Original Contributions

Postoperative Hemodynamic and Thermoregulatory Consequences of Intraoperative Core Hypothermia

Andrea Kurz, MD,* Daniel I. Sessler, MD,† Edith Narzt, MD,*, Amir Bekar, MD,‡ Rainer Lenhardt, MD,* Guenther Huemer, MD,§ Franz Lackner, MD‖

Department of Anesthesia and Intensive Care, University of Vienna, Vienna, Austria, and the Thermoregulation Research Laboratory, University of California School of Medicine, San Francisco, CA.

Study Objective: To evaluate the postoperative hemodynamic and thermoregulatory consequences of intraoperative core hypothermia.

Design: Prospective, randomized clinical trial.

Setting: Operating room and postanesthesia care unit of a university hospital.

Patients: 74 healthy, ASA status I, II, and III patients (average age 58 yrs) undergoing elective colon surgery.

Interventions: Patients were randomly assigned to be kept normothermic or ≤35°C hypothermic during surgery. Anesthesia was maintained with isoflurane, nitrous oxide, and fentanyl. Postoperatively, surgical pain was treated with patient-controlled analgesia (PCA) opioid.

Measurements and Main Results: An observer blinded to group assignment and core temperatures evaluated shivering, thermal comfort, surgical pain, heart rates (HRs), and blood pressures (BPS) during the first six postoperative hours. Morphometric characteristics, oxygen saturation, fluid balance, PCA-administered opioid, and visual analog pain scores were comparable in the two groups. Hypothermic patients felt uncomfortably cold during recovery, and their postoperative core temperatures remained significantly less than in the normothermic patients for more than four hours. Peripheral vasoconstriction and shivering were common in the hypothermic patients but rare in those kept normothermic. HRs and BPS were comparable in the two groups.

Conclusions: These data confirm that the effects of intraoperative hypothermia on postoperative HR and BP are modest in relatively young, generally healthy patients. In contrast, intraoperative hypothermia caused substantial postoperative thermal discomfort, and full recovery from hypothermia required many hours. Delayed return to core normothermia apparently resulted largely from postoperative thermoregulatory impairment.

Keywords: Anesthesia: postoperative care; blood pressure; heart rate; shivering; thermoregulation; vasoconstriction.
Introduction

Volatile anesthetics impair shivering, as evidenced by the observation that shivering is rare during surgery. Even in unparalyzed patients having core temperatures near 32°C. Similarly, all general anesthetics so far tested markedly reduce the core temperature, triggering thermoregulatory vasoconstriction.1-5 (Nonshivering thermogenesis is relatively unimportant in unanesthetized adults,6,7 and it does not contribute during general anesthesia.) Because thermoregulatory defenses are impaired by general anesthesia, most surgical patients become hypothermic unless they are actively warmed.9,10

During the initial postoperative period, brain anesthetic concentrations usually decrease rapidly,11 allowing re-emergence of thermoregulatory responses including vasoconstriction and shivering.12,13 These responses combine to decrease cutaneous heat loss,14 constrain metabolic heat to the thermal core,15 and increase metabolic heat production.13 As a result, core temperatures usually increase toward normal values once anesthesia is discontinued. One factor limiting return to normothermia is simply that some patients develop enormous heat debts during surgery. Based on the reported specific heat of humans,16 a 2.5°C reduction in mean body temperature corresponds to a debt of ~145 kcal in a 70 kg patient. This is roughly the basal heat production for 2.5 hours.17 Even when vasoconstriction is combined with vigorous shivering, some time will be required to replace this much heat. Additionally, impaired thermoregulatory responses may limit full postoperative recovery of thermal equilibrium. Such impairment may result from: (1) residual volatile anesthetic,9 (2) opioids administered to treat surgical pain,18-22 or, (3) a delayed effect of intraoperative hypothermia.

Recovery of normal core temperature in the postoperative period is a clinical priority because it may limit some hypothermia-related complications.13,23-26 The extent to which the above factors contribute—and any potential interactions among them—cannot be predicted from current thermoregulatory theories. Postoperative thermoregulatory responses also have the potential to adversely affect hemodynamic values during recovery: shivering by increasing metabolic demand15 and vasoconstriction by increasing peripheral vascular resistance.23 Accordingly, we evaluated the postoperative hemodynamic and thermoregulatory consequences of intraoperative core hypothermia.

Materials and Methods

All studies were undertaken with approval of the University of Vienna Ethics Committee and informed consent from the subjects.

