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Vital capacity induction with 8% sevoflurane and N_2O causes cerebral hyperemia

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Abstract
Purpose. Little is known about the influence of high-dose sevoflurane on cerebral volume. We evaluated induction time and cerebral blood volume with 8% sevoflurane using the "vital capacity induction" technique.
Methods. Thirty-four patients were randomly allocated into three groups. Group P received 2.0 mg·kg^{-1} of propofol i.v. and inhalation of 67% N_2O/O_2, whereas group S5 and group S8 received inhalation of primed 5% and 8% sevoflurane in 67% N_2O/O_2, respectively. Induction time was measured as the time from the start of inhalation, or from the end of injection, until loss of eyelash reflex. Near-infrared spectroscopy and bispectral index (BIS) were monitored continuously until 3 min after tracheal intubation.
Results. Induction time was less in group S8 (17.3 ± 6.4 s, mean ± SD) than in groups P (25.7 ± 8.2 s) and S5 (33.0 ± 16.8 s). There was a significant increase in cerebral blood volume after intubation in group S8, as suggested by higher cerebral oxyhemoglobin and total hemoglobin levels. There were no differences in BIS scores among the groups during the study period.
Conclusion. Vital capacity inhalation of 8% sevoflurane produces a faster loss of eyelash reflex than does 5% sevoflurane or propofol, but increases cerebral blood volume.

Key words Vital capacity induction · Sevoflurane · Induction time · Near-infrared spectroscopy · Cerebral blood volume

Introduction

The concern with induction and maintenance of anesthesia using sevoflurane inhalation solely has been growing [1,2], because sevoflurane has a low blood / gas solubility coefficient and is the least respiratory irritant of the available volatile anesthetics. In addition, it is useful for the management of the difficult airway, allowing intubation without muscle relaxants [3]. Inhalational anesthetics increase cerebral blood flow and volume [4]. However, no study has evaluated by how much cerebral blood volume would increase when using high-dose sevoflurane. In this study, we compared induction time and cerebral blood volume during anesthesia induction with 8% sevoflurane, 5% sevoflurane, or propofol.

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Materials and methods

After obtaining approval of the Institutional Review Board of the hospital and written informed consent from all patients, we recruited 34 adult patients, American Society of Anesthesiologist physical status 1 or 2, undergoing general anesthesia with tracheal intubation. We excluded patients with cardiovascular, renal, hepatic, diabetic, thyroid, or neurological diseases, and those undergoing neurosurgery. Patients were randomly assigned to induction by propofol as a 2.0 mg·kg^{-1} bolus injection (group P, $n = 12$) followed by inhalation of 67% N_2O/O_2, or inhalation induction with 5% (group S5, $n = 10$) or 8% (group S8, $n = 12$) sevoflurane in 67% N_2O/O_2. The anesthetic circuit was primed with an anesthetic gas mixture for 5 min before inhalation.

All patients were premedicated with 0.5 mg of atropine 30 min before the induction of anesthesia. A cannula was placed in a large forearm vein for an infusion of lactated Ringer's solution. In the operating room, we applied an automatic blood pressure cuff, electrocardiogram on standard lead II, pulse-oximeter, end tidal CO_2 analyzer, and bispectral index (BIS) on electroencephalogram (A-1050, Aspect Medical System, Newton, MA, U.S.A). The BIS monitor uses a proprietary algorithm

that translates electroencephalogram data into a nominal scale of 100 to 0. A BIS value of 100 indicates a fully awake state and 0 equates with an isoelectric electroencephalogram (EEG). A probe for the detection of cerebral hemoglobin volume by near-infrared spectroscopy (OM220, Shimazu Seisakujo, Kyoto, Japan) was attached on the right frontal region of the head. Near-infrared spectroscopy (NIRS) is a continuous and noninvasive bedside monitor that has been used to measure changes in cerebral hemodynamics [5]. Jobsis [6] first demonstrated that it was possible to assess cerebral oxygenation from the attenuation of near-infrared light passing through the skull underlying the brain. Changes in total hemoglobin (total-Hb) reflect fluctuations of intracranial blood volume during induction of anesthesia [7]. In the present study, we adopted oxygenated hemoglobin (oxy-Hb) and total-Hb volumes measured by the apparatus (Shimazu Seisakujo noninvasive oxygen monitor) using wavelengths of 780 and 830 nm. The reflected beam of light was analyzed and the data stored continuously on a computer. The oxy-Hb and total-Hb volumes were calculated.

