Assessment of halothane and sevoflurane anesthesia in spontaneously breathing rats

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Objective—To characterize halothane and sevoflurane anesthesia in spontaneously breathing rats.

Animals—16 healthy male Sprague-Dawley rats.

Procedure—8 rats were anesthetized with halothane and 8 with sevoflurane. Minimum alveolar concentration (MAC) was determined. Variables were recorded at anesthetic concentrations of 0.8, 1.0, 1.25, and 1.5 times the MAC of halothane and 1.0, 1.25, 1.5, and 1.75 times the MAC of sevoflurane.

Results—Mean (± SEM) MAC for halothane was 1.02 ± 0.02% and for sevoflurane was 2.99 ± 0.19%. As sevoflurane dose increased from 1.0 to 1.75 MAC, mean arterial pressure (MAP) decreased from 103.1 ± 5.3 to 67.9 ± 4.6 mm Hg, and $Paco_2$ increased from 58.8 ± 3.1 to 92.2 ± 9.2 mm Hg. As halothane dose increased from 0.8 to 1.5 MAC, MAP decreased from 99 ± 6.2 to 69.8 ± 4.5 mm Hg, and $Paco_2$ increased from 59.1 ± 2.1 to 75.9 ± 5.2 mm Hg. Respiratory rate decreased in a dose-dependent fashion from 88.5 ± 4.5 to 58.5 ± 2.7 breaths/min during halothane anesthesia and from 42.3 ± 1.8 to 30.5 ± 4.5 breaths/min during sevoflurane anesthesia. Both groups of rats had an increase in eyelid and pupillary aperture with an increase in anesthetic dose.

Conclusions and Clinical Relevance—An increase in $Paco_2$ and a decrease in MAP are clinical indicators of an increasing halothane and sevoflurane dose in unstimulated spontaneously breathing rats. Increases in eyelid aperture and pupil diameter are reliable signs of increasing depth of halothane and sevoflurane anesthesia. Decreasing respiratory rate is a clinical indicator of an increasing dose of halothane. (*Am J Vet Res* 2003;64:470–474)

Rats are commonly anesthetized in laboratory settings and increasingly so in clinical practice. Objective data on signs of general anesthesia in rats are limited. The purpose of the study reported here was to characterize halothane and sevoflurane anesthesia in spontaneously breathing rats. Recently, Imai et al[1] reported data on clinical signs of isoflurane anesthesia for rats. Our study provides similarly derived data for halothane and sevoflurane anesthesia in rats.

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Materials and Methods

Animals—Sixteen healthy male Sprague-Dawley rats were studied. Each rat was anesthetized with a single inhalation anesthetic agent and euthanatized at the end of the study. Eight rats were anesthetized with halothane, and 8 rats were anesthetized with sevoflurane. The age range for rats anesthetized with halothane was 16 to 23 weeks, and mean (± SE) body weight was 408 ± 7 g. The age range for rats anesthetized with sevoflurane was 16 to 23 weeks, and mean body weight was 473 ± 24 g. Rats were allowed ad libitum access to food and water until 4 to 8 hours prior to the onset of the study. Studies were performed between 8 AM and 6 PM. The Campus Animal Use and Care Administrative Advisory Committee of the University of California, Davis, approved the study.

Study conditions—Study conditions were similar to those reported previously.[1] Rats were weighed and placed in an acrylic induction chamber. Anesthesia was induced by delivery of the inhalation study agent (ie, 5% halothane or 7% sevoflurane in 5 L of **oxygen** [O_2]/min) into the induction chamber. After the rats became recumbent, anesthetic administration was continued via a face mask until sufficient anesthetic depth had been obtained to permit orotracheal intubation. No other pharmacologic agents were administered. Orotracheal intubation was accomplished by use of a laryngoscope and a 14-gauge IV catheter. Appropriate consideration was given to minimize respiratory dead space and prevent endobronchial intubation. Following intubation, rats were maintained with the study agent in 1 L of O_2/min, which was administered via a nonrebreathing (Bain style) circuit with a gas-sample collection device inserted between the endotracheal tube and the Bain circuit. Ventilation was spontaneous throughout the study period. Rectal temperature was maintained at 37 ± 1°C by use of a circulating water heating pad and heat lamps as needed. Rats were positioned in right lateral recumbency, and an ECG was continuously recorded on a physiograph.[a]

