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Combination of Thoracic Epidural Anesthesia Does Not Always Induce Hypothermia during General Anesthesia

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Abstract

The combination of general anesthesia and epidural anesthesia has been considered to worsen the degree of hypothermia. However, epidural anesthesia reduces cardiac output, which may prevent redistribution hypothermia. Twenty-four patients undergoing gynecologic surgery were randomly assigned to one of two groups: recipients of epidural injection of 1% ropivacaine and general anesthesia (epidural and general group, n=12) and recipients of epidural injection of saline and general anesthesia (general group, n=12). Fifteen minutes after epidural injection of 12 mL of 1% ropivacaine (epidural and general group) or saline (general group), general anesthesia was induced with propofol, and tracheal intubation was facilitated with vecuronium. Anesthesia was maintained with 35% oxygen and 0.4% to 2% isoflurane with a nitrous oxide mixture. Tympanic (core), forearm, and fingertip temperatures were recorded before the epidural injection, just before induction of general anesthesia, just after tracheal intubation, and every 15 minutes up to 90 minutes after tracheal intubation. The core temperature was significantly higher in the epidural and general group than in the general group from 30 to 90 minutes after tracheal intubation. Epidural anesthesia with 1% ropivacaine may prevent redistribution hypothermia during general anesthesia for gynecologic surgery.
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Key words: general anesthesia, epidural anesthesia, hypothermia, thermoregulation

Introduction

Mild hypothermia during anesthesia can induce severe adverse outcomes, such as wound infection and a prolonged hospital stay[1]. Anesthesia induces distribution hypothermia through vasodilation and changes in the thermoregulatory threshold. Neuraxial anesthesia, such as spinal and epidural anesthesia, combined with general anesthesia inhibits transmission to the brain of neural information about hypothermia and decreases the shivering and the vasoconstriction thresholds, thus accelerating hypothermia[2]. Joris et al. have suggested that the addition of epidural anesthesia to general anesthesia could induce more severe hypothermia than would general anesthesia alone because of a decrease in the vasoconstriction threshold[3].

On the other hand, the degree of hypothermia is

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influenced by drugs administered to induce anesthesia and vasodilators or inotropic agents administered during surgery[4,5]. Drugs that dilate peripheral arteries and increase cardiac output induce hypothermia, whereas drugs that constrict peripheral arteries and decrease cardiac output prevent the development of hypothermia[5,6].

Even though vasodilation and changes in the thermoregulatory threshold due to epidural anesthesia may induce hypothermia, the hemodynamic consequences of epidural anesthesia may affect the development of hypothermia by preventing heat from traveling to peripheral tissues. Thus, we hypothesized that thoracic epidural anesthesia with sensory block to a higher dermatomal level combined with general anesthesia contributes to the prevention of hypothermia.

Materials and Methods

Twenty-four patients who underwent gynecological laparotomy were asked to participate in this single-blind placebo-controlled trial. The Institutional Review Board approved the study, and written informed consent was obtained from each patient. Patients were randomly assigned to one of two groups; a general group and an epidural and general group. Patients in the general group received general anesthesia consisting of a high concentration of isoflurane (1.5% to 2%) during surgery and epidural injection of saline. The epidural and general group received both general anesthesia with low concentrations of isoflurane (0.5% to 1%) and an epidural injection of 1% ropivacaine. Premedication consisted of atropine sulfate, 0.5 mg; pentazocine, 15 mg; and hydroxydine, 25 mg; which were administered 30 minutes before anesthesia induction. In the operating room, the left antecubital vein was secured for infusion, and Ringer's acetate solution (kept at room temperature: approximately 25℃) was infused at a rate of 7 to 10 mL/kg/h. The ambient temperature in the operating room was monitored and maintained at approximately 25℃. Patients were covered with a cotton blanket from the time they entered the operating room until the skin was prepared for surgery. Routine monitoring included electrocardiography, noninvasive blood pressure (BP) measurement, and placement of a pulse oximeter probe. An epidural catheter was placed at the T11/12 interspace. An aural probe to measure core temperature (Monatherm, Tyco, Mansfield, MA, USA) was placed in the left external acoustic meatus. The probe was inserted by the patient until he or she felt the thermocouple touch the tympanic membrane[7]. The probe was covered with cotton gauze and taped in place. The tympanic temperature measured with a thermocouple was considered to represent the true core temperature[7]. Thermistor probes to measure skin temperature were placed on the right forearm and a fingertip. Forearm and fingertip temperatures were measured with a thermometer built into the anesthesia monitor (BP508, Nihon Kolin, Tokyo Japan), and core temperature was monitored with a thermometer (Monatherm, Tyco, Mansfield MA). The forearm-fingertip temperature gradient was used to indicate vasoconstriction, and a gradient greater than 0℃ was considered to be evidence of vasoconstriction. After monitoring equipment was placed and baseline values were recorded, patients in the general group received an epidural injection of 12 mL saline, and patients in the general and epidural group received epidural administration of 12 mL of 1.0% ropivacaine. Fifteen minutes after epidural injection, the dermatomal level of sensory block was confirmed by pinprick. General anesthesia was induced with propofol, 1.5 mg/kg, and vecuronium, 0.15 mg/kg, was used to facilitate tracheal intubation. General anesthesia in both groups was maintained with isoflurane, 0.5% to 2%; oxygen, 65%; and a nitrous oxide mixture. The concentration of isoflurane in the general group was set at 1.5% to 2% at skin incision, whereas that in the epidural and general group was set at 0.5% to 1%. After skin incision, the isoflurane concentration was titrated on the basis of the patients' vital signs. The patients' shoulders and upper extremities were covered by an insulated blanket; however, no active warming device was used during surgery.