Patients in groups S5 and S8 were instructed to breathe from a face mask to perform a "vital capacity inhaled induction" [2]: to exhale fully, inhale fully, and hold their breath as long as possible. The anesthetic circuit was primed with an anesthetic gas mixture for 5 min before inhalation. Carrier gas flows were fixed at $6 l \cdot min^{-1}$ (N_2O, 4 l; O_2, 2 l). The anesthetic machine (ADU, Datex Ohmeda, Helsinki, Finland) was equipped with a vaporizer (ADU, Datex Ohmeda) delivering 8% sevoflurane. The machine had a total capacity of 2440 ml, consisting of 340 ml in corrugated hose, 700 ml in a CO_2-absorbing cannister, and 1400 ml in bellows. The concentration of anesthetic gas in the reservoir bag was continuously analyzed (AS/3, Datex Ohmeda); it took a mean of 4.5 min to reach 8% sevoflurane. We therefore adopted a priming time of 5 min. In group P, propofol was injected quickly and an anesthesia mask was immediately placed on the patient's face without leakage.

Induction time was measured from the start of sevoflurane inhalation, or the end of propofol injection, until the loss of eyelash reflex. When eyelash reflex was absent, $0.1 mg \cdot kg^{-1}$ of vecuronium was injected intravenously. Ventilation was assisted to maintain end-tidal CO_2 at 34–37 mmHg. After 3 min, a tracheal tube was inserted using a Macintosh laryngoscope. The concentrations of anesthetic gases were unchanged for 3 min after tracheal intubation, and ventilation was controlled as before.

Vital signs were closely observed and side effects such as excessive hypotension, bucking, coughing, or body movements on placement of tracheal tubes were recorded every minute.

Statistical analysis

The data were represented by mean ± SD. Differences in the numerical data among the groups were analyzed by one-way analysis of variance. Dunnet's method was applied to the differences between the two selected groups. A P value <0.05 was considered to be statistically significant.

Results

There were no significant differences in age, weight, or height among the three groups (Table 1). Induction time in group S8 was thus significantly shorter than in the other groups. Side effects such as coughing and involuntary body movement were observed in group P during induction. In group S5, one patient showed involuntary movements. There was no significant difference in side effects among the three groups.

No significant differences were observed among the three groups in mean blood pressure or heart rate before induction (Fig. 1). There was marked hypotension 1 min after the start in groups P and S8, but blood pressure recovered slowly after intubation. There were no differences in blood pressure among the three groups throughout the study.

Heart rate increased gradually after the start of induction in groups S5 and S8, and there were significant differences between group P and groups S5 and S8 after intubation (Fig. 1).

BIS declined maximally to 15%–30% of control value, followed by a gradual recovery to 50%–70% of control (Fig. 2). There was no difference among the three groups throughout the study.

After intubation, cerebral oxy-Hb volume as detected by NIRS was significantly greater in group S8 than in the others, and remained increased during inhalation of 8% sevoflurane. Total-Hb also altered coincidently with cerebral oxy-Hb volumes (Fig. 3).

Table 1. Patient characteristics, induction time, and side effects

	Group P	Group S5	Group S8
Subjects	12	10	12
Age (years)	53 ± 11	55 ± 15	50 ± 11
Height (cm)	165 ± 7	163 ± 6	165 ± 11
Weight (kg)	57 ± 9	53 ± 5	58 ± 11
Induction time (s)	25.7 ± 8.2	33 ± 16.8	17.3 ± 6.4*
Side effects	2	1	0

Values are mean ± SD. Patient characteristics were similar among groups. The induction time in group S8 was significantly shorter than in groups P and S5 *$P < 0.05$

