Shivering during Epidural Anesthesia

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The authors tested the hypotheses that during epidural anesthesia: 1) shivering-like tremor is primarily normal thermoregulatory shivering; 2) hypothermia does not produce a subjective sensation of cold; and 3) injectate temperature does not influence tremor intensity. An epidural catheter was inserted into ten healthy, nonpregnant volunteers randomly assigned to skin-surface warming below the T10 dermatome (warmed group) or no extra warming (unwarmed group). Each volunteer was given two 30-ml epidural injections of 1% lidocaine (16.0 ± 4.7°C and 40.6 ± 0.7°C at the catheter tip), in random order separated by at least 3 h. Skin-temperature gradients (forearm–fingertip) and tympanic membrane and average skin temperatures were recorded; significant vasoconstriction was prospectively defined as a gradient ≥ 4°C. Integrated electromyographic (EMG) intensity was recorded from four upper-body muscles. Overall thermal comfort was evaluated using a visual analog scale. Tympanic membrane temperatures decreased significantly in the unwarmed group (n = 6). Tremor occurred following ten of 12 injections in unwarmed volunteers, but only following one of eight injections in the warmed group. Integrated EMG intensity did not differ significantly following epidural injection of warm and cold lidocaine: tremor started when tympanic membrane temperature decreased about 0.5°C and continued until central temperature returned to within 0.5°C of control. Tremor always was preceded by hypothermia and vasoconstriction in the arms. Thermal comfort increased in both groups after epidural injection, with maximal comfort occurring at the lowest tympanic temperatures. These data suggest that: 1) tremor during epidural anesthesia is primarily normal thermoregulatory shivering; 2) epidural injectate temperature does not influence tremor intensity; and 3) central hypothermia does not necessarily produce a subjective sensation of cold. (Key words: Anesthesia techniques: epidural. Brain: hypothalamus. Hypothermia. Temperature, measurement: epidural; skin; tympanic membrane. Temperature, regulation: setpoint; threshold.)

SHIVERING-LIKE TREMOR occurs during approximately 30% of epidural anesthetics.1–4† The etiology of this tremor remains unknown, but its effects include increased metabolic rate (=200%) and plasma catecholamine concentrations, and patient discomfort.5,6 It is commonly attributed to normal thermoregulatory shivering, but appears to occur independently of changes in central or skin-surface temperature or sensations of cold.7–9§ Proposed nonthermoregulatory etiologies include systemic absorption of epidurally administered anesthetic and central transfer of epidural anesthetic via cerebrospinal fluid.9

A possible thermoregulatory etiology for tremor during epidural anesthesia is stimulation of spinal cord temperature receptors by injection of cold local anesthetic. Changes in spinal cord temperature elicit thermoregulatory responses in all mammals and birds so far tested,10–12 and may do so in humans. Results thus far conflict: two studies report that tremor is more common after cold versus warm epidural injections,1,12 while three others demonstrate no such relationship.4,13‡

We tested the hypotheses that: 1) tremor during epidural anesthesia is primarily normal thermoregulatory shivering, resulting from central hypothermia and preceded by vasoconstriction in the arms; 2) hypothermia during epidural anesthesia does not produce a subjective sensation of cold; and 3) the temperature of epidurally injected anesthetic does not influence tremor intensity. Because thermoregulatory responses may differ among individuals and because surgery per se may be a factor, we studied healthy volunteers given epidural injections of warm and cold lidocaine. To evaluate the contribution of central hypothermia, heat loss from the epidural-induced sympathectomy was prevented by slightly warming the legs in one group.

Materials and Methods

With approval from the University of California Committee on Human Research, and written informed consent, we studied ten healthy volunteers aged 25–38 yr. None were obese, taking medication, or had a history of thyroid disease, diabetes, dysautonomia, or Raynaud’s syndrome. Volunteers refrained from coffee, tea, and food during the 8 h preceding the study.

Using sterile technique, the skin and subcutaneous tissues over the L3–4 interspace in each volunteer was infiltrated with 4 ml 0.75% bupivacaine. The tip of a 16-G Touhy needle was advanced into the epidural space, which was identified using loss of resistance to injection of air. A 30-cm-long Portex® catheter containing a calibrated thermocouple at the tip was then advanced 2–3 cm into

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the epidural space and the needle removed. The laboratory-produced, type-T (copper-constantan) thermocouple was constructed of two wires 0.11 mm in diameter and did not occlude the catheter lumen (0.8-mm ID). The epidural catheter was insulated and isolated from skin with 1-cm-thick foam. Volunteers wore shorts and a light cotton shirt and rested supine on a padded operating room table. Ambient temperatures were maintained at 22.2 ± 1.1°C (SD).