Protocol

We studied 74 ASA physical status I, II, and III patients undergoing elective colon surgery. None of the participants had a history of thyroid disease, dysautonomia, Raynaud’s syndrome, or malignant hyperthermia. Surgery started at approximately 8:30 AM in nearly all of the patients, and typically finished near noon.

Patients were premedicated with 10 mg oral diazepam. General anesthesia was induced by administration of 3 to 5 mg/kg of thiopental sodium, fentanyl 250 µg, and vecuronium 0.1 mg/kg, and the trachea was intubated, and the lungs were ventilated mechanically to maintain end-tidal CO₂ partial pressure (PET CO₂) near 35 mmHg. Anesthesia subsequently was maintained with 60% nitrous oxide (N₂O) and isoflurane. The isoflurane concentration was adjusted as necessary to keep mean arterial pressure (MAP) greater than 60 mmHg, and additional fentanyl was given at the discretion of the attending anesthesiologist. Additional vecuronium was administered as required to maintain 1 to 2 mechanical twitches in response to supramaximal electrical stimulation of the ulnar nerve at the wrist.

Patients were randomly assigned, via a computer-generated random numbers table, to routine thermal management (hypothermic group, n = 35) or extra warming (normothermic group, n = 39). (The patients were not informed as to which treatment group they were assigned.) The upper body of those assigned to extra warming was covered with a disposable Bair Hugger® forced-air cover (Augustine medical, Inc., Eden Prairie, MN) that was connected to a Model 500 heater (Augustine medical, Inc.) set on “high” (~40°C). We10 and others20 have shown that this system maintains normothermia, even during extensive surgical procedures. The other patients were allowed to become hypothermic. In those patients whose core temperature approached 34°C, forced-air warming was instituted to prevent further hypothermia. Ambient room temperature was kept at 21°C to 22°C intraoperatively and 23°C to 25°C postoperatively.

Intravenous (IV) fluids were administered at a basal rate of 10 to 15 ml/kg/hr. Additionally, 4 ml of crystalloid was given to compensate for each estimated milliliter of intraoperative blood loss. Blood was administered as necessary to maintain a hematocrit level of at least 25% to 30%. Fluid was heated to 37°C in the patients assigned to extra warming, but not in those assigned to routine thermal management.

Most patients participating in this study were relatively healthy (ASA physical status I or II). Only 2 patients in each group were assigned to ASA physical status III. Only 3 of 35 hypothermic and 5 of 39 normothermic patients took antihypertensive medications before surgery. No vasoactive drugs were used during or after surgery.

When surgery was complete, the neuromuscular block was antagonized by administration of neostigmine 2.5 mg and glycopyrrolate 0.5 mg. The tracheas were then extubated, and the patients immediately transferred to the post-anesthesia care unit, where they were monitored for six hours. Neither group was warmed during the recovery period.

During the recovery period, oxygen (O₂) was admin-
istered at an initial rate of 6 L/min via nasal prongs. Additional \( \text{O}_2 \) was given by mask if necessary to maintain oxygen saturation as measured by pulse oximetry (\( \text{SpO}_2 \)) greater than 95%. Administered \( \text{O}_2 \) was then gradually reduced to 2 to 3 L/min over the first few hours of recovery. IV fluids were administered at a rate of at least 2 ml/kg. Postoperative pain was treated with the opioid piritramide delivered via a patient-controlled analgesia (PCA) pump (background infusion of 2.2 mg/hr; bolus dose of 1.5 mg; 15-minute lockout period). (This \( \mu \)-agonist is approximately four times as potent as morphine sulphate.) The nurses and physicians administering fluid and managing pain were blinded to the patients' group assignments and core temperatures.

**Monitoring**

Ambient temperatures were measured using a thermocouple positioned at the level of the patient, well away from any heat-producing equipment. Core temperatures were measured at the tympanic membrane. The tympanic probe was inserted until the patients felt the thermocouple touch the tympanic membrane. Appropriate placement was confirmed when they easily detected a gentle rubbing of the attached wire. The probe was then taped in place, the aural canal was occluded with cotton, and the external ear was covered with a gauze pad. Tympanic membrane temperatures correlate well with distal esophageal temperatures in the perioperative period.33-30

Thermoregulatory vasoconstriction was evaluated using forearm minus fingertip, skin-temperature gradients.31 The gradients were recorded from an arm not having an IV cannula or blood pressure (BP) cuff. As in most of our previous studies,1-3 we considered a gradient exceeding 4°C to indicate significant vasoconstriction. This value was chosen because it indicates nearly maximal vasoconstriction and it was unlikely to result from inadequate vascular volume or poorly controlled surgical pain.

