

High Thoracic Segmental Epidural Anesthesia Diminishes Sympathetic Outflow to the Legs, Despite Restriction of Sensory Blockade to the Upper Thorax

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To evaluate whether, after high thoracic segmental epidural anesthesia, sympathetic blockade spreads caudally beyond sensory blockade, we assessed regional skin temperatures by infrared telethermometry in 53 nonpremedicated patients at constant ambient temperature. Either bupivacaine (4.2 ml, 0.75%, n = 10) or an equal volume of saline (placebo, n = 10) was injected at the C7-T2 epidural space in a randomized double-blinded fashion. Results were contrasted to those observed after midthoracic (T6-T9, n = 13) and lumbar (L2-T12, n = 10) epidural injection of an identical dose of bupivacaine or saline (n = 10). Despite restriction of sensory block to the upper thorax with high thoracic epidural anesthesia, skin temperatures increased significantly ($P < 0.05$ vs. saline) on the foot (great toe: $+1.2^\circ\text{C} \pm 2.9$ SD; little toe: $+0.9^\circ\text{C} \pm 2.6$) and hand (thumb: $+2.0^\circ\text{C} \pm 4.0$, digit 5: $+2.9^\circ\text{C} \pm 4.2$) but decreased after saline. Midthoracic injection also increased significantly skin temperature on the foot (great toe: $+4.0^\circ\text{C} \pm 4.9$; little toe: $+3.6^\circ\text{C} \pm 4.8$) but not on the hand. In contrast, with lumbar epidural anesthesia, skin temperature increased significantly on the foot (great toe: $+8.5^\circ\text{C} \pm 2.5$; little toe: $+8.6^\circ\text{C} \pm 2.8$) but decreased significantly on the hand (thumb: $-3.1^\circ\text{C} \pm 2.1$; digit 5: $-2.8^\circ\text{C} \pm 2.5$). Whereas the increase in foot skin temperature was greater after lumbar than after high ($P < 0.003$) or midthoracic ($P < 0.03$) segmental epidural anesthesia, there was no difference ($P = 0.6$) in foot skin temperature change between high and midthoracic injection. On the trunk, skin temperatures either did not change or decreased significantly even within analgesic dermatomes. Thus, a substantial, albeit submaximal, increase in foot skin temperature was observed with thoracic epidural anesthesia. Assuming that increased foot skin temperatures reflect diminished sympathetic outflow, we conclude that mid- and even high thoracic segmental epidural anesthesia involving only a few dermatomes can result in a widespread sympathetic block that includes the most caudal parts of the sympathetic nervous system. (Key words: Anesthetic technique, epidural: sympathetic blockade. Autonomic nervous system. Anesthetics, local: bupivacaine. Temperature: skin.)

SYMPATHETIC BLOCKADE is reported not to exceed sensory blockade in the cranial direction with lumbar epidural anesthesia.¹ Similarly, with thoracic epidural anesthesia,

it is generally believed that sympathetic blockade does not extend beyond sensory blockade in the caudal direction.² Surprisingly, there are no data to support this latter notion. In particular, it is unknown whether sympathetic tone is diminished, unchanged, or even increased caudal to the level of sensory blockade with mid- or high thoracic segmental epidural anesthesia.

We hypothesized, based on our studies in conscious dogs,³ that sympathetic blockade exceeds sensory blockade in the caudal direction with high thoracic segmental epidural anesthesia.

Accordingly, we assessed changes in regional skin temperatures as an indicator of sympathetic vasomotor tone after high thoracic segmental epidural anesthesia with a sensory block confined to the upper thorax. These results were contrasted to those observed after midthoracic and lumbar epidural anesthesia induced by an identical dose of bupivacaine. We found that with thoracic epidural anesthesia, sympathetic blockade extended down to and includes the most caudal part of the sympathetic nervous system, even with high thoracic epidural injection and despite restriction of sensory block to the upper thorax.

Materials and Methods

PATIENTS

After institutional approval and informed written consent, 53 nonpremedicated patients (ASA physical status 1 or 2) of either sex were studied on the morning prior to surgery. Patients received a constant dose of preservative free bupivacaine (4.2 ml, 0.75%) at either a high thoracic (catheter tips between C7 and T2), midthoracic (T6-T9), or lumbar (T12-L1) epidural site or an equal volume of epidural saline (placebo).

