Thermoregulatory Response Thresholds During Spinal Anesthesia

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Reportedly, during spinal anesthesia, the shivering threshold is reduced ~1°C but the vasoconstriction threshold remains normal. Such divergence between the shivering and vasoconstriction thresholds is an unusual pattern of thermoregulatory impairment and suggests that the mechanisms of impairment during regional anesthesia may be especially complex. Accordingly, we sought to define the pattern of thermoregulatory impairment during spinal anesthesia by measuring response thresholds. Seven healthy women volunteered to participate on two study days. On one day, we evaluated thermoregulatory responses to hypothermia and hyperthermia during spinal anesthesia; on the other day, responses were evaluated without anesthesia. Upper body skin temperature was kept constant throughout the study. The volunteers were warmed via the lower body and cooled by central venous infusion of cold fluid. The core temperatures triggering a sweating rate of 40 g·m⁻²·h⁻¹, a finger flow of 0.1 mL/min, and a marked and sustained increase in oxygen consumption were considered the thermoregulatory thresholds for sweating, vasoconstriction, and shivering, respectively. Spinal anesthesia significantly decreased the thresholds for vasoconstriction and shivering, and the decrease in each was ~0.5°C. The range of temperatures not triggering thermoregulatory responses (those between sweating and vasoconstriction) was 0.9 ± 0.6°C during spinal anesthesia. The synchronous decrease in the shivering and vasoconstriction thresholds during spinal anesthesia is consistent with thermoregulatory impairment resulting from altered afferent thermal input.

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The interthreshold range at constant skin temperature probably spans only a couple of tenths of a degree (2,5). Synchronous changes in all thermoregulatory response thresholds (with the interthreshold range remaining normal) is the pattern produced by fever and circadian temperature variation (10). In contrast, general anesthesia markedly increases the interthreshold range, typically to ~4°C (11,12). Sweating thresholds have not been measured during epidural or spinal anesthesia. Consequently, the interthreshold range and pattern of thermoregulatory impairment during major conduction anesthesia remain unknown. However, the sweating threshold seems to be elevated in patients with spinal cord transections (13). Similarly, these patients apparently permit wider core temperature fluctuations than individuals with intact spinal core function (14).

As a prelude to more specific investigation of the mechanisms of thermoregulatory impairment during regional anesthesia, we sought to clearly define the pattern of thermoregulatory responses during spinal anesthesia. Accordingly, we tested the hypothesis that spinal anesthesia comparably reduces the shivering and vasoconstriction thresholds. Additionally, we determined the interthreshold range during spinal anesthesia.

Methods

With approval from the University of California, San Francisco, Committee on Human Research, and written, informed consent from the volunteers, we studied seven healthy female volunteers. We restricted the population to women because sweating thresholds in women are 0.3–0.5°C higher than those in men (12).

The women were aged 20–30 yr, weighed 56 ± 7 kg, and had a height of 157 ± 11 cm. The percentage of body fat, determined by infrared interactance (Futrex 1000, Futrex, Inc., Hagerstown, MD), was 26 ± 3%. None was obese, was taking medication, or had a history of smoking, thyroid disease, dysautonomia, or Raynaud's syndrome. All volunteers were studied during the first 10 days of their menstrual cycles.

Each volunteer participated on two study days. On one day we evaluated thermoregulatory responses to hypothermia and hyperthermia during spinal anesthesia; on the other day, responses were evaluated without anesthesia. Order of the study days was assigned randomly. Also randomly assigned was whether the volunteers were initially warmed or cooled. However, to avoid confounding circadian temperature alterations (15), the order of thermal manipulations was the same on each study day in each volunteer.

Studies started approximately 9:30 AM. Volunteers fasted during the 8 h preceding each study. All volunteers were minimally clothed and reclined on their backs on a standard operating room table. A 16-gauge catheter then was inserted into the superior vena cava via the internal jugular vein using standard technique. Ambient temperature was maintained at 23.1 ± 1.1°C and ambient humidity at 38 ± 8% (model HX93 humidity transmitter, Omega Engineering, Inc., Stamford, CT).

Regardless of their randomization to initial warming or cooling, all volunteers were first warmed until fingertip vasodilation was observed. Warming assured that the volunteers started each study day in a similar thermoneutral state, and minimized subsequent redistribution hypothermia when spinal anesthesia was administered (16).

