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Effect of Halothane on Renal Hemodynamics during Normovolemia and Acute Hemorrhagic Hypovolemia

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The effects of halothane on renal hemodynamics under both normovolemic and hypovolemic conditions were investigated in chronically instrumented conscious dogs whose homeostatic mechanisms were not altered by the presence of preexisting drugs. Renal blood flow and aortic pressure were assessed by prior implantation of Doppler ultrasonic flow probes on the renal artery and a catheter in the descending aorta. Administration of halothane to conscious normovolemic dogs (Group HN) resulted in 11–26% decreases in renal vascular resistance with no significant changes occurring in renal blood flow. In a second group of animals made hypovolemic while awake (Group AH), 30% of the blood volume was removed over one-half hour. In response to hemorrhage, these conscious animals' renal blood flow did not significantly change from the normovolemic control, and renal vascular resistance significantly decreased. With no further intervention, renal vascular resistance and renal blood flow remained unchanged from the level achieved after the 30% hemorrhage. A third group of animals (Group HH) was hemorrhaged in a manner similar to that of Group AH. They also showed no significant changes in renal blood flow and a significant decrease in renal vascular resistance in response to hemorrhage. Thereafter, administration of halothane, as in Group HN, to this group produced 11–23% decreases in renal vascular resistance with no significant decline in renal blood flow from the hypovolemic control levels established after hemorrhage. The author concludes the following: 1) administration of halothane to normovolemic conscious dogs does not decrease renal blood flow; 2) a moderate degree of acute hemorrhagic hypovolemia does not decrease renal blood flow in conscious dogs; and 3) administration of halothane to acutely hypovolemic conscious dogs does not impair renal perfusion. (Key words: Anesthetics, volatile: halothane. Hemodynamics: hypovolemia. Kidney: blood flow.)

MANY INVESTIGATIONS have focused on the effects of halothane on renal perfusion and function.[1–15] These represent a variety of species, protocols, and blood flow measuring techniques. Subsequently, conclusions about halothane's effect on renal perfusion are difficult to draw. Renal blood flow has been shown to decrease,[1–3,7,12,15] increase,[4] or remain unchanged[5,6,8,11,13–15] after halothane. Additionally, the question relating to the impact of halothane on renal hemodynamics when administered during a state of acute hypovolemia has not been studied.

The present investigation was directed toward examination of the impact of halothane on renal hemodynamics in both normovolemic and acute hypovolemic situations. A chronically instrumented conscious dog preparation was used in an effort to circumvent the problems related to disturbances of cardiovascular homeostasis by background anesthetics.[16–18] The flow probe technique was chosen to enable an instantaneous and continuous visualization of renal blood flow changes.

Methods

Healthy mongrel dogs of either sex weighing 20–30 kg were used for this study. Animals underwent a laparotomy under halothane, nitrous oxide–oxygen anesthesia. A 6-mm-diameter Doppler ultrasonic flow probe (L & M Electronics, Daly City, California) was secured loosely around the left renal artery and its connective tissue without any attempt being made to clean the vessel. A small Tygon® catheter was positioned in the lower abdominal aorta via cannulation of a lumbar arterial side branch. This catheter was filled with 1,000 units/ml heparin solution. The probe wires and catheter were exteriorized through a flank wound and run subcutaneously to exit the skin between the scapulae. The abdominal incision was closed, and the animals were allowed to recover for 2–3 weeks before we proceeded with experiments. During this time the arterial catheter was cleared three times weekly and filled with fresh heparin solution, and the animals were trained to lie quietly on the experiment table on their sides. This amount of time was adequate for the tissue between the blood vessel and the flow probe to grow in, under and around the probe. This established electrical continuity and stabilized the probe's position on the vessel. Aortic pressure was measured, in mmHg, with a Statham P23ID® strain gauge manometer (Statham Instruments Inc., Oxnard, California) and the flow probe was connected, by a hardwire system, to an L & M model 1012 Doppler ultrasonic flowmeter (L & M Electronics, Daly City, California). This system has an accurate electronic zero for *in situ* use, and its calibration for volume