Measurements

Noninvasive BP, heart rate (HR), core

Table 1 Patient Characteristics

	Epidural/ general group	General group	P value
Age (y)	45 ± 10	46 ± 7.5	0.78
Height (cm)	154 ± 8	156 ± 3	0.52
Weight (kg)	54 ± 9	54 ± 6	0.97
Anesthesia time (min)	159 ± 12	155 ± 21	0.6
Total infusion (mL)	1,840 ± 202	1,750 ± 140	0.42
Total blood loss (g)	390 ± 340	410 ± 270	0.87
Number of patients who received ephedorine administration	2	0	0.12
ATH: myomectomy	9 : 3	9 : 3	1.0

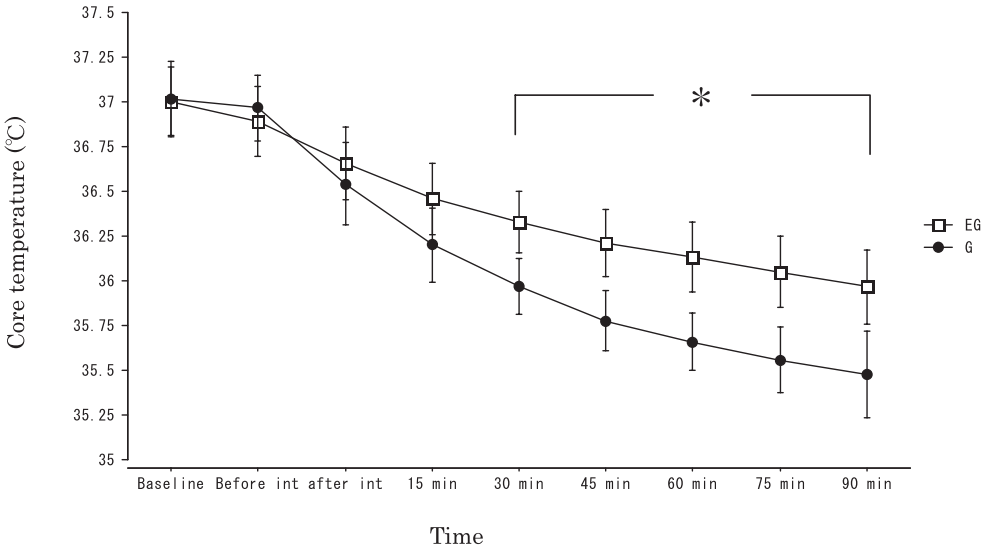

Fig. 1 Changes in core temperature after epidural administration of saline (general group: filled circles; epidural and general group: open boxes). Data are means ± 95% C.I.
*: p<0.01 between groups.

temperature, forearm temperature, and fingertip temperature were recorded just after placement of monitoring equipment (baseline), after epidural injection of saline or ropivacaine, 15 minutes after epidural injection, just after tracheal intubation, and 15, 30, 45, 60, 75, and 90 minutes after tracheal intubation. After tracheal intubation, the end-tidal concentration of isoflurane was recorded every 15 minutes. Mechanical ventilation was controlled to maintain end-tidal CO_2 pressure at 30 to 40 mmHg.

