Thermoregulatory Effects of Spinal and Epidural Anesthesia During Cesarean Delivery

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Background and Objectives: Hypothermia is likely to develop faster during spinal anesthesia than epidural anesthesia. A natural consequence of the rapid temperature decrease during spinal anesthesia is that the shivering threshold will be reached sooner and that more shivering will be required to prevent further hypothermia. We tested the hypotheses that the onset of hypothermia is more rapid and the onset and intensity of shivering earlier during spinal than epidural anesthesia. Methods: Patients undergoing cesarean delivery were randomly assigned to spinal anesthesia or epidural anesthesia. Spinal anesthesia was induced by injecting 2 mL 0.5% dibucaine into the L4-L5 interspace. Epidural anesthesia was induced with 20 mL 2% mepivacaine injected into the L2-L3 interspace. Thermal comfort and shivering were scored by a blinded observer. Results: Fifteen patients given each type of anesthesia had upper sensory levels ≥T4 dermatome. Sensation was entirely absent from the leg during spinal anesthesia, but lower block levels were near S5 during epidural anesthesia. Tympanic membrane temperatures initially decreased faster during spinal anesthesia, but subsequently decreased at a rate of 0.5°C/h in both groups. The onset and incidence of shivering (detected qualitatively) did not differ significantly between the two groups, but shivering intensity was significantly reduced during spinal anesthesia. Furthermore, the shivering thresholds were 36.4 ± 0.3°C (mean ± SD) during spinal anesthesia versus 37.1 ± 0.4°C in those given epidural anesthesia (P = .006). There were no clinically important differences in thermal comfort with the two kinds of neuraxial anesthesia. Conclusions: We failed to confirm our hypothesis, but for an unexpected reason: Thermoregulation was impaired more by spinal anesthesia than epidural anesthesia. It seems likely that in our patients spinal anesthesia inhibited thermoregulatory control more than epidural anesthesia because it better blocked sensory input from the legs. Reg Anesth Pain Med : 23: 418–423. Key words: anesthesia, anesthetic technique: epidural, spinal; hypothermia, temperature, thermoregulation: shivering.
Neuraxial block impairs thermoregulatory control, increasing the sweating threshold (triggering core temperature) and decreasing the vasoconstriction and shivering thresholds (1). Studies in volunteers and patients have shown that the vasoconstriction and shivering thresholds (triggering core temperatures) are comparably decreased by spinal and epidural anesthesia (2).

Comparable thermoregulatory response thresholds, however, do not ensure that the incidence or severity of shivering will be similar with spinal and epidural anesthesia. The onset of spinal anesthesia is faster than epidural anesthesia, and the block is usually more dense. Furthermore, spinal anesthesia usually produces a lower-body sympathectomy (3), whereas epidural anesthesia (even with a complete sensory block) may not in up to half of all cases (4,5). Each of these factors will facilitate core-to-peripheral redistribution of body heat which is the major initial cause of hypothermia during neuraxial block (4).

Hypothermia is thus likely to develop faster during spinal anesthesia than epidural anesthesia. A natural consequence of the rapid temperature decrease during spinal anesthesia is that the shivering threshold will be reached sooner and that more shivering will be required to prevent further hypothermia. Accordingly, we tested the hypotheses that (a) the onset of hypothermia is more rapid and (b) the onset of shivering is earlier during spinal anesthesia than epidural anesthesia.

**Methods**

With the approval of the Institutional Review Board at Tama-Nagayama Hospital and informed consent, we evaluated 31 women undergoing elective cesarean delivery. Indications for cesarean delivery included previous cesarean delivery (n = 5), abruptio placenta (n = 2), cephalopelvic disproportion (n = 23), and triplets (n = 1). None of the patients was in labor, and membranes were ruptured in only three cases. All were ASA Physical Status I or II. They were randomly assigned to spinal (n = 15) anesthesia or epidural (n = 16) anesthesia.

No premedication was given; patients were not given oral antacids or metoclopramide. Patients assigned to spinal anesthesia were positioned laterally, and 2 mL of hyperbaric 5% dibucaine (10 mg total) was injected into the L4-L5 interspace with a 25-gauge needle. The operating room table was then tilted as necessary to produce a sensory block just rostral to the T4 dermatome. Patients assigned to epidural anesthesia were also positioned laterally. A 17-gauge Tuohy needle was inserted between the second and third vertebrae using a paramedian approach. The epidural space was identified using loss-of-resistance to air, and 20 mL 2% mepivacaine (400 mg total) was injected at ambient temperature as a bolus through the catheter without a test dose.

No sedatives or intravenous anesthetics were administered during surgery. Unwarmed fluid was administered intravenously as necessary to maintain normal hemodynamic values. Ephedrine (5 mg boluses) was administered as necessary to maintain systolic arterial pressure $\geq$ 90 mm Hg.

We evaluated the sensory block (using cold sensation and pinprick) 5, 10, 15, and 30 minutes after induction of anesthesia. We also confirmed that sensation remained intact on the palmar surface of each hand. Abdominal preparation and surgery were delayed until the sensory block extended caudally to at least the third sacral dermatome bilaterally. The abdomen was covered with a single blanket during this period. After delivery of the baby, 0.2 mg methylergonovine maleate was injected intramuscularly; oxytocin was not administered.