Minimum alveolar concentration determination and experimental protocol—Inspiratory and expiratory gases were hand-sampled from multiple breaths via a sample collection port located between the endotracheal tube and the Bain circuit. For example, 8 to 15 consecutive end-expired gas samples of approximately 0.5 mL each were collected in a 10-mL glass syringe fitted with a stopcock. Anesthetic gas and **carbon dioxide** (CO_2) concentrations were immediately measured by use of infrared gas analyzers.[b,c] A calibration curve for each gas was obtained prior to each experiment on the basis of gases of known concentration, and calibration was rechecked during the course of each study. The **minimum alveolar concentration** (**MAC**) of anesthetic preventing gross purposeful movement in response to a noxious stimulus was determined for each rat by use of the standard tail-clamp technique of Eger et al.[2] Fifteen minutes of constant end-tidal dose was allowed for equilibrium, and stimulus was administered until purposeful movement was observed or 1 minute had elapsed. End-tidal concentration steps were performed at ± 10 to 15% of the last determined concentration. The measured agent concentration was corrected on the basis of the obtained individual anesthetic agent calibration

curves. The MAC was determined in triplicate for each rat, and the mean was recorded as the MAC for each rat. Ratios of **alveolar-to-inspired anesthetic gas concentrations (F_A/F_I)** were also determined. These data were summarized by averaging all pairs of F_A/F_I values obtained for each MAC determination in each rat (ie, at least 3 pairs/rat).

After MAC was determined, a 24-gauge catheter was placed in the distal portion of the aorta via the femoral artery by use of a cut-down technique. Continuous direct arterial pressure was recorded by use of a physiograph.[a] Individual rat-specific multiples of 0.8, 1.0, 1.25, and 1.5 MAC were calculated for halothane, and multiples of 1.0, 1.25, 1.5, and 1.75 were calculated for sevoflurane. The order in which MAC multiples were studied was randomly assigned a priori for each rat, and measurements were obtained at the end of 15 minutes of constant end-tidal agent concentration.

Anesthetic measurements—At each MAC multiple, the following measurements were recorded: heart rate and rhythm (via an ECG); arterial pressure (mm Hg); respiratory frequency (counted); arterial gas tensions[d] (**arterial partial pressure of CO_2 [$Paco_2$]** and **arterial pressure of O_2 [Pao_2]**; mm Hg); **arterial pH [pH_a]**[d]; **total plasma protein [TP]** concentration (grams/deciliters; by refractometry); PCV (%); and ocular signs including globe position, nystagmus, eyelid aperture (millimeters), pupillary aperture (millimeters), palpebral reflex, corneal reflex, and lacrimation (palpebral and corneal reflexes and lacrimation were graded as present or absent). Heart rate, systolic pressure, diastolic pressure, **mean arterial pressure (MAP)**, and respiratory frequency were determined first; ocular signs were then recorded; and arterial blood sample collection was performed last for immediate analysis of PCV, TP, Pao_2, $Paco_2$, and pH_a. At each anesthetic concentration, 0.3 to 0.4 mL of blood was collected into 0.2-mL capillary tubes for blood gas analysis and determination of PCV and TP. Ocular signs were obtained in the following order: eyelid aperture, pupillary aperture, lacrimation, globe position, nystagmus, palpebral reflex, and corneal reflex. Pupil and eyelid aperture diameters were determined under directed light stimulus and measured with calipers and a millimeter ruler. Rectal temperature was continuously monitored via a calibrated thermistor probe. After each set of measurements was taken at each MAC multiple, the rat's tidal volume was augmented on each of 3 to 5 breaths by positive-pressure ventilation to expand any areas of atelectasis. At the end of the experiment, rats were euthanatized by IV administration of pentobarbital. Necropsy was performed on all but the first 2 rats studied to verify appropriate tracheal tube positioning and evaluate the gross appearance of the lungs.

Statistical analysis—Data are reported as mean ± SEM. The repeated measures ANOVA, followed when appropriate by the Tukey test, was used to detect significant differences on the basis of anesthetic dose (MAC multiples). The level of significance was set at 5%.