Heart rate (HR) was monitored continuously using three-lead electrocardiography. \( S_p \text{O}_2 \) was measured continuously using pulse oximetry, and BP was determined oscillometrically at 5-minute intervals from the upper arm (Model 68S, Hewlett Packard, Waltham, MA). We used oscillometric rather than direct arterial BP measurements to minimize the artifact induced by thermoregulatory vasoconstriction.32-33 End-tidal isoflurane concentration was measured with a Cícero infrared monitor (Dräger, Lübeck Germany).

Postoperatively, shivering was evaluated qualitatively, using a three-point scale: grade zero indicated no shivering, grade one indicated mild or intermittent shivering, grade two indicated moderate shivering, and grade three indicated prolonged, intense shivering. Thermal comfort was evaluated using a 100-mm-long visual analog scale (VAS), with 0 mm indicating intense cold, 50 mm indicating thermal comfort, and 100 mm indicating intense warmth. Surgical pain was similarly evaluated with a VAS: 0 mm on this scale indicated no pain and 100 mm indicated the most intense pain imaginable. These qualitative assessments were made by an observer who was blinded to the patients' group assignment and core temperatures. Data were recorded at 20-minute intervals.

**Data Analysis**

Morphometric data, anesthetic parameters, and initial and final intraoperative temperatures in the two groups were compared using unpaired \( t \) tests. Postoperative data also were compared using two-tailed, unpaired \( t \) tests. As suggested by Matthews et al.,34 we limited the number of comparisons by evaluating differences only at 1-hour intervals. All values are expressed as means ± SD. A \( p \)-value less than 0.01 was considered statistically significant.

**Results**

There were no statistically significant differences in morphometric characteristics or anesthetic management in the patients assigned to hypothermia and normothermia (Table 1). Core temperatures before induction of anesthesia were similar in both groups (37.0 ± 0.2°C vs. 37.1 ± 0.3°C).

As per protocol, core temperatures at the end of surgery in the hypothermic group were significantly less than those in the warmed patients (34.4 ± 0.4°C vs. 37 ± 0.3°C, Figure 1). Initial postoperative core temperatures in the warmed patients were 36.8 ± 0.4°C, and they increased only slightly during the 6-hour recovery period. In contrast, initial core temperatures were 34.7 ± 0.5°C in those patients who were allowed to become hypothermic, and they increased at a rate of \( \approx 0.5^\circ \text{C/hr} \) for the first three hours of recovery, and at \( \approx 0.3^\circ \text{C/hr} \) for

| Table 1. Morphometric Characteristics, Anesthetic Management, and Intraoperative Temperatures in Patients Assigned to Hypothermia and Normothermia |
|--------------------|----------------|----------------|
| Hypothermia        | Normothermia   |
|--------------------|----------------|----------------|
| Age (yr)           | 59 ± 14        | 57 ± 15        |
| Weight (kg)        | 70 ± 13        | 71 ± 13        |
| Height (cm)        | 168 ± 10       | 170 ± 9        |
| Gender (M/F)       | 17/18          | 22/17          |
| Duration of Surgery (hr) | 3.5 ± 1.3     | 3.3 ± 1.1      |
| Intraoperative fluid (L) | 3.4 ± 1.0     | 3.5 ± 0.9      |
| Administered fentanyl (µg) | 650 ± 190     | 720 ± 220      |
| End-tidal (isoflurane) (%) | 0.6 ± 0.1     | 0.6 ± 0.2      |
| Final intraoperative core temp (°C) | 34.4 ± 0.4     | 37 ± 0.3*      |

*Only final intraoperative core temperatures differed significantly in the patients given each treatment.
Intraoperative Postoperative

Figure 1. Core temperatures before induction of anesthesia were similar in both groups. Core temperatures changed little in patients warmed with forced air but decreased ~2.5°C in the unwarmed patients. Core temperature in the unwarmed patients increased at a rate of ~0.5°C/hr for the first three hours (h) or recovery and at ~0.3°C/hr for the subsequent two hours. In contrast, core temperatures increased only ~0.1°C/hr for three hours and then remained constant in the patients who were warmed intraoperatively. Core temperatures in the hypothermic patients remained significantly different from those in the warmed group until more than 4 hours after surgery. Error bars indicate standard deviations; asterisks (*) indicate statistically significant differences between the groups.