Core temperatures were recorded from the tympanic membrane (Mon-a-Therm thermocouple, Mallinckrodt Anesthesiology, Inc., St. Louis, MO). 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 aural canal was occluded with cotton, the probe securely taped in place, and a gauze bandage positioned over the external ear. Mean skin-surface temperature was determined from recordings at four sites (6).

Overall thermal comfort was evaluated with a 100-mm visual analog scale (VAS) on which 0 mm was defined as worst imaginable cold, 50 mm as thermally neutral, and 100 mm as insufferably hot (7). A new, unmarked scale was used for each assessment. Shivering (above the level of the block) was evaluated qualitatively by an observer blinded to treatment and core temperature. As in previous studies (8), we used a three-point scale: 0 = no shivering; 1 = mild, intermittent shivering; and 2 = intense, continuous shivering. The core temperature triggering grade one shivering was considered the threshold for this response. All measurements were recorded at 15-minute intervals.

Thresholds in the two anesthetic groups were compared with a Mann-Whitney U test, with “missing values” (patients in whom a threshold was not detected) coded as zero. Induction of anes-
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... was considered elapsed time zero. Core and skin temperatures in the two groups were compared using two-tailed, unpaired t-tests. Shivering and VAS scores were compared using Friedman (repeated-measures, nonparametric) analysis of variance. Rates of core temperature cooling were evaluated using linear regression. Based on inspection of the data, we used temperatures between 0 and 75 elapsed minutes in patients given epidural anesthesia, whereas values between 30 and 75 elapsed minutes were used in the patients given spinal anesthesia. Data are presented in the mean ± SD; P values < .05 were considered statistically significant.

Results

Morphometric characteristics of the volunteers given each type of anesthesia were similar, as was ambient temperature (Table 1). The indications for cesarean delivery were similar in each group. Patients in both groups were given comparable amounts of intravenous fluid. Nine of the patients given spinal anesthesia required ephedrine for hypotension, whereas ephedrine was only required in three of the patients given epidural anesthesia. Surgery started approximately 25 minutes after induction of spinal anesthesia, but usually 35-40 minutes after epidural anesthesia.

One patient given epidural anesthesia was eliminated from the study because the upper block level did not reach the T4 dermatome. All results and statistical analysis are thus based on 15 patients per group. Sensation was absent from all sacral dermatomes in the patients given spinal anesthesia. Maximum lower block levels during epidural anesthesia were T5 in 11 patients, T4 in 1 patient, and T3 in 3 patients.

Core temperatures were comparable in both groups before induction of anesthesia. During the first 30 minutes of anesthesia, core temperature decreased significantly faster in the patients given spinal anesthesia. Subsequently, core temperatures decreased at virtually identical rates during epidural (0.51°C/h; r² = 0.99) anesthesia and spinal (0.50°C/h; r² = 0.99) anesthesia (Fig. 1).

Despite having significantly lower core temperatures, patients given spinal anesthesia shivered significantly less than those who had epidural anesthesia (Fig. 2). Six patients given spinal anesthesia and nine patients given epidural anesthesia shivered during the study (P = NS). Their respective thresholds were 36.4 ± 0.3°C and 37.1 ± 0.4°C (P < .05), respectively. Although thermal comfort increased after induction of anesthesia, there were no clinically important differences between treatment groups (Fig. 3). Mean skin temperatures also increased after induction of anesthesia, but were nonetheless comparable in both groups throughout the study.

Discussion

The rapid decrease in core temperature following induction of neuraxial block results mainly from an internal core-to-peripheral redistribution of body heat (4). Unlike spinal anesthesia (3), epidural anesthesia may fail to block sympathetic control in the legs (4). Spinal anesthesia rapidly produced hypo-

Table 1. Morphometric Characteristics and Ambient Temperature

<table>
<thead>
<tr>
<th></th>
<th>Epidural</th>
<th>Spinal</th>
</tr>
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<tbody>
<tr>
<td>Age (yr)</td>
<td>32 ± 5</td>
<td>28 ± 3</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>62 ± 7</td>
<td>63 ± 8</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>159 ± 8</td>
<td>160 ± 8</td>
</tr>
<tr>
<td>Ambient temperature (°C)</td>
<td>23.1 ± 0.6</td>
<td>23.1 ± 0.6</td>
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There were no statistically significant differences between the groups. Data are mean ± SD.
thermia, whereas core temperatures during epidural anesthesia decreased slowly and linearly. It thus seems likely that redistribution contributed to hypothermia during spinal anesthesia, whereas epidural anesthesia failed to produce a sympathetic block sufficient to inhibit tonic thermoregulatory vasoconstriction in the legs.

Vasodilation associated with neuraxial block only minimally increases cutaneous heat loss (4,9). Heat loss from skin to the environment was thus probably similar with each anesthetic technique. The incidence of shivering was also similar, suggesting that metabolic heat production did not differ enormously in both groups. Consistent with this theory, the rate of core temperature decrease after the first 30 minutes of anesthesia was virtually identical with spinal and epidural anesthesia.

