J Anesth (1997) 11:111–116

Journal of
Anesthesia

Circulatory and catecholamine responses to tracheal intubation and skin incision during sevoflurane, isoflurane, or halothane anesthesia*

Koh Yamada, Koh Shingu, Hiromi Kimura, Sakahiro Ikeda, Kouichi Tsushima, Toshihiro Imanishi, and Kohei Murao

Department of Anesthesiology, Kansai Medical University, 10-15 Fumizono-cho, Moriguchi, Osaka 570, Japan

Abstract: The anesthetic suppression of responses to noxious stimuli might reflect a summation of the suppression of the basal functions and the response capability. We investigated the basal suppression and response capability in hemodynamics and plasma catecholamine levels with different anesthetics at the same minimum alveolar concentration (MAC) level. Fifty-four patients were allocated to one of 6 groups to receive sevoflurane, isoflurane, or halothane at 1.25 or 2.0 MAC. Anesthesia was induced with the test agent in oxygen and the end-tidal concentration of the agent was maintained for at least 15 min at 1.25 or 2.0 MAC. The trachea was intubated under muscle relaxation with 0.1 mg·kg^{-1} vecuronium. Skin incisions were made after maintaining the end-tidal concentration of the agent for at least 15 min after tracheal intubation. The mean arterial pressure, heart rate, rate-pressure product, and plasma levels of noradrenaline and adrenaline at the prestimuli period showed no difference between agents at each MAC. The rises in these variables by tracheal intubation and skin incision were greatest in the sevoflurane group, least in the halothane group, and intermediate in the isoflurane group. Although basal hemodynamic suppression is similar at the same MAC, the suppressive action of sevoflurane on the circulatory response capability to noxious stimuli is weaker than that of isoflurane and halothane.

Key words: Halothane, Isoflurane, Sevoflurane, MAC, Response capability

Introduction

The minimum alveolar concentration (MAC) of volatile anesthetics is an index of the depth of anesthesia [1]. Body movement responses to skin incision, however, are usually abolished with recent anesthetic approaches, in which muscle relaxants are commonly used. Thus, the depth of anesthesia is clinically assessed by cardiovascular changes rather than body movements in response to surgical manipulations. Roizen et al. proposed the MAC-BAR, the MAC necessary to blocking adrenergic responses, as an index of anesthetic potency in suppressing adrenergic responses to skin incision [2]. They demonstrated that the MAC-BARs of halothane and enflurane were 1.45 and 1.6 MAC, respectively, and that there were no significant differences between agents in the ratio of MAC-BAR to MAC. An index of anesthetic suppression of body movement and coughing during tracheal intubation, MAC-intubation, has also been proposed [3]. Isoflurane or sevoflurane, in contrast to halothane, has clinically weak depressive actions on the hemodynamic responses induced by peripheral stimulation, such as inserting an intravenous line, tracheal intubation, and skin incision. We speculated that the cardiovascular changes induced by tracheal intubation or skin incision are not identical with different anesthetic agents even at the same MAC values.

The anesthetic suppression of the response to a noxious stimulus may be considered as a summation of two aspects of suppression by anesthetics. Anesthetics suppress the basal and tonic functions of a given system, e.g., the cardiovascular system, at the unstimulated state. Further, anesthetics suppress the response capability to a noxious stimulus, which is clinically assessed as the anlagesic action of anesthetics and may reflect mainly depressive actions of anesthetics on the central nervous system. These two aspects of the suppressive actions on hemodynamics can be discriminated by measuring blood pressure and heart rate before and after a noxious stimulus.

In the present study, we compared basal values and changes in mean arterial pressure (MAP), heart rate (HR) and rate-pressure product (RPP), and arterial lev-

Address correspondence to: K. Shingu

Received for publication on August 5, 1996; accepted on November 27, 1996

*This study was presented in part at the 42nd annual congress of the Japan Society of Anesthesiology, April 20, 1995

els of noradrenaline (NA) and adrenaline (A), induced by tracheal intubation and skin incision at 1.25 and 2.0 MAC of halothane, isoflurane, and sevoflurane.