With high thoracic epidural injection, ten patients each received either bupivacaine or saline in a randomized double-blinded fashion. Similarly, with midthoracic injection, either bupivacaine (n = 13) or saline (n = 10) were administered epidurally at random. For comparison with thoracic epidural anesthesia, an additional ten patients were given the same dose at a lumbar epidural injection site. Finally, to exclude the possibility that the observed effects resulted from absorbed bupivacaine in the blood rather than from sympathetic blockade, the same dose of bupivacaine also was injected intramuscularly.

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MEASUREMENTS

Regional skin temperatures were evaluated by infrared telethermometry (BioTherm model C-600M, Linear Laboratories, Los Altos, CA). This hand-held device measures radiant energy emitted by the skin; radiant energy, in turn, is directly related to skin temperature.^{4,5} The instrument is calibrated for the emissivity of human skin, linear over a temperature range from 10–50° C with a sensitivity of 0.1° C, and has a time constant of less than 1 s. The smallest target is a circle of 6-mm diameter at a distance of 2 cm. Details are reported elsewhere.^{5,6} Temperatures always were measured on the same skin areas along the body's length axis; these were the middle of the upper arm, lateral side (spinal sensory segment C4); middle of the lower arm, radial side (C5); thumb (C6); digit 5 (C8); next to the nipple (T4); xiphoid (T6); or umbilicus (T10); beside the anterior iliac spine (T12); middle of the ventral thigh (L3); middle of the ventral calf (L4); and great (L5) and little (S1) toe. On the hand and foot, temperatures always were measured on the dorsal surface 0.5 cm proximal to the nail bed. Temperatures were sampled intermittently by moving the telethermometer in a caudal-to-cranial direction along the body. Rectal and room temperatures were measured continuously with thermistors (Yellow Springs Instrument Co., Yellow Springs, OH). Rectal temperature was assessed with a model 401 (time constant 7 s), ambient temperature with a model 402 (time constant 3.2 s).

Sensory block was assessed by pin prick (20-G cannula), loss of cold sensation (alcohol spray), and analgesia (pinching the skin between thumb and index finger). Borders were defined as the most cranial and most caudal dermatomes insensitive to the various stimuli. Skin temperatures and sensory blockade were assessed on opposite body sides.

Arterial blood pressure was measured by automatic oscillometry (Dinamap) or by radial artery cannulation and transducer, as clinically indicated. Heart rate was derived from the ECG.

Sampling of a temperature profile took 60–90 s, and evaluation of sensory block required another 180–240 s. Thus, each measurement cycle took approximately 5 min.

STUDY PROTOCOL

The same induction room was used throughout the study. Average ambient temperature was 23° C (range: 22.5–24.3° C) and did not vary by more than 0.2° C for a given patient during the measurements. After insertion of vascular cannulas, saline was infused at a constant rate of 20 ml·h⁻¹. No patient was treated with vasoactive drugs, blood volume expansion, or sedatives at any time. Epidural puncture was performed with the "loss-of-resis-

tance technique" with air and a radiopaque catheter advanced into the epidural space. The patient then was turned supine with only the genital region covered by a blanket.

To minimize fluctuations in skin temperatures unrelated to the intervention, we waited for at least 45 min before epidural injection commenced. During the last 15 min before epidural injection, variables were recorded at 5-min intervals to ensure a stable baseline. After collection of baseline values, either 31.5 mg bupivacaine (4.2 ml 0.75% solution, accounting for the catheter and filter dead space of 0.8 ml) stored at room temperature or the same volume of saline were injected epidurally. To lessen potential complications of inadvertent intrathecal or intravascular injection, the dose was given in two increments, in an initial 1.5 ml and 3.5 ml 5 min later. Variables then were measured every 10 min for up to 65 min. Postoperative x-rays confirmed the position of all catheters.

The same dose of bupivacaine was injected into the deltoid muscle in two of the authors after an otherwise identical protocol.

DATA ANALYSIS

Data are presented as means ± standard deviation. Measurements immediately before epidural injection were defined as baseline. Changes from baseline at a given time were calculated and contrasted to those in the placebo group. Effects were evaluated statistically after skin temperatures had reached a plateau and sufficient time had elapsed to allow full spread of epidural anesthesia, if present. Two null hypotheses were tested: 1) changes in values of variables after epidural bupivacaine are identical to those after saline, and 2) changes in foot skin temperature after epidural anesthesia are of equal magnitude, regardless of the epidural injection site. Hypotheses were tested by the Mann-Whitney U test and rejected when the adjusted alpha-error (Bonferroni correction) was less than 5%.