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flow has been previously described.[19] However, each probe used was additionally tested in the following manner. Before implant it was examined for acoustic quality in normal saline. After each use, probes were calibrated *in vitro* on a section of canine femoral artery. Known flow, measured with a graduated cylinder and stopwatch, was supplied by a roller pump. This was compared with flow values measured by the probes and a calibration curve established for each probe. Included in this series of measurements was a zero flow point. During experiments, both the pulsatile and mean renal artery blood flow and aortic pressure were recorded on a direct writing oscillograph (Gould-Brush, Cleveland, Ohio). Renal vascular resistance was calculated as the quotient of mean arterial pressure over mean renal blood flow.

Three groups of animals were studied. In Group HN (halothane–normovolemia), control values for heart rate, aortic pressure, renal blood flow, and renal vascular resistance were obtained while the animals breathed oxygen through a conical-shaped canine anesthesia mask and a semiclosed circle absorption system. The duration of observation for controls was adequate to assure a steady state baseline had been attained. This was usually 10–15 min. Then halothane was administered in such a way as to simulate a primary induction in humans. The animals breathed spontaneously a mixture of nitrous oxide–oxygen in a ratio of 3–2 with up to 4% inspired halothane during induction (first 3 min). One to 1.5% inspired halothane in oxygen was used for maintenance (from 3–30 min). The same halothane vaporizer was used for all the experiments in this study. Its calibration was checked periodically with a Perkin-Elmer mass spectrometer. It consistently proved to be accurate and reliable. Variables were measured for a 30-min period from the beginning of induction. In Group AH (awake–hypovolemic), with the aid of local anesthesia with 1% lidocaine, a 24″ 16-gauge catheter was positioned in each animal's inferior vena cava via percutaneous entry through the saphenous vein. Awake controls were taken as in Group HN. Then the animals were hemorrhaged 30% of their estimated blood volume (EBV calculated as 10% of the body weight) over a 30-min period—that is, approximately 10% being removed each 10 min. After removal of 30% of the estimated blood volume, adequate time was again allowed for hemodynamic equilibration, usually 10–15 min. Then an additional set of control data was taken, which subsequently served as the hypovolemic controls. Hemodynamics were measured subsequently for a 30-min period in the conscious state, during which time no further interventions were made. At the end of that interval all shed blood, which had been heparinized in a beaker with 2 units of heparin/ml was reinfused over 10 min and postreinfusion measurements were taken. In Group HH (halothane–hypovolemic), normovolemic controls were measured as in the previous two groups. The animals were hemorrhaged as in Group AH, after which hypovolemic controls were taken again. Halothane then was administered as in Group HN, and renal hemodynamic data were measured for a 30-min period while the animals breathed spontaneously. Also, as in Group AH, at the end of the 30-min period shed blood was reinfused over a 10-min interval and measurements repeated postreinfusion. During the reinfusion period, the dogs in Group HH continued to breathe the maintenance concentration of halothane.

In this study, the animals in Groups HN and HH were allowed to breathe spontaneously so as to avoid the potential effects of positive-pressure ventilation on cardiac output as well as on renal hemodynamics themselves.[20] We considered the possibility of effects of halothane on ventilation and the subsequent impact of changes in Pa_{CO_2} on our results. This will be addressed below.

In all three groups, each animal served as its own control. In Group HN the changes in renal hemodynamics in response to halothane were compared with the awake normovolemic controls. In Groups AH and HH, the changes during the 30-min observation period were compared with the hypovolemic controls obtained after completion of the 30% hemorrhage. The renal hemodynamic changes after reinfusion of shed blood in Groups AH and HH were compared back to their original awake normovolemic controls, since, at that point, normovolemia had been restored. A P value of <0.05 was considered to be statistically significant. An F test was first applied to check for significance between groups. Subsequently, the Fisher's modified least-squares test was employed to examine differences between data at a particular time point.

