Reduction in the Shivering Threshold Is Proportional to Spinal Block Height

Kate Leslie, M.B.B.S., F.A.N.Z.C.A.,* Daniel I. Sessler, M.D.†

Background: Hypothermia is nearly as common, and may be as severe, during spinal and epidural anesthesia as during general anesthesia. The authors have proposed that thermoregulatory failure results when regional anesthesia increases apparent leg skin temperature to a level far exceeding actual leg skin temperature. Extensive dermatomal blocks will alter thermal input to the hypothalamus from a greater skin-surface area more than less extensive ones and thus might be expected to impair central thermoregulatory control more. Accordingly, they tested the hypothesis that reduction in the shivering threshold is directly related to the number of dermatomes blocked during spinal anesthesia.

Methods: Eleven men, aged 62 ± 6 yr (mean ± SD), undergoing urologic surgery were studied. Ice-cold lactated Ringer's solution was administered intravenously before spinal blockade, and the shivering threshold (triggering core temperature) was established. Spinal anesthesia then was induced using a randomly assigned dose of 0.5% bupivacaine (2–4 ml). Again, sufficient cold lactated Ringer's solution was given to induce shivering. Tympanic membrane, ambient and skin temperatures were measured, and extent of block was defined by loss of temperature discrimination. Presence of shivering was evaluated by a blinded observer. Mean upper-body skin and ambient temperatures, cooling rates and intravenous fluid volumes at the two thresholds were compared using paired, two-tailed t-tests (P < 0.05). Linear regression defined the relationship between reduction in shivering threshold and the number of dermatomes blocked.

Results: There were no significant differences between mean upper-body skin and ambient temperatures, cooling rates or intravenous fluid volumes at the control and spinal shivering thresholds. Spinal anesthesia reduced the shivering threshold in direct relation to the number of dermatomes blocked: Δ threshold = 0.74 – 0.06 (dermatomes blocked); r² = 0.58, P < 0.006.

Conclusions: Extensive spinal blockade impairs central thermoregulatory control more than less extensive blockade. Clinicians can thus anticipate more core hypothermia during extensive than during restricted spinal blockade. (Key words: Anesthesia: spinal. Temperature: core; hypothermia; shivering; skin; tympanic membrane. Thermoregulation: central thermoregulatory control.)

HYPOTHERMIA is nearly as common, and may be as severe, during spinal and epidural anesthesia as during general anesthesia. Core hypothermia during general anesthesia is associated with adverse outcomes, including an increased incidence of myocardial ischemia, facilitated development of wound infections, and coagulopathy. There is no reason to expect that regional anesthesia confers immunity against these hypothermia-related complications. Limiting core hypothermia during spinal or epidural anesthesia may therefore improve patient outcome. Three major factors contribute to core hypothermia during regional anesthesia: (1) internal redistribution of body heat, (2) heat loss to the environment, and (3) inhibition of central thermoregulatory control.

Both redistribution and excessive heat loss contribute significantly to core hypothermia. However, neither impairs central thermoregulatory control. Consequently, hypothermia resulting from either mechanism can potentially trigger thermoregulatory defenses including vasoconstriction and shivering (above the level of the block) and behavioral responses (such as complaining of feeling cold). These defenses can minimize further hypothermia by decreasing heat loss, increasing heat production, or prompting the application of additional insulation or initiation of active warming.

* Staff Anaesthetist, Department of Anaesthesia, Royal Melbourne Hospital, Parkville, Victoria, Australia.
† Professor, Department of Anaesthesia and Intensive Care, University of Vienna; Associate Professor of Anaesthesia, Department of Anaesthesia, University of California, San Francisco, California.

Received from the Department of Anaesthesia, Royal Melbourne Hospital, Parkville, Victoria, Australia; Department of Anaesthesia and Intensive Care, University of Vienna, Vienna, Austria; and the Thermoregulation Research Laboratory, Department of Anaesthesia, University of California, San Francisco, California. Submitted for publication October 18, 1995. Accepted for publication February 14, 1996. Supported by a research grant from the Australian and New Zealand College of Anaesthetists, the Joseph Drown Foundation (Los Angeles, CA), and National Institutes of Health Grant GM49670. The authors do not consult for, accept honoraria from, or own stock or stock options in any anesthesia-related company. Presented in part at the annual Scientific Meeting of the Australian and New Zealand College of Anaesthetists, Townsville, Australia, May 9, 1995.

