Preanesthetic Skin-Surface Warming Reduces Redistribution Hypothermia Caused by Epidural Block

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Redistribution of heat from the core to the cool peripheral compartments of the body causes hypothermia during epidural anesthesia. Diminishing the temperature gradient between the core and peripheral tissues by warming the body via the skin before anesthesia should prevent this hypothermia. We measured core temperature, skin temperatures, and cutaneous heat loss in seven volunteers who received two lidocaine epidural injections during a single study day. One epidural injection was given after the volunteer had rested in a cool room (−22°C) ("no prewarming") for 2 h, and one injection was given after the volunteer had been covered with a forced air warming mattress (−38°C) ("prewarming") for 2 h. Skin temperatures were higher after prewarming. The decrease in core temperature during epidural anesthesia was smaller after prewarming [mean within patient difference (prewarming-no prewarming): 0.41; \( P = 0.0031 \)]. However, heat loss was greater after prewarming (mean within patient difference: 26.4; \( P = 0.02 \)). Shivering was less after prewarming. We conclude that prewarming decreases redistribution hypothermia caused by epidural block. These results support the hypothesis that redistribution of heat within the body, not heat loss, is the most important etiology of hypothermia from epidural anesthesia.

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Hypothermia (a decrease in core temperature) is common in patients undergoing surgery with epidural anesthesia (1-4). Although surgical factors contribute to intraoperative hypothermia (exposure, fluid replacement), hypothermia and shivering result from the physiologic effects of epidural anesthesia alone. In the absence of anesthesia, core temperature is maintained within a normal range during exposure to a cool environment because of sympathetically mediated vasoconstriction. Vasoconstriction decreases blood flow to the skin and superficial tissues, constrains metabolic heat to the core compartment, and creates a temperature gradient between the core (normal temperature) and peripheral (relatively cool) thermal compartments. Sympathetic block from epidural anesthesia eliminates this vasoconstriction and the cool periphery is warmed at the expense of the core compartment. Thus, hypothermia from epidural anesthesia results from redistribution of heat from the core to the periphery (4).

Skin-surface warming with a forced-air cover transfers substantial amounts of heat to the body (5). Although core temperature remains well regulated, increasing body heat content decreases the temperature gradient between peripheral and core tissues. Because heat flow requires a temperature gradient, decreasing the temperature gradient between the core and the periphery should prevent redistribution hypothermia. In a nonrandomized two-period cross-over study, we tested the hypothesis that skin-surface warming before epidural anesthesia prevents redistribution hypothermia.

Methods

The study protocol was approved by the Institutional Review Board at the University of California, San Francisco, and written informed consent was obtained from seven healthy volunteers. All were without medical problems, were not taking medications, and refrained from oral intake for at least 8 h before the studies which began at 8:00 AM. The volunteers wore swim wear and rested on a padded operating room table in the “lawn chair” position during the study. Intravenous access was established in the left forearm of each volunteer, and a 20-gauge Teflon catheter was inserted into the left
radial artery for continuous blood pressure measurement and obtaining blood samples. Systolic, diastolic, and mean arterial blood pressures were displayed on a Tektronix® 414 monitor and were recorded every 15 min before anesthesia and every 10 min during anesthesia. Electrocardiogram and arterial oxygen saturation were monitored continuously. A 20-gauge catheter was placed in the epidural space through an 18-gauge Tuohy needle via the L 3-4 interspace using standard sterile and “loss of resistance” techniques. A “test dose” of 3 mL of 3% 2-chloroprocaine was injected to confirm the absence of intravenous or subarachnoid placement of the catheter. This test dose was given at least 30 min before study conditions were established and any temperature measurements were made.