Results

The renal hemodynamic and heart rate data are summarized in table 1. During the awake, normovolemic control period, there were no statistically significant differences noted between the groups for aortic pressure, renal blood flow, renal vascular resistance, or heart rate. The mean heart rates ± SEMs during this control period were 82 ± 5 (HN), 80 ± 4 (AH), and 78 ± 6 (HH) beats per min for the three groups. This variable is the best indicator of the animals' calm steady state condition before drug or hemorrhage interventions.

In the halothane–normovolemic group (HN) (table 1, fig. 1), administration of halothane resulted in a decrease in arterial pressure of 2–28% from the control value of 110 ± 3 mmHg. These changes were statistically significant from 15 min on. Renal blood flow did not change significantly from the control value of 144 ± 11 $ml \cdot min^{-1}$ during the 30-min observation period. Subsequently, calculated vascular resistance was decreased 11–26% from a control value of 0.8 ± 0.05 $mmHg \cdot ml^{-1}$ min and was

significantly decreased from 15 min on. Heartrates were significantly elevated from 1 to 30 min.

In the awake–hypovolemic group (AH) (table 1, fig. 2), arterial pressure decreased significantly from the awake normovolemic control of 108 ± 4 mmHg to 88 ± 5 mmHg after hemorrhage. Renal blood flow and heart rate in this group did not change significantly after hemorrhage. Renal vascular resistance was significantly decreased from 0.78 ± 0.07 to 0.59 ± 0.06 mmHg · ml^{-1} · min after 30% hemorrhage. Thereafter, during the 30-min observation period, aortic pressure, renal blood flow, renal vascular resistance, and heart rate did not significantly change from the awake hypovolemic control values established after 30% hemorrhage. Upon reinfusion of the shed blood in this group, aortic pressure, renal blood flow, renal vascular resistance, and heart rate all returned to levels that were comparable to the normovolemic control values.

In the hypovolemic group, where halothane was administered after hemorrhage (HH) (table 1, fig. 3), arterial pressure decreased significantly from 111 ± 4 to 91 ± 4 mmHg in response to the 30% hemorrhage. Renal blood flow and heart rate did not change significantly. Renal vascular resistance was significantly decreased from 0.75 ± 0.06 to 0.6 ± 0.06 mmHg · ml^{-1} · min in response to 30% hemorrhage. Administration of halothane after hemorrhage resulted in a 14–32% decrease in arterial pressure that was significant from 7 min on. Renal blood flow was not significantly changed. Renal vascular resistance was decreased from 11 to 23%, these changes being significant at 15–30 min. Heart rate was significantly increased at 1–7 min. After reinfusion of the shed blood, arterial pressure was significantly lower than the awake normovolemic control value. However, renal blood flow and renal vascular resistance were not significantly altered compared with the awake normovolemic controls.

Comparing between groups, the following are relevant. The significant within-group decreases in aortic pressure during halothane administration occurred earlier in Group HH than in Group HN. However, the quantitative changes in aortic pressure as well as those for renal blood flow, renal vascular resistance and heart rate were not significantly different between these two groups. In comparing Groups AH and HH, there were no significant differences for aortic pressure, renal blood flow, or renal vascular resistance during the awake hypovolemic portion of the study. In Group HH, administration of halothane resulted in significant differences in aortic pressure between Group AH and HH at 7–30 min. Renal vascular resistance was significantly lower at 15–30 min and heart rate significantly higher at 1–7 min. There were no significant differences in renal blood flow between these two groups for the entire hypovolemic period. After reinfusion, the aortic pressure in Group HH was significantly less than in Group AH, but there were no significant differences between Groups HN and HH for any of these variables.