Statistical Analysis

We performed a pilot study in 8 patients (n=4, each group), and found that the reduction in core temperature 60 minutes after induction of general anesthesia was halved in patients in the general and epidural group. By power analysis of α=0.05 and β= 0.2, 8 patients seemed to be sufficient to determine significance. We examined 12 patients in each group to increase the power. Core temperature, forearm-fingertip temperature gradient, BP, HR, and isoflurane concentration were analyzed using repeated-measures ANOVA. Fisher's protected least-significant difference test was used for post hoc analysis. Values are expressed as means and 95% confidence intervals (C.I.); a P value less than <0.05 was considered to indicate statistical significance.

Results

We performed temperature monitoring in all patients without complications. There were no significant differences in characteristics between the patient groups (**Table 1**). In the epidural and general group, the median dermatomal level of sensory block was T4 (range, T3–T6). From 30 to 90 minutes after

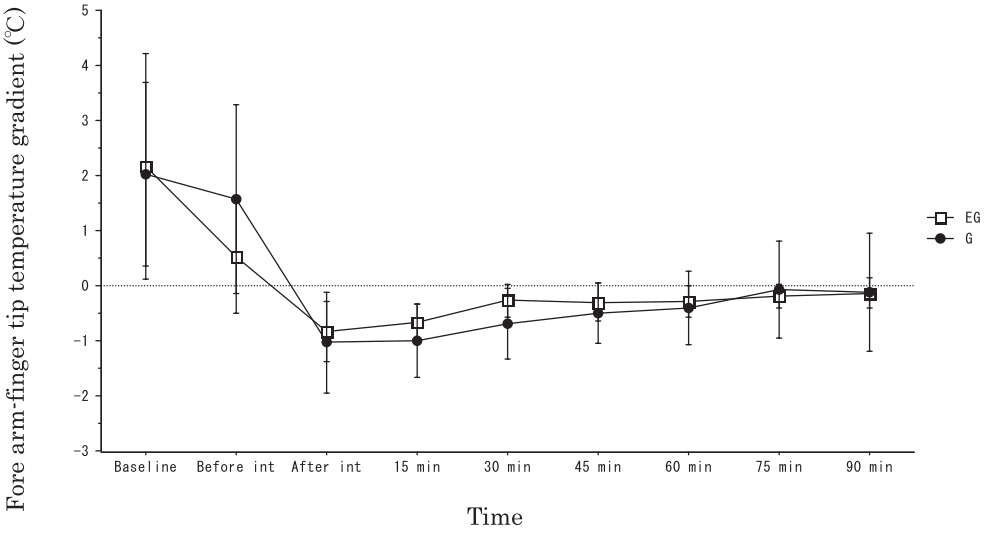

Fig. 2 Changes in the forearm-fingertip temperature gradient over time. Data are means ± 95% C.I.
There was no significant difference between the groups.

tracheal intubation, the core temperature in the epidural and general group was higher than that in the general group (**Fig. 1**). At baseline, the average value of forearm-fingertip temperature gradient in both groups was greater than 0 (vasoconstricted). After induction of general anesthesia, the fingertip temperature increased rapidly, and the forearm-fingertip temperature gradient become less than 0 (vasodilated). The values of forearm-fingertip temperature gradient from after intubation to 45 minutes after induction were significantly lower than at baseline in both groups. There was no significant difference in changes in the forearm-fingertip temperature gradient (**Fig. 2**) between the groups. Systolic blood pressure from 30 to 90 minutes after intubation was higher in the general group than in the epidural and general group, whereas heart rate 15 and 30 minutes after intubation was higher in the general group (**Fig. 3**). The isoflurane concentration was lower in the epidural and general group from just after intubation to the end of surgery (**Fig. 4**).

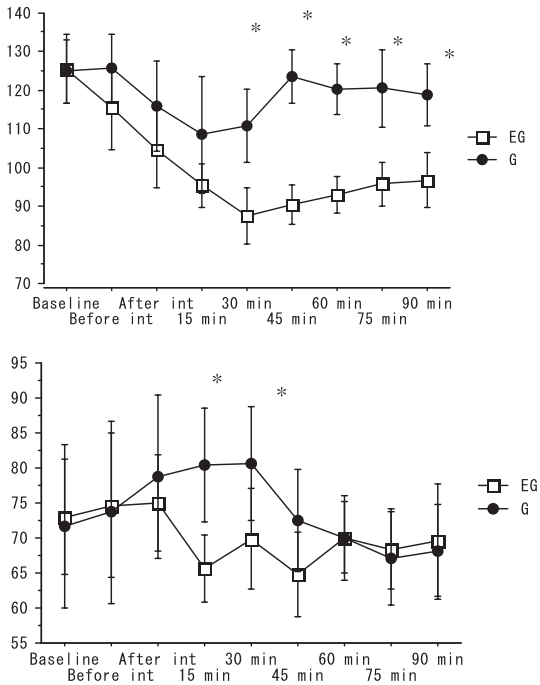

Fig. 3 Changes in systolic blood pressure (upper panel) and heart rate (lower panel).
Data are means ± 95% C.I. *: $p<0.05$ between groups