Results

Thoracic segmental epidural anesthesia induced a significant increase in foot skin temperature, even when bupivacaine was injected into the high thoracic epidural space and despite restriction of sensory block to the upper thorax.

Epidural bupivacaine evoked a sensory block with an average width of six dermatomes. The involved dermatomes shifted caudally along the body's length axis as the injection site descended from the high thoracic to the midthoracic and lumbar epidural space (fig. 1).

Regardless of the injection site, epidural anesthesia always evoked a significant increase in foot skin temperature that reached a plateau approximately 45 min after injec-

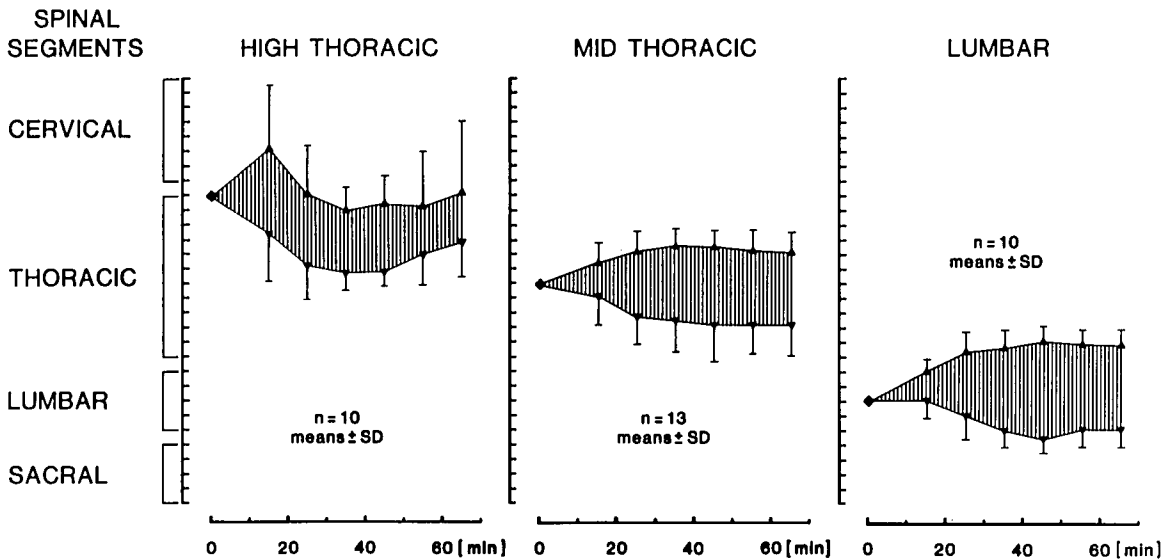


FIG. 1. The time course of dermatomal spread of sensory blockade (measured by the response to pin prick stimulation) along the spinal axis with high thoracic, midthoracic, and lumbar epidural anesthesia in supine humans. Data represent means \pm SD. Epidural injection of 4.2 ml bupivacaine 0.75% resulted in a sensory block covering an average of six dermatomes. The mean caudal level of sensory denervation shifted caudally along the spinal axis with descending epidural injection site, *i.e.*, from T6 (range: T5–T9) with high thoracic, and T10 (range: T7–L1) with midthoracic, to S1 (range: L3–S1) with lumbar epidural anesthesia. Note that with high thoracic epidural anesthesia, the mean caudal border of sensory block did not exceed dermatome T6.

tion (fig. 2). Even with high thoracic epidural anesthesia, foot skin temperature significantly increased (fig. 2), although the caudal border of sensory block for pin prick ($T6.3 \pm 1.2$), loss of cold sensation ($T6.1 \pm 2.4$), and analgesia ($T6.3 \pm 1.3$) remained confined to the upper thorax. Compared to baseline, skin temperatures had increased to a plateau 45 min after epidural bupivacaine on both the great toe ($+1.2^\circ \text{C} \pm 2.9$, $P < 0.025$ *vs.* saline) and little toe ($+0.9^\circ \text{C} \pm 2.6$, $P < 0.005$). In contrast, foot skin temperature decreased after saline, so that temperatures averaged 2.1 and 1.9°C higher (great toe and little toe, respectively) after bupivacaine than after saline. With midthoracic epidural injection of the same dose of bupivacaine, foot skin temperatures also gradually increased (fig. 2). Skin temperature significantly increased on the great ($+4.0^\circ \text{C} \pm 4.9$) and little toe ($+3.6^\circ \text{C} \pm 4.8$), whereas the caudal border of sensory block averaged $T10 \pm 2.0$ for all qualities tested. As expected with lumbar epidural anesthesia, foot skin temperatures (fig. 2) also increased significantly on the great ($+8.5^\circ \text{C} \pm 2.5$) and little toe ($+8.6^\circ \text{C} \pm 2.9$).

