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Thermoregulatory Thresholds during Epidural and Spinal Anesthesia

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Background: **There are significant physiologic differences between spinal and epidural anesthesia. Consequently, these two types of regional anesthesia may influence thermoregulatory processing differently. Accordingly, in volunteers and in patients, we tested the null hypothesis that the core-temperature thresholds triggering thermoregulatory sweating, vasoconstriction, and shivering are similar during epidural and spinal anesthesia.**

Methods: **Six male volunteers participated on three consecutive study days: epidural or spinal anesthesia were randomly assigned on the 1st and 3rd days (≈ T10 level); no anesthesia was given on the 2nd day. On each day, the volunteers were initially warmed until they started to sweat, and subsequently cooled by central venous infusion of cold fluid until they shivered. Mean skin temperature was kept constant near 36°C throughout each study. The tympanic membrane temperatures triggering a sweating rate of 40 $g \cdot m^{-2} \cdot h^{-1}$, a finger flow less than 0.1 ml/min, and a marked and sustained increase in oxygen consumption (≈ 30%) were considered the thermoregulatory thresholds for sweating, vasoconstriction, and shivering, respectively. Twenty-one patients were randomly assigned to receive epidural (n = 10) or spinal (n = 11) anesthesia for knee and calf surgery (≈ T10 level). As in the volunteers, the shivering threshold was defined as the tympanic membrane temperature triggering a sustained increase in oxygen consumption.**

Results: **The thresholds and ranges were similar during epidural and spinal anesthesia in the volunteers. However, the sweating-to-vasoconstriction (interthreshold) range, the vasoconstriction-to-shivering range, and the sweating-to-shivering range all were significantly increased by regional anesthesia. The shivering thresholds in patients assigned to epidural and spinal anesthesia were virtually identical.**

Conclusions: **Comparable sweating, vasoconstriction, and shivering thresholds during epidural and spinal anesthesia suggest that thermoregulatory processing is similar during each type of regional anesthesia. However, thermoregulatory control was impaired during regional anesthesia, as indicated by the significantly enlarged interthreshold and sweating-to-shivering ranges. (Key words: Anesthetic techniques: epidural; spinal. Temperature, regulation: setpoint; shivering; sweating; threshold; vasoconstriction. Thermoregulation.)**

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EPIDURAL anesthesia prevents surgical pain, at least in part, by decreasing conduction in spinal nerve roots. Consistent with this mechanism, segmental blocks are frequently observed in patients given epidural anesthesia. Although considerable epidurally administered local anesthetic does reach the neuroaxis,[1,2] the clinical importance of this mechanism remains unclear. In contrast, local anesthetic injected into the subarachnoid space certainly directly impairs spinal cord function. Spinal anesthesia also tends to provide a "denser" block than epidural anesthesia. Peripheral thermal afferent signals are initially integrated in the spinal cord.[3,4] Furthermore, temperature of the spinal cord *per se* is an important thermal afferent signal.[4–6] Consequently, spinal and epidural anesthesia may influence thermoregulatory processing differently. Accordingly, we tested the null hypothesis that the core-temperature thresholds triggering thermoregulatory sweating, va-

soconstriction, and shivering are similar during epidural and spinal anesthesia.

Because both skin and core temperatures contribute to thermoregulatory responses,[7-9] core temperature thresholds initiating protective thermoregulatory responses should be compared at similar skin temperatures. We recently developed a technique whereby sweating, vasoconstriction, and shivering thresholds each can be determined at constant skin temperature. Using this method in volunteers, we directly compared thermoregulatory response thresholds during epidural and spinal anesthesia to those occurring without anesthesia. Additionally, we compared the shivering thresholds under clinical conditions in patients randomly assigned to epidural or spinal anesthesia.

Materials and Methods

All studies were undertaken with approval of the local Ethics Committees and informed consent from the subjects. None of the subjects was obese, taking medication, or had a history of thyroid disease, dysautonomia, Raynaud's syndrome, or malignant hyperthermia. None of the participants was given preanesthetic medication.

Volunteers

We studied six men aged 25 ± 3 yr (mean ± standard deviation), having a height of 177 ± 5 cm, and a weight of 66 ± 5 kg. The percentage of body fat in the volunteers was 16 ± 3, as determined using infrared interactance (Futrex 1000, Futrex, Hagerstown, MD).