As expected, core temperatures initially decreased faster during spinal anesthesia than epidural anesthesia. We failed to confirm our hypothesis, however, because lower core temperatures during spinal anesthesia were associated with significantly less shivering. Furthermore, shivering thresholds were significantly less during spinal anesthesia than epidural anesthesia. These data clearly indicate that, under the settings of our study, spinal anesthesia impaired autonomic thermoregulation more than epidural anesthesia.

Neuraxial block prevents central transmission of peripheral thermal signals from the affected area. Absence of tonic cold signals may be interpreted by the thermoregulatory system as apparent (as opposed to actual) leg warming (8,10). This is consistent with the increase in thermal comfort observed after induction of neuraxial anesthesia observed here and in previous investigations (7). Because skin temperature contributes about 20% to control of shivering (11), the normal response to leg warming is a reduction in the shivering threshold. As might be expected from this mechanism, thermoregulatory inhibition during neuraxial block increases linearly with upper-body sensory block height (8). A major difference between epidural and spinal anesthesia is that spinal anesthesia in our patients fully blocked sensory input from the legs, whereas epidural anesthesia did not. Although we did not quantify block intensity, it seems likely that the intensity of the sensory block was greater during spinal anesthesia than epidural anesthesia. Our data thus suggest that inhibition of central thermoregulatory control also depends on the lower extent of sensory block.

Greater thermoregulatory inhibition during spinal anesthesia than epidural anesthesia is both surprising and interesting because previous work suggests that shivering thresholds are comparably reduced by epidural and spinal anesthesia. The thermoregulatory effects of neuraxial block have been compared in both volunteers and nonpregnant patients (2). Epidural anesthesia in the volunteers was induced with 18–23 mL local anesthesia, but then maintained with a continuous infusion of 13–18 mL/h. This dose was sufficient to maintain a complete sensory block in the legs. Epidural anesthesia in the previously evaluated patients consisted of a single 25-mL dose of local anesthetic. Sensory block in the legs was not recorded but may have

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**Fig. 2.** Shivering scores were significantly less during spinal anesthesia than epidural anesthesia (*P* < .01). Elapsed time zero identifies induction of anesthesia. Data are mean ± SD.

**Fig. 3.** There were no clinically important differences in thermal comfort scores during spinal anesthesia and epidural anesthesia. Elapsed time zero identifies induction of anesthesia. Data are mean ± SD.
been more extensive than in our current patients who were given only 20 mL of anesthetic. It thus seems likely that the thermoregulatory effects of spinal and epidural anesthesia were similar in our previous studies because sensory input from the legs was comparably impaired. In contrast, the shivering threshold was greater during epidural anesthesia in our current patients because sensory input from the legs remained relatively intact.

Spinal anesthesia completely blocked sensory perception in the legs, whereas the block was only partial during epidural anesthesia. Although it seems likely that greater thermoregulatory inhibition during spinal anesthesia than epidural anesthesia resulted from this more extensive sensory block, block level was not prospectively assigned within each type of anesthesia. Our study design thus does not preclude the possibility that greater inhibition during spinal anesthesia resulted from some specific difference between the techniques.

Our patients undergoing cesarean delivery were, naturally, all female. Even in the follicular phase of the menstrual cycle, thermoregulatory response thresholds are about 0.3°C higher in women than men. The precision of thermoregulatory control, however, is comparable in each gender (12). During the luteal phase, resting core temperatures are about 0.5°C (13). More importantly, however, our patients were pregnant and presumably had very high circulating concentrations of progesterone. Progesterone may account for elevated thresholds during the luteal phase although there is evidence that 17β-estradiol contributes (14). The effects of progesterone on minimum alveolar concentration remain controversial (15,16), and its effect on control of shivering—especially during neuraxial anesthesia—is simply unknown.

Systemic absorption of epidurally administered local anesthesia is a potential mechanism for thermoregulatory impairment during neuraxial anesthesia. However, intravenous lidocaine does not alter the shivering threshold (17). Furthermore, thermoregulatory impairment is similar to lidocaine (7) and chloroprocaine (18) anesthesia, although the plasma half-life of chloroprocaine is only approximately 20 seconds (19). These data suggest that neural block, rather than systemic absorption, is the dominant mechanism of impairment.

In summary, previous work indicates that shivering thresholds during spinal anesthesia and epidural anesthesia are similar, but that hypothermia is likely to develop more rapidly during spinal anesthesia. We thus tested the hypothesis that the onset of shivering is faster during spinal anesthesia than epidural anesthesia, and the intensity is greater. Our results, however, indicated that shivering onset and incidence differed significantly with each type of anesthesia. The intensity of shivering was reduced during spinal anesthesia, and the shivering threshold was significantly less during spinal anesthesia than epidural anesthesia. We thus failed to confirm our hypothesis, but for an unexpected reason: Thermoregulation was impaired more by spinal anesthesia than by epidural anesthesia.

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References