Thus, foot skin temperatures increased significantly after midthoracic and even after high thoracic segmental epidural anesthesia. Whereas the increase in foot skin temperatures was greater with lumbar than with high ($P < 0.003$) or midthoracic ($P < 0.03$) epidural anesthesia, there were no statistical differences ($P = 0.6$) between the high and midthoracic epidural group.

Hand skin temperatures (fig. 3) increased significantly after high thoracic epidural anesthesia, $+2.0^\circ \text{C} \pm 4.0$ on the thumb and $+2.9^\circ \text{C} \pm 4.3$ on digit 5, with a cranial border of sensory block of $T2 \pm 1.6$. Unlike high thoracic epidural injection, hand temperatures did not increase with midthoracic epidural injection. With lumbar epidural injection, a different pattern was observed: in contrast to thoracic epidural anesthesia, skin temperatures markedly and significantly decreased on the hand (fig. 3), $-3.3^\circ \text{C} \pm 2.3$ on the thumb and $-3.0^\circ \text{C} \pm 2.9$ on digit 5.

The temperature increases with sympathetic blockade were always confined to the peripheral parts of the limbs, *i.e.*, the feet and hands. No significant changes were detected on the proximal limbs (thigh, calf, and upper and lower arm) with any injection site, even within analgesic dermatomes (figs. 2 and 3). On the trunk, skin temperatures either remained unchanged—even within central areas of sensory blockade such as the nipple with high thoracic, or the xiphoid with midthoracic epidural anesthesia—or decreased significantly (fig. 4).

Rectal temperatures remained unchanged in the bupivacaine groups (high thoracic: $36.8^\circ \text{C} \pm 0.5$; midthoracic: $37.0^\circ \text{C} \pm 0.5$; lumbar: $36.7^\circ \text{C} \pm 0.5$) and in the saline groups (high thoracic: $36.8^\circ \text{C} \pm 0.5$; midthoracic and lumbar: $36.9^\circ \text{C} \pm 0.6$).

Mean arterial pressure decreased only with high thoracic epidural anesthesia ($-8 \text{ mmHg} \pm 9$, $P < 0.01$), whereas heart rate decreased with both high (-10 min^{-1}