Volunteers were randomly assigned to skin-surface warming below the T-10 dermatome (warmed group) or no extra warming (unwarmed group). Skin was warmed using a Bair Hugger® forced-air warmer (Augustine Medical Inc., Eden Prairie, Minnesota) set to "low." The Bair Hugger supplied air at ≈ 35°C to a disposable blanket, creating a shell of warm air around the lower body via flow through linear channels and small openings on the underside. Skin not covered by the Bair Hugger® or clothing was exposed to room air.

Following a 15-min control period, each volunteer was given a cold (0°C) and warm (50°C), 30-ml epidural injection of 1% lidocaine without epinephrine at a rate of 15 ml/min in random order. Neither the subjects nor the investigator assessing subjective responses were aware of the injectate temperatures; at least 3 h elapsed between injections. The extent of blockade was evaluated using thermal sensitivity to an alcohol-soaked gauze pad. Blood pressure was maintained within 20% of control values by iv administration of 500-1,000 ml lactated Ringer's solution warmed to 37°C.

Tympanic membrane, ambient, skin-surface temperature gradients (forearm–fingertip), and average skin temperatures were determined using Mon-a-Therm® (St. Louis, Missouri) thermocouples and Model 8700 electronic thermometers (Mallinckrodt, St. Louis, Missouri). Area-weighted, average skin-surface temperatures were calculated by assigning the following regional percentages: head, 6%; upper arms, 9%; forearms, 6%; palms, 2.5%; fingers, 2%; back, 19%; chest and abdomen, 19%; thighs, 19%; calves, 11.5%; feet, 6%. The laboratory-produced epidural thermocouple also was connected to one of the thermometers to determine temperature at the end of the catheter. The signals from each channel were digitized at 1 Hz in 48-s epochs using an NB-MIO-16® analog-digital converter (National Instruments, Inc., Austin, Texas) connected to a Macintosh® II computer (Apple, Inc., Cupertino, California). Temperatures were averaged, displayed on the Macintosh® screen, and recorded on a hard disk at 2-min intervals.

The tympanic thermocouples were cotton-covered flexible probes placed in contact with the tympanic membrane. Skin temperatures were measured using self-sticking, 1-cm diameter disks. Volunteers were considered hypothermic when tympanic membrane temperature decreased ≥0.4°C because this change usually provokes thermoregulatory responses in unanesthetized humans. Peripheral vasoconstriction was quantified using skin-surface temperature gradients (forearm–fingertip) on the exposed arm. This indirect measure of peripheral blood flow correlates well with blood flow measured using laser Doppler flowmetry and volume plethysmography. The iv catheter and the blood pressure cuff were placed on the opposite arm. As in our previous studies, significant vasoconstriction was prospectively defined as a skin-temperature arm gradient ≥ 4°C.

Following mild skin abrasion and degreasing, Red Dot® silver/silver chloride monitoring electrodes (3M, Inc., St. Paul, Minnesota) were positioned to record the electrical activity of the deltoidus, trapezius, pectoralis, and rhomboideus muscles. The electrodes were placed 4 cm apart and oriented in the direction of the muscle fibers. Electromyographic (EMG) signals were amplified 20,000-fold using a Grass® model P511 amplifier (Grass Instrument Company, Quincy, Massachusetts) and digitized at 512 Hz in 48-s epochs using a second NB-MIO-16® analog-digital converter. Integrated EMG intensity, in epochs free of movement and electrical artifact, was calculated in real time on a Macintosh® II computer by a program written by the authors using LabVIEW® 1.2 programming language (National Instruments, Inc.). Only these artifact-free segments are included in the data analyses. Our computerized temperature and electromyographic data acquisition system has previously been described in detail.

Overall thermal comfort was evaluated at 5–10-min intervals using 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. A new, unmarked scale was used for each assessment. When the study was finished, subjects were asked to guess which epidural injection was warm and which was cold.

Electromyographic, temperature, and subjective data for each volunteer, collected at 2-min intervals, were averaged in 15-min epochs. Unpaired, two-tailed t-tests were used for between-group comparisons of tympanic temperature and EMG intensity. Differences were considered statistically significant when P < 0.01.

Two-tailed t tests also were used to compare control skin-surface temperatures in the warmed versus unwarmed groups (unpaired) and before versus after epidural injections (paired) in both groups. The number of injections producing tremor in the warmed and unwarmed groups

† Unpublished data.
** The authors will make this program available to interested investigators.
was compared using chi-square with Yates' correction for continuity. Integrated EMG intensity, tympanic temperature changes, and visual analog scale values were averaged in 15-min periods for each volunteer and analyzed using repeated-measures ANOVA and Dunnett's test for intergroup comparison. These differences were considered statistically significant when \( P < 0.05 \). Data are expressed as means ± standard deviations.

