Thermoregulatory Response to Intraoperative Head-Down Tilt

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Thermoregulation interacts with cardiovascular regulation within the central nervous system. We therefore evaluated the effects of head-down tilt on intraoperative thermal and cardiovascular regulation. Thirty-two patients undergoing lower-abdominal surgery were randomly assigned to the 1) supine, 2) 15°–20° head-down tilt, 3) leg-up, or 4) combination of leg-up and head-down tilt position. Core temperature and forearm minus fingertip skin-temperature gradients (an index of peripheral vasoconstriction) were monitored for 3 h after the induction of combined general and lumbar epidural anesthesia. We also determined cardiac output and central-venous and esophageal pressures. Neither right atrial transmural pressure nor cardiac index was altered in the Head-Down Tilt group, but both increased significantly in the Leg-Up groups. The vasoconstriction threshold was reduced in both leg-up positions but was not significantly decreased by head-down tilt. Final core temperatures were 35.2°C ± 0.2°C (mean ± SEM) in the Supine group, 35.0°C ± 0.2°C in the Head-Down Tilt group, 34.2°C ± 0.2°C in the Leg-Up group ($P < 0.05$ compared with supine), and 34.3°C ± 0.2°C when leg-up and head-down tilt were combined ($P < 0.05$ compared with supine). These results confirm that elevating the legs increases right atrial transmural pressure, reduces the vasoconstriction threshold, and aggravates intraoperative hypothermia. Surprisingly, maintaining a head-down tilt did not increase right atrial pressure.

(Anesth Analg 2002;94:221–6)

Mild hypothermia often accompanies general and neuraxial anesthesia (1). Hypothermia is associated with numerous complications, including morbid myocardial outcomes (2), coagulopathy (3), and surgical wound infections (4). Hypothermia also prolongs postanesthetic recovery and hospitalization and is remarkably uncomfortable for patients.

The major cause of intraoperative hypothermia is core-to-peripheral redistribution of body heat. Redistribution results largely because general anesthetics reduce the vasoconstriction threshold (triggering core temperature) 2°C–4°C (1), thus defeating tonic thermoregulatory vasoconstriction. The extent to which anesthetics reduce the vasoconstriction threshold depends on the type of drug and its concentration.

However, thermoregulatory control is also affected by nonthermal factors, including signals from the cardiovascular system (5,6). We have recently reported that patients undergoing lower-abdominal surgery with their legs elevated became more hypothermic than usual, a decrease that is attenuated by positive end-expiratory pressure (7). These data suggest that baroreceptor loading augments perioperative hypothermia by reducing the core-temperature triggering peripheral vasoconstriction.

Lower-abdominal surgery is often performed with patients tilted head-down because it improves surgical visualization by moving visceral organs cephalad. Just as elevating the legs increases baroreceptor loading and reduces the vasoconstriction threshold, tilting the head downward increases central blood volume in unanesthetized subjects (8,9). The head-down position may thus impair thermoregulatory control, thereby increasing perioperative hypothermia. Accordingly, we tested the hypothesis that head-down positioning during lower-abdominal surgery increases baroreceptor loading, thereby augmenting the usual anesthetic-induced reduction in the vasoconstriction threshold and aggravating core hypothermia.

Supported by National Institutes of Health Grant GM 58273, the Joseph Drown Foundation, and the Commonwealth of Kentucky Research Challenge Trust Fund. Mallinckrodt Anesthesiology Products, Inc. (St. Louis, MO) donated the thermocouples we used. None of the authors has a financial relationship with any company related to this research.

Presented in part at the 12th World Congress of Anesthesiologists, Montreal, Canada, June 6, 2000.

Accepted for publication August 31, 2001.

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0003-2999/01

Methods

This study was approved by the Review Board on Human Experiments, Kyoto Prefectural University of Medicine, and written informed consent was obtained from each participant. Thirty-two patients were recruited for this study. All were ASA physical status I or II, between 20 and 60 yr old, and scheduled for open lower-abdominal (colorectal or gynecological) surgery. None was obese, febrile, or receiving vasodilators or medications likely to alter thermoregulation; none had a history of thyroid disease or dysautonomia.

