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Comparison of the Effects of Isoflurane and Desflurane on Cardiovascular Dynamics and Regional Blood Flow in the Chronically Instrumented Dog

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Seven mongrel dogs were chronically instrumented for the measurement of aortic and left ventricular blood pressures, cardiac output, left ventricular wall thickening, left ventricular dP/dt, and circumflex coronary, renal, hepatic and portal blood flows under the influence of desflurane (D) and isoflurane (I). Administration of the two anesthetics, was randomized, as was the order of the concentrations administered. Each dog was studied awake and at 1.2, 1.4, 1.75, and 2.0 MAC of each anesthetic on different days. Both anesthetics decreased mean arterial pressure, stroke volume, systemic vascular resistance, left ventricular dP/dt, and wall thickness. The decreases were dose-dependent for mean arterial pressure (percent of awake values: D 78, I 85 at 1.2 MAC, and D 67, I 69 at 2.0 MAC); stroke volume (D 66, I 72 at 1.2 MAC, and D 52, I 57 at 2.0 MAC); dP/dt (D 61, I 64 at 1.2 MAC, and D 46, I 49 at 2.0 MAC); and WT (D 68, I 70 at 1.2 MAC, and D 47, I 60 at 2.0 MAC). Systemic vascular resistance decreased approximately the same at 1.2 MAC (D 71, I 87%) as at 2.0 MAC (D 71, I 79%). Heart rate increased but also not in a dose-dependent fashion (percent of awake values: D 177, I 145 at 1.2 MAC, and D 176, I 155 at 2.0 MAC). Coronary blood flow was increased by both anesthetics at all concentrations (percent of awake values: I 136 at 1.2 MAC and 161 at 2.0 MAC of awake, and D 131 at 1.2 MAC and 138 at 2.0 MAC). Both anesthetics decreased coronary vascular resistance in a dose-dependent fashion. Hepatic arterial blood flows were maintained by desflurane and slightly increased by isoflurane. Hepatic vascular resistance was decreased only by isoflurane. Portal blood flow was slightly decreased at some anesthetic concentrations by both drugs. The combined total hepatic blood flow (portal plus hepatic arterial) as a result was significantly decreased by desflurane at the two high anesthetic concentrations but not significantly changed by isoflurane. Renal blood flow also was not significantly affected by either anesthetic, and consequently, renal vascular resistance was decreased by both anesthetics. There were no significant differences at any anesthetic concentrations between desflurane and isoflurane with regard to systemic or regional hemodynamics. Thus, the effects of desflurane and isoflurane on systemic, coronary, hepatic and renal hemodynamics were the same. (Key words: Anesthetics, volatile: desflurane; isoflurane. Heart: function; coronary blood flow. Kidney: blood flow. Liver: blood flow.)

FOR THE FIRST TIME in almost 20 yr,[1] a new inhalation anesthetic is undergoing clinical trials in the United States.[2,3] Desflurane is an analog of that previous new anesthetic, isoflurane, with an additional fluorine replacing the chlorine atom on the alpha ether carbon. This substitution has produced an inhalation anesthetic that differs from its parent in its lower blood solubility and decreased biotransformation.[4,5] Paired studies in the same pigs indicated that the cardiovascular effects of desflurane and isoflurane were practically identical.[6] Both drugs produced dose-dependent decreases in arterial blood pressure and stroke volume. At greater than 1 MAC anesthetic dose, cardiac output was decreased and right atrial and pulmonary capillary wedge pressures were increased in a dose-dependent manner. Systemic vascular resistance decreased and heart rate increased maximally at 0.8 MAC and then changed minimally with further increases in anesthetic concentration.

However, no studies of the effect of desflurane on regional blood flow, and especially the coronary circulation, have been published. As two editorials have suggested,[3,7] since isoflurane is a coronary vasodilator with a potential for causing unfavorable redistribution of coronary blood flow in ischemic hearts (coronary steal),[8] the effect of desflurane on the coronary circulation and its comparison with isoflurane needs to be documented. It is interesting that although isoflurane is a coronary vasodilator in dogs as well as humans,[7,9,10] such an effect evidently does not occur in swine or rats.[11,12] Consequently, the dog appears to be a more relevant model for the study of the effects of desflurane on the coronary circulation. Isoflurane appears to preserve blood flow to the liver and kidney in chronically instrumented dogs[13] and swine.[11] Therefore, we also have examined the effect of desflurane and isoflurane on renal and hepatic arterial and portal blood flow in the same animals.