Volunteers were warmed with a Bair Hugger® forced-air warmer (Augustine Medical, Inc., Eden Prairie, MN) using a lower body cover positioned over the legs and a circulating-water mattress (Blanketrol 200HL, blanket 164, Cincinnati Sub-Zero, Cincinnati, OH) placed beneath the volunteers' legs and hips. The forced-air warmer was set on "high" (~43°C) and the water mattress was set to 42°C (17).

On the spinal anesthesia day, volunteers were given 1000 mL of warmed lactated Ringer's solution before induction of anesthesia. Spinal anesthesia was induced by subarachnoid administration of 4 mL of 0.5% isobaric bupivacaine with epinephrine 1:100,000 using a 24-gauge Sprotte needle inserted via the L3-4 interspace. Arterial blood pressure remained within 15% of control values in each volunteer. The level of the resulting sensory block was determined shortly after induction of anesthesia and at 1-h intervals throughout the study, using the response to both cold sensation and pinprick response. The volunteers were observed for at least 30 min after induction of anesthesia to confirm continued peripheral vasodilation and a stable blood pressure.

Forced-air and circulating-water heating was continued in those volunteers assigned to initial warming until the sweating threshold was identified. Core hypothermia then was induced, as in previous studies (18,19), by central venous administration of lactated Ringer's solution cooled to ~4°C. Additionally, we turned off the circulating-water mattress and uncovered the legs. The fluid administration rate was adjusted to decrease core temperature ~0.8°C/h. In volunteers assigned to initial cooling, the procedure was reversed. In all cases, however, the transition from warming to cooling, or the reverse, was made gradually to minimize dynamic components of the thermoregulatory responses.

On the nonanesthetic study day, the protocol was similar, except that spinal anesthesia was not administered. Throughout core warming and cooling on each
study day, upper body skin temperature was maintained near 35°C by insulating the skin with an appropriate number of cotton blankets.

Core temperature was measured by a thermocouple (Mallinckrodt Anesthesia Products, Inc., St. Louis, MO) positioned adjacent to the tympanic membrane. The aural canal was occluded with cotton to prevent artificial cooling by the ambient environment.

Area-weighted, upper body skin-surface temperature was computed from measurements at seven sites by assigning the following regional percentage to each area: head, 11%; upper arms, 17%; forearms, 11%; anterior calves, 16%; posterior calves, 9%; feet, 9%; toes, 4% (20).

Core and skin-surface temperatures were recorded from thermocouples connected to two 16-channel Iso-Thermex electronic thermometers having an accuracy of 0.1°C and a precision of 0.01°C (Columbus Instruments International Corp., Columbus, OH).

Sweating on the chest was quantified by passing 2.0 L/min of anhydrous oxygen across a circle of skin 6-cm in diameter covered with an airtight ostomy appliance (stock #3706 and #3806, Hollister Products, Libertyville, IL). Cutaneous water loss (in g·m⁻²·h⁻¹) was calculated from the gas flow rate (model FMA-5000, Omega Engineering, Inc.), and gas temperature and relative humidity (model HX93, Omega Engineering, Inc.). We have described previously the details of this measurement technique (12). The core temperature triggering a sweating rate of 40 g·m⁻²·h⁻¹ was considered the threshold for sweating.

Absolute right middle fingertip blood flow, resulting primarily from arteriovenous shunt flow, was quantified by venous-occlusion volume plethysmography at 5-min intervals (21). Volume plethysmography is considered the most reliable measure of extremity blood flow. The core temperature triggering a finger flow of 0.1 mL/min was considered the thermoregulatory threshold for vasoconstriction.

Shivering was evaluated by indirect calorimetry at 5-min intervals during cooling. Expired respiratory gases were collected from a tightly fitted mask (Hans Rudolph Inc., Kansas City, MO) into a water-sealed respirometer. Oxygen consumption was calculated from expired gas volume and inspiratory and mixed expiratory oxygen concentration (Medspec® spectrometer, St. Louis, MO). The core temperature triggering a marked (e.g., 50%) and sustained increase in oxygen consumption was considered the threshold for shivering. Although electromyographic analysis is useful for evaluating tremor patterns, oxygen consumption is the most reliable method of quantifying shivering intensity.

Heart rate was monitored continuously by using three-lead electrocardiography. Oxyhemoglobin saturation at the right hand finger and blood pressure at the ankle of the left leg (determined oscillometrically at 5-min intervals) were measured by using a Modulus CD integrated anesthesia system (Ohmeda, Inc., Madison, WI). These data were recorded by using IdaCare®, version 1.3 (Hermes System s.a., Belgium) which is Macintosh®-based (Apple Computer, Cupertino, CA) patient information management software.

