Effects of volatile anesthetics on vagal C-fiber activities and their reflexes in anesthetized dogs

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Abstract

Effects of halothane, enflurane, isoflurane, and sevoflurane on vagal capsaicin (CAPS)-sensitive C-fibers were elucidated in anesthetized dogs. The CAPS-sensitive C-fibers were significantly stimulated by all volatile anesthetics with a significantly greater response to halothane than with sevoflurane. A significant increase in respiratory frequency (fR) and a significant decrease in tidal volume (VT) were observed with halothane and isoflurane, and a significant increase in fR was observed with sevoflurane. In contrast, a significant decrease in fR was induced by enflurane. The tachypnea induced by halothane, isoflurane, and sevoflurane was significantly reduced or no longer observed after perineural CAPS-treatment or bilateral vagotomy, whereas the slowing of respiration observed with enflurane was not affected by either of these treatments. These results suggest that vagal C-fibers play an important role in the reflex tachypnea that occurs with halothane, isoflurane, and sevoflurane. © 1998 Elsevier Science B.V. All rights reserved.

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1. Introduction

Acute inhalation of volatile anesthetics sometimes elicits excessive reactions such as coughing, inhibition of breathing (apnea), laryngospasm, and secretion (Drummond, 1988, 1993). Among the three types of sensory endings (slowly adapt-
ing stretch receptor, rapidly adapting irritant receptor, and C-fiber afferents) recognized in the lower airways, the rapidly adapting irritant receptors (RARs) and capsaicin (CAPS)-sensitive C-fibers are known to be responsible for the sensitivity to various chemical substances (Cole-ridge and Coleridge, 1984). Several studies have been performed on the responses of these vagal sensory endings to inhalation of volatile anesthetics: Coleridge et al. (1968) have pointed out that pulmonary CAPS-sensitive C-fibers are stimulated by ether, chloroform, trichloroethylene and halothane. Nishino et al. (1994) have reported that the discharge of tracheobronchial RARs is consistently inhibited by halothane, enflurane, and isoflurane.

Considering the above, it is possible to speculate that vagal C-fibers and RARs play an important role in the elicitation of airway reflexes during the inhalation of volatile anesthetics. However, electrophysiological data on these sensory endings, especially regarding the effects of new volatile anesthetics such as isoflurane or sevoflurane on vagal C-fibers, has been limited. Therefore, the aim of this study was: (1) to elucidate the effects of the volatile anesthetics on vagal CAPS-sensitive C-fiber activities; and (2) to evaluate the role of the C-fibers in the elicitation of cardiorespiratory reflexes in response to the anesthetics.

2. Methods

2.1. General

A total of 27 beagle and ten mongrel dogs of either sex were used in this study. Their mean body weight was 11.4 kg (ranging from 7.2 to 17.0 kg). Food was withheld at least 12 h before the experiments. This study was conducted under the guidelines provided by the Animal Care Committee of the Graduate School of Agricultural and Life Sciences, The University of Tokyo.

Anesthesia was induced with thiopental sodium (25 mg/kg), then maintained with a mixture of urethane (500 mg/kg) and α-chloralose (50 mg/kg) injected intravenously. A supplemental dose of urethane (200 mg/kg) and α-chloralose (20 mg/kg) was injected hourly through an intravenous catheter placed into the cephalic or saphenous vein.

2.2. Experimental protocol

2.2.1. Protocol 1: Recordings of the afferent activity of vagal C-fibers

A total of 19 beagle and four mongrel dogs were used in this experiment. The dogs were placed on an operating table in the supine position, endotracheally intubated, and ventilated spontaneously with room air. A skin incision was made in the neck to expose the vagus nerve. The left vagus nerve was transected just caudal to the nodose ganglion, and its peripheral cut end was separated to record its electrical activity. The right vagus nerve was also cut to eliminate possible secondary vagal reflexes. A pressure transducer (Toyoda, PD 104) was attached to the tracheal cannula in order to measure the intratracheal pressure (P\textsubscript{a}). Arterial blood pressure was monitored by a pressure transducer (Nihon Kohden, DX-300) connected to a catheter inserted into the femoral artery. P\textsubscript{O\textsubscript{2}} (P\textsubscript{T\textsubscript{O\textsubscript{2}}}) and (P\textsubscript{CO\textsubscript{2}}) in tidal air were sampled through the line attached to the endotracheal tube and monitored by a gas analyzer (NEC san-ei, Respina 1H26).