Materials and Methods

A description of our basic chronically instrumented dog model has been published previously.[14–16] This study was approved by both the University of Texas Medical School at Houston and Baylor College of Medicine (Houston, TX) Animal Welfare Committees. Seven dogs were instrumented as follows during tracheal intubation and

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halothane anesthesia. Through a left thoracotomy, 1) a 2.5- or 3.0-mm pulsed Doppler flow probe (Baylor College of Medicine) was placed around the circumflex coronary artery; 2) a miniature high-fidelity transducer (Konigsberg, Pasadena, CA) was inserted through a stab wound into the apex of the left ventricle; 3) a Tygon catheter (Tygon Norton, Akron, OH) was placed in the left atrium; 4) an electromagnetic flow probe (Micron, Los Angeles, CA) was placed around the main pulmonary artery; and 5) an epicardial wall thickness probe (Baylor College of Medicine) was placed on the lateral wall of the left ventricle for measurement of myocardial wall thickening. Through a left flank incision, a 3.0–4.0-mm pulsed Doppler flow probe was placed around the left renal artery, and a Tygon catheter was placed in the abdominal aorta through the iliac artery. Finally, for measurement of liver blood flow, through a midline laparotomy, a 3.0–3.5-mm pulsed Doppler flow probe was placed around the hepatic artery and a 7.0–8.0-mm probe around the portal vein. The gastroduodenal branch of the hepatic artery was ligated to ensure that the blood flow measurements reflected only hepatic perfusion. The intercostal nerves and wounds were infiltrated with 0.5% bupivacaine before closure for postoperative analgesia.

After chest closure, the pneumothorax was evacuated and the chest drain removed before the dogs awakened. The instrumentation wires and catheters were exteriorized into a specially designed pouch which was sewn to the posterior cervical region and protected by a specially designed animal jacket. The animals were nursed carefully through the first 24 h with intravenous fluids and systemic analgesics (meperidine) as necessary. The dogs were carefully trained to lie quietly in the laboratory and were studied at least 10 days after surgery when hematocrit was greater than 30% and only if body temperature, appetite, and general appearance were normal.

Each dog received both desflurane and isoflurane. The animals were randomized to receive either isoflurane or desflurane first. Studies were conducted while dogs were awake and during 1.2, 1.4, 1.75, and 2.0 MAC for the anesthetics in dogs. The MAC of desflurane was taken to be 7.2% end-tidal[17] and the MAC for isoflurane as 1.4% end-tidal. Because of its high boiling point, desflurane was delivered by a modified Ohio DM 5000 anesthesia machine with an electrically heated temperature controlled vaporizer. Inspired and expired concentrations of the anesthetics were monitored continuously from a catheter placed through the endotracheal tube at the carina of the animals with the use of a Datex Multigas analyzer modified to measure desflurane instead of halothane. The monitor was calibrated with known standards before and during the experiments. The animals were anesthetized with the test anesthetic by a mask with the aid of N_2O; the trachea of each animal was intubated; and with an Ohio volume-controlled ventilator, the lungs of each animal were ventilated with a mixture of O_2, N_2, and the appropriate anesthetic agent.

Ventilation and the proportion of O_2 in the inspired mixture were adjusted to maintain measured inspired O_2 and end-tidal CO_2 concentrations near the awake level with the Multigas analyzer. In addition, blood gases were intermittently measured to ensure that ventilation and oxygenation were similar to those in the awake animal. Lactated Ringer's solution (3–5 $ml \cdot kg^{-1} \cdot h^{-1}$) was infused *via* a foreleg vein. Rectal temperature measured with a thermocouple probe (Yellow Springs Instruments, Yellow Springs, OH) was maintained at awake levels throughout the experiment. No measurements were made until at least 15 min of constant end-tidal concentration at each anesthetic level had been maintained. The order of anesthetic concentrations were randomized in each animal.