The difference between the sweating and vasoconstriction thresholds in each individual was considered the sweating-to-constriction (interthreshold) range. Similarly, the difference between the core temperatures triggering sweating and shivering were considered the sweating-to-shivering range and those triggering vasoconstriction and shivering were considered the vasoconstriction-to-shivering range.

Upper and lower body skin-surface temperature at each threshold with, and without, spinal anesthesia were compared by repeated-measure analysis of variance and Scheffé's F tests. The thresholds and ranges on each study day were compared by using two-tailed, paired t-tests. Core cooling and warming rates were compared similarly. All values are expressed as means ± SD; differences were considered significant when P < 0.01.

Results

Spinal anesthesia usually produced a T9–10 sensory block level (range: T8–11) as measured by perception of cold and response to a pinprick. There was no clinically important regression of the block during the study period. There were no statistically significant or clinically important differences in upper body temperatures at any of the thresholds. Lower body temperatures were comparable during vasoconstriction and shivering with and without spinal anesthesia. As expected from the protocol, lower body temperatures were significantly higher during sweating (but presumably not sensed by the central regulatory system during spinal anesthesia) (Table 1).

Core cooling rates were comparable with and without spinal anesthesia: 1.0 ± 0.1°C/h and 0.8 ± 0.3°C/h, respectively. The core warming rate was statistically higher during spinal anesthesia (1.0 ± 0.2°C/h) than without anesthesia (0.6 ± 0.2°C/h); however, the difference was not clinically important.

The sweating, vasoconstriction, and shivering thresholds in each volunteer with and without spinal anesthesia are shown in Figure 1. Similarly, individual differences in the shivering threshold with and without
Table 1. Upper and Lower Body Skin-Surface Temperatures With and Without Spinal Anesthesia

<table>
<thead>
<tr>
<th>Thermoregulatory response</th>
<th>Upper body (°C)</th>
<th>Lower body (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No spinal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sweating</td>
<td>35.5 ± 0.5</td>
<td>37.8 ± 0.3*</td>
</tr>
<tr>
<td>Vasoconstriction</td>
<td>35.3 ± 0.4</td>
<td>33.8 ± 0.9</td>
</tr>
<tr>
<td>Shivering</td>
<td>35.1 ± 0.2</td>
<td>32.7 ± 0.5</td>
</tr>
<tr>
<td>Spinal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sweating</td>
<td>35.7 ± 0.5</td>
<td>38.4 ± 0.4*</td>
</tr>
<tr>
<td>Vasoconstriction</td>
<td>35.2 ± 0.5</td>
<td>34.9 ± 1.1</td>
</tr>
<tr>
<td>Shivering</td>
<td>34.8 ± 0.2</td>
<td>33.5 ± 0.7</td>
</tr>
</tbody>
</table>

There were no statistically significant or clinically important differences in upper body skin temperature throughout the study. Lower body temperatures were comparable during vasoconstriction and shivering on each study day. As expected from the protocol, lower body temperatures were significantly higher during sweating (*p*) than during the other trials. However, this high temperature presumably was not sensed by the central thermoregulatory system during spinal anesthesia.

![Figure 1](image1.png)

Figure 1. The sweating, vasoconstriction (VC), and shivering thresholds in each individual with and without spinal anesthesia. Each symbol indicates a single volunteer. Spinal anesthesia significantly decreased the thresholds for vasoconstriction and shivering, and the decrease in each was -0.5°C. Consequently, the vasoconstriction-to-shivering range also decreased -0.5°C with and without anesthesia. The sweating-to-vasoconstriction interthreshold range was 0.9 ± 0.6°C during spinal anesthesia. The sweating threshold without anesthesia, and the ranges derived from this value, should not be compared directly to the other thresholds because perceived lower body temperature differed during this trial. Please see Table 2 for additional statistical analysis.

![Figure 2](image2.png)

Figure 2. The shivering thresholds in each individual with and without spinal anesthesia. The symbols correspond to the same volunteers shown in Figure 1. The threshold was significantly lower during spinal anesthesia, by 0.5 ± 0.2°C.

Discussion

Control of shivering, like that of other thermoregulatory responses, is dominated by the hypothalamus (22). However, hypothalamic temperature per se seems relatively unimportant; instead, the regulatory system integrates thermal input from other parts of the brain, the spinal cord, deep abdominal and thoracic tissues, and the skin surface (23-25). Skin temperature apparently contributes 10-20% to control of autonomic thermoregulatory responses (23,26). It is this contribution that makes cutaneous warming an effective treatment for shivering, even in patients remaining centrally hypothermic (27).