Results

MAC—The MAC for halothane and sevoflurane was 1.02 ± 0.02 and 2.99 ± 0.19%, respectively. The F_A/F_I was 0.85 ± 0.2 (n = 26) and 0.96 ± 0.01 (24) for halothane and sevoflurane, respectively, and body temperature was 37.5 ± 0.1 and 37.3 ± 0.1°C for halothane and sevoflurane, respectively.

Cardiovascular variables—In rats anesthetized with halothane, heart rate was similar for all MAC multiples (**Table 1**). Heart rate decreased with increasing sevoflurane dose (**Table 2**), but the change in heart rate was not significant ($P = 0.772$). Systolic pressure, diastolic pressure, and MAP were similar at 1.0 times the MAC of halothane and sevoflurane, and all pressures significantly decreased as the MAC multiple increased in both anesthetic groups. For both groups, MAP was significantly less at 1.5 and 1.75 MAC, compared with 1.0 MAC, and at 1.75 MAC, compared with 1.25 MAC. Results of heart rate and MAP measurements associated with halothane and sevoflurane anesthesias were graphically (**Fig 1 and 2**, respectively) compared with earlier reported, similarly derived results from isoflurane-anesthetized rats.[1]

Respiratory variables—Respiratory frequency decreased in a dose-dependent fashion in rats anesthetized with halothane (Table 1), and the change was significant at 1.25 and 1.5 MAC, compared with both 0.8 and 1.0 MAC. Respiratory frequency also decreased in rats anesthetized with sevoflurane (Table 2), but this change was only significant at 1.75 MAC, compared with 1.0 MAC. The respiratory frequency of rats anesthetized with 1.0 MAC of sevoflurane was only approximately half of that seen in rats anesthetized with 1.0 MAC of halothane, and the subsequent reduction in rate was proportionately less during sevoflurane anesthesia. Respiratory arrest was not seen with either of the agents or at any dose of the agents studied.

The $Paco_2$ was similar at 1.0 MAC with halothane and sevoflurane anesthesia. In the halothane group, $Paco_2$ increased and pH_a decreased beginning at 1.0 MAC, and the changes reached significance at 1.25 and 1.5 MAC for pH_a and $Paco_2$, respectively. Similarly, pH_a decreased and $Paco_2$ increased with increasing sevoflurane dose; significance was observed at 1.75 MAC, compared with 1.0, 1.25, and 1.5 MAC, and at 1.5 MAC, compared with 1.0 MAC for both variables. The pH_a was also significantly decreased in the sevoflurane group at 1.25 MAC, compared with 1.0 MAC. The changes in pH_a were related to the changes in $Paco_2$, because calculated arterial base balance was consistently in the range of 1 to 3 mEq/L. Results of measurements of respiratory frequency and $Paco_2$ associated with halothane and sevoflurane anesthesia were graphically (**Fig 3 and 4**, respectively) compared with earlier reported, similarly derived results from isoflurane-anesthetized rats.[1] The Pao_2 was more than adequate and usually > 250 mm Hg with both agents.

Hematologic variables—The PCV and TP were similar at 1.0 MAC for both agents. Significant differences in PCV and TP were not found at any MAC multiple regardless of the anesthetic agent (Tables 1 and 2).

Ocular indices—A palpebral reflex was present in all rats anesthetized with halothane at 0.8 MAC, but only in 2 of 8 rats at 1.0 MAC in both anesthetic groups; this reflex was abolished in both anesthetic groups at a depth of 1.25 MAC and higher (Tables 1 and 2). The corneal reflex was absent at all MAC multiples in rats anesthetized with sevoflurane. The corneal reflex was present in 5 of 8 rats at 0.8 MAC of halothane and in 1 of 8 rats at 1.0 times the MAC of halothane, but was absent at higher MAC multiples. Lacrimation was variably present in the halothane group of rats at a variety of

MAC multiples; however, it was only observed in 1 rat anesthetized with sevoflurane at 1.0 MAC. Nystagmus was not observed in any rats anesthetized with halothane or sevoflurane. Of the 8 rats anesthetized with sevoflurane, 1 had globe rotation at 1.0 MAC, 2 had globe rotation at 1.25 MAC, and 1 had globe rotation at 1.5 MAC. Of the 8 rats anesthetized with halothane, 3 had globe rotation at 0.8 MAC, and 2 had globe rotation at 1.0 MAC. Eyelid aperture increased with an increase in anesthetic depth in both groups of rats. Eyelid aperture was significantly increased at all MAC multiples in rats anesthetized with sevoflurane, compared with 1.0