Materials and methods

Following approval by our institutional committee on human research, a total of 54 ASA physical status I and II patients, who were scheduled to undergo surgery, were enrolled in this study. Informed consent was obtained from each patient. The proposed types of surgery were gastrectomy, mastectomy, simple abdominal hysterectomy, or sigmoidal colectomy. Patients with hypertension, obesity above 20% of the body mass index, or receiving chronic medication with drugs affecting the circulation or endocrinological system were excluded. The patients were randomly allocated to one of 6 groups ($n = 9$ for each group), according to the anesthetic agents and concentrations to be studied: sevoflurane, isoflurane, or halothane, and 1.25 or 2.0 MAC, respectively. The MAC values used were 1.71% for sevoflurane, 1.2% for isoflurane, and 0.75% for halothane.

All patients were premedicated with 10mg oral diazepam 2h before anesthesia and 0.5mg intramuscular atropine sulfate 45min before anesthesia. An intravenous infusion of lactated Ringer's solution was then started and an intraarterial line was set up in a radial artery under local anesthesia with 0.5% lidocaine. The sampling catheter tip of an anesthetic gas monitor (Bruel and Kjeur Anesthetic Gas Monitor 1304, Naerum Denmark) was placed in front of the nostrils. The patient was anesthetized with a mask using the anesthetic agent to be investigated in oxygen. Following loss of verbal response, vecuronium, 0.1 $mg \cdot kg^{-1}$, was injected intravenously. The end-tidal concentration of the anesthetic agent was maintained at 1.25 or 2.0 MAC for 15min under manual controlled ventilation. End-tidal partial pressure of carbon dioxide was maintained at 30–35mmHg. Tracheal intubation was carried out by one of the authors (K.Y.). The duration of laryngoscopy was measured from the time of placing the laryngoscope into the mouth. The size of the endotracheal tube was 7.5mm in diameter for female patients and 8.5mm for male patients. Following tracheal intubation, a sampling tube for anesthetic gas monitoring was placed between the endotracheal tube and the anesthetic circuit. Skin incision was performed after maintaining the end-tidal concentration of the agent at 1.25 or 2.0 MAC for at least 15min. This concentration was maintained for a further 5min after incision.

Mean arterial blood pressure (MAP) and heart rate (HR) were monitored with a bedside monitor (Hewlett-Packard, M1166A, Andover, MA, USA). The rate-pressure product (RPP) was calculated as the heart rate multiplied by the systolic arterial pressure. Arterial blood samples of 7ml were drawn for measuring noradrenaline (NA) and adrenaline (A). Nine blood samples were taken in series from each patient; before induction of anesthesia (control); before, 1, 2, and 5min after tracheal intubation; and before, 1, 2, and 5min after skin incision. The blood samples were centrifuged immediately after collection and the resulting plasma was frozen at −20°C until assay. Catecholamines were assayed by high-performance liquid chromatography using a fluorescence detector (an automatic catecholamine analyzer, 725 CA, Tosoh, Tokyo, Japan) using diphenylethylene diamine as a fluorescent label. The lowest detectable levels of NA and A were 5 $pg \cdot ml^{-1}$.

To evaluate the effects of the anesthetics on responses induced by tracheal intubation and skin incision, any change induced by tracheal intubation or skin incision was divided by the control value for each patient. The values used for calculation after manipulation were those taken 1min after the relevant manipulation.

Values were expressed as mean ± SD. MAP, HR, RPP, and catecholamine levels in each group were analyzed by repeated measures analysis of variance (ANOVA) and Bonferroni's test. Comparisons of demographic data and variables between groups were made by ANOVA and Fisher's protected least significant difference (PLSD) test. Statistical significance was assumed at the 5% level.

Results

Demographic data for the subjects are shown in Table 1. There were no significant differences in age, body weight, distributions of sex or type of operation, and duration of laryngoscopy between the groups.

Changes in MAP during the study are shown in Table 2. Control MAP values did not differ between the groups. Anesthesia reduced MAP before tracheal intubation by 24%–37% from the control values ($P < 0.05$ in all groups), but this decrease was not statistically different between agents or concentrations of each anesthetic. MAP rose again 1min after intubation ($P < 0.05$ in all groups except 2.0 MAC isoflurane) and then decreased gradually. The rise in MAP after intubation was greater under sevoflurane anesthesia (172% and 142% of the value before intubation at 1.25 and 2.0 MAC, respectively) than isoflurane (163% and 117%) and halothane (132% and 124%) anesthesia. The rise in MAP was less at 2.0 MAC than at 1.25 MAC in all groups ($P < 0.05$). Skin incision also raised MAP in all groups after 2 and 5min. The rise in MAP 5min after incision was more marked in the sevoflurane (141% and 173% of the value before skin incision at 1.25 and 2.0 MAC, respectively) and isoflurane (171% and 157%)