**Results**

Our two study groups did not differ morphometrically (table 1). One volunteer participated on different occasions in both warmed and unwarmed groups. Volunteers were vasoconstricted (skin-temperature gradients > 4° C) before ten of 12 epidural injections in the unwarmed group and six of eight injections in the warm group.

Temperature at the end of the epidural catheter, which was within 0.1° C of tympanic temperature during the control period, decreased to 16 ± 4.7° C during the cold injections and increased to 40.6 ± 0.7° C with warm injections. The speed of onset and maximum extent of sensory blockade was similar in both groups (T9 ± 1 segment in unwarmed group, and T17 ± 3 segments in the warm). Volunteers were unable to determine whether epidural injections were warm or cold: six of ten guessed correctly after the warm injection and four of 10 guessed correctly the following cold injections.

Tympanic membrane temperatures decreased significantly in the unwarmed group (n = 6) (fig. 1). Shivering-like tremor followed ten of 12 injections (five warm and five cold) in unwarmed volunteers, but only one of eight injections in the warmed group (\( P \leq 0.05 \)). The volunteer in the warm group who demonstrated tremor following a cold injection weighed only 45 kg; she became slightly hypothermic (0.3° C) despite leg warming, was profoundly vasoconstricted (skin-temperature gradient of 8.5° C), and felt "miserably cold" during this part of the study.

The percentage increase in integrated EMG intensity did not differ significantly after warm and cold injections in unwarmed volunteers (fig. 2). Inclusion of the single volunteer in the warm group in whom tremor occurred did not alter the statistical significance of these results. Thermal comfort increased following each epidural injection in the unwarmed volunteers; maximal comfort occurred at the lowest central temperature (fig. 3). Thermal comfort in the warmed volunteers averaged 49 ± 1 mm and did not change significantly through the study.

Because responses were similar following warm and cold epidural injections, these data were combined during the rest of the analysis. EMG patterns were not formally analyzed in this study. Nonetheless, the typical, slow "waxing and waning" pattern of normal shivering was common, while clonic tremor patterns were not.

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**Table 1. Morphometric Characteristics of the Volunteers**

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>M/F</th>
<th>Age (y)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Warm</td>
<td>4</td>
<td>2/2</td>
<td>29 ± 2</td>
<td>62 ± 14</td>
<td>162 ± 9</td>
</tr>
<tr>
<td>Cold</td>
<td>6</td>
<td>4/2</td>
<td>29 ± 6</td>
<td>65 ± 9</td>
<td>165 ± 7</td>
</tr>
</tbody>
</table>

None of the morphometric characteristics were statistically different among the groups.

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**Fig. 1.** Changes in tympanic membrane temperatures in volunteers given no extra warming (unwarmed legs: 12 injections in six volunteers) and those whose legs were warmed with air at ∼35° C (warmed legs: eight injections in four volunteers). Warm and cold epidural injections (grouped in this figure) were given after a 15-min control period (mean ± SD). *Tympanic temperatures that were significantly lower in the group with unwarmed legs (\( P < 0.01 \)).

**Fig. 2.** The percentage increase in integrated EMG intensity following epidural injection of warm (∼41° C at tip of catheter) and cold (∼17° C) lidocaine following 12 epidural injections in six volunteers with unwarmed legs (mean ± SD). Lidocaine (50 ml, 1%) was injected after a 15-min control period. Shivering-like tremor occurred following ten of 12 injections and its intensity was not dependant on injectate temperature. Tremor intensity was similar following warm and cold injections at all times (\( P = NS \)).
SHIVERING DURING EPIDURAL ANESTHESIA

![Graph showing changes in tympanic membrane temperatures, percentage increase in tremor intensity, and thermal comfort (millimeter on a visual analog scale) following 12 epidural lidocaine injections in six volunteers with unwarmed legs (mean ± SD). Epidural injections were given after a 15-min control period. Changes in these variables were similar following warm and cold epidural injections, so the data are combined in this figure. Tremor started when tympanic temperature decreased about 0.5°C and continued until central temperature returned to within 0.5°C of control. Thermal comfort increased following each epidural injection in each volunteer; maximal comfort occurred at the lowest central temperature. *Statistically significant changes from control (P < 0.01) for each parameter.]

Tremor started when tympanic membrane temperature decreased by approximately 0.5°C and continued until tympanic temperature returned to within 0.5°C of control. In every case, tremor was preceded by significant thermoregulatory peripheral vasoconstriction (skin-temperature gradient > 4°C).