The volunteers each participated on three consecutive study days: epidural anesthesia and spinal anesthesia were randomly assigned on the 1st and 3rd days, and no anesthesia was given on the 2nd day. On each study day, the volunteers were warmed *via* the skin surface until sweating was detected and then cooled by central venous administration of cold lactated Ringer's solution until vasoconstriction and shivering were triggered.

Protocol. Volunteers fasted during the 8 h preceding each study, which started at approximately 9:30 AM. They were minimally clothed and reclined on their backs on a standard operating room table. A circulating water blanket set at 40°C (Blanketrol II, Maxi-Therm blanket 276, Cincinnati Sub-Zero, Cincinnati, OH) was positioned beneath the upper body. Ambient temperature was maintained at 21.3 ± 0.9°C throughout the studies.

On one of the study days, a catheter was advanced 2–3 cm into the L3–L4 epidural space. The catheter was then injected with 3 ml of 2% 2-chloroprocaine (Chloroprocaine HCl, *United States Pharmacopeia*, Abbott Laboratories, North Chicago, IL) with epinephrine 1:100,000. This test dose was followed in 5 min by slow administration of 15–20 ml 2% 2-chloroprocaine without epinephrine. During induction of epidural anesthesia, 1,000 ml lactated Ringer's solution warmed to 37°C was administered intravenously to minimize sympathectomy-induced vascular volume shifts.

The initial volume of 2-chloroprocaine was chosen based on each volunteer's height and calculated to produce a dermatomal level of sensory blockade near T10, as determined by loss of cutaneous cold sensation and response to pinprick. Subsequently, a continuous infusion of 2% 2-chloroprocaine was administered at a rate of 13–18 ml/h to maintain a comparable sensory block level. 2-Chloroprocaine was chosen as the epidural anesthetic because it is rapidly metabolized in plasma. Systemic absorption of the anesthetic, and subsequent recirculation to the brain, thus were unlikely to influence centrally mediated thermoregulatory responses. The volunteers were observed for at least 30 min after induction of epidural anesthesia to ensure hemodynamic stability.

On the day on which spinal anesthesia was to be given, volunteers were given 1,000 ml warmed lactated Ringer's solution intravenously. Anesthesia then was induced by subarachnoid administration of 4 ml 0.5% isobaric bupivacaine through a 24-G Sprotte needle inserted *via* the L3–L4 intervertebral space. The level of the resulting sensory blockade was determined shortly after induction of anesthesia and at 1-h intervals throughout the study, using the response to cold sensation. The volunteers were observed for at least 30 min after induction of anesthesia to confirm continued peripheral vasodilation and a stable blood pressure.

Volunteers were warmed by increasing the temperature of the circulating-water mattress to 42°C and adding a Bair Hugger forced-air warmer set on "medium" (≈ 40°C) (model 525 cover and model 200 warmer, Augustine, Eden Prairie, MN). The forced-air warmer was modified to allow continuous temperature control by a rheostat, and the temperature was increased as necessary to maintain upper-body skin temperature constant. Active warming was applied only to upper body; the lower body was covered by cotton blankets. Forced-air and circulating-water heating was

continued until the sweating threshold was identified. Mean skin temperature at the sweating threshold subsequently was maintained for the duration of the study by adjusting the circulating-water and forced-air warmers. Core hypothermia then was induced by central venous administration of lactated Ringer's solution cooled to approximately 4°C.[10] Core warming and cooling was restricted to less than 1.7°C/h, because such rates to not trigger dynamic thermoregulatory responses.[11]

On the 2nd study day (no anesthesia), the protocol was similar except anesthesia was not administered.

Monitoring. Core temperature was measured at the right tympanic membrane using Mon-a-Therm thermocouples (Mallinckrodt, St. Louis, MO). The aural probe then was inserted by volunteers until they felt the thermocouple touch the tympanic membrane; appropriate placement was confirmed when volunteers easily detected a gentle rubbing of the attached wire. The probe was then securely taped in place, the aural canal occluded with cotton, and a gauze bandage positioned over the external ear. Tympanic membrane temperatures correlate well with distal esophageal temperatures during anesthesia.[12,13]

Area-weighted, upper-body skin temperature was computed from measurements at 7 sites and lower-body skin temperature was similarly derived from 8 sites as previously reported.[14] Core and skin-surface temperatures were recorded from thermocouples connected to two calibrated 16-channel electronic thermometers (Iso-Thermex, Columbus Instruments, Columbus, OH) having an accuracy of 0.1°C and a precision of 0.01°C. Individual and computed average temperatures were displayed at 1-s intervals.