Figure 2. All hypothermic patients demonstrated significant thermoregulatory vasoconstriction (gradients > 4°C) during the first three postoperative hours (h). Subsequently, gradients in most of these patients decreased but never became negative. None of the normothermic patients had postoperative gradients exceeding 4°C, and the mean was 2°C during the first 2 hours of recovery; subsequently, most of these patients developed negative (vasodilated) gradients. The hypothermic patients reported feeling unpleasantly cold, and the sensation persisted for more than 2 hours. Only a few of the warmed patients shivered at any time during the recovery period. In contrast, most of the hypothermic patients demonstrated grades 2 or 3 tremor during the first postoperative hour. By the end of the first elapsed hour, however, shivering was rare, even in the hypothermic patients. Error bars indicate standard deviations; asterisks (*) indicate statistically significant differences between the groups.

Discussion

As expected from previous studies, internal redistribution of body heat reduced core temperature ~1°C
During the first hour of anesthesia and surgery, the initial reduction in core temperature was comparable in both groups, but forced-air warming subsequently increased core temperature in treated patients whereas core temperature continued to decrease in those not actively warmed. Consequently, all patients assigned to forced-air warming were normothermic at the end of surgery, despite having undergone large abdominal operations in a cool environment. Such efficacy is consistent with previous studies showing that forced-air warming transfers more heat than circulating-water or infrared heating, and maintains intraoperative core temperature better than circulating-water or inspiratory gas heating and humidification.

As expected from previous studies, none of the normothermic patients vasoconstricted during surgery. Isoflurane (0.75% end-tidal) decreases the thermoregulatory threshold for vasoconstriction to approximately 35°C. Our current patients were given less N₂O (60%) and less isoflurane (≈0.6%) than those in our previous study, but they were also given fentanyl. Thus, it is not surprising that the vasoconstriction threshold in these patients was similar.

Core temperatures between 36°C and 37.5°C define the normothermic range in typical populations. However, the range of core temperatures tolerated without triggering thermoregulatory responses (interthreshold range) in a given individual at a given time is only ±0.2°C. This “setpoint” range varies diurnally, usually having a minimum near 3:00 AM and a maximum, =1°C greater, near 3:00 PM. Initial postoperative core temperatures in the warmed patients averaged 36.8 ± 0.4°C. Although this value is well within the normothermic range, it is likely that the lower boundary of the interthreshold range was near 37°C at noon when most of these patients finished surgery. It is thus not surprising that core temperatures, even in the “normothermic” patients, increased slightly during the initial hours of postanesthetic recovery.

The initial autonomic thermoregulatory response to hypothermia is peripheral vasoconstriction, mediated primarily by arteriovenous shunts located in the fingers, toes, and nose. The next response in infants is nonshivering thermogenesis, but nonshivering thermogenesis does not occur in adults given isoflurane, and it probably contributes little to thermoregulation in unanesthetized adults. And finally, shivering is initiated when core temperature is sufficiently less than the “setpoint” range. Most hypothermic patients both vasoconstricted and shivered. Despite the shivering and vasoconstriction, core temperatures in the hypothermic patients increased relatively slowly during the first five postanesthetic hours.

Core temperatures in the hypothermic group required more than 4 hours to reach values comparable to those in the patients kept normothermic during surgery. The relatively slow 0.5°C/hr increase in core temperature suggests that thermoregulatory compensations were blunted by residual anesthetic or a time-dependent factor. Far more rapid increases in core temperatures have been observed under other circumstances, suggesting that prolonged hypothermia did not result simply from the magnitude of the heat debt, but rather from inadequate activation of thermoregulatory compensations. Consistent with this theory is our observation that shivering stopped long before core temperature returned to normal.

