalamic suppression and the effects of muscle relaxants.¹ Although anesthetics are believed to inhibit normal thermoregulatory responses to cold, the magnitude of this effect is unknown.² Thermoregulatory inhibition is thought to be profound at surgical concentrations of anesthesia,¹ but, presumably, the magnitude of inhibition depends on both the choice of anesthetic and the dose administered.³ Patients who remain relatively warm (undergoing operations involving minimal heat loss to the environment) may reach a passive thermal steady state without activating thermoregulation.

Core temperature may be better maintained during lighter levels of anesthesia, *e.g.*, nitrous oxide (N₂O)/narcotic administration, than during the deeper levels usually produced by the potent inhalation agents.^{1,4} Alternatively, core temperatures may be similar during both anesthetics.^{5,6} Existing studies⁴⁻⁶ are difficult to interpret because of variability in patient selection, premedication, anesthetic management, and the extent of visceral exposure during surgery. These studies also are confounded by the inclusion of N₂O (which is known to

suppress the thermostatic setpoint⁷) in both narcotic and volatile anesthetic regimens. In the present study, the effects of halothane/O₂ and N₂O/fentanyl anesthesia on temperature regulation are compared in surgical patients experiencing minimal heat loss to the environment.

METHODS

With approval from the UCSF Committee on Human Subjects and written informed consent, we studied 16 ASA Physical Status I or II volunteers [age: 50 ± 16 yr (SD)] undergoing elective eye surgery scheduled to last at least 3 h. Nine of these subjects were male, and differences in average ages were not significant. Subjects had no history of fever, infection, dysautonomia, thyroid disease, obesity, or susceptibility to malignant hyperthermia. They were not premedicated, and arrived in the operating room clothed in a hospital gown and covered by a thin blanket. They remained covered with the blanket during induction of anesthesia and throughout the study. The surgical drapes were made of paper, and the patient's head was covered during surgery.

Anesthesia was induced with thiopental (4 mg/kg iv) and pancuronium (0.1 mg/kg iv), and the trachea was intubated. Patients were randomly assigned to an anesthetic regimen of halothane/O₂ or 70% N₂O/O₂/fentanyl. The inspired halothane concentration was adjusted to maintain arterial blood pressure at 85% of the pre-induction blood pressure value. A full anesthetic dose of halothane was maintained until the end of surgery, at which time tracheas were extubated while patients remained deeply anesthetized. Fentanyl was administered as an initial loading dose of 5–7 µg/kg, followed by an infusion of 2–3 µg · kg⁻¹ · h⁻¹. The infusion rate was determined by each patient's response to the loading dose; no further adjustments were made during the study period. Two patients given fentanyl also received propranolol (1–3 mg iv) when intraoperative blood pressures were 20% greater than the preinduction value.

Ventilation was controlled to maintain end-tidal CO₂ concentrations at 35–40 mmHg. Fresh gas flow was 5 l/min, and a Humid-Vent® artificial nose was added to the circuit between the circle and the endotracheal tube. Heating blankets were not used. Intravenous

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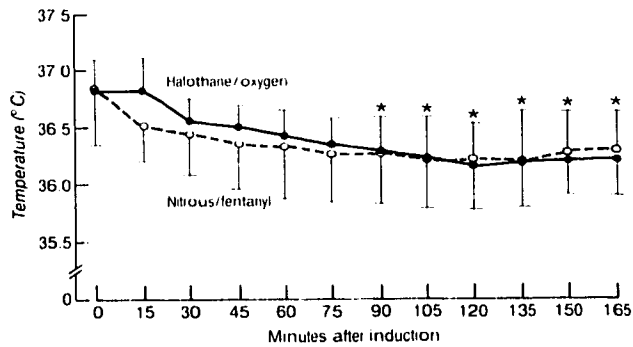


FIG. 1. Mean rectal temperatures (\pm SD) in patients who received N_2O /fentanyl (O, $n = 8$) or halothane/ O_2 (●, $n = 8$) anesthesia during eye surgery. Time zero is just prior to induction. Thermal steady state was reached after 2 h of anesthesia at $0.6^\circ C$ below control ($P < 0.05$). Temperatures at more than 90 min after induction (*) were significantly lower than at time zero in both groups. There is no statistically significant difference between the two curves.

crystalloid solutions were not warmed, but no patient received more than 250 ml/h (this amount will decrease body temperature $< 0.15^\circ C$ during the study period).

Rectal temperatures were measured before induction of anesthesia, and every 15 min thereafter with disposable Mon-a-Therm[®] thermocouples accurate to $0.1^\circ C$. The probes were inserted 10 cm into the rectum and allowed to equilibrate at least 4 min prior to the first reading.