The rate of sweating was determined using a ventilated capsule situated on the chest. Anhydrous oxygen was flushed across a 6-cm-diameter circle of skin, surrounded by an air-tight adhesive ostomy appliance (stock 3706 and 3806, Hollister Products, Libertyville, IL), at a rate of 2.0 l/min. Cutaneous water loss (in grams per squared meter per hour) was calculated from the gas flow rate (FMA-5000, Omega Engineering, Stamford, CT) and gas temperature and relative humidity (HX93, Omega Engineering). We have previously described the details of this measurement technique.[15] As in previous studies,[16] we considered the core temperature triggering a sweating rate of 40 $g \cdot m^{-2} \cdot h^{-1}$ to be the threshold for sweating.

Absolute right middle fingertip blood flow, resulting primarily from arteriovenous shunt flow, was quantified using venous-occlusion volume plethysmography at 5-min intervals. Volume plethysmography is considered the most reliable measure of extremity blood flow; we previously described our technique for determining finger blood flow in detail.[17] The vasoconstriction threshold was defined as the core temperature triggering a sudden and sustained decrease of finger flow, usually to less than 0.1 ml/min.

Shivering was evaluated by oxygen consumption, which was measured using a Deltatrac (Datex Instrumentarium, Helsinki, Finland). The monitor measures the oxygen concentration in exhaust gas drawn at a constant flow of 40 l/min through a clear plastic canopy placed over the volunteers head. Oxygen uptake is determined from the difference in oxygen content between the mixed exhaust gas and the inspired ambient air. Measurements were averaged over 1-min intervals, and recorded every five min. Although electromyographic analysis is useful for evaluating tremor patterns, oxygen consumption is the most reliable method of quantifying shivering intensity. The tympanic membrane temperature triggering a marked (*i.e.*, 30%) and sustained increase in oxygen consumption was considered the threshold for shivering. The threshold in each volunteer was determined by an investigator masked to core temperature and treatment.

Heart rate was monitored continuously using three-lead electrocardiography. Oxyhemoglobin saturation was measured continuously using pulse oximetry and blood pressure was determined oscillometrically at 5-min intervals at the left upper arm using the Modulus CD Anesthesia System (Ohmeda, Madison, WI). Analog and serial thermoregulatory data were recorded at 5-min intervals, using a modification of a previously described data-acquisition system.[14] Anesthetic data were recorded using IdaCare version 1.3 (Hermes Systems, Premier Anesthesia Systems, Atlanta, GA), which is Macintosh (Apple, Cupertino, CA)-based patient information management software. The two systems operated asynchronously on a Macintosh II fx computer.

Data Analysis. The average upper- and lower-body skin temperatures at the onset of sweating were considered baseline values. The difference between the sweating and vasoconstriction thresholds in each individual defined the sweating-to-vasoconstriction (interthreshold) range. Similarly, the difference between the core temperatures triggering sweating and shivering were considered the sweating-to-shivering range and the difference between the vasoconstriction and shiv-

ering thresholds was considered the vasoconstriction-to-shivering range.

Upper- and lower-body skin temperatures at each threshold for the control, epidural anesthesia, and spinal anesthesia days were compared using repeated-measure analysis of variance and Scheffé's F tests. The thresholds and ranges on each study day and core cooling and warming rates were similarly compared. All values are expressed as means ± standard deviations; differences were considered significant when $P < 0.01$.

Patients

We studied 21 ASA physical status 1–3 patients who shivered while undergoing elective knee and calf surgery. We studied approximately 10 patients in each group because, based on the typical variability in thermoregulatory responses, this number would be sufficient to detect clinically important differences between the anesthetic techniques. Sequential qualifying and consenting patients were enrolled into the study. The operations started between 8:00 AM and noon.

Protocol. Upon arrival to the operating suite, 10 ml/kg of unwarmed intravenous fluid was administered. The patients were randomly assigned to receive epidural or spinal anesthesia. Epidural anesthesia was induced with 25 ml of 0.5% bupivacaine, and no further anesthetic was administered. Spinal anesthesia consisted of a single intrathecal bolus of 4 ml of 0.5% bupivacaine. An additional 500 ml of crystalloid and two mg ephedrine was administered intravenously to a single patient given spinal anesthesia whose systolic blood pressure decreased to less than 100 mmHg. The level of the resulting sensory blockade was determined shortly after induction of anesthesia and at 1-h intervals throughout the study, using the response to cold sensation.

