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Epidural Anesthesia Reduces the Gain and Maximum Intensity of Shivering

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Background: Shivering can be characterized by its threshold (triggering core temperature), gain (incremental intensity increase), and maximum intensity. The gain of shivering might be preserved during epidural or spinal anesthesia if control mechanisms compensate for lower-body paralysis by augmenting the activity of upper-body muscles. Conversely, gain will be reduced approximately by half if the thermoregulatory system fails to compensate. Similarly, appropriate regulatory feedback might maintain maximum shivering intensity during regional anesthesia. Accordingly, the gain and maximum intensity of shivering during epidural anesthesia were determined.

Methods: Seven volunteers participated on two randomly ordered study days. On one day (control), no anesthesia was administered; on the other, epidural anesthesia was maintained at a T8 sensory level. Shivering, at a mean skin tempera-

ture near 33°C, was provoked by central-venous infusion of cold fluid; core cooling continued until shivering intensity no longer increased. Shivering was evaluated by systemic oxygen consumption and electromyography of two upper-body and two lower-body muscles. The core temperature triggering an increase in oxygen consumption identified the shivering threshold. The slopes of the oxygen consumption *versus* core temperature and electromyographic intensity *versus* core temperature regressions identified systemic and regional shivering gains, respectively.

Results: The shivering threshold was reduced by epidural anesthesia by $\approx 0.4^\circ\text{C}$, from 36.7 ± 0.6 to $36.3 \pm 0.5^\circ\text{C}$ (means \pm SD; $P < 0.05$). Systemic gain, as determined by oxygen consumption, was reduced from -581 ± 186 to $-215 \pm 154 \text{ ml} \cdot \text{min}^{-1} \cdot ^\circ\text{C}^{-1}$ ($P < 0.01$). Lower-body gain, as determined electromyographically, was essentially obliterated by paralysis during epidural anesthesia, decreasing from -0.73 ± 0.85 to -0.04 ± 0.06 intensity units/ $^\circ\text{C}$ ($P < 0.01$). However, upper-body gain had no compensatory increase: -1.3 ± 1.1 units/ $^\circ\text{C}$ control *versus* -2.0 ± 2.1 units/ $^\circ\text{C}$ epidural. Maximum oxygen consumption was decreased by one third during epidural anesthesia: 607 ± 82 *versus* $412 \pm 50 \text{ ml/min}$ ($P < 0.05$).

Conclusions: These results confirm that regional anesthesia reduces the shivering threshold. Epidural anesthesia reduced the gain of shivering by 63% because upper-body muscles failed to compensate for lower-body paralysis. The thermoregulatory system thus fails to recognize that regional anesthesia reduces metabolic heat production, instead responding as if lower-body muscular activity remained intact. (Key words: Core temperature; electromyography; hypothermia; regional anesthesia; thermoregulation.)

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THERMOREGULATORY shivering can be characterized by its threshold (triggering core temperature), gain (incremental intensity increase with further reduction in core temperature), and maximum intensity.¹ Spinal² and epidural³ anesthesia decrease the vasoconstriction and shivering thresholds, possibly by producing a substantial increase in apparent (rather than actual) leg temperature.³

The gain of shivering might be well preserved during regional anesthesia if control mechanisms compensate for lower-body paralysis by augmenting activity of upper-body muscles. In this scenario, feedback systems would recognize that heat production was insufficient to prevent additional hypothermia and proportionately

increase upper-body muscular activity. Conversely, gain will be reduced approximately by half if the thermoregulatory system fails to compensate. This is the response that would be expected if upper-body muscular activity was determined by body temperature without regard to actual heat production.

Similarly, it might seem logical that lower-body paralysis will reduce the maximum intensity of shivering during regional anesthesia. However, even during maximum shivering,⁴ muscular activity is usually considerably less intense than during exercise.⁵ Appropriate regulatory feedback of heat production thus might maintain maximum shivering intensity even during regional anesthesia.