Address correspondence to Dr. Leslie: Department of Anaesthesia, Royal Melbourne Hospital, Parkville, Victoria, 3050, Australia.

Anesthesiology. V 84, No 6, Jun 1996
The final major factor contributing to core hypothermia during regional anesthesia is a failure of normal centrally mediated thermoregulatory control. Thermoregulatory inhibition is insidious compared with redistribution and excessive heat loss because substantial hypothermia can develop without symptoms to alert either the patient or the anesthesiologist. We have proposed that failure results when regional anesthesia increases apparent leg skin temperature to a level far exceeding actual leg temperature. Increased apparent skin temperature then decreases the threshold (triggering core temperature) at which vasoconstriction and shivering are initiated. Extensive regional blocks will alter thermal input to the hypothalamus from a greater skin-surface area than less extensive ones, and thus might be expected to impair central thermoregulatory control more. Accordingly, we tested the hypothesis that reduction in the shivering threshold during spinal anesthesia is directly related to the number of dermatomes blocked.

Methods

With approval from the Board of Medical Research at the Royal Melbourne Hospital, we studied 11 consenting men, of ASA physical status 1 or 2, undergoing urologic surgery during spinal anesthesia. The sample size was determined on the basis of typical interindividual variation in thermoregulatory responses, and our previous experience with this type of study. The patients' age was 62 ± 6 yr (mean ± SD), height 170 ± 7 cm and weight 75 ± 14 kg. None had a history of ischemic heart disease, cerebrovascular disease, or any specific contraindication to shivering or infusion of cold fluid. None of the patients reported any systemic diseases, or was taking medication, known to affect thermoregulation. Patients were randomly assigned to receive 2–4 ml 0.5% spinal bupivacaine in 7.5% dextrose.

Protocol

No sedatives were administered. Patients rested supine, covered with a single sheet, and routine anesthesia variables, including blood pressure, heart rate, and hemoglobin oxygen saturation, were monitored continuously. A 14-G, 57-mm long cannula was inserted into an antecubital vein of the nondominant arm. Core hypothermia was induced by intravenous administration of \( \approx 4^\circ{C} \) lactated Ringer's solution at a rate of \( \approx 50 \text{ ml/min} \). The antecubital area was covered with a bag of warmed fluid to minimize perception of fluid temperature. Core cooling rates were restricted to \(<2^\circ{C}/h \) because such rates are unlikely to trigger dynamic thermoregulatory responses. After 5 min of continuous shivering, the cold fluid infusion was stopped, and the patients were rewarmed using forced air (Bair Hugger, Augustine Medical, Eden Prairie, MN) until shivering ceased and core temperatures were restored to control values.

Spinal anesthesia was then induced. The dura mater was punctured at the L2–L3 vertebral level with a 27-G needle and bupivacaine was injected slowly. Correct needle placement was confirmed by repeated aspiration. When anesthesia was adequate, patients were placed in the lithotomy position, covered with one sheet, and surgery was started. Bladder irrigation fluid was warmed to \( 37^\circ{C} \). No sedatives or oxygen were given. The shivering threshold was then redetermined, using the same technique. The effect of circadian variations was minimized by determining control and spinal shivering thresholds within a 1-h period. Once the shivering threshold was determined, patients were again rewarmed using forced air.

Measurements

Core temperatures were measured at 5-min intervals at the tympanic membrane. The aural probes were inserted until felt by the patients next to their tympanic membranes; appropriate placement was confirmed by gently rubbing the wire. The probes then were taped in position and the canals were occluded with cotton. Mean upper-body skin temperatures were computed from measurements at three sites: \( 0.24(T_{\text{forehead}}) + 0.37(T_{\text{cheek}}) + 0.39(T_{\text{arm}}) \) as described previously. Ambient temperatures were measured from probes positioned near the patients, but well away from heat-generating devices. All temperatures were recorded using Mon-a-Therm thermocouple probes (Mallinckrodt Anesthesiology Products, St. Louis, MO).