The tip of a central-venous catheter was placed at the right atrium the day before surgery, and its position was confirmed radiologically. Patients fasted for 8 h before the study. All the operations were performed between 8:00 AM and noon. The operating room was maintained near 24°C at a relative humidity of approximately 40%. Participating patients were kept in the operating room at least 30 min before surgery so they could become accustomed to the environment. During this time and at least 30 min before the induction of anesthesia, an 18-gauge catheter was inserted into the left antecubital vein; at least 10 $mL \cdot kg^{-1} \cdot h^{-1}$ of lactated Ringer's solution at ambient temperature was given. A 22-gauge catheter was inserted into the left radial artery for pressure monitoring and blood sampling. Additionally, an epidural catheter was inserted via the L1-2 or the L2-3 vertebral interspaces with the patient in a lateral position.

General anesthesia was induced by IV administration of 2 mg/kg propofol, and tracheal intubation was performed after IV administration of 0.15 mg/kg vecuronium. For maintenance of anesthesia, patients inhaled 0.4% isoflurane and 66% nitrous oxide in oxygen. An IV infusion of vecuronium, initially set to 0.025 $mg \cdot kg^{-1} \cdot h^{-1}$, was adjusted to maintain one or two twitches in response to supermaximal stimulation of the ulnar nerve at the wrist. Mechanical ventilation was adjusted to maintain end-tidal P_{CO_2} between 35 and 40 mm Hg. After an initial dose of 7 mL of 1% lidocaine without epinephrine into the epidural catheter, 0.25% bupivacaine at a rate of 5 mL/h was infused for the remainder of surgery to obtain epidural analgesia.

Patients were randomly assigned to one of four positions: supine ($n = 8$), 15°–20° head-down tilt (Trendelenburg position, $n = 8$), leg-up (lithotomy position, $n = 8$), and leg-up combined with head-down tilt ($n = 8$). The designated positions were initiated 10 min after the induction of general anesthesia and were maintained for 3 h.

Blood pressure, heart rate, oxygen saturation, end-tidal P_{CO_2}, and end-tidal isoflurane concentrations were recorded at 5-min intervals. Upper- and lower-body sensory block levels were evaluated after emergence from general anesthesia by response to cold sensation.

Core temperature was measured at the tympanic membrane by using Mon-a-Therm® thermocouples (Mallinckrodt; St. Louis, MO). The aural probes were inserted by the patients until they felt the thermocouple touch the tympanic membrane; appropriate placement was confirmed when they easily detected a gentle rubbing of the attached wire. The aural canal was occluded with cotton and taped in place. We also recorded forearm minus fingertip skin-temperature gradients (10).

We considered the difference between central-venous and esophageal pressures at end-expiration to represent right atrial transmural pressure. Esophageal pressure was measured with a fluid-filled intraesophageal balloon (TY-102 U; Nihon Kohden) that was advanced into the distal third of the intrathoracic esophagus; proper position was confirmed by appropriate changes in esophageal pressure with respiration after the induction of anesthesia. Both pressures were referenced to the midaxillary line, and the transducers were attached to the skin at the level where the tips of the central venous pressure catheter or esophageal balloon were most likely located. Both pressures were recorded every 5 min.

Cardiac output was measured 10 (before postural change), 30, 60, 90, 120, 150, and 180 min after the induction of anesthesia. Cardiac output was derived from the average of three separate 1-mL boluses of 5 mg indocyanine green (ICG) that were injected into the antecubital vein. Dye concentration was measured with pulse dye-densitometry (DDG analyzer; Nihon Kohden, Tokyo, Japan). Serum ICG concentration becomes negligible within 20 min; consequently, measurements at the designated time points were not affected by previous ICG injections (11).

Blood samples were collected from the radial artery at 20, 90, and 180 min after the induction of anesthesia. Samples were immediately centrifuged at 4°C, and aliquots of the plasma were stored at −80°C until assayed. Plasma epinephrine and norepinephrine were measured by high-performance liquid chromatography with an electrochemical detector after alumina extraction. Radioimmunoassay kits were used to evaluate plasma concentrations of angiotensin II (Angiotensin II; Nichols Institute, CA) and renin (Renin RIABEAD; Dainabot, Tokyo, Japan).