Table 1—Mean (± SEM) values of variables measured in eight rats anesthetized with halothane

	Multiples of the MAC			
Variables	**0.8 MAC**	**1.0 MAC**	**1.25 MAC**	**1.5 MAC**
Respiratory rate (breaths/min)	88.5 ± 4.5	79.5 ± 3.2	64.5 ± 3.2[a,b]	58.5 ± 2.7[a,b]
Heart rate (beats/min)	302 ± 8	294 ± 5	296 ±7	296 ± 9
Systolic arterial pressure (mm Hg)	123 ± 7	113 ± 5	104 ± 6[a]	88 ± 5[a,b,c]
Mean arterial pressure (mm Hg)	99 ± 6	89 ± 4	81 ± 4[a]	70 ± 5[a,b]
Diastolic arterial pressure (mm Hg)	83 ± 5	74 ± 4	68 ± 3[a]	59 ± 4[a,b]
Arterial pH	7.32 ± 0.01	7.31 ± 0.01	7.28 ± 0.01[a,b]	7.23 ± 0.02[a,b,c]
Arterial Pco_2 (mm Hg)	59.1 ± 2.1	59.0 ± 1.6	66.0 ± 2.1	75.9 ± 5.2[a,b,c]
Arterial Po_2 (mm Hg)	321 ± 66	284 ± 83	293 ± 75	285 ± 59
PCV (%)	45.5 ± 1	44.4 ± 1.1	43.9 ± 0.9	43.8 ± 1.15
Total protein (g/dL)	5.5 ± 0.2	5.3 ± 0.2	5.3 ± 0.2	5.1 ± 0.3
Rectal temperature (°C)	37.6 ± 0.12	37.4 ± 0.09	37.5 ± 0.08	37.5 ± 0.12
Eyelid aperture (mm)	3.0 ± 0.3	3.3 ± 0.2	3.7 ± 0.2[a]	4.0 ± 0.1[a,b]
Pupillary aperture (mm)	1.6 ± 0.2	1.9 ± 0.3	2.2 ± 0.2	2.6 ± 0.2[a]
Rotated globe position (No. of rats)	3	2	0	0
Central globe position (No. of rats)	5	6	8	8
Positive palpebral reflex (No. of rats)	8	2	0	0
Negative palpebral reflex (No. of rats)	0	6	8	8
Positive corneal reflex (No. of rats)	5	1	0	0
Negative corneal reflex (No. of rats)	3	7	8	8
Positive lacrimation (No. of rats)	2	4	4	5
Negative lacrimation (No. of rats)	6	4	4	3
Positive Nystagmus (No. of rats)	0	0	0	0
Negative Nystagmus (No. of rats)	8	8	8	8

[a]Significantly ($P < 0.05$) different from value at 0.8 MAC. [b]Significantly ($P < 0.05$) different from value at 1.0 MAC. [c]Significantly ($P < 0.05$) different from value at 1.25 MAC.
MAC = Minimum alveolar concentration.

Table 2—Mean (± SEM) values of variables measured in eight rats anesthetized with sevoflurane