Shivering was evaluated by observation of pectoralis major muscles. The observer, who was blinded to the patients’ core temperatures and dermatomal block levels, was the same for each study. As in previous investigations, \(^{15}\) shivering was graded: 0 = none, 1 = mild, 2 = vigorous. The shivering threshold was defined as the core temperature at the onset of sustained grade 1 shivering.

Block heights were determined using loss of temperature discrimination, at 10-min intervals.
Data Analysis
The difference between the shivering threshold before and after the spinal block was determined in each patient. The relationship between the number of blocked dermatomes and the reduction in the shivering thresholds was determined using linear regression. Mean upper-body skin temperatures, ambient temperatures, core cooling rates, and infused fluid volumes at the two thresholds, and core and mean upper-body skin temperatures before the start of each phase were compared using two-tailed, paired t tests. Results are expressed as mean ± SD; P < 0.05 was considered statistically significant.

Results
Mean upper-body skin temperatures were not significantly different before the control or spinal phases of the study (33.0 ± 0.8 vs. 33.2 ± 0.8; P = NS). Core temperature was slightly lower before the spinal than before the control phase (36.6 ± 0.5 vs. 36.9 ± 0.2°C; P = 0.02), but was greater than the spinal shivering threshold in all patients.

There were no statistically significant or clinically important differences in mean upper-body skin temperatures, ambient temperatures, core cooling rates, or infused fluid volumes at the two thresholds (Table 1).

Spinal anesthesia reduced the shivering threshold in direct relation to the number of dermatomes blocked, and residual analysis revealed this relationship to be linear: ∆ threshold = 0.74 − 0.06(dermatomes blocked); r² = 0.58, P < 0.006. The regression line crossed the ordinate when 12 segments were blocked (10th thoracic dermatome; figure 1).

Discussion
Three major factors contribute to core hypothermia during regional anesthesia. The first is redistribution hypothermia, which results when regional anesthesia blocks sympathetic nerves normally maintaining a core-to-peripheral temperature gradient.5,6 The magnitude of core hypothermia after peripheral nerve block depends on the extent of sympathetic block and the initial temperature difference between core and peripheral tissues.18 Although extensive dermatomal block levels are more likely to produce a nearly complete sympathectomy,19 sympathetic nerve block levels during regional anesthesia may be difficult to predict.6 The preinduction difference between core and peripheral tissue temperatures also is variable, and depends mostly on environment—and the patient’s response to that environment—during the hours preceding anesthesia.18

The second major cause of hypothermia during regional anesthesia is environmental heat loss exceeding metabolic heat production. Regional anesthesia per se minimally decreases heat production,5 but heat loss can be large in minimally covered11,12 patients undergoing large operations20 in a cold operating-room environment.21,22 The extent to which heat loss exceeds production depends largely on the ambient temperature,21,22 patient insulation (or active warming),11-13 incision size,20 and the amount of unwarmed intravenous fluid administered.23,24

Table 1. Skin and Ambient Temperatures, Core Cooling Rates, and Administered Fluid Volumes

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Spinal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean upper-body skin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>temperature (°C)</td>
<td>32.9 ± 0.7</td>
<td>33.1 ± 0.8</td>
</tr>
<tr>
<td>Ambient temperature</td>
<td>22.9 ± 1.5</td>
<td>23.2 ± 1.6</td>
</tr>
<tr>
<td>Cooling rate (°C/h)</td>
<td>1.1 ± 0.6</td>
<td>1.5 ± 1.0</td>
</tr>
<tr>
<td>Intravenous fluid</td>
<td>0.9 ± 0.5</td>
<td>1.1 ± 0.6</td>
</tr>
</tbody>
</table>