Afferent activity of the nerve filament was recorded with a pair of platinum electrodes. The nerve trunk was dissected into several thin filaments until a single unit activity was clearly discriminated. These nerve preparations were performed within a pool of paraffin oil using fine forceps with the aid of a binocular microscope (Olympus, SZ 60). The signal was amplified by a low noise DC-amplifier (DIA Med., DPA 201) and a biophysical amplifier (DIA Med., DPA 200), and displayed on an oscilloscope (Iwatsu, SS 5762) in parallel with a loudspeaker (NEC san-ei, Model 7747). All the signals were displayed on a thermal-array recorder (NEC san-ei, RT 3100N) and recorded by a magnetic tape recorder (Sony, PC 204A). Once an acceptable single unit was found, CAPS-sensitive C-fibers and RARs were identified by their characteristic patterns of discharge, responses to lung inflation (2–3 V\textsubscript{T}) and deflation, and to right and left atrial injections of CAPS (10 μg/kg) according to the previous meth-
ods (Coleridge and Coleridge, 1977, 1984). The CAPS injections were performed either: (1) through a 5-French Swan-Ganz catheter (Baxter Healthcare, Model 93-132-5F) positioned at the right atrium via the left jugular vein; or (2) through a 20 G soft polyvinyl catheter (Toray Medical, Anthron) advanced into the left atrium via the common carotid artery.

After a control period of > 1 min with 100% oxygen at a flow rate of 6 L/min, each fiber was challenged with 5% halothane, enflurane, isoflurane, and sevoflurane for 1 min each. The order of administration of each anesthetic was random. The anesthetic vaporizer was the specific type for each anesthetic (BOC, FLUOTEC 3 for halothane; Cyprane, ENFLURATEC for enflurane; Muraco Medical, FORAWIC for isoflurane; Kimura Medical, S-3 for sevoflurane). The intratracheal anesthetic concentration was monitored with an infrared gas monitor (Datex, Capnomac) which sampled through a line connected to the uppermost sidearm. Anesthetic gas was delivered in 95% oxygen by using a semi-closed circle system (Kimura Medical, COMPACT 15).

At the end of the experiment, the dogs were chest-opened and artificially ventilated by a ventilator (Kimura Medical, KV – 1 + 1) to investigate each receptive field by exploring the external surface of the lower airways with a cotton stick. The nerve filament on the electrodes was covered by cotton pledgets soaked in mineral oil, and the thoracotomy was performed carefully with the electrical scalpel (Silver Medical, Electrosurgical Unit SL-5) so as not to disturb the vagal filament during this surgery.

2.2.2. Protocol 2: Perineural CAPS treatment

Eight beagle and six mongrel dogs were used in this experiment. To evaluate the reflex contribution of C-fibers by inhalation of volatile anesthetic, a method that had previously been used successfully in dogs (Schelegle et al., 1995) for the perineural CAPS treatment of the vagus nerves was applied in this study. Briefly, the midcervical segment of each vagus nerve was isolated, and cotton pledgets soaked in a digestive solution consisting of Krebs solution (136.9 mM NaCl, 5.4 mM KCl, 5.5 mM glucose, 23.8 mM NaHCO₃, 1.5 mM CaCl₂, 1.0 mM MgCl₂, and 0.001–0.01 mM EDTA) containing collagenase (1000 U/ml) and hyaluronidase (1000 U/ml) were placed on each segment for 20 min to remove connective tissue and to increase segment permeability. The digestive solution was removed by washing with saline after 20 min, and cotton pledgets containing a 1% CAPS solution in a vehicle of 10% ethanol, 10% Tween 80, and 80% saline were then placed on the vagal segments for 15–20 min to block C-fiber activity. Each anesthetic was then administered according to the procedure described in protocol 1. Changes in tidal volume (Vₜ), respiratory frequency (fR), and ventilation (VE) were recorded by a respirometer (Bear Medical Systems, NVM-1). The efficacy of C-fiber blockade was evaluated by the preservation of responses to lung inflation (Hering-Breuer reflex) and lack of response to the right atrial injection of CAPS (10 µg/kg) before and after the trial.