As in previous studies (7), we defined the vasoconstriction threshold as the tympanic temperature that triggered a rapid increase in the skin-temperature gradient.

Table 1. Patient Demographics and Preinduction Values

Variable	Group			
	Supine	Head-Down Tilt	Leg-Up	Leg-Up and Head-Down Tilt
Age (yr)	50 ± 3	49 ± 4	47 ± 4	52 ± 6
Weight (kg)	63 ± 5	62 ± 5	58 ± 4	60 ± 4
Height (cm)	161 ± 3	160 ± 3	162 ± 3	165 ± 5
Sex (M/F)	4/4	3/5	3/5	4/4
Mean arterial pressure (mm Hg)	86 ± 2	92 ± 5	91 ± 4	90 ± 5
Pulse pressure (mm Hg)	50 ± 4	52 ± 5	53 ± 3	54 ± 4
Central venous pressure (mm Hg)	5.1 ± 0.8	4.9 ± 0.6	5.0 ± 0.8	5.4 ± 0.5
Heart rate (bpm)	88 ± 4	85 ± 3	82 ± 4	88 ± 4
Tympanic (core) temperature (°C)	36.8 ± 0.1	36.7 ± 0.1	36.9 ± 0.2	36.8 ± 0.1

Values are expressed as mean ± SEM, $n = 8$ per group. There was no statistically significant difference among the groups.

The threshold was determined individually for each patient by an investigator blinded to treatment. Once the threshold was reached, thermal responsiveness (gain) was defined by the slope of a regression between the skin-temperature gradient and core temperature.

Baseline values were averaged over the final 30 min before the induction of general anesthesia. Intraoperative values were presented over time or first averaged within each patient and then averaged among the patients in each group. Thermal responsiveness (gain) and the core temperature threshold data for vasoconstriction were analyzed with general linear regression models for one-way analysis of variance (ANOVA; one between factor), followed by Scheffé multiple-comparison tests.

The effects of postural differences and time on the cardiovascular, thermogenic, and hormonal responses were analyzed by general linear regression model procedures for two-way ANOVA with repeated measures (one between, one within factors), followed by Scheffé multiple comparison tests. Results are presented as mean ± SEM; $P < 0.05$ was considered statistically significant.

Results

The patients in the four groups were comparable with respect to sex, age, weight, and height. Preinduction cardiovascular and thermal values were also similar (Table 1). Anesthetic management did not differ significantly among the groups (Table 2), and no patient received a blood transfusion.

Although slight fluctuations in arterial blood pressure were observed at the induction of anesthesia and during surgery, no appreciable difference was demonstrated in the mean arterial pressure or pulse pressure throughout the study among the groups. Heart rate decreased by approximately 15–20 bpm during surgery in all groups, but no significant difference was shown among the groups.

Patients assigned to either Leg-Up group vasoconstricted at lower core temperatures than the Supine group ($P < 0.05$). In contrast, vasoconstriction occurred at similar core temperatures in the Supine and the Head-Down groups (Fig. 1). Core body temperature in the Leg-Up groups decreased significantly more than in the Supine Control group starting 85 min after the induction of anesthesia ($P < 0.05$) (Fig. 2, Table 3). The onset of peripheral vasoconstriction was delayed in both Leg-Up groups compared with the Supine group ($P < 0.05$) but was similar in the Supine and Head-Down Tilt groups (Fig. 2).

The gain of vasoconstriction (slope of the forearm minus fingertip temperature gradient/core temperature relationship below the threshold) was 11.1 ± 3.5 in the Supine group, 9.5 ± 3.1 in the Head-Down group, 3.2 ± 2.8 in the Leg-Up group, and 4.1 ± 3.6 in the Head-Down Plus Leg-Up group (Fig. 1). There was thus a trend toward lower gain in the Leg-Up ($P = 0.09$ compared with Supine) and the Head-Down Plus Leg-Up ($P = 0.15$ compared with Supine) groups, but these differences were not statistically significant.