The patients were covered with a single layer of surgical draping; no other warming measures were taken during the initial portion of the study. No sedatives were administered until the patients shivered. Once patients did shiver, they were actively rewarmed using a Bair Hugger forced-air system. Subsequent anesthetic management was left to the discretion of the responsible anesthesiologist.

Monitoring. Ambient temperature was measured using a bare-wire thermocouple positioned at the level of the patient, well away from any heat-producing equipment. Core temperature was measured at the tympanic membrane, as described above. Temperatures were measured using Yellow Springs Instruments thermistors (Yellow Springs, OH) and recorded at 5-min intervals.

Shivering was evaluated using whole-body oxygen consumption, as described above. A sustained 30% increase in oxygen consumption identified the shivering threshold. Heart rate was monitored continuously using three-lead electrocardiography. Oxyhemoglobin saturation was measured continuously using pulse oximetry, and blood pressure was determined oscillometrically at 5-min intervals.

Data Analysis. The preinduction fluid bolus was not considered part of the intraoperative fluid balance. Fluid administered during surgery was divided by the duration of surgery to produce the fluid administration rate. Ambient temperature for each patient was averaged over time, starting from induction of anesthesia.

Morphometric data, ambient temperature, fluid administration rate, initial core temperature, time to onset of shivering, the shivering threshold, and mean skin temperature at the threshold were compared using two-tailed, unpaired t tests. All values are expressed as means ± standard deviation; differences were considered significant when $P < 0.05$.

Results

Volunteers

Epidural anesthesia usually produced a T10 dermatome (range T8–T12) sensory block level. Similarly spinal anesthesia typically produced a T10 level sensory blockade (range T8–T11) throughout the study. Core warming and cooling rates were comparable on the epidural anesthesia, spinal anesthesia, and no anesthesia days (table 1). There were no statistically significant differences in upper- or lower-body skin temperatures at any of the thresholds (table 2).

The vasoconstriction threshold was unchanged by epidural and spinal anesthesia. Regional anesthesia

Table 1. Core Cooling and Warming Rates in the Volunteers

Rate	No Anesthesia (°C/h)	Epidural (°C/h)	Spinal (°C/h)
Cooling	1.1 ± 0.2	0.8 ± 0.2	1.2 ± 0.2
Warming	0.7 ± 0.3	1.1 ± 0.1	0.7 ± 0.2

Values are mean ± SD.

There were no statistically significant or clinically important differences in the core cooling and warming rates on the different study days.

Table 2. Upper Body and Lower Body Skin-surface Temperatures in the Volunteers

	No Anesthesia (°C)	Epidural (°C)	Spinal (°C)
Upper body			
Sweating	36.5 ± 0.6	36.7 ± 0.3	36.9 ± 0.4
Vasoconstriction	36.6 ± 0.4	36.6 ± 0.2	36.6 ± 0.3
Shivering	36.2 ± 0.5	36.1 ± 0.4	35.8 ± 0.6
Lower body			
Sweating	35.6 ± 0.7	36.3 ± 0.4	36.4 ± 0.8
Vasoconstriction	36.0 ± 0.3	36.2 ± 0.5	36.2 ± 0.6
Shivering	35.4 ± 0.4	35.8 ± 0.6	35.8 ± 0.8

Values are mean ± SD. Average upper body and lower body, skin-surface temperatures at the sweating, vasoconstriction, and shivering thresholds on each study day. There were no statistically significant differences in upper body or lower body skin temperatures at the thresholds on each study day.

slightly increased the sweating threshold and slightly decreased the shivering threshold. Consequently, the sweating-to-vasoconstriction (interthreshold) range, the vasoconstriction-to-shivering range, and the sweating-to-shivering range all increased significantly compared to the no anesthesia day. The interthreshold ranges were 0.6 ± 0.2 and 0.6 ± 0.1°C, respectively, in the volunteers during epidural and spinal anesthesia; in contrast, the range was only 0.3 ± 0.1°C without anesthesia. The thresholds and ranges were similar during epidural and spinal anesthesia (table 3). Two of the volunteers complained of mild back pain, which was relieved by administration of oral analgesics.