Table 1. Patient demographics

	Sevoflurane		Isoflurane		Halothane	
MAC	1.25	2.0	1.25	2.0	1.25	2.0
Sex (M/F)	2/7	3/6	1/8	3/6	2/7	3/6
Age (years)	47 ± 6	49 ± 6	50 ± 7	49 ± 5	47 ± 7	49 ± 7
Weight (kg)	55 ± 8	57 ± 8	51 ± 6	59 ± 9	55 ± 4	56 ± 14
Operation						
Gastrectomy	3	3	3	3	3	3
Mastectomy	3	3	3	3	3	3
Hysterectomy	1	3	2	2	1	0
Colectomy	2	0	1	1	2	3
Duration of laryngoscopy (s)	16 ± 10	14 ± 9	13 ± 9	17 ± 10	13 ± 13	15 ± 17

Values for sex distribution and type of surgery are numbers of patients. Values for age, weight, and duration of laryngoscopy are mean ± SD.

Table 2. Changes in mean arterial pressure (mmHg, $n = 9$ in each group, mean ± SD)

	MAC	Control	1	2	3	4	5	6	7	8
Sevoflurane	1.25	93 ± 12	71 ± 5*	122 ± 15*§	108 ± 16§	84 ± 10	80 ± 13	117 ± 18*¶	115 ± 15*¶	113 ± 17*¶
	2.0	96 ± 12	69 ± 8*	98 ± 13§a	94 ± 16§	81 ± 12	59 ± 11*a	93 ± 10¶a	102 ± 8¶a	102 ± 8¶
Isoflurane	1.25	99 ± 14	67 ± 12*	109 ± 16§	96 ± 18§	77 ± 14*	63 ± 13*b	103 ± 18¶b	106 ± 18¶	108 ± 13¶
	2.0	101 ± 10	64 ± 18*	75 ± 24*a,b	74 ± 21*a,b	67 ± 19*b	58 ± 16*	68 ± 16*a,b	77 ± 16*¶a,b	91 ± 17¶a
Halothane	1.25	100 ± 21	71 ± 6*	94 ± 18§b	88 ± 17§b	75 ± 10*	66 ± 8*b	82 ± 9*b,c	84 ± 10*¶b,c	93 ± 11¶b,c
	2.0	92 ± 12	62 ± 6*	77 ± 13*§a,b	74 ± 13*b	68 ± 9*b	55 ± 8*a	64 ± 5*a,b	71 ± 6*¶a,b	78 ± 9*¶a,b

1, before tracheal intubation; 2, 1 min after intubation; 3, 2 min after intubation; 4, 5 min after intubation; 5, before skin incision; 6, 1 min after skin incision; 7, 2 min after skin incision; 8, 5 min after skin incision.
* $P < 0.05$ *vs* control; § $P < 0.05$ *vs* 1; ¶ $P < 0.05$ *vs* 5; a $P < 0.05$ *vs* 1.25 MAC of the same agent; b $P < 0.05$ *vs* the same MAC of sevoflurane; c $P < 0.05$ *vs* the same MAC of isoflurane.

Table 3. Changes in heart rate (beats·min^{-1}, $n = 9$ in each group, mean ± SD)

	MAC	Control	1	2	3	4	5	6	7	8
Sevoflurane	1.25	83 ± 23	81 ± 15	99 ± 14*§	90 ± 18	90 ± 18	83 ± 15	113 ± 16*¶	107 ± 18*¶	103 ± 15¶
	2.0	84 ± 14	81 ± 16	101 ± 16*§	92 ± 17§	92 ± 17	78 ± 12	108 ± 9*¶	105 ± 12*¶	97 ± 19¶
Isoflurane	1.25	87 ± 11	85 ± 13	99 ± 7*§	88 ± 10	88 ± 10	77 ± 10	102 ± 12*¶	100 ± 12¶	97 ± 14¶
	2.0	83 ± 21	91 ± 22	98 ± 21*	94 ± 21	94 ± 21	79 ± 16	90 ± 16a,b	93 ± 13b	95 ± 19
Halothane	1.25	91 ± 22	77 ± 13	89 ± 20§	81 ± 18	81 ± 18	69 ± 11*	78 ± 13b,c	78 ± 12b,c	77 ± 11b,c
	2.0	80 ± 18	76 ± 14	85 ± 14§	80 ± 14	80 ± 14	73 ± 13	82 ± 12b	85 ± 10b,c	82 ± 9b