Right atrial transmural pressure was significantly increased in the Leg-Up and the Leg-Up combined with Head-Down Tilt groups than in the Supine group ($P < 0.05$). However, right atrial transmural pressures in the Supine and the Head-Down Tilt groups were similar, because central venous pressure and esophageal pressure increased comparably in the Head-Down Tilt group. Cardiac index increased significantly in the Leg-Up and Leg-Up combined with Head-Down groups ($P < 0.05$), whereas in the Head-Down group, cardiac index did not differ significantly from that of the Supine group (Fig. 3).

No significant difference was found among the groups in plasma epinephrine or norepinephrine concentrations during surgery (Fig. 4). Plasma renin activity and plasma angiotensin II concentrations 3 h after anesthetic induction were similar in the Supine and Head-Down groups, but

Table 2. Anesthetic Management During Surgery

Variable	Group			
	Supine	Head-Down Tilt	Leg-Up	Leg-Up and Head-Down Tilt
Ambient temperature (°C)	24.0 ± 0.4	23.9 ± 0.4	24.1 ± 0.4	24.2 ± 0.3
Blood loss at 3 h (mL)	322 ± 35	289 ± 32	271 ± 29	250 ± 42
Fluid replacement at 3 h (mL)	2401 ± 150	2351 ± 165	2340 ± 194	2249 ± 152
End-tidal isoflurane (%)	0.4 ± 0.1	0.4 ± 0.1	0.4 ± 0.1	0.4 ± 0.1
Sensory block (median, range)	T10, T7–T12	T9, T7–T12	T10, T8–T12	T10, T7–T12

Values are expressed as mean ± SEM unless otherwise noted; $n = 8$ per group. There was no statistically significant difference among the groups.

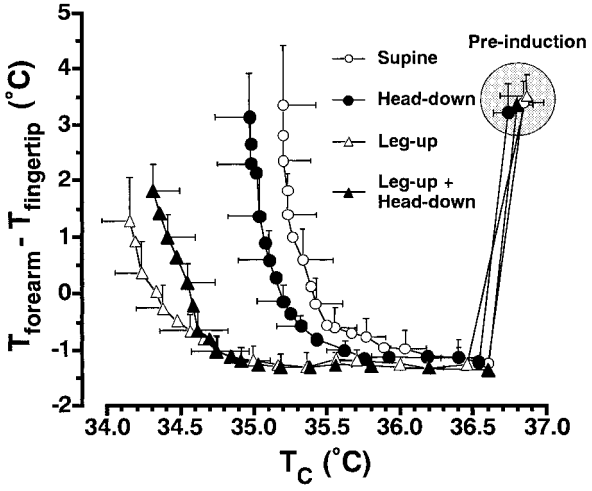

Figure 1. Forearm minus fingertip skin-temperature gradient ($T_{forearm} - T_{fingertip}$) as a function of tympanic membrane core temperature (T_C). Data are presented as mean ± SEM.

were both significantly less in the Leg-Up position group than in the Supine group ($P < 0.05$).

Discussion

We recently reported that positioning patients with their legs up during lower-abdominal surgery augments perioperative hypothermia by attenuating peripheral vasoconstriction. In contrast, applying positive end-expiratory pressure prevents hypothermia by enhancing peripheral vasoconstriction. The level of baroreceptor loading thus alters the threshold for vasoconstriction and thermal responsiveness (gain) of this defense (7). Lower-abdominal surgery is often performed with the head tilted downward to improve surgical visualization by dislocating the visceral organs cephalad. We hypothesized that using a head-down tilt position during lower-abdominal surgery would enhance hypothermia by reducing the vasoconstriction threshold, just as it does during the leg-up position.

A head-down tilt increases central blood volume in unanesthetized humans (8,9). Curiously, we failed to