Table 3. Thresholds and Ranges in the Volunteers

	No Anesthesia (°C)	Epidural (°C)	Spinal (°C)
Sweating	36.9 ± 0.2	37.1 ± 0.3	37.4 ± 0.3*
Vasoconstriction	36.6 ± 0.2	36.5 ± 0.2	36.7 ± 0.3
Shivering	35.7 ± 0.4	35.4 ± 0.5	35.5 ± 0.5
Sweating-to-constriction	0.3 ± 0.1	0.6 ± 0.2*	0.6 ± 0.1*
Vasoconstriction-to-shivering	0.9 ± 0.4	1.2 ± 0.5*	1.2 ± 0.4*
Sweating-to-shivering	1.2 ± 0.4	1.7 ± 0.5*	1.9 ± 0.4*

Values are mean ± SD. The sweating, vasoconstriction, and shivering thresholds under three different conditions: no anesthesia, epidural anesthesia, and spinal anesthesia. Shown also are the sweating-to-vasoconstriction (interthreshold) range, the vasoconstriction-to-shivering range, and the sweating-to-shivering range.

* Statistically significant differences *versus* no anesthesia.

Table 4. Morphometric Characteristics, Ambient Temperatures, Block Levels, and Fluid Administration Rates in the Patients

	Epidural	Spinal
Age (yr)	35 ± 12	33 ± 9
Weight (kg)	76 ± 14	75 ± 17
Height (cm)	172 ± 12	177 ± 10
Gender (M/F)	7/3	7/4
Ambient temperatures (°C)	21.7 ± 0.8	21.7 ± 0.9
Block levels (median, range)	T11, T8–T12	T9, T8–T12
Fluid administration rates (l/h)	0.5 ± 0.1	0.5 ± 0.2

Values are mean ± SD. There were no statistically significant differences between the patients given each type of anesthesia.

Patients

Morphometric characteristics, ambient temperatures, block levels, and fluid administration rates were similar in the patients given epidural and spinal anesthesia (table 4).

Initial tympanic membrane temperatures were similar with each type of anesthesia. The shivering thresholds were virtually identical in patients given epidural (36.4 ± 0.6°C) and spinal (36.3 ± 0.4°C) anesthesia (table 5). Power analysis indicated that this study had a 70% chance of detecting a 0.6°C difference in the shivering thresholds (chosen because that was the interthreshold range during both spinal and epidural anesthesia in the volunteers).

Discussion

Epidural anesthesia probably functions, at least in part, by blocking spinal nerve roots whereas spinal anesthesia directly prevents signal conduction in the *cauda equina* and spinal cord *per se*. Because spinal cord temperature itself is an important thermal input[4–6] and because thermal afferent signals are initially

Table 5. Initial Core Temperature, Time Elapsed to Shivering, and the Shivering Thresholds in the Patients

	Epidural (n = 10)	Spinal (n = 11)
Initial core temperatures (°C)	37.1 ± 0.5	37.0 ± 0.4
Time to shivering (min)	61 ± 37	51 ± 28
Shivering thresholds (°C)	36.4 ± 0.6	36.3 ± 0.4

Values are mean ± SD. There were no statistically significant differences between the patients given each type of anesthesia.

integrated in the spinal cord,[3,4] spinal and epidural anesthesia might be expected to influence thermoregulatory processing differently. Nonetheless, the sweating, vasoconstriction, and shivering thresholds were comparable in volunteers during epidural and spinal anesthesia. Similarly, the shivering thresholds were virtually identical during each type of anesthesia in patients. These data suggest that the two types of regional anesthesia comparably alter thermoregulatory processing. We have previously proposed that thermoregulatory impairment induced by regional anesthesia results largely from blocked afferent temperature signals.[16] This mechanism remains attractive because peripheral thermal input would be comparably impaired by epidural and spinal anesthesia.

Autonomic thermoregulatory responses are mostly determined by core temperature. Nonetheless, skin temperature contributes substantially.[7-9] Although the thermoregulatory contribution of upper-body skin during regional anesthesia remains unknown, sentient skin temperature surely continues to modify regulatory responses to core thermal perturbations. Upper-body (sentient) skin temperature was not recorded in the patients but typically would be approximately 33–34°C in an approximately 22°C ambient environment.[18] In contrast, skin temperatures were 36–37°C in the volunteers. Consistent with this difference, the shivering thresholds were approximately 1°C less in the volunteers than in the patients.