Most physiologists believe that temperature, rather than heat, is the major input to the central thermoregulatory control systems.^{6,7} That is, thermoregulatory responses are controlled only by tissue temperature rather than by body heat content or heat flow. However, heat control has its supporters.^{8,9} Current thermoregulatory theory is thus insufficiently detailed to predict the effect of regional anesthesia on either aspect of shivering. Accordingly, we tested the hypothesis that epidural anesthesia reduces the gain and maximum intensity of shivering.

Methods

With approval from the Committee on Human Research at the University of California, San Francisco, and informed consent, we studied seven healthy female volunteers. None was obese, was taking medication, or had a history of thyroid disease, dysautonomia, or Raynaud's syndrome. Women not using hormonal contraceptives were studied during the first 10 days of their menstrual cycles. Morphometric characteristics included height 164 ± 7 cm, weight 61 ± 10 kg, and age 27 ± 5 yr.

Treatment Protocol

Studies started at approximately 8:30 A.M. to minimize circadian changes in body temperature. The volunteers fasted 8 h before each study, and rested supine on a standard operating room table. During the studies, they were minimally clothed and ambient temperature was maintained near 22°C. After standard anesthetic safety monitors were positioned, a 16-gauge catheter was inserted into the superior vena cava *via* the right internal jugular vein. A 14-French Foley catheter was inserted into the bladder.

On one randomly-chosen day, no anesthesia was given (control). On the other, a catheter was inserted into the epidural space *via* the L3-L4 interspace. The epidural catheter was then injected with 2 ml of 1% lidocaine with epinephrine 1:100,000. This test dose was followed in 5 minutes by 15-17 ml of 2% mepivacaine (Astra Pharmaceuticals, Westborough, MA) without epinephrine. The initial anesthetic dose was based on the volunteers' heights and calculated to produce a dermatomal level of sensory blockade near T8 bilaterally as determined by loss of cutaneous cold sensation and response to pinprick. If necessary, we injected additional mepivacaine to maintain the target level of sensory blockade.

Induction of epidural anesthesia was preceded by 1 h of active prewarming¹⁰ designed to minimize redistribution hypothermia.¹¹ Active warming was continued for an additional 20-30 min, as necessary to return the core temperature to preinduction levels. Upper- and lower-body mean skin-surface temperatures were adjusted to 33°C, a typical value, using forced air (Augustine Medical, Eden Prairie, MN) and circulating water (Cincinnati Sub-Zero Products, Cincinnati, OH).

The core was cooled by central-venous administration of lactated Ringer's solution at $\approx 3^\circ\text{C}$. The cooling rate was restricted to $\leq 2^\circ\text{C/h}$, because such rates do not trigger dynamic thermoregulatory responses.¹² Fluid was typically administered at ≈ 10 -20 ml/min during the initial portion of the study but increased to ≈ 80 ml/min by the end. Cooling ceased when shivering intensity no longer increased, despite a continued decrease in core temperature, or when the volunteer requested it. The cooling period typically lasted 1 h on control days and 2 h with epidural anesthesia.

Measurements

Core temperature was recorded from the tympanic membrane using Mon-a-Therm thermocouples (Mallinckrodt Anesthesiology Products, St. Louis, MO). The aural probes were 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 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 calculated from measurements at 15 area-weighted sites.¹³ Upper- and lower-body skin temperatures were calculated as previously described.³ Temperatures were recorded at 1-min intervals from thermocouples con-

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nected to calibrated Iso-Thermex thermometers (Columbus Instruments, Columbus, OH) that have an accuracy of 0.1°C and a precision of 0.01°C .

Heart rate was measured continuously using pulse oximetry, and blood pressure was determined oscillographically at 5-min intervals at the left ankle. Oxygen consumption, as measured by a DeltaTrac metabolic monitor (SensorMedics Corp., Yorba Linda, CA), quantified shivering. The system was used in the canopy mode, with a 40 l/min flow through a plastic "bubble" around the volunteers' heads. Measurements were averaged over 1-min intervals and recorded every minute.