Each volunteer was given two epidural injections during the study day. One epidural injection followed a 2-h period in which the volunteer rested uncovered in the laboratory (ambient temperature ~ 22°C; study condition: no prewarming). The other epidural injection followed a 2-h period in which the volunteer was covered with a full length forced-air warming cover (Bair Hugger®, model 200, Augustine Medical, Inc., Eden Prairie, MN) set on “medium” (~38°C; study condition: prewarming). This setting prevents nearly all heat loss to the environment and substantially increases skin temperature (5). The temperature of the warming cover was reduced if the volunteer became uncomfortably warm or began to sweat. Warming was discontinued and the cover was removed at induction of epidural anesthesia. The order of the study conditions was alternated between subjects to assure even distribution of the order of study conditions.

Before each anesthetic, the subjects were hydrated with 8 mL/kg of intravenous Ringer’s lactate solution warmed to ~40°C. Forty milliliters of 1.5% lidocaine were injected into the epidural space in 5-mL doses during 10 min. Anesthetic level was determined every 5 min by loss of sensation to pinprick. Hypotension (decrease in systolic blood pressure of >30%) was treated by giving an additional 300-mL bolus of intravenous Ringer’s lactate and, if necessary, intravenous ephedrine, 10 mg. Before the second study period was initiated, resolution of the first epidural anesthetic was confirmed by the absence of anesthetic level, the absence of postural changes in blood pressure, the ability to ambulate, and return of toe skin temperature to pre-anesthetic values.

Core temperature was measured at the tympanic membrane using a cotton-tipped probe placed adjacent to the tympanic membrane and taped in place. Skin temperature was determined from the weighted average of 10 skin surface temperature sites: 0.06 (head) + 0.09 (upper arm) + 0.06 (forearm) + 0.045 (hand) + 0.19 (back) + 0.095 (chest) + 0.095 (abdomen) + 0.19 (thigh) + 0.115 (calf) + 0.06 (foot) (4). All temperature probes were attached to Mallinkrodt® model 8700 thermometers (St. Louis, MO) calibrated before each use and accurate to within 0.1°C. Toe skin temperature was measured by an infrared skin temperature monitor (Exergen Corp., Newton, MA). Temperatures were recorded every 15 min during the 2-h period preceding each epidural injection, and then every 10 min for 2 h after anesthetic injection.

Heat flux (W/m²) was measured from the 10 skin surface temperature sites using thermal flux transducers (Concept Engineering, Old Saybrook, CT). Heat flux was converted to watts/site by multiplying by the calculated body surface area [area (m²) = weight(0.425) (kg)·height(0.725) (cm)·0.007184] of each volunteer and assigning the same weighted value as the skin temperature measurements. Heat flux was defined as positive when heat traversed skin to the environment. The details of thermal flux measurements have been described previously (5). Total heat loss during anesthesia was calculated by integrating the heat flux values over time and converting watts to kcal (1 W = 0.86 kcal/h). Shivering was noted as present or absent every 5 min during anesthesia.

Arterial blood samples were obtained for analysis of serum lidocaine concentrations during each anesthetic. Samples were drawn just before and then every 10 min for 60 min after injection of epidural lidocaine. Samples were kept on ice, centrifuged, and the serum frozen until analysis by fluorescent polarization immunoassay (specificity, 99%; between-test variability, <3%) (6,7).

Group data [mean±SD] are presented to show the magnitude of the outcome measurements. The significance of differences in outcome measurements after prewarming or no prewarming was assessed using two-tailed Student’s t-test for paired data. Mean within patient differences were calculated by subtracting the “prewarming” values from the “no prewarming” values and are presented as mean (SD). The outcome measurements included: skin temperature before anesthesia, skin temperature at lowest core temperature during anesthesia, core temperature before anesthesia, lowest core temperature during anesthesia, greatest decrease in core temperature during anesthesia, heat loss during anesthesia, peak serum lidocaine concentrations, and duration of shivering. Serum lidocaine concentrations also were compared between the first and second epidural injections. The incidence of shivering was compared by using McNemar’s test with Williams’ continuity correction (8). That the order of study conditions did not affect the study results was determined by calculating the differences in response (change in core temperature) between the prewarming and no prewarming study conditions and testing (two-tailed Student’s t-test for unpaired data) that this difference did not vary as a function of order of study conditions (9).
Results

The descriptive data of the seven volunteers are shown in Table 1. The first study condition was "prewarming" for three subjects. The order of study conditions did not affect the study results (Table 2) (9). All volunteers obtained mid- to high thoracic levels of anesthesia after each epidural injection. Anesthesia completely resolved within 2.5 h after lidocaine injection. No volunteer experienced hypotension that required treatment with additional intravenous fluids or ephedrine.