TABLE 1. Renal Hemodynamics with Halothane, Hemorrhage, and Hemorrhage plus Halothane

Variable	Mean Actual Values ± SEM		Mean Per Cent Changes ± SEM from Respective Controls							Mean Actual Values ± SEM After Reinfusion
	Awake Normovolemic Controls	Awake Hypovolemic Controls after Hemorrhage	1 Min	3 Min	5 Min	7 Min	10 Min	15 Min	20 Min	30 Min	
$\overline{AoP}$ (mmHg)	HN 110 ± 3	—	10 ± 6	−2 ± 6	−10 ± 7	−11 ± 7	−15 ± 5	−22 ± 3*	−27 ± 2*	−28 ± 2*	—
	AH 108 ± 4	88 ± 5*	3 ± 3	2 ± 4	1 ± 4	0 ± 3	−1 ± 3	−2 ± 3	−5 ± 4	0 ± 5	113 ± 2
	HH 111 ± 4	91 ± 4*	10 ± 6	3 ± 14	−14 ± 13	−20 ± 9*†	−24 ± 9*†	−30 ± 6*†	−32 ± 6*†	−30 ± 6*†	97 ± 5*†
$\overline{RBF}$ (ml · min^{-1})	HN 144 ± 11	—	5 ± 5	0 ± 4	4 ± 5	6 ± 6	6 ± 8	6 ± 7	1 ± 7	0 ± 8	—
	AH 149 ± 14	162 ± 17	1 ± 1	−1 ± 2	1 ± 2	−2 ± 2	−4 ± 3	−4 ± 3	−5 ± 3	−3 ± 4	147 ± 17
	HH 151 ± 9	158 ± 10	8 ± 3	−4 ± 7	−4 ± 7	−5 ± 6	−6 ± 8	−4 ± 8	−9 ± 8	−10 ± 7	171 ± 16
$\overline{RVR}$ (mmHg · ml^{-1} · min)	HN 0.80 ± 0.05	—	6 ± 7	0 ± 7	−11 ± 8	−14 ± 7	−17 ± 6	−24 ± 4*	−26 ± 4*	−25 ± 4*	—
	AH 0.78 ± 0.07	0.59 ± 0.06*	3 ± 2	2 ± 3	0 ± 3	1 ± 2	2 ± 2	2 ± 3	−1 ± 4	2 ± 4	0.85 ± 0.10
	HH 0.75 ± 0.06	0.60 ± 0.06*	2 ± 5	11 ± 16	−11 ± 12	−16 ± 8	−17 ± 8	−23 ± 8*†	−20 ± 9*†	−19 ± 8*†	0.62 ± 0.09
$\overline{HR}$ (beats · min^{-1})	HN 82 ± 5	—	47 ± 12*	68 ± 10*	60 ± 9*	57 ± 10*	48 ± 10*	44 ± 11*	41 ± 13*	34 ± 13*	—
	AH 80 ± 4	96 ± 9	0 ± 4	−1 ± 4	−1 ± 5	0 ± 5	−1 ± 7	1 ± 5	−3 ± 7	−6 ± 6	88 ± 6
	HH 78 ± 6	92 ± 6	36 ± 12*†	37 ± 12*†	33 ± 13*†	33 ± 14*†	23 ± 10	19 ± 8	17 ± 10	15 ± 9	93 ± 2

$\overline{AoP}$ = mean aortic pressure; $\overline{RBF}$ = mean renal blood flow; $\overline{RVR}$ = mean calculated renal vascular resistance; HR = mean heart rate; HN = halothane normovolemic group (n = 12); AH = awake hemorrhage group (n = 9); HH = hemorrhage plus halothane group (n = 8). In Group HN, the per cent changes are from the awake normovolemic controls. In Groups AH and HH the per cent changes are from the awake hypovolemic controls after 30% hemorrhage. Also, in Groups AH and HH, the actual values after reinfusion were compared back with the awake normvolemic controls.