1, before tracheal intubation; 2, 1 min after intubation; 3, 2 min after intubation; 4, 5 min after intubation; 5, before skin incision; 6, 1 min after skin incision; 7, 2 min after skin incision; 8, 5 min after skin incision.
* $P < 0.05$ *vs* control; § $P < 0.05$ *vs* 1; ¶ $P < 0.05$ *vs* 5; a $P < 0.05$ *vs* 1.25 MAC of the same agent; b $P < 0.05$ *vs* the same MAC of sevoflurane; c $P < 0.05$ *vs* the same MAC of isoflurane.

anesthesia groups than in the halothane anesthesia group (141% and 142%).

Changes in HR are shown in Table 3. Control HR values were not significantly different between the groups. Anesthesia did not change HR significantly in any group before tracheal intubation. Tracheal intubation increased HR by 8%–25% from the values before intubation. The rise in HR caused by intubation was significant in all groups except the 2.0 MAC isoflurane group. HR after intubation was significantly higher than the control values in all groups except the halothane group. Skin incision also significantly increased HR in the sevoflurane (by 36% and 38% from before skin incision, respectively) and 1.25 MAC isoflurane (32%) groups, but the change was not significant in the 2.0 MAC isoflurane (14%) or halothane (13% and 12%) groups. Heart rate after skin incision during sevoflurane anesthesia was higher than that during isoflurane and halothane anesthesia.

The percentage change in RPP after tracheal intubation is shown in Fig. 1. The rise in RPP was greater during sevoflurane than isoflurane or halothane anesthesia at both concentrations ($P < 0.05$). Differences in the rise in RPP after tracheal intubation between the 1.25 and 2.0 MAC levels were significant in the sevoflurane and isoflurane, but not significant in the

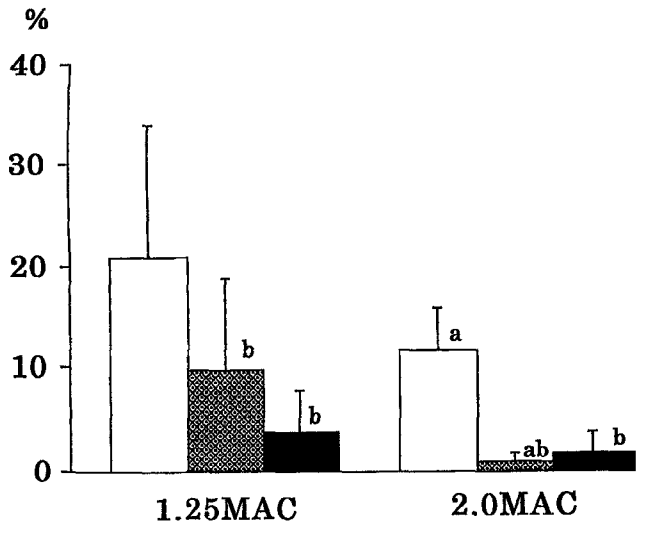

Fig. 1. Change in rate-pressure product (RPP) after tracheal intubation. *a*, $P < 0.05$ *vs* 1.25 MAC of the same agent; *b*, $P < 0.05$ *vs* sevoflurane at the same MAC. Values are mean ± SD, $n = 9$. The percentage change was calculated by dividing the value obtained 1 min after tracheal intubation by that obtained before tracheal intubation. *Open bars*, sevoflurane; *hatched bars*, isoflurane; *solid bars*, halothane