	Multiples of the MAC			
Variables	**1.0 MAC**	**1.25 MAC**	**1.5 MAC**	**1.75 MAC**
Respiratory rate (breaths/min)	42.3 ±0.8	40.3 ±1.7	37.7 ±1.8	30.5 ±4.5[a]
Heart rate (beats/min)	324 ±10	318 ±7	316 ±9	303 ±6
Systolic arterial pressure (mm Hg)	131±9	111 ±3[a]	100 ±7[a]	88 ±7[ab]
Mean arterial pressure (mm Hg)	103 ±5	90 ±2	79 ±6[a]	68 ±5[ab]
Diastolic arterial pressure (mm Hg)	91 ±6	79 ±3	68±5[a]	56 ±4[ab]
Arterial pH	7.33 ±0.02	7.27 ±0.01[a]	7.23 ±0.02[a]	7.15 ±0.03[a,b,c]
Arterial Pco_2 (mm Hg)	58.8 ±3.1	67.8 ±3.9	76.3 ±4.9[a]	92.2 ±9.2[a,b,c]
Arterial Po_2 (mm Hg)	500 ±22	462 ±19	405 ±33[a]	420 ±35
PCV (%)	42.4 ±0.6	42.5 ±0.8	1.2 ±1.2	40.8 ±1.3
Total protein (g/dL)	5.4 ±0.1	5.4 ±0.1	5.2 ±0.2	5.1 ±0.2
Rectal temperature (°C)	37.3 ±0.1	37.6 ±0.1	37.8 ±0.1	37.5 ±0.2
Eyelid aperture (mm)	3.1 ±0.3	3.7 ±0.3[a]	3.9 ±0.2[a]	4.2 ±0.2[a,b]
Pupillary aperture (mm)	1.6 ±0.2	2.6 ±0.5[a]	3.1 ±0.4[a]	9 ±0.4[a,b,c]
Rotated globe position (No. of rats)	1	2	1	0
Central globe position (No. of rats)	7	6	7	8
Positive palpebral reflex (No. of rats)	2	0	0	0
Negative palpebral reflex (No. of rats)	6	8	8	8
Positive corneal reflex (No. of rats)	0	0	0	0
Negative corneal reflex (No. of rats)	8	8	8	8
Positive lacrimation (No. of rats)	1	0	0	0
Negative lacrimation (No. of rats)	7	8	8	8
Positive Nystagmus (No. of rats)	0	0	0	0
Negative Nystagmus (No. of rats)	8	8	8	8

See Table 1 for key.

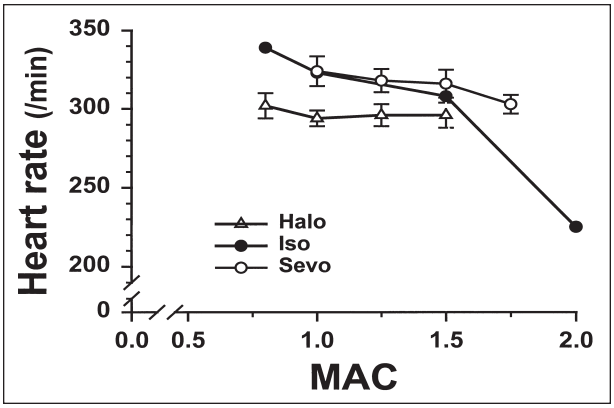

Figure 1—Mean (± SEM) heart rate (beats/min) in rats in response to changing doses of halothane (Halo) and sevoflurane (Sevo) in the present study, compared with results from rats similarly anesthetized with isoflurane (Iso) in a previous study[1] from the same laboratory. MAC = Minimum alveolar concentration.

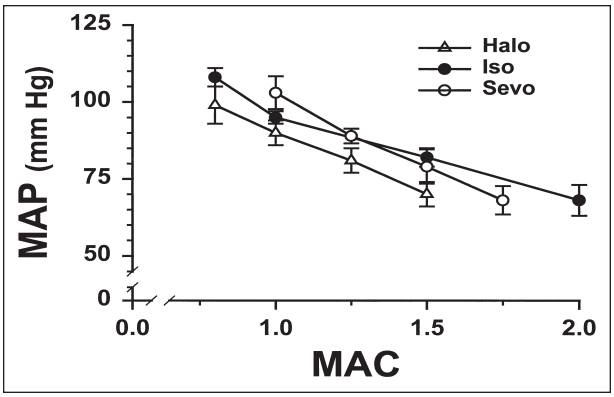

Figure 2—Mean (± SEM) value of mean arterial pressure (MAP; mm Hg) in rats in response to changing doses of Halo and Sevo in the present study, compared with results from rats similarly anesthetized with Iso in a previous study[1] from the same laboratory. *See* Figure 1 for remainder of key.