All the dogs were euthanized at the end of the experiment by an intravenous injection of pentobarbital sodium (50 mg/kg).

2.3. Data analysis

The discharge frequencies of single units were counted for 1 min before the trial with a spike height discriminator (DIA Medical, DSE 425), and the data were then averaged every 10 sec. The averaged discharge frequency for 1 min before the challenge was taken as the control value, and the maximum or minimum discharge frequency out of the values averaged every 10 sec after the beginning of the challenge was taken as the peak value. The latency (sec) of each receptor’s response was evaluated as the point when the number of discharges increased or decreased. Changes in heart rate (HR) and mean arterial blood pressure (ABP) were obtained from the tracings before and after the onset of each trial. The ABP was estimated as the sum of the diastolic pressure and one-third of the pulse pressure. The HR was calculated from the arterial blood pressure tracings. In all the cardiorespiratory parameters, control values were averaged for 1 min before the trial, and peak values were taken after the onset of each trial.
For the comparison between the data, Wilcoxon’s signed rank sum test or Mann–Whitney’s U-test was used. To compare the differences among anesthetic groups, a one-way ANOVA test was run, followed, when necessary, by Tukey’s multiple comparison test. $P < 0.05$ were taken as statistically significant. All data were expressed as mean $\pm$ SE.

3. Results

3.1. Subtypes and pulmonary location of CAPS-sensitive C-fibers and RARs

Single units were recorded from a total of 21 pulmonary C-fibers and 15 bronchial CAPS-sensitive C-fibers and 15 RARs. Changes in the discharge pattern of both types of endings in response to the right atrial injection of CAPS are shown in Fig. 1. Stimulation of CAPS-sensitive C-fibers by right atrial injection of CAPS coincided with an inhibition of breathing (apnea), followed by hypotension and bradycardia, while the activity of the RARs was unaffected (Fig. 1).

The CAPS-sensitive C-fibers observed in this study were divided into two types of receptors according to the criteria reported by Coleridge and Coleridge (1977; 1994). The latency in response to the right atrial injection of CAPS was $2.7 \pm 0.5$ sec for pulmonary C-fibers and $7.4 \pm 1.8$ sec for bronchial C-fibers. The pulmonary C-fibers were not stimulated by the left atrial injection of CAPS, whereas the bronchial C-fibers were activated with a latency of $3.5 \pm 1.2$ sec.

The pulmonary location of each ending was determined in 17 CAPS-sensitive C-fibers (10 pulmonary C-fibers and 7 bronchial C-fibers) and five RARs. In this study, all the RARs (5/5, 100%) and three bronchial C-fibers (3/7, 43%) were located in the extra-pulmonary bronchi. On the other hand, all pulmonary C-fibers (10/10, 100%) and four bronchial C-fibers (4/7, 57%) were found within the left lung. Most pulmonary C-fibers were located in the central or peripheral part of the lung, while the majority of bronchial C-fibers were located in the proximal part.

3.2. Effects of volatile anesthetics on CAPS-sensitive C-fibers and RARs and their cardiorespiratory reflexes

The effects of volatile anesthetics on vagal CAPS-sensitive C-fibers and RARs are illustrated in Figs. 2 and 3. Peak-averaged discharges of 36 CAPS-sensitive C-fibers (21 pulmonary C-fibers and 15 bronchial C-fibers) and 15 RARs are shown in Fig. 4.

The discharge frequency of C-fibers was markedly increased after the onset of inhalation of all the anesthetics (Figs. 2 and 3), while that of the RARs was inhibited (Fig. 2). The increase in both pulmonary and bronchial C-fiber activity was significantly greater with halothane than with sevoflurane ($P < 0.05$, Fig. 4). The effects of enflurane and isoflurane were midcourse between halothane and sevoflurane. The latency of pulmonary C-fibers was $18.5 \pm 2.1$ sec for halothane, $29.5 \pm 2.4$ sec for enflurane, $27.3 \pm 1.9$ sec for isoflurane, and $42.5 \pm 3.1$ sec for sevoflurane. The latency of bronchial C-fibers was $7.3 \pm 1.0$ sec for halothane, $11.2 \pm 1.8$ sec for enflurane, $9.8 \pm 1.6$ sec for isoflurane, and $16.5 \pm 2.2$ sec for sevoflurane. With all anesthetics, a significantly shorter latency was observed in bronchial C-fibers than in pulmonary C-fibers ($P < 0.05$). The latency of each C-fiber response was significantly shorter with halothane than with sevoflurane ($P < 0.05$).