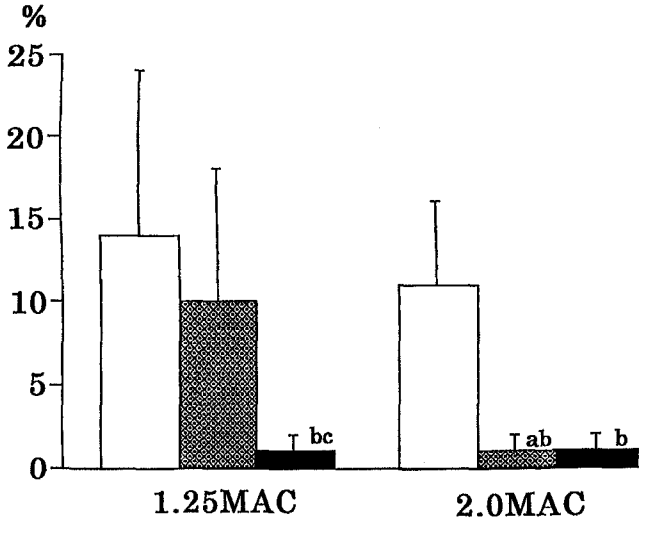

Fig. 2. Change in RPP after skin incision. *a*, $P < 0.05$ *vs* 1.25 MAC of the same agent; *b*, $P < 0.05$ *vs* sevoflurane at the same MAC; *c*, $P < 0.05$ *vs* isoflurane at the same MAC. Values are mean ± SD, $n = 9$. The percentage change was calculated by dividing the value obtained 1 min after skin incision by that obtained before skin incision. *Open bars*, sevoflurane; *hatched bars*, isoflurane; *solid bars*, halothane

halothane anesthesia groups. The rise in RPP after skin incision is shown in Fig. 2. At 1.25 MAC, the rise in RPP after skin incision was greater in the sevoflurane and isoflurane groups than in the halothane anesthesia group ($P < 0.05$). At 2.0 MAC, the rise was greater with sevoflurane anesthesia than with isoflurane or halothane anesthesia ($P < 0.05$).

Changes in NA and A levels are shown in Tables 4 and 5. Noradrenaline levels increased significantly after induction of anesthesia in the 2.0 MAC sevoflurane and isoflurane groups ($P < 0.05$). Tracheal intubation increased NA levels further and a significant increase from control values ($P < 0.05$) was apparent in all groups except the 1.25 MAC halothane group. Skin incision also increased NA levels, but there was no significant difference in the rise in NA levels between groups. Adrenaline decreased after induction of anesthesia and the rise in A levels after tracheal intubation was not significant. The rise in A levels after incision was also not significant except in the 2.0 MAC sevoflurane and 1.25 MAC isoflurane groups.

Discussion

The present study indicates that the suppressive actions of volatile anesthetics on the cardiovascular changes induced by tracheal intubation and skin incision differ between agents even if compared on the same MAC basis, and that these suppressive actions are most potent with halothane, least with sevoflurane, and intermediate with isoflurane.

Roizen et al. defined the potency of an anesthetic in blocking adrenergic response to skin incision as MAC-BAR [2], and demonstrated no difference between anesthetics in the ratio of MAC-BAR to MAC. The value of MAC-BAR was 1.45 MAC for halothane and 1.60 MAC for enflurane. The patients in their study, however, received 60% nitrous oxide with the anesthetics tested. Nitrous oxide has a potent analgesic action and might therefore mask any differences between the potencies of the anesthetics in suppressing adrenergic and cardiovascular responses to noxious stimuli. The present study was performed without analgesics or nitrous oxide and revealed that the ratios of MAC-BAR to MAC were different with different agents. We assumed the MAC value of sevoflurane to be 1.71% [4], but the other investigator reported it as being 2.05% [5]. Even if the MAC values of sevoflurane we used were low, the suppressive action of sevoflurane was the least. The results for sevoflurane in this study are consistent with those of a recent report which indicated that the MAC-BAR for sevoflurane is above 7% (4.3 MAC) [6].

The present study indicates that the suppressive actions of anesthetic agents at the same MAC concentration can differ if different responses are observed. MAC is based on body movement in response to noxious

Table 4. Changes in noradrenaline concentrations ($pg \cdot ml^{-1}$, $n = 9$ in each group, mean ± SD)

	MAC	Control	1	2	3	4	5	6	7	8
Sevoflurane	1.25	131 ± 96	173 ± 87	236 ± 125*	174 ± 98	166 ± 96	151 ± 79	269 ± 135*¶	249 ± 125*¶	222 ± 118*
	2.0	151 ± 43	281 ± 86*[a]	358 ± 110*[a]	311 ± 71*[a]	296 ± 110*[a]	196 ± 76	359 ± 130*¶	351 ± 112*¶	338 ± 72*¶
Isoflurane	1.25	169 ± 33	241 ± 67	578 ± 432*§	303 ± 114[b]	241 ± 56	171 ± 43	347 ± 139	337 ± 120	334 ± 69
	2.0	136 ± 90	340 ± 195*	336 ± 175*[a]	309 ± 157*	271 ± 148	188 ± 140	292 ± 178	334 ± 219*	360 ± 195*¶
Halothane	1.25	170 ± 97	211 ± 59	275 ± 109[c]	236 ± 66	202 ± 37	152 ± 40	201 ± 45	227 ± 47	305 ± 101*¶
	2.0	164 ± 92	305 ± 73	391 ± 169*	347 ± 116*[a]	322 ± 91[a]	182 ± 45	274 ± 89	349 ± 148*¶	406 ± 167*¶