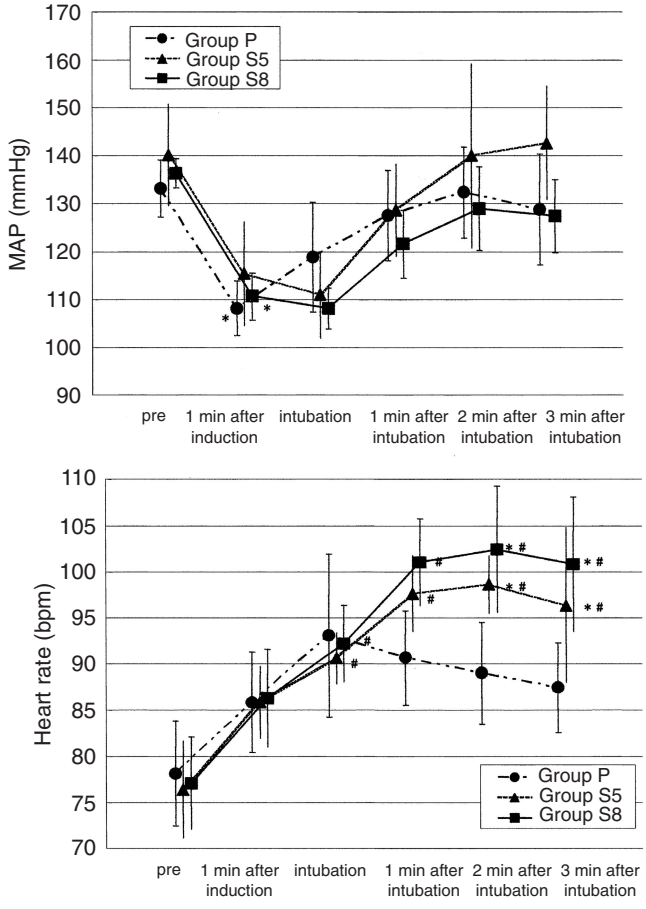

Fig. 1. Time course of changes in mean arterial blood pressure (*MAP*) and heart rate. Values are expressed as mean ± SD. *Circles* indicate group P. *Triangles* indicate group S5. *Squares* indicate group S8. MAP was significantly decreased at 1 min in group P (108.3 ± 19.6) and group S8 (110.7 ± 15.6) compared with pre-MAP ($*P < 0.05$). Heart rate was increased in groups S5 and S8 by induction and intubation ($\#P < 0.05$). Heart rate in group S5 and group S8 was significantly greater than in group P at 2 min and 3 min after intubation ($*P < 0.05$)

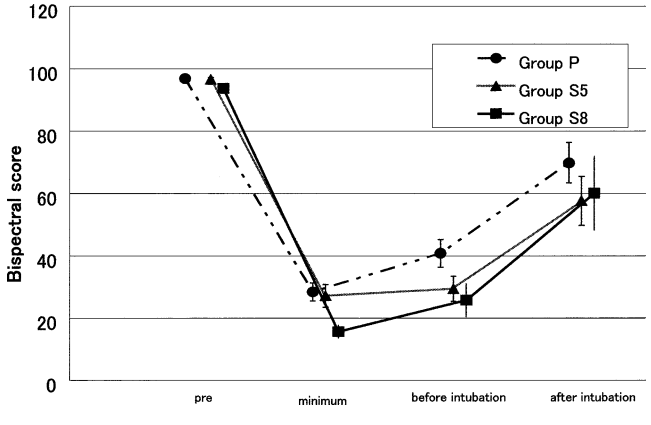

Fig. 2. Time course of changes in bispectral index. There was no significant difference among the three groups

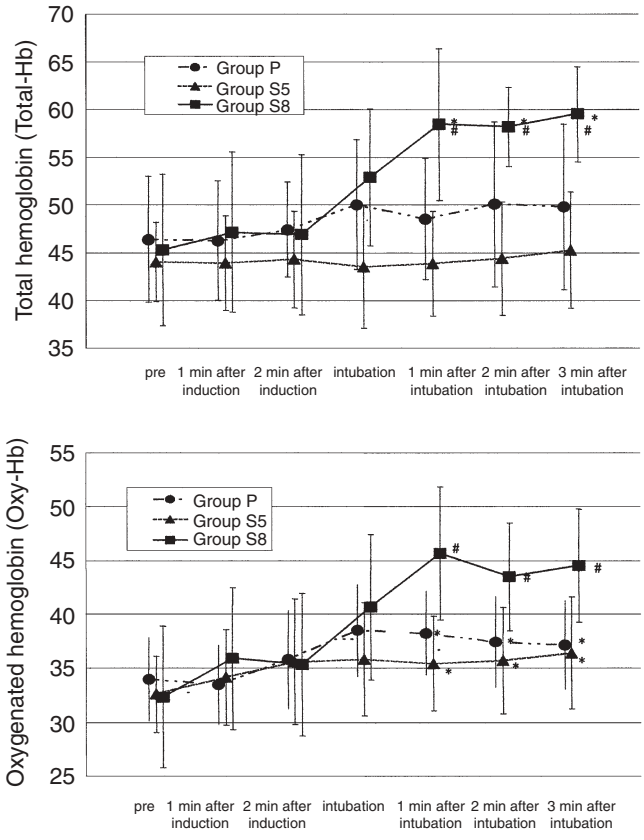

Fig. 3. Time course of changes in cerebral total hemoglobin (*total-Hb*) and oxygenated hemoglobin (*oxy-Hb*). Total-Hb was increased in group S8 after intubation ($\#P < 0.05$ vs pre). Total-Hb in group S8 was higher than in group S5 after intubation ($*P < 0.05$). Oxy-Hb was increased in group S8 after intubation ($\#P < 0.05$ vs pre). Oxy-Hb in group S8 was higher than in groups P and S5 after intubation ($*P < 0.05$)