Data are mean ± SD. There were no significant differences between treatments.
The third major cause of hypothermia is anesthetic-induced inhibition of centrally mediated thermoregulatory control. Our results confirm that the shivering threshold is reduced during epidural and spinal anesthesia.\textsuperscript{2,25} We have proposed that this abnormal tolerance for hypothermia results from increased apparent (as opposed to actual) lower-body skin temperature.\textsuperscript{9} Sympathectomy-induced vasodilatation does increase lower-body skin temperature \(\approx 1^\circ\text{C}\) during regional anesthesia, but this increase is insufficient to account for the observed thermoregulatory impairment.\textsuperscript{a} A corollary of this hypothesis is that more extensive blocks will increase apparent skin temperature over a larger surface area, and thus produce greater inhibition of thermoregulatory defenses. Our data are consistent with this expectation and support the theory that regional anesthesia increases tolerance for core hypothermia by increasing apparent lower-body skin temperature.

A curious facet of our results was that interruption of afferent thermal input from at least half the skin surface (=T10) did not impair thermoregulation. Similarly, no alteration in vasodilatation thresholds was observed during caudal anesthesia (=T12) in children.\textsuperscript{26} This result contrasts with previous studies in which the vasodilatation and shivering thresholds were reduced \(\approx 0.6^\circ\text{C}\) by epidural or spinal blocks to \(\approx T10\). Kurz et al.\textsuperscript{7} studied spinal anesthesia in young female volunteers, and reported decreases in shivering thresholds of \(\approx 0.5^\circ\text{C}\) at sentient skin temperatures near 35\(^\circ\text{C}\). Elevated sentient skin temperatures in Kurz’s volunteers likely account for their greater tolerance of hypothermia. Ozaki et al.\textsuperscript{25} studied spinal anesthesia in patients and volunteers. The patients had cool skin temperatures (not measured), whereas skin temperatures in the volunteers were near 36\(^\circ\text{C}\). The shivering thresholds in both groups were only slightly below normal. Why the reduction in the shivering threshold should vary in these populations remains unclear.

Thermoregulatory responses must be compared at a specific sentient skin temperature, because cutaneous sensation is an important afferent to the central regulatory system, accounting for \(\approx 20\%\) of input.\textsuperscript{27–30} We fulfilled this requirement in our study, because sentient skin temperatures were similar at the control and spinal and shivering thresholds, and were typical of patients in the operating room.

Our patients were older (\(\approx 60\) yr) than subjects in previous studies. During general anesthesia, the vasodilatation threshold is \(\approx 1^\circ\text{C}\) less in patients aged \(\approx 70\) yr than patients aged \(\approx 40\) yr.\textsuperscript{31} However, shivering thresholds during spinal anesthesia do not decrease until patients exceed age 80 yr.\textsuperscript{32} Age therefore is unlikely to have been an important confounding factor.

In conclusion, extensive spinal block impairs central thermoregulatory control more than less extensive block. Clinicians can thus anticipate more core hypothermia during extensive than restricted blocks. Temperature monitoring and thermal management may be necessary to avoid the complications of hypothermia in patients having extensive blocks—especially when they are administered for procedures large enough to significantly increase heat loss.

The authors thank Critical Assist, for the loan of a Bair Hugger forced air warmer, and Mallinckrodt Anesthesiology Products, for providing the thermocouples and thermometers used in this study. They also appreciate the assistance of staff at the Essendon campus of the Royal Melbourne Hospital in data collection, and thank the Department of Urology, for allowing recruitment of their patients.

References


15. Glosen B, Sessler DI, Faure EAM, Stuen R, Thisted RA, Karl L. Central temperature changes are not perceived during epidural anesthesia. ANESTHESIOLOGY 1992; 77:10-6


30. Lim T: Central and peripheral control mechanisms of shivering and its effects on respiration. J Appl Physiol 1960; 15:567-74


32. Vassieff N, Rosencher N, Sessler DI: Conseiller C: shivering threshold during spinal anesthesia is reduced in the elderly. ANESTHESIOLOGY 1995; 83:1162-6