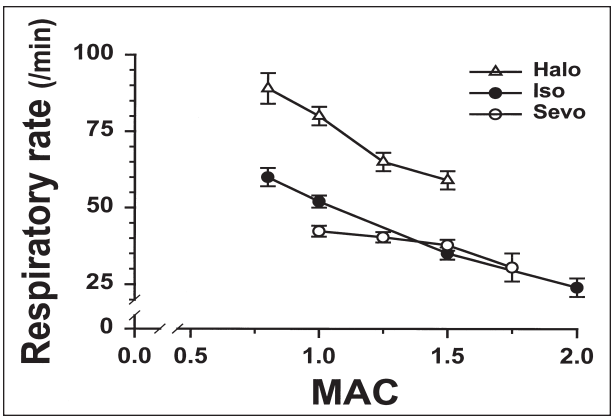

Figure 3—Mean (± SEM) respiratory rate (breaths/min) in rats in response to changing doses of Halo and Sevo in the present study, compared with results from rats similarly anesthetized with Iso in a previous study[1] from the same laboratory. *See* Figure 1 for remainder of key.

MAC (Table 2). Eyelid aperture was significantly increased at 1.5 MAC in rats anesthetized with halothane, compared with 1.0 MAC. Pupillary aperture also increased with increasing MAC multiples in both

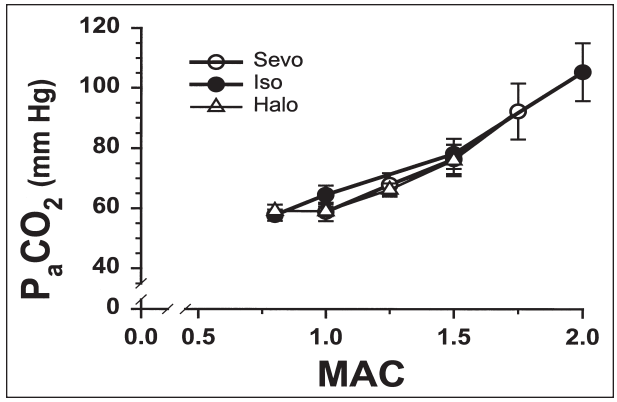

Figure 4—Mean (± SEM) arterial partial pressure of carbon dioxide ($Paco_2$; mm Hg) in rats in response to changing doses of Halo and Sevo in the present study, compared with results from rats similarly anesthetized with Iso in a previous study[1] from the same laboratory. *See* Figure 1 for remainder of key.

groups. For sevoflurane, a significant increase in pupillary aperture was present at 1.75 MAC, compared with all other MAC multiples, and at 1.25 and 1.5 MAC, compared with 1.0 MAC. For halothane, however, a significant increase in pupillary aperture was present only at 1.5 MAC, compared with 0.8 MAC.

Necropsy—Necropsies were performed to verify appropriate tracheal tube positioning and evaluate the gross appearance of the lungs. No obvious gross abnormalities of lung parenchyma were observed in either cohort.

Discussion

In our study, the MAC determined in 16- to 23-week-old Sprague-Dawley rats was 1.02 and 2.99% for halothane and sevoflurane, respectively. These end-tidal anesthetic concentrations agree favorably with a comparable study with halothane by White et al[3] (1.11 and 0.90% for young vs old) and a study with sevoflurane by Taheri et al[4] (2.8%) in Sprague-Dawley rats of similar age and body weight. Variability, although generally small, from other reported results[5-9] is explained on the basis of differences in animals (eg, breed and age) and study design (eg, use of inspired vs end-tidal and alveolar breath samples).

In a previously reported study,[1] a significant decrease in heart rate and MAP accompanied an increase in end-tidal concentration of isoflurane. Results of our present study indicate little to no significant change in heart rate, but a similar anesthetic dose-related decrease in MAP with halothane and sevoflurane. Further visual comparison of data (Fig 2) suggests that at the deepest level of anesthesia, halothane is the most depressing to MAP (ie, lower anesthetic dose causing similar MAP), compared with isoflurane (highest dose) and sevoflurane (intermediary). Values reported here for MAP are in close agreement to previously published[6,8-10] values from rats anesthetized only with halothane or sevoflurane. However, presently reported values for heart rate are slightly less than some of those observed in the previous reports. The discrepancy in reported heart rate likely relates to younger rats being studied in the pre-

vious report.[6] Present results indicate that a decrease in MAP is a useful guide to increased inhalation anesthetic dose, whereas change in heart rate is of little value in defining anesthetic dose, at least in the range of 1.0 to 1.5 MAC among the 3 inhalation agents.