With the use of a Gould polygraph (Gould, Cleveland, OH), hemodynamic variables were recorded continuously at 25 $mm \cdot min^{-1}$ during the entire experiment except for times of data collection (25 $mm \cdot s^{-1}$) and for the left ventricular dP/dt calibration (200 $mm \cdot s^{-1}$). Measurements (heart rate, aortic blood pressure, cardiac output, left ventricular dP/dt, coronary, hepatic, portal, and renal blood flow, and arterial blood gases) were obtained prior to anesthesia and during a brief apneic period 15 min after obtaining a constant end-tidal anesthetic concentration. In addition to the recorded variables, we calculated the systemic and regional vascular resistance as the ratio between mean arterial pressure and cardiac output or regional blood flow. The pulsed Doppler flows were calibrated in terms of Doppler frequency shift.[18] Mean values are presented. Baseline zero and the linear relationship between volume flow and frequency shift have been established *in vivo*.[19]

In order to compare the observations between the two anesthetics in the same dog, a two-way analysis of variance (ANOVA) for repeated measure design (anesthetic × MAC) was used. For the comparison of changes related to the different concentrations of each anesthetic, a one-way ANOVA was used followed by Dunnett's *t* test when the one-way ANOVA indicated statistical significance. In addition, in order to determine whether or not the changes produced by the anesthetics were dose-dependent, data were fitted to a linear model related to the MAC level. Alpha was set up at a level of 0.05. Data are presented as mean ± SD.[20]

Results

Ventilation and Oxygenation

Although arterial CO_2 tension (Pa_{CO_2}) was significantly lower and *p*Ha and arterial O_2 tension (Pa_{O_2}) significantly

TABLE 1. Arterial Blood Gases at Various Anesthetic Concentrations

		MAC			
	Awake	1.2	1.4	1.75	2.0
pH_a					
D	7.36 ± 0.03	7.39 ± 0.03*	7.41 ± 0.04*	7.40 ± 0.03*	7.40 ± 0.04*
I	7.39 ± 0.02	7.38 ± 0.01	7.37 ± 0.03	7.36 ± 0.02*	7.37 ± 0.02
Pa_{CO_2}					
D	34 ± 7	27 ± 4*	29 ± 4*	27 ± 3*	26 ± 4*
I	31 ± 3	30 ± 4	29 ± 2	31 ± 4	30 ± 3
Pa_{O_2}					
D	110 ± 18	128 ± 15*	131 ± 13*	130 ± 16*	126 ± 12
I	109 ± 17	126 ± 19	127 ± 24*	120 ± 18	124 ± 25
ET %					
D	0	8.4 ± 0.1*	10.3 ± 0.3*	12.7 ± 0.1*	14.2 ± 0.1*
I	0	1.6 ± 0.02*	1.8 ± 0.06*	2.3 ± 0.1*	2.6 ± 0.1*

Means ± SD.
D = desflurane, I = isoflurane.
ET % = percent in end-tidal gas. *$P < 0.05$ *versus* awake.

higher during desflurane than during awake periods, the magnitude of the change was small and not significantly different from the values during equipotent concentrations of isoflurane in the same animals (table 1).

Systemic Hemodynamics

At no time, awake or anesthetized, were there any significant differences between systemic hemodynamics in the animals anesthetized with desflurane or isoflurane (table 2). Both anesthetics produced a dose-related decrease in arterial blood pressure (systolic, diastolic, and mean). Heart rate was increased by both anesthetics without a dose dependency. Although the mean heart rate was greater at all anesthetic concentrations with desflurane, the differences were not statistically significant because of the variability (standard deviation [SD] ± 30 with isoflurane). Cardiac output was not significantly changed by desflurane, whereas cardiac output was reduced at the two highest concentrations of isoflurane. Again, there was no significant difference between cardiac output at any concentration of desflurane *versus* isoflurane. However, both anesthetics produced a dose-related decrease in stroke volume. Systemic vascular resistance was reduced significantly at 1.2 MAC desflurane but did not change with further increases in anesthetic concentration. The decrease in systemic vascular resistance produced by isoflurane was not significantly different from awake until 1.4 MAC. The contractile performance of the heart as indicated by both wall thickening and left ventricular dP/dt was progressively decreased by increasing concentrations of both anesthetics.

Regional Hemodynamics

Coronary blood flow was increased and coronary vascular resistance was decreased by both anesthetics, although the blood flow effect was not significant with desflurane until 1.4 MAC (table 2). The blood flow effect was not dose-dependent, whereas the decrease in coronary vascular resistance was dose-related (because of the dose-related decrease in arterial blood pressure). Again, there were no significant differences between desflurane and isoflurane at any anethetic dose.

Although at any given MAC there were no statistically significant differences between the effects of desflurane and isoflurane on hepatic and portal blood flow, there were some differences as far as the effect compared to control was concerned (table 3). Isoflurane produced a slight increase in hepatic arterial blood flow at MAC multiples of 1.4 and above, whereas there was no change at any anesthetic concentration produced by desflurane. As a consequence, calculated hepatic arterial vascular resistance was significantly decreased by the higher concentrations of isoflurane but not statistically different from awake with the higher concentrations of desflurane. In contrast, portal blood flow was significantly reduced at 1.75 MAC desflurane and 1.2 MAC isoflurane. Therefore, total hepatic blood flow (the sum of portal and hepatic arterial blood flow) was significantly decreased by the two higher concentrations of desflurane but was not changed by isoflurane.