Shivering was also evaluated with electromyography as previously described.¹⁴ Silver-silver chloride monitoring electrodes were positioned to record the electric activity of the right pectoralis, trapezius, lower rectus abdominis (within the blocked region), and the quadriceps femoris after mild skin abrasion and degreasing. The active electrodes were positioned 4 cm apart and oriented in the direction of the muscle fibers.¹⁴ After appropriate amplification (model P511; Grass Instruments, Quincy, MA), the signals were recorded on a thermoelectric printer having a linear resolution to 1,000 Hz (Dash-4; Astro-Med, West Warwick, RI). Signals were also digitized for computer analysis at 500 Hz (models NB-MIO-16H, NB-DMA-2800, and LabVIEW 3.11; National Instruments, Austin, TX), and root-mean square values of 1-min acquisition intervals were computed.

Data Analysis

A sustained increase in oxygen consumption identified the shivering threshold. The maximum intensity of shivering was identified by an oxygen consumption that failed to increase further despite continued reduction in core temperature. The shivering threshold and maximum intensity were determined *post hoc* by an investigator blinded to treatment and core temperature.

Our primary measure of the systemic (whole-body) gain of shivering was considered to be the average slope of the individual oxygen consumption *versus* the core temperature regressions. Gains in shivering in the upper body (pectoralis, trapezius) and lower body (rectus abdominis, quadriceps femoris) were similarly determined as the average slopes of the individual integrated electromyographic intensity *versus* core temperature regressions.

For graphical display purposes, the group response was characterized as previously described.¹⁵ (1) The core temperatures triggering shivering on the control

and epidural days were designated thresholds in each volunteer. (2) Oxygen consumption values were calculated relative to the individual threshold temperatures under each condition. Because oxygen consumption measurements were taken at specific time intervals rather than at fixed temperature intervals, available data from each volunteer within 0.25°C core temperature increments were averaged. Subsequently, the population means were calculated from these individual averages. (3) Finally, the average oxygen consumption values for the population were plotted relative to the mean thresholds with and without epidural anesthesia, and the slopes were determined by linear regression.

Hemodynamic responses and ambient temperature on each study day were first averaged within each volunteer, and values between the shivering threshold and maximum shivering intensity were included in this average. The resulting values were averaged among volunteers. Results for the two study days were compared using two-tailed paired *t* or Wilcoxon tests. Results are presented as mean \pm SD; probability values <0.05 were considered significant.

Results

The total volume of 2% mepivacaine administered was 23 ± 5 ml. Epidural anesthesia produced a sensory block to the level of the $\text{T8} \pm 1$ dermatome, which remained constant during the cooling period. Maximum shivering intensity could be identified only in four of the seven volunteers because the others requested that the study be discontinued before the maximum was reached. Maximum intensity was thus calculated using the data from only four volunteers; other results are based on data from all seven volunteers. Mean skin temperature, and upper- and lower-body skin temperatures, remained near 33°C throughout the experiments. The total volume of administered Ringier's lactate solution was also similar on the two study days, as were environmental conditions and hemodynamic responses (table 1).

The shivering threshold was reduced $\approx 0.4^{\circ}\text{C}$ by epidural anesthesia, from 36.7 ± 0.6 to $36.3 \pm 0.5^{\circ}\text{C}$ ($P < 0.05$). The linear relationship between oxygen consumption and core temperature during shivering in individual volunteers was good. Correlation coefficients (r^2) averaged 0.83 ± 0.14 on the control days and 0.82 ± 0.17 on the epidural days. Systemic gain of shivering, as determined by oxygen consumption, was reduced

Table 1. Temperatures, Administered Fluid, Hemodynamic Responses, and Environmental Conditions