There was no statistically significant difference between anesthetics with regard to the discharge of RARs ($P = 0.17$, Fig. 4). The delay of the onset of inhibition was $5.7 \pm 0.9$ sec for halothane, $12.8 \pm 1.6$ sec for enflurane, $8.6 \pm 1.5$ sec for isoflurane, and $14.3 \pm 2.2$ sec for sevoflurane. There were no significant differences among the latencies to the different anesthetics ($P = 0.09$).

The peak percent changes of $f_R, V_T$, and $V_E$ after the inhalation of each anesthetic in dogs with non-treatment ($n = 14$), CAPS-treatment ($n = 7$), and bilateral vagotomy ($n = 7$) are shown in Fig. 5. A significant increase in $f_R$ and a significant decrease in $V_T$ was observed during the inhalation of halothane ($P < 0.01$) and isoflurane ($P < 0.05$), and a significant increase in $f_R$ was observed with sevoflurane ($P < 0.05$). In contrast,
Fig. 1. Response of a pulmonary C-fiber (smaller spikes) and a rapidly adapting receptor (larger spikes) (upper panel) and a bronchial C-fiber (lower panel) to the right atrial injection of capsaicin (10 μg/kg) in an anesthetized spontaneously breathing dog. Note the pulmonary C-fiber was activated immediately after the capsaicin injection but the bronchial C-fiber had a longer latency, whereas in both cases apnea was followed by hypotension and bradycardia. Rapidly adapting receptor activity was unaffected by the capsaicin injection. The pulmonary C-fiber ending was located in the central part of the left lower lobe and the rapidly adapting receptor ending was in the left main bronchus. The bronchial C-fiber ending was located at the hilum of the left lower lobe. ENG, electroneurogram; Peso, esophageal pressure;Ptr, intratracheal pressure; PTO2, PO2 in tidal air; PTCO2, PCO2 in tidal air. The injection time is marked on the bottom trace.

A significant decrease in fR was observed during the inhalation of enflurane (P < 0.05) (Fig. 5).

The latency of the respiratory responses was 15.6 ± 1.8 sec for halothane, 26.3 ± 2.2 sec for enflurane, 21.5 ± 2.5 sec for isoflurane, and 28.4 ± 2.5 sec for sevoflurane. No statistically significant differences were found among these latencies (P = 0.26). The increase in fR and the decrease in Vt were significantly reduced by both perineural CAPS-treatment and bilateral vagotomy only for halothane (P < 0.05) (Figs. 5 and 6). Significant changes in these parameters from the control level were no longer observed during the administration of either isoflurane or sevoflurane after perineural CAPS-treatment or bilateral vagotomy. In contrast, the effect of enflurane on fR and Vt are the opposite of those of the other three anesthetics. No significant dif-
Fig. 2. Responses of a pulmonary C-fiber and a rapidly adapting receptor (the same endings as in Fig. 1, upper panel) to 5% volatile anesthetics in an anesthetized spontaneously breathing dog. The pulmonary C-fiber (smaller spike) was stimulated especially by halothane, whereas the rapidly adapting receptor (larger spike) was inhibited by all anesthetics. Shallow breathing was introduced by the inhalation of halothane, isoflurane, and sevoflurane, but slowing of breathing was observed during the administration of enflurane. The horizontal lines show the inhalation time of each volatile anesthetic. Abbreviations as in Fig. 1.
Fig. 3. Responses of a bronchial C-fiber (the same ending as in Fig. 1, lower panel) to 5% volatile anesthetics in an anesthetized spontaneously breathing dog. The bronchial C-fiber was stimulated by all anesthetics, most strongly by halothane. The horizontal lines show the inhalation time of each volatile anesthetic. Abbreviations as in Fig. 1.
Fig. 4. Responses of pulmonary C-fibers (n = 21), bronchial C-fibers (n = 15), and rapidly adapting receptors (n = 15) to 5% volatile anesthetics. Control and peak values (mean ± SE) were obtained from recordings for 1 min before inhalation and for 10 sec at maximum response during inhalation. Hal, halothane; Enf, enflurane; Iso, isoflurane; Sevo, sevoflurane. * P < 0.05, ** P < 0.01 vs. control, † P < 0.05 vs. sevo.