We chose to monitor rectal, rather than esophageal, temperatures because: 1) most patients would object to a control esophageal temperature measurement prior to the induction of anesthesia, and 2) esophageal temperature measurements are accurate only when the probe position is maintained in the lower third of the esophagus.⁸ We avoided tympanic temperatures to prevent perforation of the tympanic membrane.⁹ When compared with tympanic membrane temperatures, the accuracy of rectal and esophageal temperatures is similar (in patients not subject to deliberate cooling).¹⁰

Patient temperatures at each measurement point were averaged within each anesthetic group. The two groups were compared using two-tailed Student's *t* tests. Changes within groups were evaluated with repeated-measures ANOVA and Dunnett's test. Differences were considered statistically significant when $P < 0.05$.

RESULTS

Rectal temperatures during induction and maintenance of anesthesia did not differ significantly between groups (fig. 1). The initial average temperature in patients given halothane was $36.8 \pm 0.3^\circ C$ ($n = 8$, SD),

and, in those given N_2O /fentanyl, $36.9 \pm 0.5^\circ C$ ($n = 8$, SD). In both groups, rectal temperatures decreased by $0.3^\circ C$ during the first 30 min, followed by a slow decline of $0.15^\circ C/h$ for 1.5 h.

Two hours after induction, the average rectal temperatures in the halothane group and in the N_2O /fentanyl group were each $36.2 \pm 0.4^\circ C$ (SD). Analysis of type II error, 2 h after induction, indicates a 75% chance that the true population means differed by $< 0.6^\circ C$, and a 90% chance that they differed by $< 1.0^\circ C$. Both temperatures represented a significant and parallel decrease from the respective control core temperatures ($P < 0.05$), and no further changes in mean rectal temperature occurred after 2 h. Neither the slope of the temperature decline nor the steady-state temperatures correlated with patient age. Room temperatures in the halothane group ($20.5 \pm 1.4^\circ C$) and N_2O /fentanyl group ($21.0 \pm 0.8^\circ C$) were not significantly different. Temperatures in the two patients who received propranolol did not differ significantly from those in other patients.

DISCUSSION

To ascertain the cause of intraoperative hypothermia, the effects of hypothalamic suppression must be separated from decreased metabolic rate and from exposure to a cold environment. Ophthalmic surgery is ideal for evaluating the effects of anesthetics on hypothalamic function because incision size is small and patients are completely draped. In this study, heat loss was further reduced by maintaining a moderate room temperature and decreasing loss through respiratory evaporation. Avoidable heat losses to the environment were therefore minimal and similar, suggesting that any differences between core temperatures in the N_2O /fentanyl and halothane/ O_2 groups would result primarily from the direct effects of anesthetics.

In both groups, core temperatures decreased rapidly during the first 30 min of anesthesia. The increase in skin temperature after induction of general anesthesia and initial decrease in core temperature probably result from central redistribution of cool peripheral blood.¹¹ In both groups, initial rapid cooling was followed by a slow, linear decline in rectal temperature that continued until thermal steady state was reached 2 h after induction. The time required to reach steady state was similar to that reported in previous studies,¹² although patient temperatures in the present study remained higher. This difference is not surprising, in that earlier trials included patients undergoing procedures with greater exposure to the cold operating room environment.

Body temperature is in steady state when heat loss to the environment (determined by cutaneous blood flow

and other factors held constant during this study) is equal to internal heat production. After 2 h of anesthesia, mean steady-state temperatures were identical in both groups, demonstrating that halothane/O₂ and N₂O/fentanyl anesthesia have comparable effects on heat balance. It is unlikely that central control (thermoregulatory inhibition) caused virtually identical hypothermia during two dissimilar anesthetic regimens. However, these data do not exclude the possibility that the degree of anesthetic-induced thermoregulatory inhibition depends on anesthetic depth, not type.

In summary, we compared changes in rectal temperatures in patients undergoing eye surgery during N₂O/fentanyl or halothane/O₂ anesthesia, and found no significant differences between groups. In both, average temperatures decreased 0.6° C and reached a thermal steady state at 36.2° C after 2 h of anesthesia.

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Changes in EEG Spectral Edge Frequency Correlate with the Hemodynamic Response to Laryngoscopy and Intubation

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Laryngoscopy and intubation of the trachea frequently produce tachycardia, hypertension, and increased serum concentrations of catecholamines.^{1,2} These responses may be exaggerated or lead to in-

creased morbidity in patients with hypertension,³ coronary artery disease,⁴ intracranial aneurysm,⁵ or increased intracranial pressure.^{6,7} However, these responses may be blunted either by ensuring an adequate depth of anesthesia prior to laryngoscopy or administering an additional, nonanesthetic drug to block the sympathetic response.⁸⁻¹⁰

Because the electroencephalogram (EEG) is an indicator of the electrical activity of the cerebral cortex, it may provide a clinically useful measure of the degree of cerebral depression following a dose of anesthetic. Moreover, the EEG signal may be processed to provide a quantitative value which represents underlying electrical activity. One such parameter is the Spectral Edge Frequency (SEF),¹¹ which reflects the highest frequency present in the EEG signal and is particularly sensitive to

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