	Control	Epidural
Mean skin temperature (°C)	32.7 ± 0.8	32.7 ± 0.9
Δ Mean skin temperature (°C)	-0.1 ± 0.1	-0.2 ± 0.2
Upper-body skin temperature (°C)	32.9 ± 0.6	32.7 ± 0.9
Lower-body skin temperature (°C)	32.5 ± 1.2	32.8 ± 0.8
Ringer's lactate (L)	2.3 ± 0.9	2.3 ± 0.6
Mean blood pressure (mmHg)	91 ± 6	87 ± 6
Heart rate (beats/min)	72 ± 4	73 ± 11
Ambient temperature (°C)	21.5 ± 1.0	22.3 ± 0.5
Ambient humidity (%)	43 ± 12	44 ± 7

Data are mean ± SD. Temperatures and hemodynamic responses on each study day were first averaged within each volunteer, and then among volunteers; values between the shivering threshold and maximum shivering intensity were included in this average. Change (Δ) in mean skin temperature was computed from the shivering threshold to the end of gain. There were no statistically significant differences between the treatments.

63% from -581 ± 186 to -215 ± 154 ml · min⁻¹ · °C⁻¹ ($P < 0.01$; fig. 1).

Individual correlation coefficients between electromyographic intensity and core temperature in the upper and lower body averaged 0.75 ± 0.22 and 0.72 ± 0.28 on the control day and 0.83 ± 0.09 and 0.35 ± 0.28 on the epidural day. Lower-body gain was reduced from -0.73 ± 0.85 to -0.04 ± 0.06 units/°C during epidural anesthesia due to paralysis of the lower body ($P < 0.01$). However, there was no compensatory increase in upper-body gain during epidural anesthesia: -1.3 ± 1.1 for control versus -2 ± 2.1 for epidural (fig. 2). Maximum shivering intensity during epidural

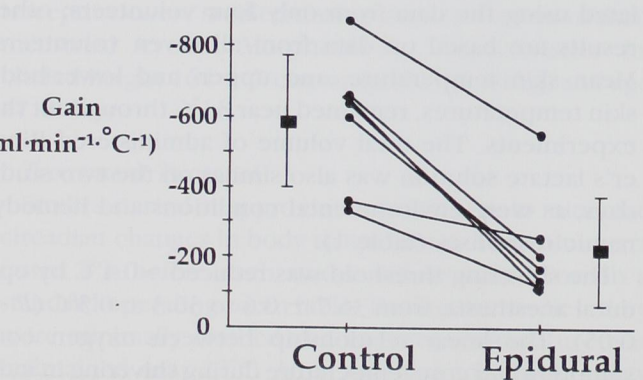


Fig. 1. Circles identify individual shivering gains without (control) and with epidural anesthesia. Squares show the mean values (±SD). The gain of shivering was reduced by 63% during epidural anesthesia from -581 ± 186 to -215 ± 154 ml · min⁻¹ · °C⁻¹ ($P < 0.01$).

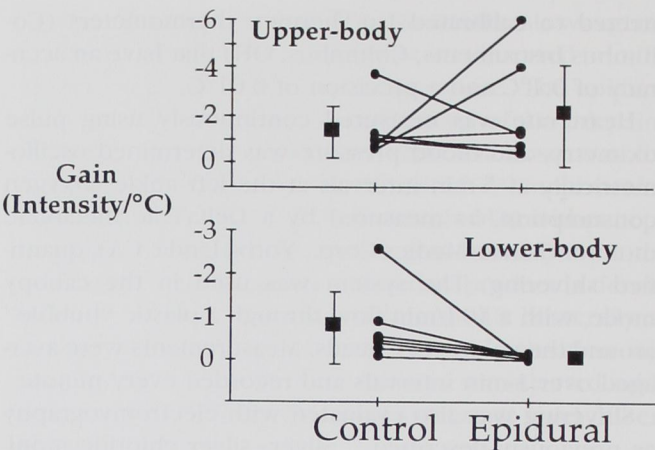


Fig. 2. Circles identify the gain of shivering, as determined by electromyography, of the upper body (pectoralis, trapezius) and lower body (rectus abdominis, quadriceps femoris) without (control) and with epidural anesthesia. Squares show the mean values (±SD). Lower-body gain was reduced from -0.73 ± 0.85 to -0.04 ± 0.06 intensity units/°C during epidural anesthesia because of the paralysis of the lower body ($P < 0.01$). However, there was no compensatory increase in upper-body gain during epidural anesthesia.