Core temperatures were similar before the induction of anesthesia with both study conditions (Table 3). Skin temperatures were much higher after prewarming, but decreased when the warming cover was removed at the time of epidural injection (Figure 1). In contrast, after no prewarming, skin temperature increased slightly with epidural anesthesia (Figure 1) (Table 3). Along with these observed changes in skin temperature, heat loss during anesthesia was greater after prewarming [217 (27) kcal] than after no prewarming [191 (26) kcal; mean within patient difference, 26.4 (23.3); P = 0.02] (Figure 2). Despite greater heat loss during anesthesia, core temperatures decreased less after prewarming than after no prewarming in all seven subjects (Figure 3) (Table 3). The maximum decrease in core temperature during anesthesia after prewarming was less than half of that measured after no prewarming [prewarming, -0.3 (0.3)°C; no prewarming, -0.7 (0.3); mean within patient difference, 0.41 (0.23); P = 0.003].

Shivering was observed during anesthesia in four volunteers after prewarming and in all seven volunteers after no prewarming (P = 0.049) (Figure 3). The duration of shivering was less when prewarming was used [31.0 (32) min] compared to no prewarming [57.2 (23) min; mean within patient difference, -26.1 (16); P = 0.005].

Peak serum lidocaine concentrations during epidural anesthesia did not differ as a function of preanesthetic study condition. The highest concentrations measured were 4.2 (0.6) μg/mL after prewarming and 4.6 (0.5) μg/mL without prewarming [mean within patient difference, -0.4 (0.6); P = 0.16]. Peak lidocaine concentrations also were similar during the first and second anesthetics [first injection, 4.3 (0.4) μg/mL; second injection, 4.5 (0.7) μg/mL; mean within patient difference, -0.2 (0.8); P = 0.58].

Discussion

Our results support the hypothesis that redistribution of heat is the most important etiology of hypothermia caused by epidural anesthesia. We have shown that increasing body heat content by skin surface warming helps prevent this hypothermia. Similar results have been obtained using skin surface warming before administering general anesthesia (10).

Our results agree with previous findings that heat loss to the environment does not contribute significantly to hypothermia during epidural anesthesia (4). Despite more heat loss from the skin during anesthesia, core temperature decreased less after prewarming. The greater heat loss after prewarming can be explained by the higher heat content of the skin and the relatively large temperature gradient between the environment (~22°C) and skin (~36°C) after removal of the warming cover at anesthetic induction. Core temperature decreased less after prewarming because the heat content of the superficial tissues was relatively high and the temperature gradient between the core and superficial tissues was small. In contrast, vasoconstriction during no prewarming decreased the heat content of the superficial tissues and the skin. Heat loss was less during epidural anesthesia because the gradient between ambient temperature and skin temperature was less. However, the temperature gradient between the core and superficial tissues was higher and core temperature fell from heat redistribution. Our findings confirm that heat loss is not an important determinant of the degree of hypothermia caused by epidural anesthesia. Core temperature changes are determined largely by the temperature of the superficial tissues and subsequent heat redistribution.
Table 3. Core and Skin Temperatures (°C) [mean (SD)] Before and During Epidural Anesthesia