The sweating-to-vasoconstriction interthreshold range during spinal and epidural anesthesia, 0.6°C, did not differ significantly from that we reported previously during spinal anesthesia.[16] In that study, however, sentient skin temperature with and without anesthesia differed considerably at the sweating thresholds and consequently the sweating thresholds could not be directly compared under the two circumstances. Skin temperatures were well controlled in the present study so all thresholds during epidural anesthesia, spinal anesthesia, and no anesthesia can be directly compared.

The threshold changes induced by regional anesthesia were relatively small, and as a result, none of the thresholds differed significantly from those without anesthesia except the sweating threshold during spinal anesthesia. This result contrasts with our previous study, in which the vasoconstriction and shivering thresholds during spinal anesthesia were comparably decreased approximately 0.5°C.[16] The mechanism by which regional anesthesia decreases cold-response thresholds remains unclear, but may result from an apparent (as opposed to actual) increase in lower-body temperature. Specifically, at typical leg skin temperatures (≈ 33°C), cold-receptor activity predominates.[19] Thus, when regional anesthesia blocks all thermal input, the regulatory system may largely perceive loss of tonic cold signals and interpret the change as relative leg warming.

In this scenario, reduction in the vasoconstriction and shivering thresholds by regional anesthesia will depend critically on the lower-body skin temperature during the trial and be obliterated by a sufficiently high skin temperature. Consistent with this theory, vasoconstriction and shivering thresholds were not significantly decreased at mean lower-body skin temperatures near 36°C in our current volunteers—a value far exceeding that typical in patients who are not actively warmed[18] and that in our previous study (≈ 33°C).[16] (Such high temperatures were necessary to trigger sweating and then were kept constant for the remainder of the studies.) It is thus likely that threshold changes induced by regional anesthesia in our current volunteers were smaller than reported previously because their lower-body skin temperatures were maintained at a higher level.

Although a variety of thermoregulatory models have been proposed, the actual mechanisms by which thermal afferent signals initiate efferent responses remains virtually unknown. Consequently, it is unclear whether central control systems respond to integrated warm and cold afferent input, or whether warm and cold responses are independently processed from their respective afferent signals. Decreased vasoconstriction and shivering thresholds would be expected in each case during regional anesthesia with unwarmed legs; similarly, little change in the cold-response threshold would be expected when the legs are sufficiently warmed. However, warm responses such as sweating would be affected differently: the threshold would be reduced if central control systems respond to integrated warm and cold afferent input, but increased if warm and cold responses are independently processed. This increase would result because anesthetic-induced inhibition of warm signals (even if they constitute a small fraction of the total thermal input) would *decrease* leg temperature apparent to the system integrating warm input and controlling sweating. The observed increases in the sweating thresholds during regional anesthesia thus are most consistent with independent thermoregulatory processing of warm and cold afferent signals. The magnitude of the increase in the sweating threshold

presumably is directly related to leg skin temperature, as is the decrease in vasoconstriction and shivering thresholds. The net result is, therefore, that regional anesthesia likely expands the interthreshold range at any leg temperature. This pattern of thermoregulatory impairment is similar to that produced by isoflurane[12,20] and enflurane[15,21] anesthesia, although its magnitude is far less.

We made no effort to evaluate the incidence of shivering during epidural *versus* spinal anesthesia (only shivering patients were admitted to the study). Although the thresholds were comparable in the two groups, it remains possible that the clinical incidence of shivering differs with the two techniques because the core hypothermia might develop more rapidly after induction of spinal anesthesia. However, hypothermia more likely develops at similar rates despite the generally more rapid onset of spinal anesthesia because redistribution hypothermia[22] is probably largely limited by convective transfer of heat from the thermal core to peripheral tissues. A prospective, randomized trial evaluating the incidence of shivering during epidural and spinal anesthesia has yet to be published.

In summary, comparable sweating, vasoconstriction, and shivering thresholds during epidural and spinal anesthesia suggest that thermoregulatory processing is similar during each type of regional anesthesia. However, thermoregulatory control was impaired during regional anesthesia, as indicated by the significantly enlarged interthreshold and sweating-to-shivering ranges.

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