* $P < 0.05$ from the respective within group controls.

† $P < 0.05$ for Group AH *versus* Group HH.

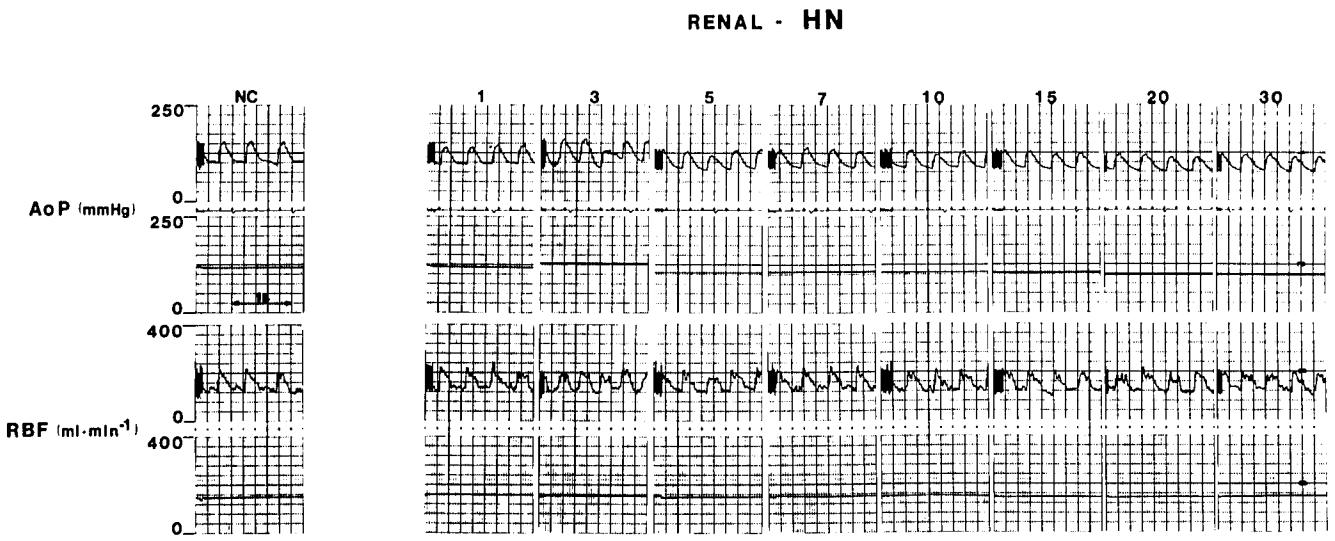

FIG. 1. Recordings are shown of phasic and mean arterial pressures (AoP) and renal blood flows (RBF) in conscious, normovolemic animals given halothane (Group HN). Data were recorded at control while awake (NC) and for 30 min after halothane administration. Paper speed was 25 mm/s for fast wave forms. Note that RBF remained at control despite a decline in AoP with halothane.

ferences in renal blood flow, vascular resistance, or heart rate at that time.

Discussion

Because of the lack of toxicity to the kidney, halothane has been considered an excellent anesthetic choice for situations involving compromised renal function. Almost paradoxically, however, this drug has traditionally been considered to decrease renal perfusion.[1-3,7,12,15] More recent studies disagree with those findings and have shown that halothane does not decrease renal blood flow.[5,6,8,11,13-15] The reason for this discrepancy probably relates to two factors. Older data were obtained with methods for which the accuracy is questionable, *i.e.*, PAH extraction techniques. Also, much of these data were obtained in animals previously anesthetized with other drugs. Thus, the true impact of halothane itself probably was misassessed.