1, before tracheal intubation; 2, 1 min after intubation; 3, 2 min after intubation; 4, 5 min after intubation; 5, before skin incision; 6, 1 min after skin incision; 7, 2 min after skin incision; 8, 5 min after skin incision.
* $P < 0.05$ *vs* control; § $P < 0.05$ *vs* 1; ¶ $P < 0.05$ *vs* 5; [a] $P < 0.05$ *vs* 1.25 MAC of the same agent; [b] $P < 0.05$ *vs* the same MAC of sevoflurane; [c] $P < 0.05$ *vs* the same MAC of isoflurane.

Table 5. Changes in adrenaline concentrations ($pg \cdot ml^{-1}$, $n = 9$ in each group, mean ± SD)

	MAC	Control	1	2	3	4	5	6	7	8
Sevoflurane	1.25	58 ± 40	14 ± 12	24 ± 18	17 ± 18	15 ± 20	16 ± 23	63 ± 68	44 ± 54	51 ± 66
	2.0	66 ± 48	12 ± 10	14 ± 11	12 ± 8	10 ± 8	9 ± 9	24 ± 16[a]	20 ± 11	78 ± 87¶
Isoflurane	1.25	63 ± 31	8 ± 3	17 ± 12	12 ± 5	8 ± 3	6 ± 2	29 ± 30[b]	29 ± 32	67 ± 86¶
	2.0	58 ± 47	13 ± 6	12 ± 6	10 ± 4	9 ± 3	6 ± 3	16 ± 13	31 ± 50	100 ± 163
Halothane	1.25	91 ± 52	18 ± 16*	21 ± 11*	17 ± 10*	14 ± 10*	13 ± 9*	15 ± 10*[b]	21 ± 15*	35 ± 38*
	2.0	63 ± 60	9 ± 4*	10 ± 2*	9 ± 2*	9 ± 3*	7 ± 2*	11 ± 4*	12 ± 5*	23 ± 34

1, before tracheal intubation; 2, 1 min after intubation; 3, 2 min after intubation; 4, 5 min after intubation; 5, before skin incision; 6, 1 min after skin incision; 7, 2 min after skin incision; 8, 5 min after skin incision.
* $P < 0.05$ *vs* control; ¶ $P < 0.05$ *vs* 5; [a] $P < 0.05$ *vs* 1.25 MAC of the same agent; [b] $P < 0.05$ *vs* the same MAC of sevoflurane.

stimuli. The suppression of body movement in response to noxious stimuli caused by anesthetics may involve suppression of the sensory systems, the central integrating systems, and the motor nervous system as well as the effector, skeletal muscles. If a different response is evaluated, the systems involved in the evoked responses are different. Volatile anesthetics do not have identical actions on the different networks in the nervous system, and different anesthetics may have different actions on a given system. Thus, the suppressive actions of anesthetic agents may vary if different responses are observed. Furthermore, the suppressive actions of anesthetics might reflect the summation of the basal suppression, which is indicated at the unstimulated state, and the suppression of the response capability of the system involved. Body movement responses are usually evaluated as either positive or negative as a result of the summation. On the other hand, the variables measured in this study were evaluated quantitatively. Thus, we could discriminate suppressions on the response capabilities from basal suppressions at the unstimulated states in the circulatory and hormonal systems. The basal suppressions by the agents of blood pressure and heart rate before tracheal intubation and skin incision did not differ between agents. However, changes of blood pressure and heart rate caused by intubation or incision were greatest with sevoflurane anesthesia.

