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Short communications

Increased fingertip vascular tone leads to a greater fall in blood pressure after induction of general anesthesia

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Abstract
General anesthesia causes peripheral vasodilation. We thus hypothesized that patients with increased peripheral vascular tone would become more hypotensive after the induction of general anesthesia compared to those without increased peripheral vascular tone. To test this hypothesis, we compared the decrease in blood pressure after anesthetic induction between patients with increased peripheral vascular tone and those without increased peripheral vascular tone. Twenty-seven adult patients (10 men and 17 women) who underwent abdominal surgery with general anesthesia were enrolled in this study. In each patient, the peripheral vascular tone was assessed by either the fingertip skin-surface temperature (FSST) or the forearm-fingertip skin-surface temperature gradient (FFSSTG; forearm skin-surface temperature minus FSST). The decrease in blood pressure 15 min after anesthetic induction was larger in patients with an FSST of 29°C or less (FSST = 27.3 ± 1.6°C; FFSSTG = 5.2 ± 1.6°C) than in those with an FSST of more than 29°C (FSST = 30.8 ± 1.0°C; FFSSTG = 1.6 ± 1.2°C). In conclusion, increased fingertip vascular tone (presumably due to thermoregulatory vasoconstriction) before anesthetic induction leads to a greater fall in blood pressure after anesthetic induction.

Key words Fingertip skin-surface temperature · Blood pressure · General anesthesia

General anesthesia causes peripheral vasodilation [1] either directly or indirectly (through effects on the nervous system). We thus hypothesized that patients with increased peripheral vascular tone would become more hypotensive after the induction of general anesthesia compared to those with normal or decreased peripheral vascular tone. To test this hypothesis, we compared decreases in blood pressure after the induction of general anesthesia between patients with increased peripheral vascular tone and those without increased peripheral vascular tone.

The protocols of the present study were approved by the Ethics Committee of our institution, and informed consent was obtained from each patient. Twenty-seven American Society of Anesthesiologists (ASA)-1 patients (10 men and 17 women; age, 50–85 years) scheduled for open abdominal surgery were allocated to two groups based on fingertip skin-surface temperature (FSST) measured before the induction of general anesthesia: those with a pre-induction FSST of more than 29°C were allocated to group A (n = 13) and those with a pre-induction FSST of 29°C or less were allocated to group B (n = 14). Our rationale for this allocation was a previous finding that FSST below 29°C indicated a state in which fingertip blood flow had nearly ceased [2,3].

In each patient, an epidural catheter was placed at the lower thoracic level, and 2 ml of 1% mepivacaine was epidurally injected as a test dose to confirm its effectiveness more than 2 h before the induction of anesthesia. Subsequently, no drugs were epidurally injected until the end of this study. Famotidine, 20 mg IV, was administered, as premedication, 2 h before the induction of general anesthesia. With the standard anesthetic safety monitors, anesthesia was induced with thiamylal (3–4 mg·kg^{-1} IV) and sevoflurane 5% (inhalation concentration), and tracheal intubation was facilitated with vecuronium (0.12 mg·kg^{-1} IV). Anesthesia was subsequently maintained with sevoflurane 1.5% (end-tidal concentration) in oxygen (40%). In each patient, the peripheral vascular tone was assessed by either the FSST or the forearm-fingertip skin-surface temperature gradient (FFSSTG; forearm skin-surface temperature minus FSST), both of which have been suggested to serve as indices of peripheral blood flow or vascular tone [2,3]. The arm used for measuring temperature was

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contralateral to that used for infusion. The forearm temperature was measured at the radial side of the arm midway between the wrist and the elbow; the fingertip temperature was measured at the bulb of the middle finger. The fingertip and forearm skin-surface temperatures were measured with a skin thermometer (ST-717; Scalar, Tokyo, Japan), and tympanic membrane temperature (TMT) was measured with a tympanic thermometer (Genius; Nippon Sherwood, Tokyo, Japan). The FFSSTG was calculated from the formula: FFSSTG = forearm surface temperature—fingertip surface temperature. ΔSystolic blood pressure (ΔSBP) and Δdiastolic blood pressure (ΔDBP) were calculated from the formulae: ΔSBP = systolic blood pressure—systolic blood pressure before anesthetic induction and ΔDBP = diastolic blood pressure—diastolic blood pressure before anesthetic induction. The ambient temperature was maintained between 21°C and 23°C. The legs were warmed with a forced-air warming machine. The arms were covered with an aluminum foil blanket.

Comparisons between groups were evaluated by the Kruskall-Wallis test followed by the Mann-Whitney *U*-test. Physiologic data were compared using analysis of variance (ANOVA), followed, when significant, by Bonferroni's multiple comparison test. $P < 0.05$ was considered significant. Results were expressed as means ± SD.