Discussion

In the present study, core temperature was better maintained in the epidural and general group than in the general group. The vasoconstriction or shivering threshold is reduced by inhalational, intravenous, and neuraxial anesthesia[8,9]. Joris et al. have reported that the vasoconstriction threshold during combined epidural/enflurane anesthesia is less than that during general anesthesia alone[3]. This result indicates that in patients who have received approximately the same concentration of inhalational anesthetics, the addition of epidural anesthesia may

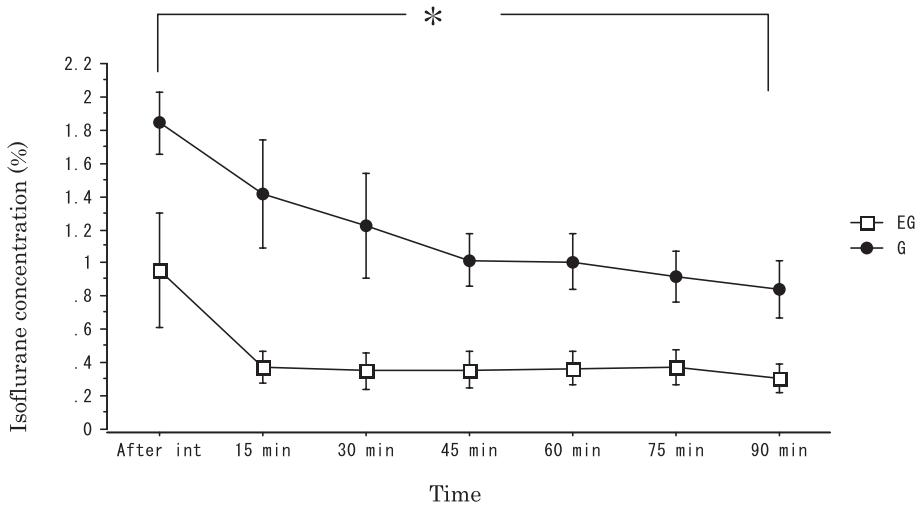

Fig. 4 Changes in isoflurane concentration over time after tracheal intubation.
Data are means ± 95% C.I. *: $p<0.05$ between groups.

induce severe hypothermia due to a further reduction in the vasoconstriction threshold. However, in the present study, there was no significant difference between the groups in the forearm-fingertip temperature gradient, even though the vasoconstriction threshold in the epidural and general group might be lowered by epidural anesthesia. The vasoconstriction threshold is negatively correlated with the isoflurane concentration[8]. In the general group, patients received a significantly higher concentration of isoflurane than in the epidural and general group. The vasoconstriction threshold in the general group might be reduced by a higher concentration of inhalationl anesthetics. In the present study, the forearm-fingertip temperature gradient showed similar changes in the two groups, indicating the vasoconstriction threshold might be similar in both groups.

Hypothermia after induction of general anesthesia results largely from core-to-peripheral redistribution of body heat[10]. Most induction anesthetics produce vasodilation, which facilitates rapid distribution of heat, whereas ketamine maintains vasoconstriction, which reduces the magnitude of redistribution hypothermia[4]. An increase in cardiac output may be responsible for redistribution hypothermia. Shitara et al. have demonstrated that dobutamine infusion exacerbates intraoperative hypothermia after induction of general anesthesia[5]. The apparent mechanism is an increase in cardiac output which in turn augments convective transfer of heat from core to peripheral tissues. Under isoflurane anesthesia, cardiac output is maintained even though relatively higher concentrations of isoflurane are administered, whereas thoracic epidural anesthesia that spreads to a level higher than the T7 dermatome decreases cardiac output through sympathetic nerve blockade (10–12). In this study, in general group higher concentrations of isoflurane were administered around the time of skin incision (1.2%–1.5%), and in the epidural and general group a large volume of ropivacaine was administered, and the spread of epidural anesthesia reached the T4 dermatomal level. Heart rate was lower in epidural and general group 15 and 30 minutes after induction, and the number of patients who received ephedrine was similar in both groups. We speculate that during this period cardiac output was maintained in the general group but decreased in the epidural and general group. The dose and potency of the local anesthetics we used were higher than those reported by Jorris et al. A decrease in cardiac output may be the main reason for maintenance of normothermia in the epidural and general group. Although the lumbar sympathetic nerve blockade increases blood flow in the lower extremities, and heat loss from the skin might be increased by vasodilation, in the present study, the decrease in cardiac output by thoracic sympathetic nerve blockade might have decreased

heat transfer.