Skin temperatures during the control periods were significantly higher in the warmed than unwarmed group (P ≤ 0.01). Average leg skin temperature increased significantly following epidural injection in both groups. However, average arm (and back, chest, and head) temperatures decreased enough in the unwarmed group to decrease average total skin temperature significantly (P ≤ 0.05) (fig. 4).

**Discussion**

Our volunteers felt warmer during the 45 min following epidural injections than in the preceding control period. Increased thermal comfort was expected because a warm sensation in the legs typically accompanies major conduction anesthesia. However, tympanic temperatures (in the unwarmed group) decreased significantly during the same period, and maximum thermal comfort coincided with minimum central temperature and onset of shivering.

Subjective thermal sensation and physiologic responses (e.g., vasoconstriction, shivering) are controlled by different hypothalamic structures and do not necessarily respond synchronously. For example, monkeys can be trained to modify their environment behaviorally in response to skin-surface temperature perturbations (e.g., push a lever to receive a blast of cool air in a warm environment). In contrast, they respond poorly to isolated hypothalamic temperature changes produced by an implanted thermode. These observations suggest that subjective thermal sensation is mostly controlled by skin-surface temperature.

It is therefore surprising that our subjects reported improved thermal comfort (warmth) in spite of a decrease in average skin-surface temperature following induction of epidural anesthesia. This discrepancy may be due to blockade of afferent input via A-delta fibers (cold sensation) and C-fibers (warm sensation) by epidural anesthesia because cutaneous cold receptors fire tonically at comfortable ambient temperatures whereas warm receptors are quiescent. Anesthetic-induced absence of tonic cold input may thus be perceived as a warm sensation.

Although our study does not eliminate other causes of tremor during epidural anesthesia, it supports a thermoregulatory mechanism because tremor in all but one...
instance was preceded by central hypothermia. Furthermore, EMG patterns were qualitatively similar to those produced by normal thermoregulatory shivering in cold-exposed volunteers. It is thus likely that most tremor during epidural anesthesia in nonpregnant subjects is normal shivering.

Previous studies and our current results do not support an influence of epidural injectate temperature on tremor intensity in nonpregnant subjects. In contrast, tremor during epidural anesthesia for cesarean delivery was significantly more common after cold (4°C) than warm (37°C) bupivacaine administration. Additionally, epidural injection of warm anesthetic reportedly stops shivering induced by cold injections in postpartum women. The possibility remains, therefore, that pregnancy enhances the contribution of spinal cord thermoregulatory input. The most attractive hypothesis is that tremor during epidural anesthesia is not only caused by the convergence of several thermal stimuli, as previously suggested, but that there is a synergistic effect of pregnancy and local anesthesia.

Hypothermia during epidural anesthesia is thought to result from anesthetic-induced sympatholysis that increases leg temperature and thus heat loss to the environment. Although leg temperature in our volunteers increased following induction of epidural anesthesia, skin temperature in the arms, head, and back decreased enough to make area-weighted, average skin temperature decrease slightly in the unwarmed group. (Decreased skin perfusion in the upper body is partially thermoregulatory in response to central hypothermia, but also involves mechanisms not yet fully characterized.) It is thus likely that total heat loss to the environment changed little.

Central hypothermia can occur without an increase in environmental heat loss if metabolic heat production decreases. Nonshivering thermogenesis (an increase in metabolic heat production) increases the basal metabolic rate only 25–40% in adult humans. Our study did not evaluate nonshivering thermogenesis, which requires measurement of systemic oxygen consumption. However, a small decrease in heat production alone would not account for the hypothermia we observed.

Most likely, hypothermia in our volunteers resulted from redistribution of heat within the body. Mean body temperature remains constant when metabolic heat production equals environmental heat loss. Skin temperatures are typically near 33°C, and central temperatures are not reached until ~2.5 cm below the surface. Consequently, anatomic mean body temperature must include a significant correction for relatively cool tissues near the skin surface. Peripheral warming can therefore decrease central temperature (without increasing environmental heat loss) via redistribution of heat within the body.

In summary, central hypothermia was not accompanied by a subjective sensation of cold, probably because epidural lidocaine inhibited tonic cutaneous cold receptors input to hypothalamic thermoregulatory centers. Shivering followed central hypothermia and vasoconstriction in the arms, suggesting a thermoregulatory etiology, but not one mediated by epidural thermal receptors. Hypothermia following epidural injections may, in part, result from redistribution of heat within the body.

References


**Physiologic mean body temperature (weighted for thermoregulatory responses rather than heat content) has different coefficients.**