There were no differences in age, height, weight, or body mass index (BMI) between the groups. The ages in group A (6 men and 7 women) and group B (4 men and 10 women) were 63 ± 15 and 65 ± 10 years, respectively. The BMI values in groups A and B were 22.2 ± 3.8 and 21.4 ± 3.2 $kg \cdot m^{-2}$, respectively.

The FSST immediately before anesthetic induction was lower ($P < 0.05$) in group B (27.3 ± 1.6°C) than in group A (30.8 ± 1.0°C; Table 1). However, the FSST increases after anesthetic induction in both groups, and the FSST at 15 and 30 min after anesthetic induction were identical in the two groups (Table 1). In addition, the FFSSTG immediately before anesthetic induction was larger ($P < 0.05$) in group B (5.2 ± 1.6°C) than in group A (1.6 ± 1.2°C; Table 1). However, the FFSSTG decreased ($P < 0.05$) after anesthetic induction in both groups (Table 1). Furthermore, the TMT decreased ($P < 0.05$) after anesthetic induction in both groups (Table 1). The results suggest that in both groups, anesthetic induction caused peripheral vasodilation resulting in redistribution hypothermia (i.e., a decrease in core temperature).

Both systolic and diastolic blood pressure decreased after anesthetic induction in both groups (Table 1). The heart rate also decreased after anesthetic induction in both groups; however, the decrease reached significance only in group A (30 min after anesthetic induction; Table 1).

The present results suggest that patients with increased fingertip vascular tone (i.e., FSST ≤ 29°C) become more hypotensive with the induction of general anesthesia compared to those with normal or decreased fingertip vascular tone (i.e., FSST > 29°C). Although the observed difference in the degree of systemic hypotension was relatively small, it would be prudent to exercise more caution during the induction of general anesthesia in patients with increased fingertip vascular tone. In addition, because the measurement of skin-surface temperature is noninvasive, it would be beneficial to estimate

Table 1. Hemodynamics, FSST, FFSSTG, and tympanic membrane temperature

Parameter	Group	0 Min	15 Min	30 Min
SBP (mmHg)	Group A	154 ± 21	125 ± 14**	121 ± 18**
	Group B	162 ± 21	115 ± 12**	126 ± 29**
DBP (mmHg)	Group A	82 ± 13	68 ± 10**	68 ± 10**
	Group B	84 ± 11	62 ± 9**	71 ± 17**
HR ($beat \cdot min^{-1}$)	Group A	71 ± 11	68 ± 8	63 ± 8**
	Group B	71 ± 10	69 ± 14	68 ± 17
FSST (°C)	Group A	30.8 ± 1.0	33.6 ± 1.0**	33.9 ± 1.0**
	Group B	27.3 ± 1.6*	33.8 ± 0.7**	33.8 ± 0.8**
FASST (°C)	Group A	32.4 ± 0.7	32.3 ± 0.9	32.5 ± 0.9
	Group B	32.4 ± 0.9	32.1 ± 0.7	32.3 ± 1.1
FFSSTG (°C)	Group A	1.6 ± 1.2	–1.3 ± 1.0**	–1.4 ± 1.1**
	Group B	5.2 ± 1.6*	–1.7 ± 0.8**	–1.6 ± 1.1**
TMT (°C)	Group A	36.9 ± 0.3	36.5 ± 0.3**	36.3 ± 0.3**
	Group B	36.9 ± 0.4	36.6 ± 0.4**	36.3 ± 0.4**

* $P < 0.05$, significantly different from group A; ** $P < 0.05$, significantly different from 0 min
Data values are means ± SD
SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; FSST, fingertip skin-surface temperature; FASST, forearm skin-surface temperature; FFSSTG, forearm-fingertip skin-surface temperature gradient; TNT, tympanic membrane temperature. Group A, FSST was more than 29°C at 0 min; group B, FSST was 29°C or less