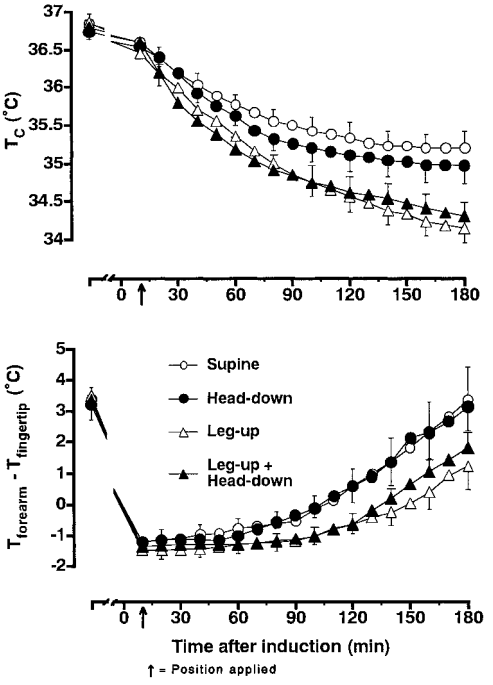

Figure 2. Core body temperature (T_C) and forearm minus fingertip skin temperature gradient ($T_{forearm} - T_{fingertip}$) after the induction of anesthesia. The leg-up position and/or the head-down tilt position started 10 min after the induction of anesthesia. All T_C values after 85 elapsed minutes in the Leg-Up Position group and the Leg-Up Combined Head-Down Tilt Position group differed significantly compared with the Supine group. All forearm minus fingertip skin temperature gradients after 120 elapsed minutes in the Leg-Up Position group were smaller than in the Supine group, as were those in the combined Leg-Up and Head-Down Tilt group. Values are shown as mean ± SEM.

detect any effect of the head-down tilt position on our indexes of venous return: the right atrial transmural pressure and cardiac index. Our observation that a head-down tilt does not increase central blood volume during general anesthesia combined with epidural anesthesia was surprising. However, a head-down tilt does not augment venous return in patients in hemorrhagic shock (12,13), and placing patients in the leg-up, but not the head-down, position increases mean arterial pressure during hypotension induced by spinal anesthesia (14,15). Furthermore, one report

Table 3. Thermoregulatory Responses

Variable	Group			
	Supine	Head-Down Tilt	Leg-Up	Leg-Up and Head-Down Tilt
Vasoconstriction threshold (°C)	35.7 ± 0.2	35.4 ± 0.2	34.8 ± 0.2*	34.8 ± 0.2*
Gain of vasoconstriction	11.1 ± 3.5	9.5 ± 3.1	3.2 ± 2.8	4.1 ± 3.6
Core temperature at 3 h (°C)	35.2 ± 0.2	35.0 ± 0.2	34.2 ± 0.2*	34.3 ± 0.2*

Values are expressed as mean ± SEM, $n = 8$ per group.
* $P < 0.05$ compared with the Supine group.

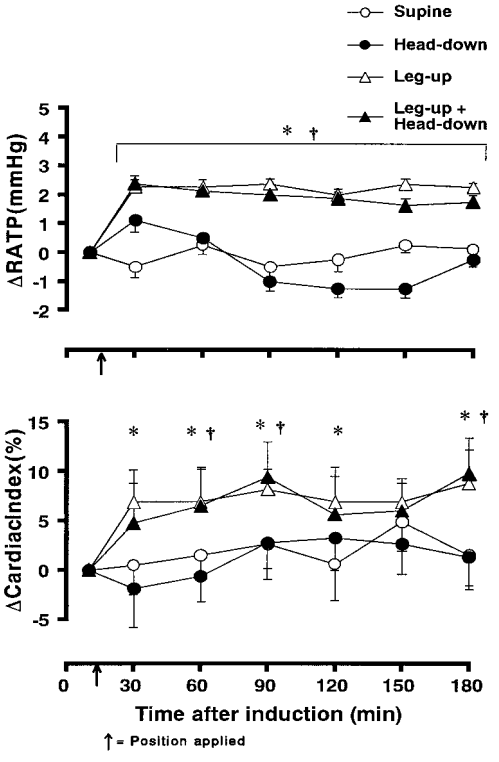

Figure 3. The top graph plots the right atrial transmural pressure (RATP), and the bottom graph plots the changes in cardiac index compared with shortly after the induction of anesthesia. The leg-up position and/or head-down tilt positions started 10 min after the induction of anesthesia. Values are shown as mean ± SEM. *$P < 0.05$ for the Leg-Up group compared with the Supine group. †$P < 0.05$ for the Combined Leg-Up and Head-Down group compared with the Supine group.