At least three factors may have contributed to prolonged postoperative thermoregulatory inhibition. The first is residual volatile anesthetic. The dose-response curve for thermoregulatory inhibition by isoflurane appears linear at surgical doses. Inhibition has not been specifically tested at the low concentrations typical during recovery. But if impairment is indeed a linear function of dose in this range, representative early postoperative anesthetic concentrations (e.g., 0.2% isoflurane) might decrease the vasoconstriction threshold to 0.6°C. Although we did not measure postoperative

Table 2. Postoperative Oxygen Saturation (SpO₂) Fluid Balance, Analgesia, and Temperatures During the Initial Six Hours of Postanesthetic Recovery

<table>
<thead>
<tr>
<th>Hypothermia</th>
<th>Normothermia</th>
</tr>
</thead>
<tbody>
<tr>
<td>SpO₂ (%)</td>
<td>98 ± 1</td>
</tr>
<tr>
<td>Administered fluid (L)</td>
<td>1.4 ± 0.6</td>
</tr>
<tr>
<td>Urate output (L)</td>
<td>0.6 ± 0.4</td>
</tr>
<tr>
<td>Administered piritramid (mg)</td>
<td>19 ± 9</td>
</tr>
<tr>
<td>Ambient temperature (°C)</td>
<td>25.1 ± 0.6</td>
</tr>
<tr>
<td>Initial core temperature (°C)</td>
<td>34.7 ± 0.5</td>
</tr>
<tr>
<td>Core temperature after 6 hr (°C)</td>
<td>37.2 ± 0.4</td>
</tr>
</tbody>
</table>

Note: *Initial postoperative core temperatures were less in the patients assigned to intraoperative hypothermia.

Figure 3. Heart rates and blood pressures were similar in the normothermic and hypothermic patients throughout the 6-hour recovery period. Error bars indicate standard deviations; none of the differences was statistically significant.
end-tidal isoflurane concentrations, values exceeding 0.2% surely did not persist beyond the first half hour of recovery. Such concentrations would not be sufficient to explain the observed prolonged hypothermia in the unwarmed patients.

Opioids represent an additional factor perhaps impairing postoperative thermoregulatory control. Not only were opioids used as an anesthetic adjuvant, but the patients were given systemic opioids postoperatively to treat surgical pain. Opioids cause a variety of species-specific and dose-specific effects in animals, most typically hyperthermia at low doses and hypothermia at higher doses. The thermoregulatory consequences of opioid administration in humans remain unclear, but they certainly do not generally include hyperthermia. Many opioids inhibit postanesthetic shivering, and it is probable that these drugs also cause generalized thermoregulatory impairment in humans. It is likely that such impairment reduced thermoregulatory efficacy and, consequently, prolonged hypothermia in our postoperative patients. Consistent with this mechanism is our observation that recovery of core normothermia was considerably more rapid in a previous study in which opioids were not administered. A final factor potentially contributing to impaired postoperative thermoregulatory responses is a prolonged effect of intraoperative core hypothermia. The extent to which postoperative thermoregulatory control is altered by intraoperative thermal disturbances remains unknown. However, patients recovering from hypothermic cardiopulmonary bypass frequently develop a postoperative fever that appears to be a compensation for the period of intraoperative hypothermia. Our data do not allow us to quantify the contribution—if any—of prior thermal perturbations to postoperative regulatory impairment.

Hemodynamic responses were similar in the two groups, with no statistically significant or clinically important differences in HRs or BPs between the normothermic and hypothermic patients. In our previous evaluation of hemodynamic responses in adults recovering from isoflurane anesthesia, HRs usually did not differ significantly or were slightly greater when the volunteers were normothermic than when they were hypothermic. In that study, however, BPs were significantly greater after hypothermic anesthesia than during normothermic recovery. A critical difference between that study and our current one is that we previously measured BPs at the radial artery, whereas in this study we made oscillometric measurements at the brachial artery.

We have recently described a vasoconstriction-induced increase in radial arterial systolic blood pressures (SBPs) relative to brachial and femoral arterial pressures. In all likelihood, the exaggerated radial arterial pressures that we observed previously were artfactually increased as a result of thermoregulatory vasoconstriction and did not reflect an increase in central BP. Consistent with this possibility, hemodynamic responses (measured oscillometrically) were comparable in normothermic and hypothermic infants and children recovering from general anesthesia. Little or no change in central BPs during thermoregulatory vasoconstriction is also consistent with the relatively small fraction of the cardiac output traversing arteriovenous shunts.

In contrast to our findings of comparable HRs and comparable or increased BPs during recovery from hypothermic anesthesia, Gorman et al. reported decreased HRs and increased SBPs and radial arterial BPs in hypothermic postoperative patients. Important differences between these studies include the facts that Gorman et al. studied an elderly group of relatively sick patients and that those patients were admitted directly to the intensive unit and quite likely were heavily sedated. Consistent with thermoregulatory inhibition in the elderly and impairment of shivering induced by opioid administration, little shivering was observed even in their hypothermic patients. Nonetheless, HRs and BPs may have been greater in the hypothermic patients than in the normothermic patients studied by Gorman et al. because thermoregulatory vasoconstriction had more hemodynamic effect in their elderly population.