Another misunderstood aspect of renal blood flow, for the same reasons cited above, relates to how it is altered during hypovolemia. Traditional thinking is that renal blood flow decreases with hemorrhage. This assumption has been based on the fact that during hemorrhage, urine output decreases or actually ceases. One study of humans attempted to measure renal blood flow during hemor-

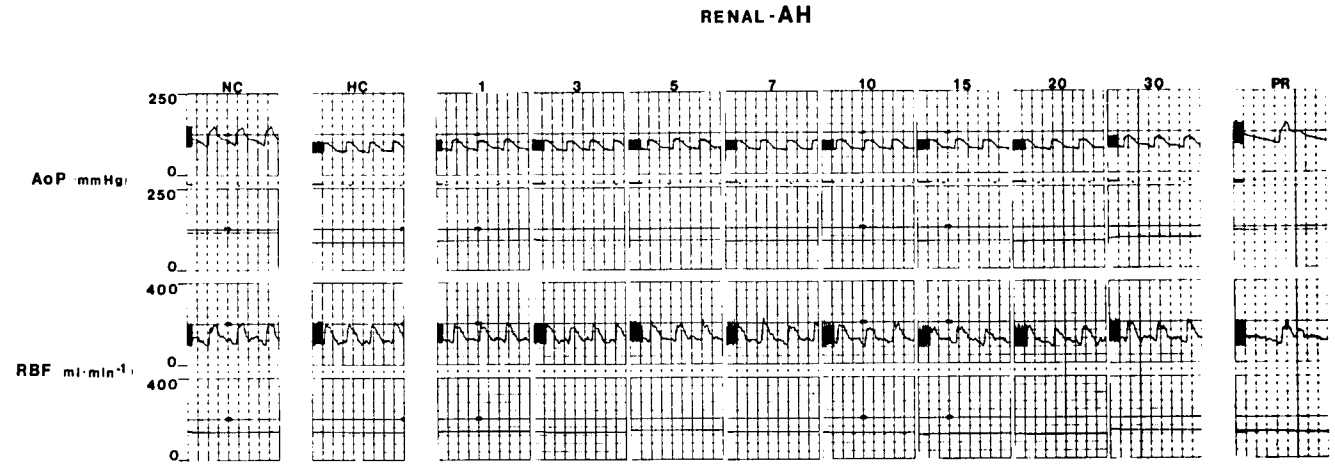

FIG. 2. Recordings are shown of phasic and mean arterial pressures (AoP) and renal blood flows (RBF) first in normovolemic conscious animals (NC), then after hemorrhage to establish a conscious hypovolemic control (HC). Data were recorded thereafter for 30 min, with the animals remaining awake but hypovolemic (Group AH). Shed blood was reinfused and the conscious post reinfusion (PR) values noted. Paper speed was 25 mm/s for fast wave forms. Note that with this degree of hemorrhage RBF remains at control levels.