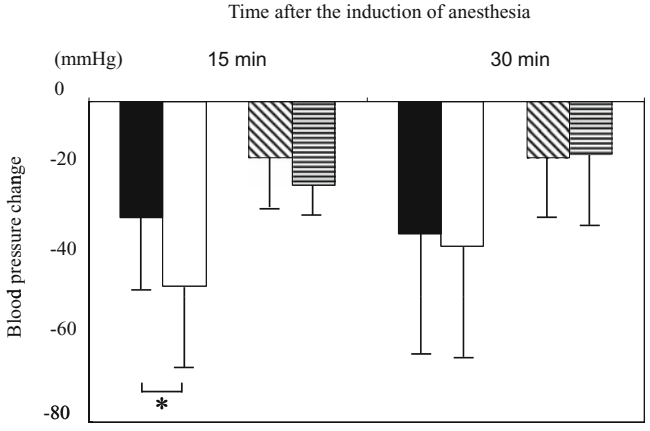

Fig. 1. Systolic and diastolic blood pressure changes. ΔSystolic blood pressure (ΔSBP) was defined as the change in systolic blood pressure during the induction of anesthesia. ΔDiastolic blood pressure (ΔDBP) was defined as the change in diastolic blood pressure during the induction of anesthesia. Group A, fingertip skin-surface temperature (FSST) was more than 29°C at 0 min. Group B, FSST was 29°C or less. *$P < 0.05$, significantly different from group A. *Black bars*, ΔSBP-group A; *white bars*, ΔSBP-group A; *diagonally striped bars*, ΔDBP-group A; *horizontally striped bars*, ΔDBP-group B

the fingertip vascular tone by measuring the FSST before the induction of general anesthesia.

Because arteriovenous anastomoses, as well as countercurrent heat-exchange systems, both of which are prominently involved in thermal regulation, are numerous in the digits but virtually absent in the forearm or calf, skin-surface temperature gradients such as the FFSSTG would be reasonably useful in detecting thermoregulatory vasomotion [4,5]. In this study, the FFSSTG was significantly increased in the patients with increased fingertip vascular tone (i.e., FSST > 29°C; group B). However, no significant differences were observed in the forearm skin-surface temperature between the two study groups (Table 1). These findings suggest that the increase in FFSSTG in group B largely reflected the decrease in FSST. In other words, thermoregulatory vasoconstriction had emerged at the induction of general anesthesia in the patients with increased FSST (i.e., those belonging to group B. We therefore postulate that the anesthetic-induced increase in the interthreshold range and the resultant disappearance of the thermoregulatory vasoconstriction was responsible for the greater fall in blood pressure observed in the patients with increased FSST. Thus, in such patients with increased fingertip vascular tone, increasing their mean body temperature (or body heat content) in advance before anesthetic induction would be effective in attenuating the degree of systemic hypotension after anesthetic induction. Specifically, in these patients, preoperative warming using a forced-air warmer or an electric warming blanket [6], and/or preoperative administration of vasodilators [7] or amino acids [8] (for the induction of thermogenesis) would be effective in increasing the mean body temperature and thereby releasing thermoregulatory vasoconstriction. However, releasing the thermoregulatory vasoconstriction could lead to significant systemic hypotension [9] in patients whose blood pressure is maintained around the lower limit of the normal range with the aid of thermoregulatory vasoconstriction (e.g., hypothermic patients with decreased intravascular volume). In such patients, intravenous infusion of fluids before and during the above-described preoperative attempts to release the thermoregulatory vasoconstriction would be effective in preventing dangerous decreases in blood pressure.

In conclusion, the present findings suggest that increased fingertip (or digital) vascular tone or the presence of thermoregulatory vasoconstriction before anesthetic induction leads to a greater fall in blood pressure after anesthetic induction.

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References

1. Howie MB, Gravlee GP. Induction of general anesthesia. In: Hensley FA, editor. A practical approach to cardiac anesthesia (Japanese edition). Philadelphia: Lippincott Williams & Wilkins; 2003. p. 183.
2. Rubinstein EH, Sessler DI. Skin-surface temperature gradients correlate with fingertip blood flow in humans. Anesthesiology. 1990;73:541–5.
3. T. Akata, T. Kanna, J. Yoshino, M, Higashi, K. Fukui, S. Takahashi. Reliability of fingertip skin-surface temperature and its related thermal measures as indices of peripheral perfusion in the clinical setting of the operating theatre. Anaesth Intensive Care. 2004;32:519–29.
4. Sessler DI, Olofsson CI, Rubinstein EH, Beebe JJ. The thermoregulatory threshold in humans during halothane anesthesia. Anesthesiology. 1988;68:836–42.
5. Siegel MN, Gravenstein N. More on thermoregulatory thresholds with halothane. Anesthesia. 1989;70:370–1.
6. Hyson JM, Sessler DI, Moayeri A, McGuire J, Schroeder M. The effects of preinduction warming on temperature and blood pressure during propofol/nitrous oxide anesthesia. Anesthesiology. 1993;79:42–8.
7. Vassilieff N, Rosencher N, Sessler DI, Conseiller C, Lienhart A. Nifedipine and intraoperative core body temperature in humans. Anesthesiology. 1994;80:123–8.
8. Sellden E, Lindahl SG. Amino acid-induced thermogenesis reduces hypothermia during anesthesia and shortens hospital stay. Anesth Analg. 1999;89:1551–6.
9. Greif R, Laciny S, Rajek A, DoufasAG, Sessler DI. Blood pressure response to thermoregulatory vasoconstriction during isoflurane and desflurane anesthesia. Acta Anaesthesiol Scand. 2003;47:847–52.