anesthesia, as determined by systemic oxygen consumption, decreased 32% from 607 ± 82 to 412 ± 50 ml/min ($P < 0.05$; table 2).

Systemic gain in the entire study population, as determined by oxygen consumption, was reduced from -412 to -112 ml · min⁻¹ · °C⁻¹ (fig. 3). Gain, as determined by electromyography, was similar in the upper body with (-109 units/°C) and without (-151 units/°C) epidural anesthesia. In contrast, epidural anesthesia markedly reduced gain from -70 units/°C to 4.5 units/°C (fig. 4).

Table 2. Shivering Threshold, Gains, and Maximum Intensity

	Control	Epidural	P/Ratio
Shivering threshold (°C)	36.7 ± 0.6	36.3 ± 0.5	<0.05
Systemic gain (ml · min ⁻¹ · °C ⁻¹)	-581 ± 186	-215 ± 154	<0.01
Upper-body gain (intensity/°C)	-1.31 ± 1.08	-2.03 ± 2.14	NS
Lower-body gain (intensity/°C)	-0.73 ± 0.85	-0.04 ± 0.06	<0.01
Maximum intensity (ml/min)	607 ± 82	412 ± 50	<0.05

NS = not significant.
Data are mean ± SD. Gains are based on individual regressions.

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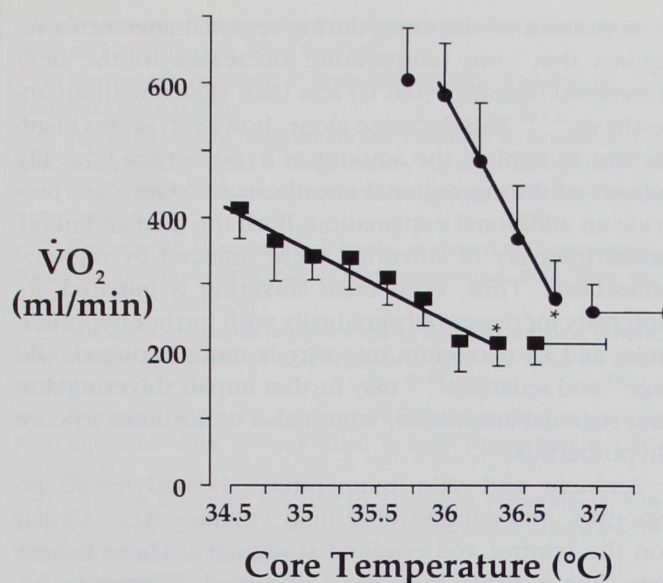


Fig. 3. Systemic oxygen consumption without (circles) and with (squares) epidural anesthesia. The horizontal standard deviation bars indicate variability in the thresholds (*) among the volunteers; although error bars are shown only once in each series, the same temperature variability applies to each data point. The slopes of the oxygen consumption *versus* core temperature relations (solid lines) were determined using linear regression. These slopes defined the gain of shivering with and without epidural anesthesia. Gain was reduced 3.7 times, from $-412 \text{ ml} \cdot \text{min}^{-1} \cdot ^\circ\text{C}^{-1}$ ($r^2 = 0.99$) to $-112 \text{ ml} \cdot \text{min}^{-1} \cdot ^\circ\text{C}^{-1}$ ($r^2 = 0.96$). These gains are lower, and the ratio of the gains are higher, than those shown in figure 1. This is entirely an artifact of averaging oxygen consumption among the volunteers before computing the regression, and it illustrates the importance of calculating the gain for each volunteer. Data are presented as mean \pm SD.