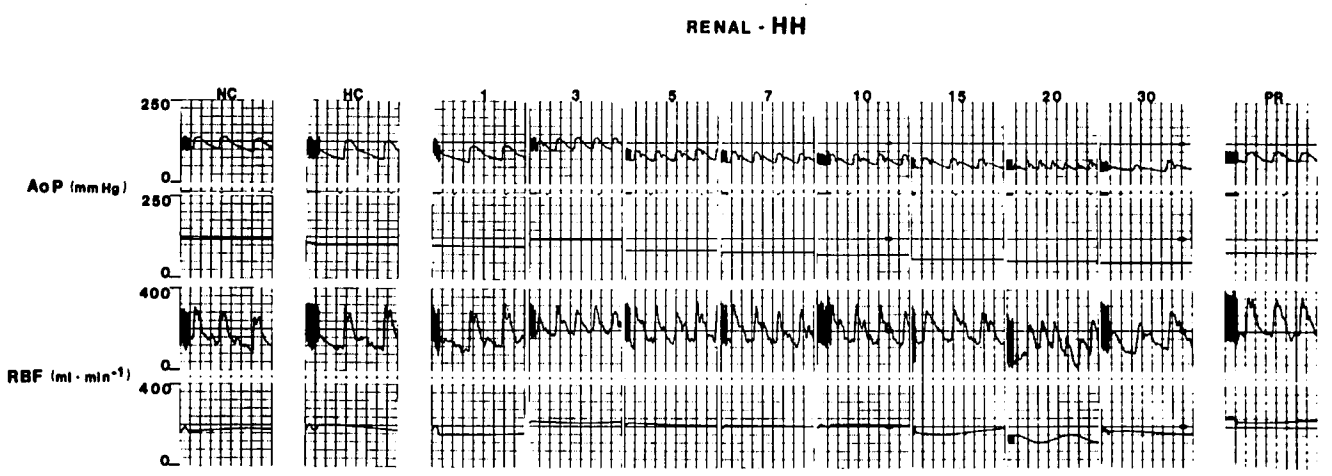

FIG. 3. Recordings are shown of phasic and mean arterial pressures (AoP) and renal blood flows (RBF) first in normovolemic conscious animals (NC), then after hemorrhage to establish a conscious hypovolemic control (HC). Halothane then was administered to these hypovolemic animals (Group HH) and data recorded for 30 min. Shed blood was reinfused and the anesthetized postreinfusion (PR) values noted. Paper speed was 25 mm/s for fast wave forms. Note that despite halothane administration, RBF remains at control levels.

rhage (15–22% of EBV) with a PAH extraction technique and reported it to decrease.[21] This method is particularly inaccurate in hypovolemic situations.[22,23] Hemorrhage studies in conscious dogs and primates, using flow probe techniques to measure renal blood flow, have shown that with mild–moderate degrees of hemorrhage (14–26% EBV) renal blood flow does not decrease.[24] However, renal perfusion is not totally beyond compromise in this regard and can decrease with several hemorrhage (50% EBV). This is true in both dogs and primates.[24]

The present study has reconfirmed that a moderate level of halothane does not significantly decrease renal blood flow in a conscious dog. In this same experimental setting, it has again shown that hemorrhage of up to 30% of the EBV does not decrease renal blood flow. In fact, in the conscious animal, renal vascular resistance significantly decreases with hemorrhage so as to maintain renal blood flow near control levels. However, this study also has gone one step further. It has examined the impact of halothane on renal perfusion when administered after the onset of acute hypovolemia. Following hemorrhage in our conscious dogs, blood pressure remained in a normal range primarily because of homeostatic mechanisms that compensate for hypovolemia. Halothane administration then decreased aortic pressure by as much as 32% to levels that would be considered hypotensive, *i.e.*, 60–70 mmHg mean pressure. Yet, renal blood flow was maintained at levels not significantly different from that group's (HH) hypovolemic control obtained after 30% hemorrhage. The renal blood flow changes in Group HH also were not significantly different from those in Group AH or the normovolemic–halothane group (HN). The heart rate changes seen in these groups deserve comment. In Groups HN and HH, heart rate increased after halothane. This has been noted before in conscious dogs and primates.[24] The heart rate changes in these two groups were comparable. Despite 30% less blood volume in Group HH, and presumably a lower cardiac output, RBF was similar to Group HN. In Groups AH and HH, where blood volumes were presumably equivalent, the heart rates were different at 1–7 min. Recognizably, the maintenance of RBF in Group HH may have been due to a larger cardiac output than in Group AH at these times. However, in the 7–30-min phase of the study, heart rates were comparable, as were renal blood flows. The maintenance of flow was due to the renal vascular dilation from halothane that was not seen in Group AH. These data would indicate that the kidney retains its ability to pressure autoregulate its blood flow not only in the face of hypovolemia but also after hypotension due to a superimposed anesthetic, in this case halothane. It is important to realize that this might not be the case with other anesthetics.