The magnitude of respiratory frequency of rats anesthetized with halothane (Table 1) was approximately double that observed in rats anesthetized with sevoflurane (Table 2). This difference in response to the 2 anesthetics has also been observed with anesthetized horses.[11] An increase in anesthetic dose causes a decrease in respiratory frequency, although it is notably less dramatic at lighter levels of sevoflurane anesthesia (Fig 4).

In the study reported here, rats in both anesthetic groups (halothane, sevoflurane) had a dose-dependent increase in Pa_{CO_2} and an accompanying decrease in pH_a. The magnitude of hypoventilation, dose-related change in Pa_{CO_2}, and resultant respiratory acidosis (calculated base balance did not change in relation to pH_a) were similar to that seen with rats anesthetized under like conditions in our laboratory with isoflurane (Fig 4).[1] The Pa_{CO_2} measured in rats anesthetized with halothane in the study reported here (with allowances for differences in study conditions) compare favorably with results of the previous study of White et al.[3] However, the magnitude of hypercapnia in the present study in rats anesthetized with sevoflurane was greater (10 to 20 mm Hg, depending on dose) than that reported by Crawford et al[8,9] in rats over the range of 1.0 to 1.5 MAC. There are no obvious reasons for this discrepancy in sevoflurane-associated data other than the absolute doses of sevoflurane studied were less than those in the present investigation.

The Pa_{O_2} was generally > 250 mm Hg in rats receiving anesthetic. However, the Pa_{O_2} was notably less in magnitude and more variable among rats anesthetized with halothane as opposed to those anesthetized with sevoflurane. Review of results from individual rats indicated that most of the reduction in the mean value (and thus the associated larger variability) of Pa_{O_2} during anesthesia could be traced to the first 3 rats anesthetized with halothane. In these rats, there was less regular application of periodic mechanical sighs (intermittent manual rebreathing bag compression to increase tidal volume) to slow or reverse any potential for lung atelectasis. The presence of atelectasis and its progression with time would increase venous admixture and, as a result, reduce Pa_{O_2}.

Anesthesia-induced changes in eye-related behavior are commonly used clinical measures of anesthetic depth. Present results provide evidence in support of this practice. For example, eye aperture and pupil diameter in rats anesthetized with sevoflurane and halothane increased with increasing anesthetic dose. These findings, especially of dose-dependant mydriasis, are similar to findings[1] for rats anesthetized with isoflurane. Unfortunately, pupil diameter is likely to be difficult to consistently evaluate empirically in a clinical setting because of the rats' small pupil size. The corneal and palpebral reflexes are also of limited value, because they are lost early as anesthetic dose is increased beyond light levels. For example, the corneal reflex was abolished in rats anesthetized with sevoflurane (regardless the dose), but was present in 5 of 8 rats anesthetized with halothane at 0.8 MAC (dose not measured with sevoflurane) and in 1 of 8 rats anesthetized with halothane at 1.0 MAC. The corneal reflex was abolished at 1.25 MAC in all rats anesthetized with either agent. In halothane-anesthetized rats, the palpebral reflex was present in all rats at 0.8 MAC, but mostly abolished at 1.0 MAC and completely abolished by 1.25 MAC. A similar finding was observed in sevoflurane-anesthetized rats with abolishment of the palpebral reflex at 1.25 MAC. Nystagmus was not observed in any rats in either anesthetic group. Lacrimation was observed in only 1 rat anesthetized with sevoflurane at 1.0 MAC. Lacrimation was more commonly observed in rats anesthetized with halothane, but the response was inconsistent for individual rats and from 1 rat to another. No changes associating lacrimation with anesthetic depth were observed. From these limited observations, only the corneal and palpebral reflexes appear to be useful in judging anesthetic depth and identifying light from deeper levels of anesthesia.

[a]Grass model 7 polygraph, Grass Instruments Co, Quincy, Mass.
[b]LB2 anesthetic analyzer, Sensormedics Corp, Anaheim, Calif.
[c]LB2 carbon dioxide analyzer, Sensormedics Corp, Anaheim, Calif.
[d]ABL 330 acid-base laboratory, Radiometer, Copenhagen, Denmark.
[e]Clintubes, Radiometer, Copenhagen, Denmark.

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