The order of potency of these volatile anesthetics in suppressing cardiovascular changes is similar to that for depressing evoked potentials in cats. Ogawa et al. [7] showed that halothane was the most potent depressant of the photic evoked response in cats, and that enflurane augmented rather than suppressed the amplitude of this response. Isoflurane depressed the amplitude but shortened the latency of the photic evoked response. Sevoflurane has similar actions on the central nervous system to enflurane in that it depresses spontaneous neuronal activity in the midbrain reticular formation but enhances somatosensory evoked potentials under deep anesthesis [8]. We previously reported a difference between the effects of halothane and enflurane on the bradykinin-induced rise in neuronal activity in the spinal lateral funiculus [9]. Halothane depressed this response significantly, whereas enflurane had no effect in spinal cats. These findings all indicate that halothane has more potent suppressing actions on sensory input to the cerebral cortex induced by peripheral stimulation than isoflurane, sevoflurane, or enflurane.

Although the hemodynamic responses differed between anesthetic agents, catecholamine levels were not significantly different. The arterial levels of catecholamines may not be reliable indices of the amounts of catecholamines released from the sympathetic nervous system since anesthetics can modify the catecholamine

reuptake process into the nerve terminal as well as the releasing process [10,11].

MAC is defined as an index of the depth of anesthesia. However, cardiovascular responses, such as arterial blood pressure and heart rate, induced by noxious stimuli are used as indices of the depth of anesthesia in clinical practice. To suppress the hemodynamic responses, higher concentrations of sevoflurane and isoflurane than of halothane are required on a MAC basis. However, higher concentrations of volatile anesthetics depress hemodynamics during periods without surgical stimuli. The hemodynamic variables showed no significant difference between agents in the nonstimulated state in this study when the same MAC levels were used. These considerations suggest that sevoflurane and isoflurane should be used with other analgesic agents, such as nitrous oxide or opioids, to stabilize hemodynamic variables during anesthesia. We investigated hemodynamic variables in normotensive patients. In hypertensive or elderly patients, we speculate that greater hemodynamic changes could occur in response to noxious stimuli.

In conclusion, the basal hemodynamic suppressions by the three anesthetics studied were similar at the same MAC, whereas the suppressive action of sevoflurane on the hemodynamic response to noxious stimuli was weaker than that of isoflurane or halothane.

Acknowledgments. This study was supported in part by Grants-in-Aid for Scientific Research No. 07771275 and 08457415, from the Ministry of Education, Science and Culture, Japan.

References

1. Eger EI II, Saidman LJ, Brandstater B (1965) Minimum alveolar anesthetic concentration: A standard of potency. Anesthesiology 26:756–763
2. Roizen MF, Horrigan RW, Frazer BM (1981) Anesthetic doses blocking adrenergic (stress) and cardiovascular responses to incision-MAC BAR. Anesthesiology 54:390–398
3. Yakaitis RW, Blitt CD, Angiulo JP (1977) End-tidal halothane concentration for endotracheal intubation. Anesthesiology 47:386–388
4. Katoh T, Ikeda K (1987) The minimum alveolar concentration (MAC) of sevoflurane in humans. Anesthesiology 66:301–303
5. Scheller MS, Saidman LJ, Partridge BL (1988) MAC of sevoflurane in humans and the New Zealand white rabbit. Can J Anaesth 35:153–156
6. Ura T, Higuchi H, Sato T (1995) Minimum alveolar concentration of sevoflurane for blocking adrenergic response to tracheal intubation and surgical incision in women. Anesthesiology 83:A327
7. Ogawa T, Shingu K, Shibata M, Osawa M, Mori K (1992) The divergent actions of volatile anaesthetics on spontaneous neuronal activity and reactive capability in central nervous system in cats. Can J Anaesth 39:862–872
8. Osawa M, Shingu K, Murakawa M, Adachi T, Kurata J, Seo N, Murayama T, Nakao S, Mori K (1994) Effects of sevoflurane on central nervous system electrical activity in cats. Anesth Analg 79:52–57
9. Shingu K, Osawa M, Omatsu Y, Komatsu T, Urabe N, Mori K (1981) Naloxone does not antagonize the anesthesic-induced depression of nociceptor-driven spinal cord response in spinal cats. Acta Anaesth Scand 25:526–532
10. Deegan R, He HB, Wood AJJ, Wood M (1991) Effects of anesthesia on norepinephrine kinetics: Comparison of propofol and halothane anesthesia in dogs. Anesthesiology 75:481–488
11. Deegan R, He HB, Wood AJJ, Wood M (1993) Effect of enflurane and isoflurane on norepinephrine kinetics: a new approach to assessment of sympathetic function during anesthesia. Anesth Analg 77:49–54