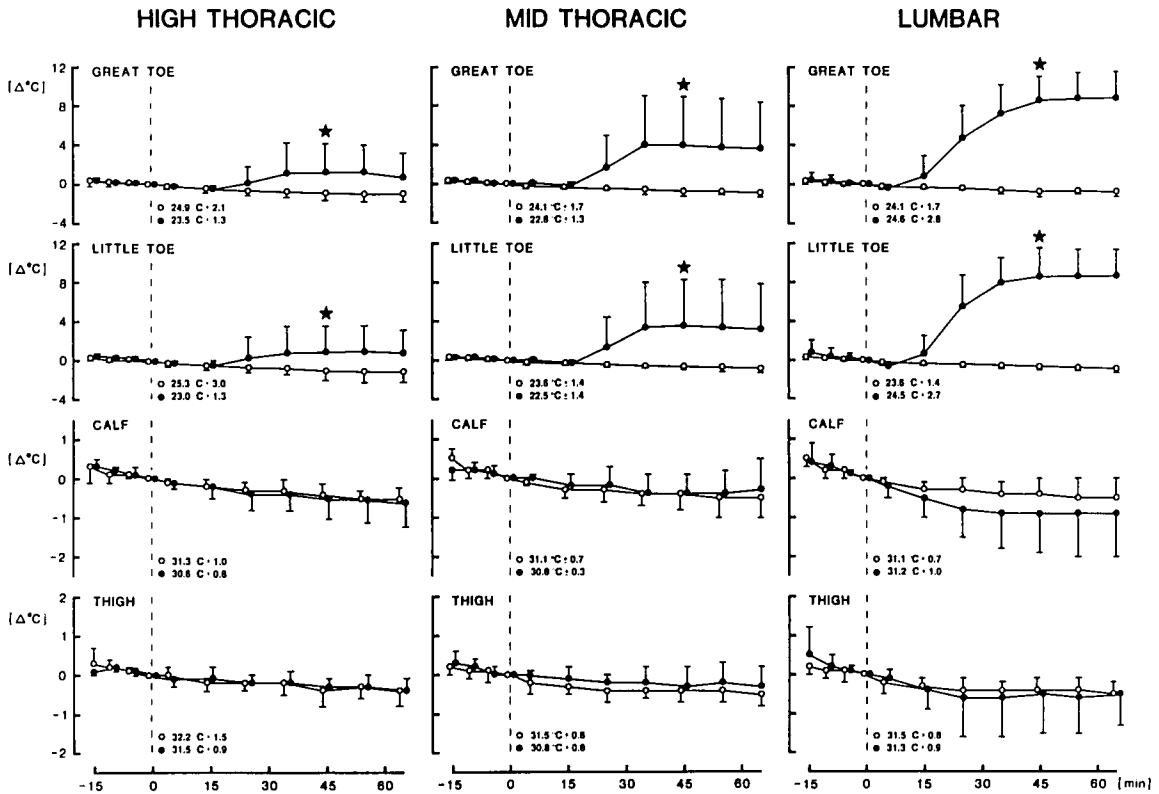


FIG. 2. Changes in lower limb skin temperatures from baseline with high thoracic, midthoracic, and lumbar epidural injection of either bupivacaine (filled symbols) or saline (open symbols). Data represent means \pm SD. Baseline values of absolute skin temperature for each group and site also are shown. At 0 min, indicated by the vertical dashed line, solutions were injected epidurally as described in the methods section. Regardless of the injection site, temperatures on the toes gradually increased after epidural bupivacaine to reach a plateau around 45 min, but decreased after epidural saline. Note that skin temperature on the foot, *i.e.*, on both the great and little toe, significantly increased with mid- and even with high thoracic epidural bupivacaine injection, though to a lesser degree than after lumbar epidural bupivacaine. The increase was seen despite restriction of sensory block to the thorax. Also note that no temperature changes were observed with epidural anesthesia on the proximal limbs, *i.e.*, on the thigh and calf. Even with lumbar epidural anesthesia and sensory denervation of the thigh and calf, skin temperatures remained unchanged. (Star represents $P < 0.05$ compared to saline.)

± 7 , $P < 0.001$) and midthoracic epidural bupivacaine ($-7 \text{ min}^{-1} \pm 6$, $P < 0.025$). No complications resulted from the study, and in all patients, the epidural catheter was used subsequently to provide intra- or postoperative analgesia.

With intramuscular bupivacaine no increase in regional skin temperatures was detected at any site.

Discussion

Segmental thoracic epidural anesthesia evoked an increase in foot skin temperature not only after midthoracic, but also after high thoracic injection of bupivacaine, *i.e.*, when sensory block was confined to the upper thoracic dermatomes. Thus, sympathetic blockade extends to and involves the most caudal portion of the sympathetic nervous system even with high thoracic segmental epidural anesthesia.

We have used changes in skin temperatures on the foot and hand as an indicator of altered sympathetic outflow, as have others.⁷⁻¹⁰ Whereas changes in peripheral skin temperature provide only an indirect measure of changes in skin blood flow and sympathetic drive, increases in skin temperatures on feet and hands are well documented when sympathetic drive is diminished, such as with spinal, epidural, or brachial plexus anesthesia.¹¹⁻¹³

Our conclusions rest on the tenable premise that the temperature increase on the foot after thoracic epidural anesthesia results from diminished sympathetic outflow rather than from the presence of absorbed bupivacaine in the blood. First, the mass of bupivacaine injected epidurally was very small and unlikely to exert cardiovascular effects after absorption. In fact, the same dose given intramuscularly did not evoke an increase in foot skin temperature although equal plasma bupivacaine concentrations were observed after epidural and intramuscular in-