Shivering-like tremor during epidural anesthesia is normal thermoregulatory shivering (28), triggered by core hypothermia and preceded by vasoconstriction above the level of the block (8). Although epidural anesthesia does not directly impair central thermoregulatory control (6), the core temperature triggering shivering is ~0.5°C below normal values (8). Why epidural anesthesia should induce regulatory tolerance for hypothermia that normally would trigger vigorous shivering remains unknown. Tolerance may result because, at the lower-body skin temperatures observed in this study, cutaneous cold receptors fire tonically (29). Anesthetic-induced block of cold receptors might thus be perceived by the cold-defense portion of the central regulatory system as a markedly increased leg skin temperature. Such an increase in apparent (as opposed to real) leg temperature is consistent with the observation that induction of epidural anesthesia increases overall perception of warmth (8).

Warm receptors also fire tonically at this temperature but probably contribute less to cold defenses than do cold receptors. It thus is quite likely that the perceived increase in skin temperature applies only to the portion of the regulatory system controlling cold responses. In

anesthesia are shown in Figure 2. The thermoregulatory response thresholds under each condition, and the differences produced by spinal anesthesia, are summarized in Table 2.

Spinal anesthesia significantly decreased the thresholds for vasoconstriction and shivering, and the decrease in each was ~0.5°C. Consequently, the vasoconstriction-to-shivering range was not altered by anesthesia. The sweating-to-vasoconstriction interthreshold range was 0.9 ± 0.6°C during spinal anesthesia.
The vasoconstriction and shivering thresholds indeed Cohn (9) tested thermoregulatory responses to spinal and Cohn (9). Again, observed differences may reflect were reduced comparably in our volunteers, indicating that patterns of thermoregulatory impairment are similar during spinal and epidural anesthesia. The sweating threshold without anesthesia, and the ranges derived from this value, should not be compared directly to the other thresholds because perceived lower body temperature differed during this trial. Asterisks (*) indicate statistically significant differences between the two study days.

<table>
<thead>
<tr>
<th>Thermoregulatory response (°C)</th>
<th>No spinal</th>
<th>Spinal</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sweating</td>
<td>37.3 ± 0.3</td>
<td>37.6 ± 0.3</td>
<td>-0.3 ± 0.3</td>
</tr>
<tr>
<td>Vasoconstriction</td>
<td>37.0 ± 0.2</td>
<td>36.6 ± 0.4*</td>
<td>0.4 ± 0.3</td>
</tr>
<tr>
<td>Shivering</td>
<td>36.5 ± 0.3</td>
<td>36.0 ± 0.5*</td>
<td>0.5 ± 0.2</td>
</tr>
<tr>
<td>Sweating-to-constriction</td>
<td>0.3 ± 0.3</td>
<td>0.9 ± 0.6</td>
<td>-0.6 ± 0.6</td>
</tr>
<tr>
<td>Constriction-to-shivering</td>
<td>0.5 ± 0.2</td>
<td>0.6 ± 0.4</td>
<td>-0.1 ± 0.2</td>
</tr>
<tr>
<td>Sweating-to-shivering</td>
<td>0.7 ± 0.3</td>
<td>1.5 ± 0.6*</td>
<td>-0.8 ± 0.5</td>
</tr>
</tbody>
</table>

Spinal anesthesia significantly decreases the thresholds for vasoconstriction and shivering, and the decrease in each was ~0.5°C. Consequently, the vasoconstriction-to-shivering range also decreased ~0.5°C with and without anesthesia. The sweating-to-vasoconstriction interthreshold range was 0.9 ± 0.6°C during spinal anesthesia. The sweating threshold without anesthesia, and the ranges derived from this value, should not be compared directly to the other thresholds because perceived lower body temperature differed during this trial. Asterisks (*) indicate statistically significant differences between the two study days.

Table 2. Thresholds and Ranges With and Without Spinal Anesthesia

To the extent that regional anesthesia induces an apparent increase in leg skin temperature, the cold-response portion of the thermoregulatory system would tolerate more hypothermia than usual. The critical expression of such tolerance would be an unusually low shivering threshold. Our data confirm Roe and Cohn's (9) critical observation that the shivering threshold is reduced significantly during spinal anesthesia. The slightly greater reduction they observed (~1°C vs ~0.5°C) likely reflects variability in the study populations or protocols. For example, we evaluated young, unsedated female volunteers, whereas Roe and Cohn (9) tested thermoregulatory responses to spinal anesthesia in patients of unspecified age, who perhaps were given sedative adjuvants.

