Less Core Hypothermia when Anesthesia Is Induced with Inhaled Sevoflurane Than with Intravenous Propofol

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Hypothermia after the induction of anesthesia results initially from core-to-peripheral redistribution of body heat. Sevoflurane and propofol both inhibit central thermoregulatory control, thus causing vasodilation. Propofol differs from sevoflurane in producing substantial peripheral vasodilation. This vasodilation is likely to facilitate core-to-peripheral redistribution of heat. Once heat is dissipated from the core, it cannot be recovered. We therefore tested the hypothesis that the induction of anesthesia with IV propofol causes more core hypothermia than induction with inhaled sevoflurane. We studied patients undergoing minor oral surgery randomly assigned to anesthetic induction with either 2.5 mg/kg propofol (n = 10) or inhalation of 5% sevoflurane (n = 10). Anesthesia in both groups was subsequently maintained with sevoflurane and 60% nitrous oxide in oxygen. Calf minus toe skin temperature gradients <0°C were considered indicative of significant vasodilation. Ambient temperature and end-tidal concentrations of maintenance sevoflurane were comparable in each group. Patients in both groups were vasodilated throughout most of the surgery. Nonetheless, core temperatures in patients who received propofol were significantly lower than those in patients who received inhaled sevoflurane. These data support our hypothesis that even a brief period of vasodilation causes substantial redistribution hypothermia that persists throughout surgery. Implications: Core temperatures in patients who received IV propofol were consistently lower than those in patients who received inhaled sevoflurane, although anesthesia was subsequently maintained with sevoflurane in nitrous oxide in both groups. This suggests that even a brief period of propofol-induced vasodilation during anesthetic induction causes substantial redistribution hypothermia that persists throughout surgery.

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Core body temperature poorly estimates mean body temperature because peripheral tissues are normally 2–4°C cooler than the core (1,2). This normal core-to-peripheral tissue temperature gradient is maintained by tonic thermoregulatory vasoconstriction. All anesthetics, including sevoflurane (3,4) and propofol (5,6), profoundly inhibit thermoregulation. In particular, these anesthetics decrease thresholds for vasoconstriction and shivering (triggering core temperatures). Induction of anesthesia thus inhibits tonic vasoconstriction and facilitates a rapid core-to-peripheral redistribution of body heat. This redistribution of body heat is the major cause of core hypothermia during the first hour of anesthesia (2).

Propofol differs from sevoflurane in producing profound, peripheral arterial and venous dilation (7). Although not a central thermoregulatory action, vasodilation is nonetheless likely to facilitate core-to-peripheral redistribution of heat. For example, acute administration of the vasodilator nifedipine increases hypothermia after the induction of anesthesia (8). Once heat is lost from the core, it cannot be recovered from the periphery because flow of heat up a temperature gradient would violate the second law of thermodynamics.

These data suggest that even a brief period of vasodilation, such as during anesthetic induction, may

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have substantial and prolonged effects on body temperature. Accordingly, we tested the hypothesis that a reduction in core temperature would be greater after the induction of anesthesia with propofol versus sevoflurane.

**Methods**

With approval of the ethics committee of the Hamamatsu University School of Medicine and informed written consent, we studied 20 ASA physical status I or II patients undergoing minor oral surgery. None was obese, was taking medication, or had a history of thyroid disease, dysautonomia, or Raynaud’s syndrome.

Patients fasted for 10 h before arriving in the operating room. They were premedicated IM with 0.5 mg of atropine and 20 mg of famotidine 30 min before the induction of anesthesia. A 20-gauge catheter was inserted into a left forearm vein for fluid and drug administration. The patients were then preoxygenated for 5 min at a fresh gas flow of 6 L/min.

The patients were then randomly assigned, based on computer-generated codes, to anesthetic induction with 2.5 mg/kg IV propofol (n = 10) or inhaled 5% sevoflurane in oxygen (n = 10). The face mask in the sevoflurane group was briefly removed and occluded while the entire circle circuit was equilibrated with 5% sevoflurane. Patients were then asked to take three large breaths from the mask; thereafter, ventilation was manually assisted with 5% sevoflurane in oxygen until tracheal intubation (9).