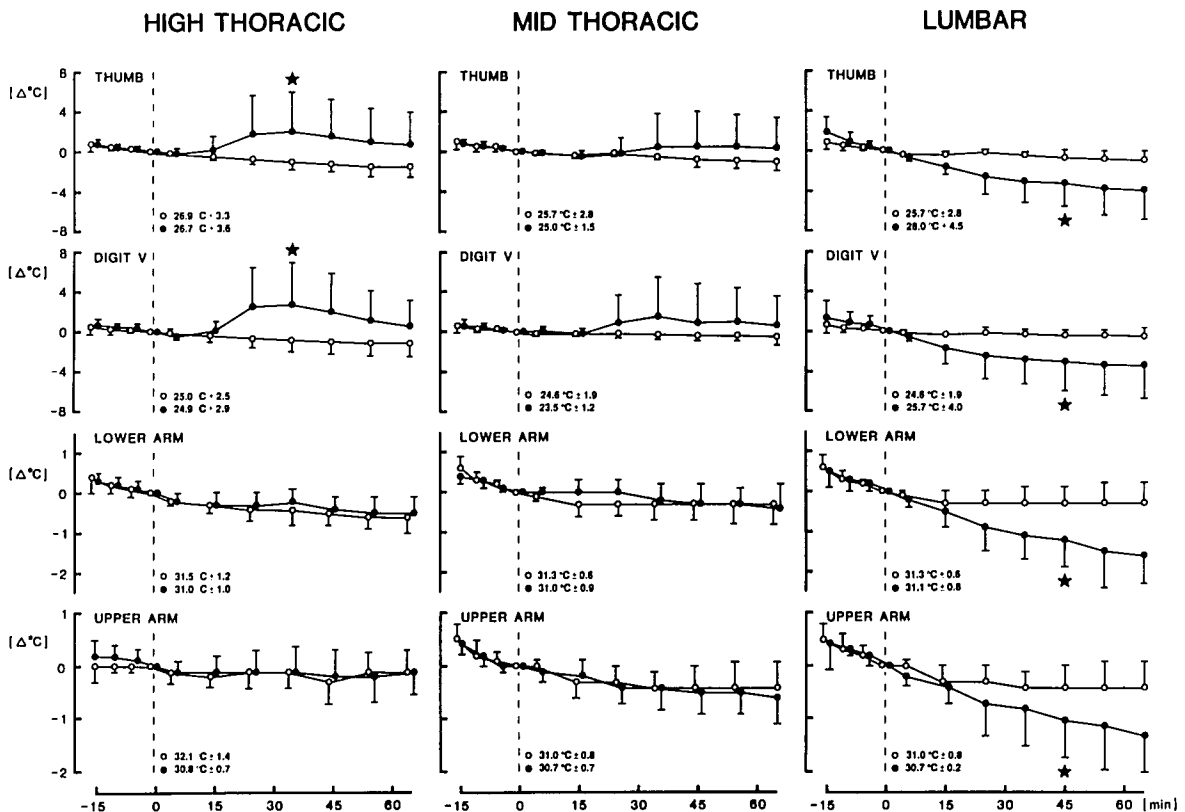


FIG. 3. Changes in upper limb skin temperatures from baseline with high thoracic, midthoracic, and lumbar epidural injection of either bupivacaine (filled symbols) or saline (open symbols). Data represent means \pm SD. Baseline values of absolute skin temperature for each group and site also are shown. At 0 min, indicated by the vertical dashed line, solutions were injected epidurally as described in the methods section. Skin temperatures on the hand, *i.e.*, on the thumb and on digit 5, increased significantly with high thoracic epidural anesthesia, but decreased with saline. Note that, similar to those in the legs (fig. 2), increases in skin temperatures were confined to the distal part of the limb, *i.e.*, the hand, whereas no changes were observed on the lower and upper arm. With midthoracic epidural bupivacaine neither hand nor arm skin temperatures changed significantly. In contrast, with lumbar epidural anesthesia, finger and lower and upper arm skin temperatures decreased significantly, suggesting compensatory vasoconstriction in the upper limb. (Star represents $P < 0.05$ compared to saline.)

jection, respectively.¹⁴ Second, if absorbed bupivacaine alone rather than a decreased efferent sympathetic drive were responsible for the increased skin temperatures on the feet after thoracic epidural anesthesia, hand skin temperatures also would be expected to increase after lumbar epidural injection. Instead, hand skin temperatures decreased significantly, most likely because of reflex vasoconstriction. Thus, increased foot skin temperatures after epidural anesthesia in all likelihood result from diminished sympathetic outflow.

Regional skin temperatures can reflect changes in blood flow and sympathetic drive only if both ambient and body temperatures are constant.¹⁵ These preconditions were met in our study.

We compared changes in foot skin temperature after epidural injection at the various sites under the assumption that changes in skin temperature correlate with changes in skin blood flow. Indeed, in humans, finger and toe skin

temperature changes correlate closely with those of skin blood flow over a skin temperature range of 21 to 34°C when measured at ambient temperatures between 20 and 25°C.^{16,17} These ranges were not exceeded in our study, so that a greater increase in foot skin temperature after epidural anesthesia most likely reflects a greater diminution of sympathetic drive.