Although statistically significant differences in IIRs and BPs were reported in some of the above studies, the differences usually were small. These data suggest that aggravated cardiac ischemia in hypothermic postoperative patients may be mediated by a mechanism more subtle than gross hemodynamic alterations. We did not look for myocardial ischemia in this study. Another limitation of our study is that we did not evaluate plasma catecholamine concentrations, which might have provided a better indication of hypothermia-induced "stress."

All hypothermic patients demonstrated significant thermoregulatory vasoconstriction postoperatively. Mean skin-temperature gradients exceeded 4°C for more than 3 hours during recovery. Few of these patients vasodilated fully (gradient <0°C), even when core temperature returned to normal values. This prolonged hypothermia is a consequence of large reductions in body heat content. Based on the reported specific heat of humans, a 3°C reduction in mean body temperature corresponds to a depth of 175 kcal in a 70 kg patient. This roughly equals the basal heat production for three hours. Even when vasoconstriction is combined with vigorous shivering, considerable time may be required to replace so much heat.

The average skin-temperature gradient in the patients kept normothermic was positive for the first several hours of recovery, and then decreased to less than 0°C, indicating full vasodilation. It is likely that the initial vasoconstriction was nonthermoregulatory and resulted from stress and poorly controlled surgical pain.

Shivering was common in the patients who were allowed to become hypothermic, with most patients shivering at some time during the first postoperative hour. In contrast, shivering was rare in the patients kept normothermic during surgery. These data are consistent with our previous observations that all spontaneous oscillatory muscular activity following isoflurane anes-
sia is thermoregulatory. In that study, we identified two tremor patterns, the most common having electromyographic patterns similar to those produced by cold-induced shivering in the absence of anesthesia. Far less common was a phasic pattern resembling pathologic clonus. Although we did not evaluate tremor patterns in this study, it is likely that both patterns contributed to the observed muscular activity. Measurements of $O_2$ consumption would have quantified physiologic stress of shivering better than our qualitative assessments. However, such measurements have been reported previously.

$SpO_2$ values were comparable in the two groups, as would be expected since $O_2$ administration was titrated to maintain $SpO_2$ greater than 95%. Nonetheless, $SpO_2$ was easily maintained in all patients, suggesting that hypothermia and the resulting shivering did not significantly predispose to hypoxemia in the relatively healthy patients we studied. (Thermoregulatory vasoconstriction per se slightly increases $SpO_2$, but the increase is only $\approx 2\%$, which is not clinically important.) These results are consistent with our observations in hypothermic young adults. They also are consistent with our previous observation that desaturation is rare in hypothermic infants and children during the postoperative period, even when they are given no supplemental $O_2$. It remains likely that patients having preexisting severe pulmonary compromise would tolerate postoperative hypothermia and shivering less successfully, and at least one such episode has been reported.

We have postulated that shivering per se aggravates pain simply because vigorous muscular activity will jar areas in which surgical incisions were made. It also is possible that hypothermia directly increases pain perception, or that it alters the pharmacodynamics of opioids. Our hypothermic and normothermic patients required comparable amounts of opioid (administered via PCA) to maintain similar VAS pain scores. However, it remains likely that hypothermia delayed opioid clearance and that plasma concentrations were greater in the unwarmed patients. Since we did not measure plasma opioid concentrations, we cannot directly evaluate the extent to which postoperative hypothermia contributes to surgical pain.

In summary, we randomly assigned 74 patients to be kept normothermic or $\approx 2.5^\circ C$ hypothermic during colon surgery. Morphometric characteristics, $SpO_2$, fluid balance, PCA-administered opioid, and VAS pain scores were comparable in the two groups. Hypothermic patients felt uncomfortably cold during recovery, and their postoperative core temperatures remained significantly less in the normothermic patients for more than four hours. Shivering was common in the hypothermic patients, but rare in those kept normothermic. HRs and BPs were comparable in the two groups. These data indicate that intraoperative hypothermia causes postoperative thermal discomfort and that full recovery from hypothermia requires many hours. Delayed return to core normothermia appears to result largely from postoperative thermoregulatory impairment. It is likely that impairment results, at least in part, from opioids given to treat surgical pain.

Acknowledgment

We thank Gepa-Med, Inc. Vienna, Austria, for donating the disposable Bair Hugger® forced-air covers that we used.

References