Endotracheal intubation was facilitated by the IV administration of 0.1 mg of vecuronium bromide. Anesthesia in both groups was subsequently maintained with approximately 2% end-tidal sevoflurane and 60% nitrous oxide in oxygen. Ventilation was controlled to maintain Petco₂ near 35 mm Hg. Heat- and moisture-exchanging filters were positioned between the endotracheal tube and the breathing circuit. IV fluids were warmed to 37°C, and ambient temperature was maintained near 25–26°C. Patients were covered with a single cotton blanket and one layer of surgical drape according to our routine management.

Ambient temperature was measured by using a Mon-a-Therm® thermocouple (Mallinkrodt Anesthesiology Products, Inc., St. Louis, MO) positioned at the level of the patient, well away from any heat-producing equipment. Mean skin temperature was calculated from four sites: 0.3 (Tchest + Taxm) + 0.2 (Thigh + Tcalf) (10). Calf and toe skin temperatures were measured to determine calf minus toe skin-surface temperature gradients, which were used as an index of foot arteriovenous shunt perfusion. As in previous studies, we considered a leg gradient <0°C to indicate vasodilation (11). All skin temperatures were measured using thermocouples.

Core temperature was measured at the tympanic membrane before the induction of anesthesia. The aural probes were inserted by patients until they felt the thermocouple touch the tympanic membrane; appropriate placement was confirmed when patients easily detected a gentle rubbing of the attached wire. The aural canal was occluded with cotton. The probe was securely taped in place, and a gauze bandage was positioned over the external ear. All skin and tympanic membrane temperature probes were positioned on the patients’ right sides. After the induction of anesthesia, core temperature was measured at the distal esophagus by using thermocouples.

Heart rate was measured from a three-lead electrocardiogram. Blood pressure was determined oscillometrically at the left ankle. End-tidal sevoflurane and carbon dioxide concentrations were recorded by using an Ultima monitor (Datex, Helsinki, Finland). Oxyhemoglobin saturation (SpO₂) was monitored by pulse oximetry. All values were recorded at 15-min intervals, starting immediately before the induction of anesthesia (elapsed time zero).

Hemodynamic responses, end-tidal sevoflurane and carbon dioxide concentrations, ambient temperature, relative humidity, and mean skin temperatures were first averaged for each patient; the resulting values were then averaged among patients. Differences between the two groups were compared by using two-tailed, unpaired t-tests. All results are presented as means ± SD. P < 0.01 was considered statistically significant.

**Results**

Morphometric characteristics were comparable between the two groups. Duration of surgery, fluid replacement, ambient temperature, relative humidity, end-tidal sevoflurane and carbon dioxide concentrations, mean skin temperature, heart rate, and mean blood pressure were also similar between the two groups (Table 1).

Initial core temperatures were virtually identical in the two groups. However, core temperatures in the patients who received propofol were subsequently significantly lower than those in the patients who received sevoflurane (Fig. 1). One hour after the induction of anesthesia, the core temperature had decreased 1.5 ± 0.3°C in the propofol group but only 0.8 ± 0.2°C in the sevoflurane group (P < 0.001).

Patients in both groups were intensely vasoconstricted before the induction of anesthesia, as indicated by calf minus toe skin temperature gradients near 8°C. Sevoflurane and propofol both induced arteriovenous shunt vasodilation, as indicated by negative calf minus toe skin-surface temperature gradients.
There were, however, no clinically important or statistically significant differences in vasomotor status between the groups at any time (Fig. 2).

### Discussion

Hypothermia after the induction of general anesthesia develops with a characteristic pattern consisting of three distinct phases: 1) an initial rapid decrease in core temperature largely caused by an internal core-to-peripheral redistribution of body heat (2); 2) a slower, linear decrease in core temperature that results from heat loss exceeding metabolic heat production (12); and 3) a core temperature plateau resulting from decreased cutaneous heat loss (13) and constraint of metabolic heat to the core thermal compartment in patients who become sufficiently hypothermic to trigger thermoregulatory vasoconstriction (14).

Our major finding is that core temperature decreased significantly more after induction with propofol than with sevoflurane and that hypothermia persisted for the duration of surgery. Because anesthesia was subsequently maintained with sevoflurane in nitrous oxide in both cases, the observed temperature difference seems to result exclusively from the choice of induction anesthetic.