Discussion

Regional anesthesia significantly decreased the shivering threshold by $\approx 0.4^\circ\text{C}$, which is similar to the $\approx 0.6^\circ\text{C}$ reduction reported previously.^{2,3,16} Our data thus confirm that peripheral regional anesthesia produces a central inhibition of thermoregulatory control. This central inhibition is, of course, compounded by direct peripheral inhibition of neuronally mediated thermoregulatory defenses.^{17,18}

The simplest model of thermoregulatory control would be one in which tissue temperatures are the only inputs and responses are entirely controlled by these temperature signals. Although widely adapted,^{6,7} this model is not the only one advocated. A competing hypothesis is that mammals also sense body heat and cutaneous flow of heat.^{8,9} Similarly, the system might well include feedback control of metabolic rate, just as

sweating can be inhibited by skin wetting.¹⁹ These models differ in that the regulatory system might compensate for lower-body paralysis in the latter two cases but not if temperature was the only input.

Epidural anesthesia decreased the gain of shivering by two thirds (based on individual responses) and decreased maximum intensity by one third. The thermoregulatory system thus could not compensate for lower-body paralysis. Both results suggest that the human thermoregulatory system fails to discern or compensate when one half of the body is paralyzed during epidural

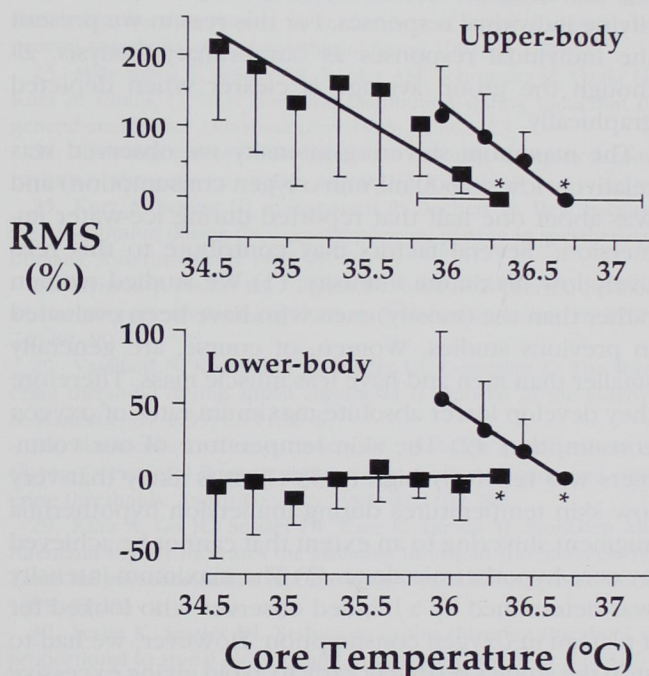


Fig. 4. Root-mean-square electromyographic intensity from the upper and lower body without (circles) and with (squares) epidural anesthesia. The horizontal standard deviation bars indicate variability in the thresholds (*) among the volunteers; although error bars are shown only once, the same temperature variability applies to each data point in each series. The slopes of the electromyographic intensity *versus* core temperature relations (solid lines) were determined using linear regression. These slopes defined the gain of shivering with and without epidural anesthesia. Gain was similar in the upper body with ($-109 \text{ units}/^\circ\text{C}$, $r^2 = 0.97$) and without ($-151 \text{ units}/^\circ\text{C}$, $r^2 = 0.85$) epidural anesthesia, for a ratio of 1.4. In contrast, epidural anesthesia markedly reduced gain in the lower body from $-70 \text{ units}/^\circ\text{C}$ ($r^2 = 0.99$) to $4.5 \text{ units}/^\circ\text{C}$ ($r^2 = 0.21$). These gains are lower, and the ratio of the gains higher, than those shown in figure 2. This is entirely an artifact of averaging oxygen consumption among the volunteers before computing the regression, and it illustrates the importance of calculating the gains for each volunteer. Data are presented as percentage changes from the shivering threshold, with error bars indicating SD.

anesthesia. That is, the regulatory system responds as if lower body muscular activity was normal. Because the alternative hypothesis would require that the central regulatory system detect heat production,^{8,9} our data are most consistent with a system in which the major input is tissue temperature.