Neither anesthetic significantly affected renal blood flow. Renal vascular resistance was significantly decreased by isoflurane doses greater than 1.4 MAC, whereas a statistically significant effect was produced by desflurane at 1.7 and 2.0 MAC. However, as with all the other effects, there were no significant differences between desflurane and isoflurane at any MAC multiple.

Discussion

As might have been suspected by the striking similarity between the systemic cardiovascular effects of desflurane

TABLE 2. Systemic and Coronary Hemodyamics

	n	Awake	MAC 1.2	MAC 1.4	MAC 1.75	MAC 2
SAP (mmHg)						
D†	7	138 ± 13	107 ± 13*	103 ± 17*	98 ± 15*	89 ± 20*
I†	7	135 ± 18	112 ± 17*	105 ± 22*	100 ± 20*	95 ± 22*
DAP (mmHg)						
D†	7	67 ± 6	57 ± 10	54 ± 15*	50 ± 9*	48 ± 15*
I†	7	71 ± 11	61 ± 9	57 ± 8*	54 ± 11*	50 ± 14*
MAP (mmHg)						
D†	7	93 ± 8	73 ± 12*	71 ± 14*	67 ± 12*	62 ± 16*
I†	7	93 ± 12	79 ± 11*	72 ± 8*	69 ± 9*	64 ± 17*
HR (beats per min)						
D	7	79 ± 14	140 ± 13*	138 ± 23*	143 ± 16*	139 ± 18*
I	7	82 ± 14	119 ± 30*	123 ± 33*	127 ± 23*	127 ± 23*
CO (l/min)						
D	6	2.25 ± 0.4	2.53 ± 0.5	2.30 ± 0.3	2.35 ± 0.4	1.98 ± 0.3
I	6	2.10 ± 0.1	2.03 ± 0.2	2.00 ± 0.3	1.88 ± 0.2*	1.78 ± 0.3*
SV (ml)						
D†	6	27.6 ± 0.3	18.3 ± 0.4*	16.5 ± 0.4*	16.6 ± 0.4*	14.4 ± 0.03*
I†	6	24.7 ± 0.3	17.7 ± 0.6*	16.7 ± 0.4*	15.1 ± 0.4*	14.1 ± 0.4*
SVR ($mmHg \cdot l^{-1} \cdot min^{-1}$)						
D	6	43.1 ± 10.05	30.5 ± 11.4*	30.7 ± 10.9*	29.4 ± 11.9*	30.7 ± 11.9*
I	6	45.1 ± 7.5	39.3 ± 5.3	36.0 ± 4.0*	35.6 ± 3.9*	35.7 ± 8.6*
dP/dt (mmHg/s)						
D†	5	2856 ± 340	1755 ± 490*	1671 ± 356*	1508 ± 283*	1326 ± 378*
I†	5	2820 ± 458	1807 ± 475*	1855 ± 562*	1504 ± 421*	1384 ± 445*
WT (%)						
D†	5	25.8 ± 6	17.5 ± 2*	12.6 ± 4*	12.6 ± 3*	12.2 ± 3*
I†	5	25.8 ± 10	18.0 ± 5*	17.6 ± 8*	16.3 ± 9*	15.6 ± 7*
CBF (ml/min)						
D	7	42 ± 8	55 ± 7	57 ± 10*	58 ± 14*	58 ± 21
I	7	41 ± 12	56 ± 22*	58 ± 23*	61 ± 25*	66 ± 22*
CR ($mmHg \cdot ml^{-1} \cdot min^{-1}$)						
D†	7	2.29 ± 0.42	1.35 ± 0.21*	1.27 ± 0.29*	1.21 ± 0.30*	1.18 ± 0.45*
I†	7	2.39 ± 0.5	1.54 ± 0.5*	1.38 ± 0.5*	1.30 ± 0.6*	1.06 ± 0.4*

Means ± SD.

SAP = systolic arterial pressure; DAP = diastolic arterial pressure; MAP = mean arterial pressure; HR = heart rate. CO = cardiac output; SV = stroke volume; SVR = systemic vascular resistance; dP/dt = left ventricular dP/dt; WT = left ventricular wall thickening; CBF = coronary blood flow; CR = coronary vascular resistance.