In all groups (non-treatment, CAPS-treatment, and bilateral vagotomy), ABP decreased significantly with all anesthetics (P < 0.05), while HR increased significantly only with isoflurane (P < 0.05) (Table 1). No statistically significant differences were found in HR and ABP values among the anesthetics within and between groups, although HR was higher in dogs with vagotomy (Table 1).

4. Discussion

A rapid shallow breathing pattern represented by an increase in fR and a decrease in VT was consistently observed after the onset of inhalation of halothane, isoflurane, and sevoflurane, which coincided with an increase in the number of action potentials for the bronchial and pulmonary C-fibers. Moreover, most of the effects on breathing pattern produced by these inhalants were reduced or eliminated by either perineural CAPS treatment or bilateral vagotomy. Perineural CAPS-treatment provides a complete abolition of cardiorespiratory reflexes via the vagal CAPS-sensitive C-fibers without affecting the Hering–Breuer inflation reflex via the slowly adapting pulmonary stretch receptors (Schlegle et al., 1995). Such reflex responses do not include any upper airway reflex because the anesthetics were administered by method that bypassed the larynx. These observations are consistent with the findings that the reflex tachypnea, at least that evoked by the inhalation of halothane, isoflurane, and sevoflurane observed in the present study, is mediated primarily by the stimulation of vagal C-fibers.

In this study, a greater stimulation of the vagal C-fibers was observed with halothane, accompanied by a greater tachypnea than that induced by other anesthetics, which is consistent with the significant reduction in response after CAPS-treatment or vagotomy (Fig. 5). This finding, along with clinical observations that the administration of halothane results in an increased incidence of tachypnea as compared with enflurane, isoflurane, and sevoflurane during the induction and maintenance of anesthesia (Mutoh et al., 1995, 1997), suggests the important role of the C-fibers during the inhalation of halothane.

In contrast to the tachypnea which occurs in response to the inhalation of these three inhalants, a slowing of respiration is observed during the inhalation of enflurane. Because these respiratory responses to enflurane were equally observed in all the groups with or without CAPS-treatment and bilateral vagotomy, it is possible that the
reflex responses mediated by vagal C-fiber afferents might have been masked or blocked by the depression of the central nervous system via the anesthetic absorbed into the systemic circulation. Halogenated volatile anesthetics generally depress medullary inspiratory and expiratory neurons by blocking synaptic transmission, the effect of which is greater with enflurane than halothane (Kasaba et al., 1987).

In the present study, differences in the latencies between pulmonary and bronchial C-fibers to the right atrial CAPS injection were recognized, the results of which mostly agree with those of a previous study (Coleridge and Coleridge, 1977, 1984). In contrast, the latencies of each C-fiber to inhalation of volatile anesthetics were reversed. This is because the preferential stimulation of bronchial C-fibers by inhaled substances is more likely due to the greater accessibility of the agents.

In any event, it is still unclear which type of C-fibers are more likely to be associated with the tachypnea that occurs during halothane, isoflurane, and sevoflurane administration; it can be presumed, however, that the bronchial C-fibers make an important contribution to the reflex tachypnea based on the coincidence of the latencies between the two responses.

The inhibitory effects of volatile anesthetics on RARs in the present study were not consistent with findings for other inhaled chemical irritants such as NH₃ (Mills et al., 1969; Bergren and Sampson, 1982; Matsumoto, 1989) and cigarette smoke (Sellick and Widdicombe, 1971; Kou and Lee, 1990), which induced a stimulation of RARs in various species. The inhibition of tracheobronchial RARs by the inhalation of volatile anesthetics has also been reported in a previous study (Nishino et al., 1994), as well as the inhibi-

![Fig. 5. Summary of ventilatory responses to volatile anesthetics with or without capsaicin (CAPS)-treatment and bilateral vagotomy in 14 anesthetized spontaneously breathing dogs. The increase in fR and the decrease in VT with halothane were significantly depressed after CAPS-treatment and vagotomy. Significant changes in these respiratory parameters with isoflurane and sevoflurane were no longer observed after the treatments. In contrast, no significant changes were observed with enflurane among treatments. Each value is expressed as the peak percent change from control (mean ± SE). * P < 0.05, ** P < 0.01 vs. control, # P < 0.05 vs. non-treatment.](image-url)
Fig. 6. Time-course of changes in ventilation during the inhalation of halothane with or without capsaicin (CAPS)-treatment and bilateral vagotomy in 14 anesthetized spontaneously breathing dogs. Values are expressed as mean ± SE. The horizontal lines show the onset of the inhalation of halothane.