An ambient temperature (23°C) below the thermoneutral range of naked resting humans¹⁵ was chosen because the diminution of peripheral vasoconstrictor tone evoked by epidural anesthesia might have been difficult to demonstrate under conditions of absent or low basal tone, *i.e.*, at or above the thermoneutral range. To compensate for the effects of any residual time dependent fluctuations in skin temperature, we contrasted prospectively the effects of epidural bupivacaine with those of epidural saline. Thus, the study protocol was sufficiently sensitive to detect changes in skin temperatures, if present,

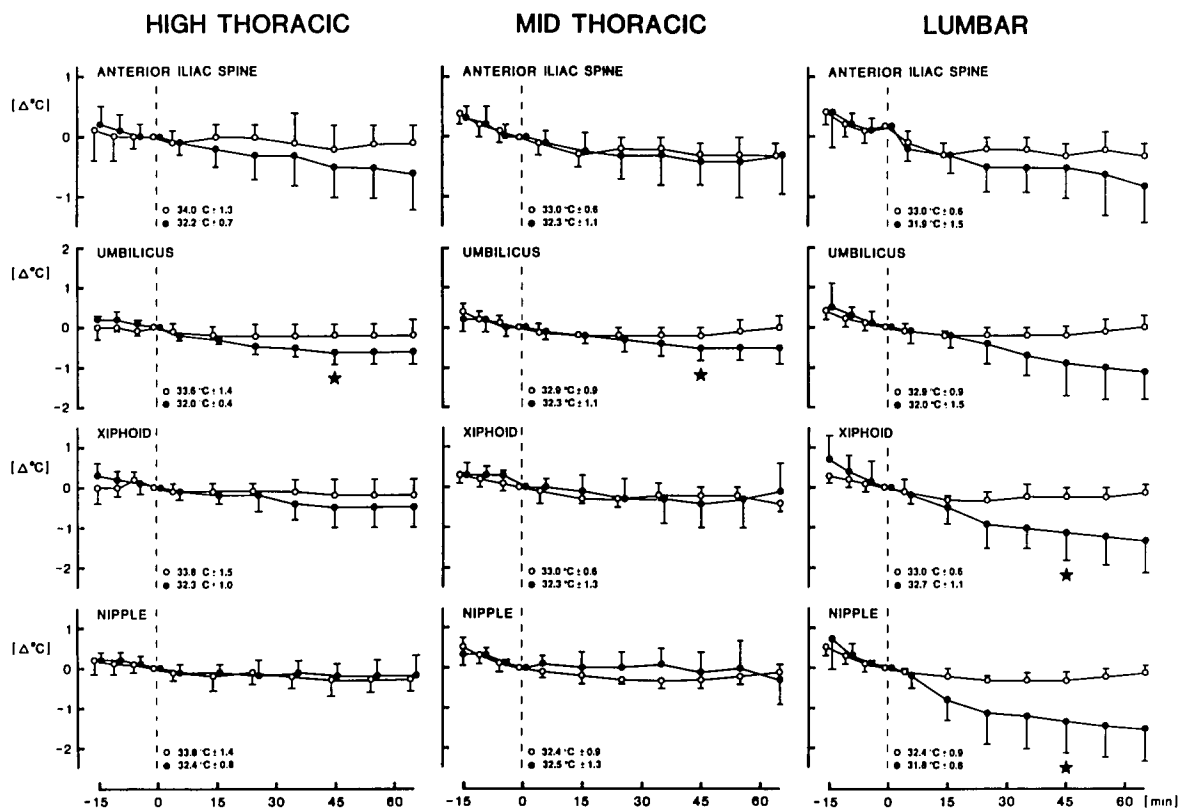


FIG. 4. Changes in trunk skin temperatures from baseline with high thoracic, midthoracic, and lumbar epidural injection of either bupivacaine (filled symbols) or saline (open symbols). Data represent means \pm SD. Baseline values of absolute skin temperature for each group and site also are shown. At 0 min, indicated by the vertical dashed line, solutions were injected epidurally as described in the methods section. Regardless of epidural injection site, trunk skin temperatures either did not change or decreased significantly after epidural bupivacaine. With high thoracic epidural anesthesia, trunk skin temperatures remained unchanged within the area of sensory block (nipple and xiphoid) and decreased significantly below this area on the umbilicus. Midthoracic epidural anesthesia resulted in decreased trunk skin temperatures within (umbilicus), but unchanged skin temperatures above (nipple) or below (anterior iliac spine) the sensory denervated dermatomes. With lumbar epidural anesthesia, skin temperatures decreased above the level of sensory block (umbilicus, xiphoid, and nipple), possibly indicating vasoconstriction in unblocked regions. Thus, on the trunk skin, temperatures remained unchanged even within areas deprived of sensory innervation. (Star represents $P < 0.05$ compared to saline.)

and by evaluating effects of epidural saline as a control, also was suitable to account for any alterations in skin temperature unrelated to epidural anesthesia.