* $P < 0.05$ *versus* awake.

† Significant correlation with MAC multiples.

and isoflurane in swine,[6] we likewise found no difference between the general cardiovascular effects of the two anesthetics in chronically instrumented dogs. In addition, measuring coronary blood flow simultaneously with pulsed Doppler flow probes, we have shown that both anesthetics produce an increase in coronary blood flow that appears to be nearly maximal at the low anesthetic concentrations (1.2 MAC). This plateau effect at the higher concentration is probably related to the dose-dependent decrease in mean arterial pressure (coronary perfusion pressure) that counters the decrease in coronary vascular resistance seen with both anesthetics.

From a pharmacologist's viewpoint, then, both desflurane and isoflurane are coronary vasodilators of about equal potency. Physiologically, this coronary vasodilation should serve to maintain myocardial oxygen supply equal to demand with no change in coronary arteriovenous extraction (or coronary venous oxygen content).[21] Therefore, if a drug is a true coronary vasodilator, this relationship should be disturbed, and there should there be a decrease not only in calculated coronary vascular resistance, but also in arteriovenous oxygen extraction. This has certainly been shown for isoflurane in both dogs[7] and humans.[10] However, our preparation did not allow for the sampling of coronary venous blood, and hence we could not document this effect. Further experiments with measurement of arterial and coronary venous oxygen contents are necessary to completely characterize the coronary vascular effect of desflurane. However, given the results in the literature for isoflurane and the similarity of the effects of desflurane as reported in the current publication, we believe that the coronary vascular effects of desflurane are essentially the same as those of isoflurane.

The effects of isoflurane on the coronary circulation appear to be species-related. Gelman *et al.* have shown essentially the same effects of isoflurane in their dog model, although they used radiolabeled microspheres for

TABLE 3. Hepatic and Renal Hemodyamics

	n	Awake	MAC 1.2	MAC 1.4	MAC 1.75	MAC 2
HBF (ml/min)						
D	6	149 ± 68	154 ± 77	164 ± 69	157 ± 67	155 ± 77
I	6	144 ± 47	167 ± 45	195 ± 37*	185 ± 59*	192 ± 62*
HRes (mmHg · ml^{-1} · min^{-1})						
D	6	0.80 ± 0.5	0.61 ± 0.4	0.50 ± 0.2	0.49 ± 0.3	0.53 ± 0.4
I	6	0.71 ± 0.3	0.51 ± 0.2	0.39 ± 0.1*	0.40 ± 0.2*	0.38 ± 0.2*
PBF (ml/min)						
D	6	475 ± 134	421 ± 59	424 ± 65	357 ± 108*	385 ± 78
I	6	481 ± 145	387 ± 81*	422 ± 75	425 ± 115	399 ± 101
Total HBF (ml/min)						
D	6	624 ± 85	587 ± 77	603 ± 64	514 ± 111*	527 ± 103*
I	6	626 ± 189	553 ± 108	617 ± 110	614 ± 162	591 ± 128
RBF (ml/min)						
D	6	117 ± 30	134 ± 41	126 ± 38	122 ± 30	117 ± 36
I	6	122 ± 36	116 ± 47	131 ± 59	131 ± 63	125 ± 43
RRes (mmHg · ml^{-1} · min^{-1})						
D	7	0.86 ± 0.3	0.62 ± 0.2	0.61 ± 0.3	0.57 ± 0.2*	0.58 ± 0.3*
I	7	0.85 ± 0.2	0.81 ± 0.2	0.61 ± 0.2*	0.62 ± 0.2*	0.54 ± 0.2*

Means ± SD.

HBF = hepatic arterial blood flow; HRes = hepatic arterial vascular resistance; PBF = portal blood flow; Total HBF = total hepatic blood flow; RBF = renal arterial blood flow; RRes = renal arterial vascular resistance.

* $P < 0.05$ *versus* awake.

the blood flow measurements, in contrast to our Doppler ultrasound technique.[13] Seyde *et al.* in the rat[12] and Lundeen *et al.* in the pig[11] also used radiolabeled microspheres. However, in neither the rat nor the pig was there significant coronary vasodilation at 1 MAC isoflurane anesthesia. Unfortunately, there was no dose response studied in the rat. Isoflurane at 1.5 MAC in the pig, however, produced a major decrease in coronary blood flow and consequently little change in coronary vascular resistance. Several studies in humans have indicated that isoflurane does produce significant coronary vasodilation, although all studies have been in patients with documented coronary artery disease, and hence concentrations of isoflurane greater than approximately 1 MAC have not been studied.[10,22,23]

In like manner, the other new inhalation anesthetic, sevoflurane, appears to have a different effect on the coronary circulation in pigs.[24] In our chronically instrumented dogs, sevoflurane was practically indistinguishable from isoflurane, producing a significant increase in coronary blood flow and a decrease in coronary vascular resistance.[16] In chronically instrumented swine, 1 and 1.5 MAC sevoflurane decreased myocardial blood flow by 24 and 33%, respectively, from awake controls, a result that correlated with a similar decrease in the rate–pressure product.