We did not find any significant differences in the cardiovascular responses to the volatile anesthetics with or without treatment. This suggests that the cardiovascular change in response to the anesthetics is more likely attributable to direct peripheral vasodilation (Bernard et al., 1990; Pagel et al., 1991), than the stimulation of vagal C-fibers. Higher HR and slightly lower ABP values of isoflurane might be due to the direct action on the sympathetic output and vasodilator motor neurons. In fact, the stimulation of efferent sympathetic activities, rather than the stimulation of vagal sensory afferents, is primarily responsible for the cardiovascular responses to a rapid increase in isoflurane concentration (Okamoto et al., 1996). The vasodilator action of isoflurane is known to be greater than other anesthetics (Bernard et al., 1990; Pagel et al., 1991).

Clinically, various complications associated with the induction of anesthesia with volatile anesthetics have been presumed to be caused by an irritation of the airway mucosa (Doi and Ikeda, 1993). In our previous study, laryngeal CAPS-sensitive receptors were consistently stimulated by halogenated volatile anesthetics (Mutoh et al., 1998), and the degree of change in discharge frequency observed for volatile anesthetics clearly corresponded to the degree of airway irritation by volatile anesthetics as experienced clinically in humans, i.e. a higher incidence of complications such as coughing, laryngospasm, inhibition of breathing (apnea), and excessive secretions with halothane, enflurane, and isoflurane than with sevoflurane during the induction of anesthesia (Yurino and Kimura, 1992, 1993a,b, 1994; Funk et al., 1996).

The present study demonstrates the important role of vagal CAPS-sensitive C-fibers in the reflex hyperpnea induced by halothane, isoflurane, and sevoflurane. Such findings may be an indicative of the functional importance of these fibers in the defensive or protective airway reactions to volatile anesthetics.
Table 1
Changes in heart rate (HR) and mean arterial blood pressure (ABP) by inhalation of volatile anesthetics

<table>
<thead>
<tr>
<th>Anesthetic</th>
<th>Condition</th>
<th>HR (beats/min)</th>
<th>ABP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control/Peak</td>
<td>Control/Peak</td>
<td></td>
</tr>
<tr>
<td>Halothane</td>
<td>Non-treatment</td>
<td>125 ± 9</td>
<td>116 ± 9</td>
</tr>
<tr>
<td></td>
<td>Capsaicin-treatment</td>
<td>122 ± 8</td>
<td>118 ± 10</td>
</tr>
<tr>
<td></td>
<td>Vagotomy</td>
<td>141 ± 10</td>
<td>136 ± 12</td>
</tr>
<tr>
<td>Enflurane</td>
<td>Non-treatment</td>
<td>120 ± 11</td>
<td>124 ± 8</td>
</tr>
<tr>
<td></td>
<td>Capsaicin-treatment</td>
<td>128 ± 11</td>
<td>131 ± 12</td>
</tr>
<tr>
<td></td>
<td>Vagotomy</td>
<td>144 ± 10</td>
<td>146 ± 11</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>Non-treatment</td>
<td>125 ± 9</td>
<td>133 ± 8*</td>
</tr>
<tr>
<td></td>
<td>Capsaicin-treatment</td>
<td>123 ± 8</td>
<td>132 ± 10*</td>
</tr>
<tr>
<td></td>
<td>Vagotomy</td>
<td>138 ± 9</td>
<td>148 ± 7*</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>Non-treatment</td>
<td>123 ± 11</td>
<td>129 ± 10</td>
</tr>
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<td>Capsaicin-treatment</td>
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<td>135 ± 10</td>
</tr>
<tr>
<td></td>
<td>Vagotomy</td>
<td>143 ± 10</td>
<td>149 ± 12</td>
</tr>
</tbody>
</table>

No statistical significant differences among anesthetics were found in HR and ABP within and between treatments. All data were expressed as mean ± SE.
* Significantly different from control value (P<0.05), n = 14.

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