Discussion

We compared induction times between patients receiving propofol and sevoflurane. The mean induction time for 8% sevoflurane with 67% N_2O was 17.3 ± 6.4 s, significantly shorter than that following propofol injection followed by N_2O inhalation, and for 5% sevoflurane/N_2O inhalation.

Philip et al. [1] compared induction times between vital capacity inhalation of 8% sevoflurane in 75% N_2O/O_2 with intravenous propofol 2.0 mg·kg^{-1}. Time to loss of eyelash reflex was 56 s for sevoflurane and 92 s for propofol, much greater than in our study, even with the same nominal gas concentrations. However, the two studies used different techniques of anesthesia circuit priming. In the previous study, only 45 s was spent priming the anesthesia circuit [1]. This was probably not enough to achieve the desired concentration. The capacity of the circle system of the anesthetic machine, including tubes, CO_2-absorbing cannister, and reservoir

bag, was 2.4 l in the present study. The concentration of gas in the circuit approached 8% after 4.5 min of priming; hence, we waited for 5 min to fill the circle system. Yurino and Kimura [2] , using 4.5% sevoflurane in N_2O to induce anesthesia by vital capacity inhalation, found a mean induction time of 54 s. They adopted times from the start of inhalation to loss of response to verbal command as induction times. That report supported the result obtained in the present study that induction by inhalation of 8% sevoflurane with N_2O was faster than induction by intravenous propofol.

Mean blood pressure was markedly depressed after induction, but recovered gradually after tracheal intubation in the three groups. It declined more in groups P and S8 than in group S5 after induction. Inhalation of a high concentration of sevoflurane is known to induce hypotension [8,9], mainly by decreased after load and depressed cardiac contractility [10]. Propofol also lowered mean blood pressure, probably by a reduction in after load through sympathetic inhibition [11]. Heart rate increased after induction in groups S5 and S8, especially after intubation, but did not change significantly in group P. Blood pressure and heart rate can, however, decrease within 3 min after bolus injection of propofol [11]. The reduction in blood pressure did not interrupt the course of anesthetic induction in any patient.

Glass et al. [12] concluded that a BIS value of <50 for a variety of clinically used anesthetic agents equated with adequate depth of anesthesia. In our study, BIS declined to a value <30 after induction of anesthesia in all three groups. However, it gradually recovered to 68 in group P, 58 in group S5, and 60 in group S8 at the end of intubation. There were no differences among the three groups at any time. Wilder-Smith et al. [13] and Mi et al. [14] both reported EEG arousal during laryngoscopy and intubation during induction of anesthesia with various anesthetics. However, BIS was maintained at about 50 during laryngoscopy in groups S5 and S8, suggesting that the arousal response was eliminated. We have also shown adequate depth of anesthesia in groups S5 and S8 during and after the induction periods. Propofol was injected by one shot, not continuously. This is the reason that the BIS value in group P became 68 after intubation.

Changes in total-Hb reflect fluctations of intracranial blood volume [7]. Total-Hb increased significantly in group S8, which received 8% sevoflurane and N_2O. This suggests that brain vessels dilated considerably at this concentration. Total-Hb with 8% sevoflurane/N_2O started to increase after intubation. The increase can be explained by the following reasons. First, it took time to saturate with sevoflurane from pulmonary alveoli through blood vessels to brain. Second, heart rate increased by the intubation maneuver. As a result, cerebral blood volume increased per unit time. These two reasons explain the fact that total-Hb increased after intubation in group S8. Injection of propofol did not affect intracranial hemoglobin volume. Therefore, it is suggested that great care should be taken for the induction of anesthesia with 8% sevoflurane in patients with elevated intracranial pressure.

In conclusion, a vital capacity inhalation techno ue with 8% sevoflurane and 67% N_2O induced anesthesia more rapidly than did 2.0 $mg \cdot kg^{-1}$ of propofol intravenously. The depth of anesthesia was well maintained during the intubation procedure, as observed by BIS. However, induction with 8% sevoflurane and 67% N_2O caused cerebral hyperemia, something to be borne in mind when dealing with patients with elevated intracranial pressure.

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