Although desflurane did produce slight but significant decreases in total hepatic blood flow at 1.75 MAC as a consequence of a similar decrease in portal blood flow, there was no significant difference between hepatic, portal, or total hepatic blood flow at any of the MAC concentrations of desflurane and isoflurane. This is particularly significant since each anesthetic was studied in the same dogs on different days. In addition, this is the first investigation in which coronary, systemic, hepatic and renal hemodynamics have been studied simultaneously in a chronically instrumented conscious dog. In a similar investigation in our laboratory, with some of the same animals, although enflurane resulted in greater systemic cardiovascular depression (mean arterial pressure and cardiac output) compared to isoflurane, effects on the hepatic circulation were qualitatively similar to those seen in this investigation with desflurane.[25] However, 2 MAC enflurane significantly decreased hepatic arterial blood flow in contrast to desflurane, and lower anesthetic doses (1.2 MAC) of enflurane decreased portal and total hepatic blood flow.[25] As in the current study, there was no effect of isoflurane on hepatic arterial, portal, or total hepatic blood flow. In their chronically instrumented dog model, using radiolabeled microspheres to measure hepatic circulation, Gelman *et al.* also reported that isoflurane preserved total hepatic blood flow.[13] However, this was related to a major increase in hepatic arterial blood flow and equal decreases in the "preportal" blood flow. The effects of halothane and isoflurane on preportal blood flow were essentially the same, but halothane also produced a dose-related decrease in hepatic arterial blood flow and hence a huge decrement in total hepatic flow. Thus, it appears that the fluorinated methyl ethyl ethers—enflurane, desflurane and isoflurane—all are splanchnic vasodilators, with isoflurane the most potent and enflurane the least.

Although previous studies in basally anesthetized acute animal preparations suggested that inhalation anesthetics decreased renal blood flow,[26] our studies investigating halothane,[14] isoflurane, enflurane,[15] sevoflurane (unpublished data), and desflurane in the chronically instrumented dog with the Doppler ultrasound technique have indicated that all five anesthetics are renal vasodilators and preserve renal blood flow even at high anesthetic concentrations. Gelman *et al.* showed the same effects for both halothane and isoflurane in chronically instrumented dogs with radiolabeled microspheres.[13] Lundeen *et al.*[11] Manohar and Parks,[24] and Jarnberg *et al.*[27] also found maintenance of renal blood flow and renal vasodilation during anesthesia with isoflurane, sevoflurane and enflurane, respectively. Finally, although Seyde *et al.* reported that renal blood flow was well maintained with isoflurane and halothane anesthesia in the rat, enflurane did produce significant decrease in renal blood flow at 1 MAC anesthesia.[12] However, in the latter experiments, the animals were studied shortly after recovering from an ether anesthetic and breathed spontaneously during the experiments. In the large animal experiments, instrumentation was accomplished many days prior to experimentation and all animals were ventilated to normal CO_2 and O_2 levels. Consequently, it seems fair to conclude that in properly studied animals, inhalation anesthetics have minimal effect on renal blood flow. Whether or not it is correct to say that "autoregulation" of the renal circulation is maintained during inhalation anesthesia is somewhat controversial, inasmuch as renal blood flow and kidney function are directed towards water and salt homeostasis rather than responding to general cardiovascular dynamics.

In summary, the new inhalation anesthetic desflurane appears to produce essentially the same effects on the systemic, coronary, hepatic, and renal circulation in dogs as those produced by isoflurane. Although dose-related decreases in mean arterial pressure result from desflurane administration, cardiac output and the above-mentioned regional blood flows are well preserved, even at very high concentrations. Previous studies with the currently available inhalation anesthetics (halothane, enflurane and isoflurane) in chronically instrumented dogs have shown effects similar to those seen in humans.[28–31] Consequently, it is likely that the clinical cardiovascular effects of desflurane are similar to those of isoflurane.

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