Sevoflurane has a low blood-gas partition coefficient and is nonpungent, which permits rapid inhaled induction of anesthesia in children and adults (15). The speed of inhaled induction of anesthesia is virtually identical to that of sevoflurane and IV anesthetics, and inhaled sevoflurane is associated with little hypotension (9). In contrast, induction with propofol frequently causes hypotension. Both arteriolar and venous dilation contribute to hypotension, with the dominant mechanism being inhibition of sympathetic vasoconstrictor nerve activity rather than a direct action on vascular smooth muscle (16).

The most likely explanation for exaggerated hypothermia in the patients who received propofol is that the drug caused a brief period of systemic vasodilation that facilitated core-to-peripheral redistribution of body heat. Once redistribution occurred, heat that escaped to peripheral tissues could not be recovered by the core because heat cannot move up a temperature gradient. Relative hypothermia in the propofol patients thus persisted for the duration of surgery, although anesthesia was subsequently comparable in both groups. These data suggest that even brief periods of vasodilation associated with the choice of anesthetic induction drugs can have substantial and prolonged effects of core temperature.

Skin temperature gradients indicated that patients who received sevoflurane and propofol were both

### Table 1. Morphometric and Anesthetic Data

<table>
<thead>
<tr>
<th></th>
<th>Sevoflurane</th>
<th>Propofol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (M/F)</td>
<td>5/5</td>
<td>4/6</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>32 ± 14</td>
<td>31 ± 16</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>56 ± 7</td>
<td>56 ± 9</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>162 ± 11</td>
<td>161 ± 8</td>
</tr>
<tr>
<td>Duration of surgery (min)</td>
<td>114 ± 99</td>
<td>125 ± 99</td>
</tr>
<tr>
<td>Fluid replacement (L)</td>
<td>1.0 ± 0.5</td>
<td>1.3 ± 1.0</td>
</tr>
<tr>
<td>Ambient temperature (°C)</td>
<td>25.7 ± 1</td>
<td>25.8 ± 0.6</td>
</tr>
<tr>
<td>Relative humidity (%)</td>
<td>37 ± 11</td>
<td>35 ± 5</td>
</tr>
<tr>
<td>End-tidal sevoflurane (%)</td>
<td>2.0 ± 0.2</td>
<td>1.9 ± 0.2</td>
</tr>
<tr>
<td>End-tidal CO₂ (mm Hg)</td>
<td>37 ± 2</td>
<td>36 ± 2</td>
</tr>
<tr>
<td>Mean skin temperature (°C)</td>
<td>34.3 ± 0.5</td>
<td>33.9 ± 0.8</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>80 ± 13</td>
<td>84 ± 8</td>
</tr>
<tr>
<td>Mean blood pressure (mm Hg)</td>
<td>80 ± 9</td>
<td>81 ± 10</td>
</tr>
</tbody>
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Values are means ± sd.
vasodilation throughout most of their anesthesia. Dilation is consistent with the thermoregulatory inhibition that each drug produces (3–6). Gradients accurately measure arteriovenous shunt vasomotor status (17). However, the method is relatively specific for shunt flow because the subtraction procedure incorporated into the measure was designed, in part, to compensate for generalized vasomotor changes. Comparable and negative skin temperature gradients in our two patient groups thus only indicate that arteriovenous shunts were dilated in each case. These data cannot be considered as an index of systemic vasomotor status. We thus lack quantification in our patients of the vasodilation associated with each induction drug.

Clinical experience suggests that systemic vasodilation during anesthetic induction with propofol is dose-dependent. In the current study, we administered 2.5 mg/kg propofol; smaller doses of propofol may be associated with less intraoperative hypothermia but are also unlikely to be sufficient for endotracheal intubation. Several aspects of our protocol and thermal management presumably influenced patient temperature. Our patients were premedicated with 0.5 mg of atropine and 20 mg of famotidine. Famotidine premedication is associated with intraoperative hypothermia (18), and atropine has numerous thermoregulatory actions (19–21). Ambient temperature was maintained near 25–26°C, which is higher than that in most other studies. Finally, heat- and moisture-exchanging filters were used in the breathing circuit. However, all these factors were similarly applied to all patients in each group. The observed differences in intraoperative core temperature are thus most likely due to the induction technique.

In conclusion, core temperatures in patients who receive IV propofol for anesthetic induction were consistently lower than those in patients who received inhaled sevoflurane for anesthetic induction. This suggests that even a brief period of propofol-induced vasodilation during anesthetic induction causes substantial redistribution hypothermia that persists throughout surgery.

References