The increase in foot skin temperatures with thoracic segmental epidural anesthesia even when sensory block was restricted to the upper thorax is a new observation and indicates that sympathetic blockade extends below the area of sensory block. Several reasons may explain this finding. First, the sympathetic system lacks distinct segmental organization: postganglionic sympathetic fibers overlap several dermatomes relative to the entrance of sensory fibers into the spinal cord.^{18,19} Efferent nerves supplying the legs are believed to emerge from the cord only below T12²⁰ or T10.²¹ However, some authors have speculated that efferents from higher segments may contribute to vasomotion in the legs since toe temperatures begin to decrease as the cranial border of sensory block

regressed to T6–7 or T8–11 during recovery from lumbar spinal or epidural anesthesia, respectively.^{11,22} Thus, block of sympathetic fibers in the high thoracic epidural space may have diminished sympathetic drive to the foot and yet still have preserved sensory innervation in the lower limbs.

Second, attenuation of peripheral sympathetic outflow may have resulted from penetration of the local anesthetic into the spinal cord,^{23,24} which may diminish the tonically active nerve traffic from supraspinal sympathetic neurons in the brain to spinal preganglionic neurons.^{18,25–27}

Finally, subarachnoid caudal diffusion of bupivacaine also may explain the increase in foot skin temperature after thoracic epidural anesthesia, since preganglionic sympathetic B fibers may be more susceptible to local anesthetics²⁸ than are sensory fibers, and since minimum blocking concentrations are reached in the subarachnoid

space 10–30 min after an epidural injection.²⁹ Regardless of the responsible mechanism, mid- and even high thoracic segmental epidural anesthesia increased skin temperature as far caudal as the foot despite restriction of sensory blockade to the upper thorax. Whereas no statistically significant difference in the magnitude of the foot skin temperature increase was found between high and mid-thoracic segmental epidural anesthesia, foot skin temperature increased most with lumbar epidural anesthesia. This indicates that sympathetic outflow to the legs is diminished with midthoracic and even with high thoracic segmental epidural anesthesia, albeit not to the maximum extent possible. This finding is at variance with the traditional concept that, in contrast to spinal anesthesia,³⁰ the extent of sensory and sympathetic blockade is congruent with epidural anesthesia.^{1,2}

Sympathetic blockade increased skin temperature only on feet and hands, but not on the proximal portion of the limbs or on the trunk, even in dermatomes deprived of sensation. Similar to our observations, calf skin temperatures have been reported to be unchanged after both lumbar spinal⁷ and epidural³¹ anesthesia with a sensory block up to T4 and even when foot skin temperatures were markedly increased. On the trunk, skin temperature was found by most^{7,8} but not all⁹ studies to decrease significantly after spinal anesthesia.

The lack of temperature increase on these regions is hardly surprising, since the ability of a body part to alter its temperature (and so to emit heat) in response to blood flow changes is mainly determined by its surface/volume ratio.^{32,33} This ratio is 22-fold greater for toes and fingers than for the trunk.³³ Furthermore, the skin circulation of hands and feet, but not of the trunk, is under the influence of tonically active sympathetic vasoconstrictor fibers, particularly below thermoneutral temperature.^{15,34–36} All of these factors may explain why increases in skin temperature in response to diminished sympathetic drive were detected only on the feet and hands.

Assuming that increased foot skin temperatures reflect diminished sympathetic outflow, we conclude that thoracic segmental epidural anesthesia can result in a widespread diminution of sympathetic outflow extending to and including the most caudal part of the sympathetic system. Even high thoracic segmental epidural anesthesia attenuates sympathetic activity on the legs, despite a sensory block restricted to the upper thorax. Therefore, our results do not support the widely held view that sympathetic blockade does not exceed sensory block with epidural anesthesia.^{1,2} Rather, it appears impossible to induce a segmental sensory block by thoracic epidural anesthesia and yet leave sympathetic vasomotor control caudal to the border of sensory blockade unimpaired.

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