The gain of shivering was reduced nearly twice as much when calculated based on the average oxygen consumption *versus* core temperature regression than when it was based on the average of individual regressions. This difference is entirely an artifact of averaging oxygen consumption among individuals before computing the regression and illustrates the importance of identifying individual responses. For this reason we present the individual responses as our primary analysis, although the group average is clearer when depicted graphically.

The maximum shivering intensity we observed was relatively low (≈ 600 ml/min oxygen consumption) and was about one half that reported during ice-water immersion.⁴ Several factors may contribute to this relatively low maximum intensity. (1) We studied women rather than the (mostly) men who have been evaluated in previous studies. Women, of course, are generally smaller than men and have less muscle mass. Therefore they develop lower absolute maximum rates of oxygen consumption. (2) The skin temperature of our volunteers was relatively high ($\approx 33^\circ\text{C}$). It is likely that very low skin temperatures during immersion hypothermia augment shivering to an extent that cannot be achieved by core hypothermia alone. (3) The maximum intensity was determined by a blinded observer who looked for a plateau in oxygen consumption. However, we had to stop the studies relatively early to avoid giving excessive fluid. It thus remains possible that with additional cooling, oxygen consumption would have increased more. Our maximum intensity estimates are, therefore, somewhat less reliable than the gain measurements.

Hypothermia during regional anesthesia is common²⁰ and can be nearly as severe as that observed during general anesthesia.^{21,22} As during general anesthesia,²³ hypothermia during regional anesthesia results initially from vasodilation-induced core-to-peripheral redistribution of body heat.¹¹ Redistribution is followed in both cases by a period when heat loss exceeds metabolic heat production.²⁴ The decrease in core temperature persists until sufficient hypothermia triggers effective defenses. This is usually arteriovenous shunt vasoconstriction during general anesthesia²⁵ but is primarily shivering during epidural or spinal anesthesia.²⁶

Activation of shivering during regional anesthesia requires that core temperature decreases to the new threshold (usually $\approx 0.6^\circ\text{C}$) less than values without anesthesia.^{2,3,16} This decrease alone, however, seems insufficient to explain the amount of hypothermia typically observed during regional anesthesia.^{21,22} Our data provide an additional explanation: Both the gain and maximum intensity of shivering were reduced by regional anesthesia. Thus, even once shivering is initiated, its intensity increases only gradually with further hypothermia, and its maximum intensity is much reduced. Old age²⁷ and sedatives^{28,29} may further impair shivering during regional anesthesia, which also contributes to core hypothermia.

Ambient and skin temperature, hemodynamic responses, and administered fluid volumes were similar on the control and epidural study days. These factors are thus unlikely to explain observed differences between the treatment days. We evaluated only a single epidural anesthetic, and only at one relatively low block level. Reduction in the shivering threshold during regional anesthesia is proportional to block level.³⁰ It is also likely that even greater reductions in the shivering gain and maximum intensity would be observed with higher block levels.

In conclusion, our results confirm that epidural anesthesia significantly reduces the shivering threshold. The gain of shivering was reduced by 63% during epidural anesthesia because upper-body muscles failed to compensate for lower-body paralysis; the maximum intensity was also reduced by one third. The thermoregulatory system thus fails to recognize that regional anesthesia prevents the increase in regional metabolic heat production, instead responding as if lower-body muscular activity remained intact. These data support a thermoregulatory model in which tissue temperatures are the dominant input. They also explain why shivering so often fails to prevent core hypothermia during regional anesthesia.

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