<table>
<thead>
<tr>
<th></th>
<th>Prewarming</th>
<th>No prewarming</th>
<th>Mean within patient difference</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin temperature before anesthesia</td>
<td>36.2 (0.5)</td>
<td>31.5 (0.6)</td>
<td>4.6 (0.7)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Core temperature before anesthesia</td>
<td>37.0 (0.4)</td>
<td>36.7 (0.2)</td>
<td>0.23 (0.3)</td>
<td>0.1</td>
</tr>
<tr>
<td>Lowest core temperature during anesthesia</td>
<td>36.7 (0.7)</td>
<td>36.0 (0.5)</td>
<td>0.64 (0.47)</td>
<td>0.01</td>
</tr>
<tr>
<td>Skin temperature at time of lowest core temperature</td>
<td>33.7 (0.7)</td>
<td>31.8 (0.6)</td>
<td>1.9 (0.91)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Warming the body via the skin reduced thermoregulatory shivering during epidural anesthesia. Shivering is a troublesome side effect of anesthetic-related hypothermia and it contributes to patient discomfort (11), increases oxygen consumption (12,13), and interferes with patient monitoring. Previous studies have confirmed that tremor during epidural anesthesia is thermoregulatory shivering prompted by hypothermia (4). However, both skin and core temperatures influence the shivering response (14,15); a greater (or lesser) decrease in core temperature is required to trigger shivering when skin temperature is very high (or very low), and sudden and extreme increases or decreases in skin temperature can suppress or trigger shivering. Because skin temperatures were higher after prewarming, we can not simply conclude that less shivering was observed after prewarming because of smaller changes in core temperature. Rather, our results show that shivering during epidural anesthesia is less when both skin temperature is relatively high and the decrease in core temperature is small. Our data suggest that more shivering occurred during anesthesia after no prewarming because of a larger decrease in core temperature. More shivering after no prewarming cannot be attributed to a decrease in skin temperature because such a change was not observed (Figure 1) (Table 3).

It is unlikely that systemic absorption of epidural lidocaine interfered with thermal responses in this volunteer study. The levels obtained in this study were similar to those we measured in a previous study in which intravenous lidocaine did not affect core temperature, vasoconstriction, or shivering in response to cold exposure (7).

The results of this study probably would have been more dramatic if we had completely eliminated any temperature gradient between the core and superficial tissues. Despite the 2-h period of prewarming, it is likely that body heat content differed among the volunteers. The fact that core temperature decreased more than 0.2°C in four of the volunteers after prewarming suggests that being covered by a forced air warmer at ~38°C for 2 h was not always adequate to obliterate temperature gradients between the core and peripheral compartments.

Our study conditions differ from those in the operating room in several ways, and caution must be used to extrapolate these data to the care of patients. First, these data do not necessarily support the hypothesis that increasing operating room temperature during the induction of anesthesia will help limit intraoperative hypothermia. The brief exposure of the patient to an increased ambient temperature would have minimal effects on body heat content and therefore would minimally affect redistribution hypothermia from anesthesia. (However, patient comfort may be increased by the sensation of warmth on the skin, and shivering triggered by sudden exposure of the skin to a cold environment may be prevented.) A more rational clinical application of these study results would be to make sure that patient rooms or places in which patients spend several hours before receiving an anesthetic are warm.
Second, our study volunteers differed from surgical patients in that thermal responses occurred as a result of anesthesia only. They did not have surgical conditions that also contribute to intraoperative hypothermia such as exposure of body cavities to cool ambient temperatures, lavage of body cavities with relatively cool solutions, and infusion of large volumes of room temperature intravenous fluids (16,17).

Third, our study volunteers received two anesthetics in 1 day. The thermoregulatory and metabolic effects of receiving two epidural anesthetics in 1 day are unknown. Further, normal circadian fluctuations in body
temperature may have altered the thresholds for thermoregulatory responses during the course of the day (18,19). The study conditions were alternated between volunteers to assure that these potential effects were distributed evenly between the two study conditions. Our study results may have been more dramatic if the study volunteers had received the epidural anesthetics at the same time of day on two different days, but the nonsignificant test for interaction between the order of study conditions and outcome shows that the subjects’ responses were not affected by the order of the two study conditions.

In summary, we have found that warming the body via the skin for 2 h before inducing epidural anesthesia raises skin temperature (but not core temperature) and helps prevent hypothermia during the induction of epidural anesthesia. This occurs despite increased heat loss and supports the hypothesis that redistribution of heat is the most important cause of hypothermia resulting from epidural anesthesia.

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References