The autonomic thermoregulatory responses to cold are usually similarly integrated. A reduced shivering threshold should, therefore, be accompanied by a comparable reduction in the threshold for vasoconstriction. The vasoconstriction and shivering thresholds indeed were reduced comparably in our volunteers, indicating that patterns of thermoregulatory impairment are similar during spinal and epidural anesthesia.

Our vasoconstriction data contrast with those of Roe and Cohn (9). Again, observed differences may reflect the populations studied or contrasting experimental protocols. But more importantly, patients in that study may not have been given sufficient intravenous fluid to fully compensate for anesthetic-induced vasodilation. Peripheral vasoconstriction is not uniquely a thermoregulatory response: there is a substantial synergy between hypovolemia and cold-induced vasoconstriction (30). Thus, even slight relative hypovolemia after induction of spinal anesthesia might have triggered vasoconstriction, producing an artifically high threshold. To avoid potentially confounding effects of relative hypovolemia, we administered 1000 mL of fluid during induction of spinal anesthesia. Additionally, we confirmed that peripheral vasodilation persisted at least 30 min before proceeding with the study. An additional potential problem with the previous report is that forehead skin temperature was used as an index of vasoconstriction. This measure is not particularly specific and lacks the precision of plethysmography.

Thermoregulatory response thresholds must be considered at a particular skin temperature, because cutaneous sensation is an important afferent to the central regulatory system (1,23,31). Consequently, we actively controlled skin temperatures throughout the study. Upper body skin temperature was kept nearly constant throughout all study periods. Lower body skin temperatures were comparable during vasoconstriction and shivering, with and without spinal anesthesia.

To prevent cutaneous cold input during our evaluation of vasoconstriction and shivering thresholds, the core was cooled directly by central venous administration of cold fluid. However, we have yet to devise a method for comparably warming the core in humans without increasing skin temperature. Consequently, lower body skin temperatures were elevated significantly during both sweating trials, but presumably cutaneous warming was not perceived directly by the central thermoregulatory system during spinal anesthesia. The results necessary to answer our study questions (all thresholds during spinal anesthesia and the vasoconstriction and shivering threshold without anesthesia), can thus be freely compared.

However, our evaluation of the sweating threshold without anesthesia is confounded by a perceived difference in lower body skin temperature and should be compared to values during spinal anesthesia with caution. The contribution of increased lower body temperature to regulatory integration is not known precisely, but probably is less than 10%. The ~5°C increase in lower body temperature during this study thus may have reduced the measured sweating threshold ~0.5°C. Consequently, the sweating threshold without anesthesia may have been ~0.5°C higher than actually recorded, were lower body skin temperature similar to that during the other trials. Applying this theoretical compensation, the interthreshold ranges would have been comparable with or without spinal anesthesia. Nonetheless, the observed interthreshold range during
spinal anesthesia is high compared with the entire previously reported sweating-to-shivering range (5). It thus remains likely that spinal anesthesia increases the interthreshold range, rather than synchronously decreasing all thermoregulatory response thresholds. The observed difference in response thresholds with and without spinal anesthesia were small, but statistically significantly different at the P < 0.01 level. Seven volunteers were, therefore, sufficient to answer our study questions. Hypothermia during regional anesthesia results from several interacting causes. Impaired central thermoregulation is only one of these factors and, in some cases, not the most important. These data should, therefore, be extrapolated to clinical situations with recognition of the other factors contributing to observed thermal perturbations. The extent to which responses would differ in men remains unknown, but the general pattern of impairment produced by spinal anesthesia likely is similar in men and women.

In summary, the sweating-to-vasoconstriction range was 0.9 ± 0.6°C during spinal anesthesia, but these data cannot be compared directly with the interthreshold without anesthesia because of differences in perceived lower body temperature. Spinal anesthesia reduced the threshold for shivering = 0.5°C, which is similar to the reduction produced by epidural anesthesia. The vasoconstriction threshold also was decreased = 0.5°C, as expected from the normally similar integration of cold responses. The synchronous decrease in the shivering and vasoconstriction thresholds during spinal anesthesia is consistent with thermoregulatory impairment resulting from altered afferent thermal input.

We would like to thank Ohmeda, Inc., for loan of a Modulus® CD integrated anesthesia machine; Mallinkrodt Anesthesia Products, Inc., for donation of the thermocouples we used; and Hermes Systems, Inc., for loan of an IdaCare™ automatic record-keeping system.

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