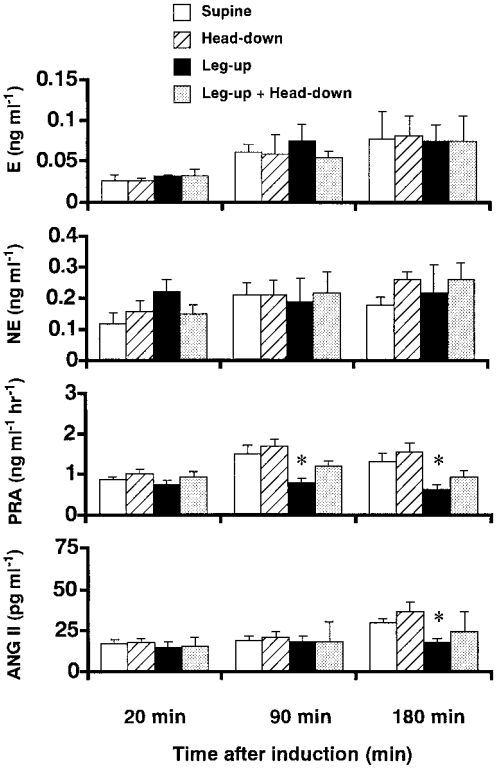

Figure 4. Plasma epinephrine (E), norepinephrine (NE), renin activity (PRA), and angiotensin II (ANGII) concentrations 20, 90, and 180 min after anesthetic induction. Data are presented as mean ± SEM. *$P < 0.05$ for the Leg-Up group compared with the Supine group.

suggests that the hemodynamic consequences of the head-down tilt position are questionable—even in normovolemic and normotensive patients (16).

The critical factor may be that we included epidural anesthesia as part of our management. Although epidural anesthesia did not induce significant hypotension in any of the groups, the resulting sympathetic block may have prevented an increase in central blood volume. Alternatively, the normal hemodynamic response to head-down may have been prevented in our patients by another mechanism that remains to be elucidated.

Another possibility is that central blood volume was increased in the head-down tilt position, but that esophageal pressure was comparably increased, possibly by visceral organs shifting the diaphragm upward. It is likely that such a shift would inhibit augmentation of venous return and cardiac output. This theory is supported by studies showing that cardiac output is reduced despite an increase in central venous pressure in patients undergoing laparoscopy in the head-down tilt position (17,18). Right atrial transmural pressure was not directly measured in those studies, but the results suggested that it was reduced, because the diaphragm was dislocated upward by a

pneumoperitoneum. Additionally, in mechanically ventilated paralyzed patients, thoracic volume is reduced by an upward shift of the diaphragm (19). Although disagreement persists regarding the change in the central blood volume during mechanical ventilation (20,21), enhanced positive thoracic pressure resulting from pressure by visceral organs might also have prevented any increase in right atrial transmural pressure in the Head-Down Tilt position group in our study.

Plasma renin activity and angiotensin II levels in the Head-Down Tilt group did not differ from those of the Supine Control group, whereas they were decreased in the Leg-Up group. Baroreceptor loading reduces plasma renin concentration and renal sympathetic nervous activity in humans (9). These hormonal data indirectly support our conclusion that the head-down position does not increase venous return in normovolemic, anesthetized, paralyzed patients.

Because baroreceptor loading was not influenced by head-down tilt, it is not surprising that this position had little thermoregulatory consequence. This finding is consistent with a single previous study in which the head-down tilt position during surgery also failed to enhance perioperative hypothermia. However, the patients in that study did not become sufficiently hypothermic to trigger thermoregulatory vasoconstriction. That study also lacked accurate measurement of the vasoconstriction threshold, cardiovascular variables, and hormonal responses (22). The data that were presented here are thus the first to fully evaluate the effects of head-down tilt on baroreceptor loading, thermoregulatory vasoconstriction, and core hypothermia.

In summary, we evaluated thermoregulatory vasoconstriction and core temperature in patients given combined epidural and general anesthesia for lower-abdominal surgery. Head-down tilt did not accentuate venous return and, therefore, did not augment baroreceptor loading. Consequently, the threshold for thermoregulatory vasoconstriction remained unchanged, and the patients became no more hypothermic than those maintained supine. The head-down tilt position thus does not require special perioperative thermal precautions or management unless the leg-up position is used simultaneously.

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