Throughout the present study, the forearm-fingertip temperature gradient showed similar changes in the two groups and the forearm-fingertip temperature gradient after intubation was less than 0 in both groups. The core temperature in many of the patients in both groups may have been higher than the vasoconstriction threshold throughout the study. Again, vasodilation occurred in many of these patients. In this setting, heat can distribute from core to peripheral tissue by an increase in cardiac output at anesthesia induction or skin incision. The difference in core temperature was significant from 30 to 90 minutes after induction. In both groups, heat distributed from the core to the periphery throughout the study. We did not measure cardiac output directly; however, in this population, a slow and stable heart rate may suggest lower cardiac output in the epidural and general group. The cardiac output at skin incision in the general group may have been greater than that in the epidural and general group, in which nociceptive input was completely blocked. The effect of cardiac output on heat distribution may be greater at skin incision than at anesthesia induction.

A limitation of this study is the short observation time and the high ambient temperature. Although the core temperature was better maintained in the epidural and general group than in the general group, the difference between the groups was small. Also, in both groups, the core temperature decreased linearly over time. In prolonged operations, vasoconstriction in the entire body might have occurred in the general group, whereas in the epidural and general group, vasodilation in the lower extremities and body trunk might have continued, and, consequently, severe hypothermia might have been induced.

In conclusion, in gynecological procedures, maintained core temperatures were higher in patients who received both general and epidural anesthesia than in patients who received general anesthesia alone.

References

1. Kurz A, Sessler DI, Lenhardt R: Perioperative normothermia to reduce the incidence of surgical-wound infection and shorten hospitalization. Study of wound infection and temperature group. N Engl J Med 1996; 334: 1209–1215.
2. Emerick TH, Ozaki M, Sessler DI, et al.: Epidural anesthesia increases apparent leg temperature and decrease the shivering threshold. Anesthesiology 1994; 81: 289–298.
3. Joris J, Ozaki M, Sessler DI, et al.: Epidural anesthesia impairs both central and peripheral thermoregulatory control during general anesthesia. Anesthesiology 1994; 80: 268–277.
4. Ikeda T, Kazama T, Sessler DI, et al.: Induction of anesthesia with ketamine reduces the magnitude of redistribution hypothermia. Anesth Analg 2001; 93: 934–938.
5. Shitara T, Wajima Z, Ogawa R: Dobutamine infusion modifies themoregulation during general anesthesia. Anesth Analg 1996; 83: 1154–1159.
6. Ikeda T, Ozaki M, Sessler DI, et al.: Intraoperative phenylephrine infusion decreases the magnitude of redistribution hypothermia. Anesth Analg 1999; 89: 462–465.
7. Cattaneo CG, Frank SH, Hesel TW, El-Rahmany HK, Kim LJ, Tran KM: The accuracy and precision of body temperature monitoring mathods during regional and general anesthesia. Anesth Analg 2000; 90: 938–945.
8. Støen R, Sessler DI: The thermoregulatory threshold is inversely proportional to isoflurane concentration. Anesthesiology 1990; 72: 822–827.
9. Ozaki M, Kurz A, Sessler DI, et al.: Therrmoregulatory thresholds during epidural and spinal anesthesia. Anesthesiology 1994; 81: 282–288.
10. Matsukawa T, Sessler DI, Christensen R, Ozaki M, Schroeder M: Heat flow and distribution during epidural anesthesia. Anesthesiology 1995; 83: 961–967.
11. Graves CL, McDermott RW, Bidwai A : Cardiovascular effects of isoflurane in surgical patients. Anesthesiology 1974; 41: 486–489.
12. Kajimoto Y, Nishimura N: Metaraminol and dobutamine for the treatment of hypotension associated with epidural block. Resuscitation 1984; 12: 47–51.
13. Kerkkamp HEM, Gielen MJM: Hemodynamic monitoring in epidural blockade: Cardiovascular effects of 20ml 0.5% bupivacaine with and without epinephrine. Reg Anesth 1990; 15: 137–141.

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