These data regarding halothane and normovolemic animals are consistent with previous results in intact dogs, primates, and rats,[6,9,11,13] as well as isolated dog kidneys[8] in that halothane decreased renal vascular resistance, thus maintaining renal blood flow, despite a decreased perfusion pressure. This differs from results obtained with halothane in pigs where renal blood flow was measured with microspheres.[12] In that study also, the pigs actually were anesthetized twice on the same day and positive-pressure ventilation was used. The microsphere technique has its own set of problems related to accuracy, such as inadequate mixing, capillary shunting, and rheologic and hemodynamic changes.[25–27] An interesting example of this is the recent study of halothane by Gelman *et al.*[15] There, renal blood flow, measured by the microsphere technique in the dog, was not found to change significantly from

control using two different sizes of microspheres, but the changes that did occur were in opposite directions. Another possibility to explain the differing results is species differences. For decades the standards for studying renal physiology and pharmacology have been the dog and rat. The advent of the porcine species for cardiovascular—and particularly renal—investigations is fairly recent. Much remains to be learned relative to the validity of extrapolating porcine data to humans. This is not to suggest that canine data can be extrapolated confidently to humans. However, a vast amount of data exist that indicate a functional similarity between the physiology of the canine and human cardiovascular systems. Indirect evidence of this for renal physiology is seen in the results from canines and primates dealing with hemorrhage.[24] Probably even more important to obtaining good regional blood flow data than flow measuring techniques and species is the use of a subject whose cardiovascular function is not altered by prior anesthetics and sedation.[16-18] The pigs used in Tranquilli's study were anesthetized and reawakened just before reanesthetization with halothane and measurement of its effects on renal blood flow.[12] In addition, a higher concentration of halothane was used (2.25%) than in our study (1–1.5%) or the one of Gelman *et al.* (0.9–1.8%).[15]

Another important feature to consider is the presence or absence of positive-pressure ventilation, which we did not use. Although studies in dogs given "anesthetizing" doses of pentobarbital have shown that intermittent positive-pressure ventilation did not significantly alter renal blood flow,[20] we wanted to avoid this variable, feeling it was more of a concern than the potential Pa_{CO_2} changes. Halothane did not depress ventilation in conscious rats[11] or dogs[15] and is not felt to be a ventilatory depressant in humans when 0.5–1.5% inspired concentrations are employed.[28] We did not measure Pa_{CO_2} in this study. However, in other studies in our laboratory, employing similar concentrations of halothane in the spontaneously ventilating dog, Pa_{CO_2} changed from a mean ± SEM of 37 ± 1.4 mmHg awake to 41 ± 2.9 mmHg anesthetized, a nonsignificant change. Additionally, we have previously shown that hypercarbia does not alter renal hemodynamics in conscious dogs.[29]

During hypovolemic conditions, homeostatic mechanisms defend the integrity of blood pressure. This is achieved through baroreceptor and volume receptor-mediated reflexes, which result in increased levels of both neurogenic and humoral agents, whose effects are of a vasoconstrictive nature.[30-33] It is known that increases in systemic vascular resistance in response to hemorrhage, after inhalation anesthesia, are less than those seen after intravenous anesthetic techniques.[34] After reinfusion, arterial pressure in Group HH was significantly less than in the awake group, AH. This was probably due to the fact that halothane, being a complete anesthetic, obtunds compensatory cardiovascular reflexes that can result in vasoconstriction.[35] Despite this, blood flow to the kidney was not impaired. The focus should be on organ perfusion rather than perfusion pressure. If adequate organ perfusion can be achieved despite a lower perfusion pressure, that is an acceptable situation. This assumes that perfusion pressure is not too low for consideration of other vital organs such as heart and brain. While these organs do require an adequate perfusion pressure, they probably autoregulate their blood flow more on a metabolic basis and do not seem to pressure autoregulate as strongly as the kidney.

Consideration also was given in this study as to whether animals being studied during hemorrhage should have a prior splenectomy. Since the blood storage function of the dog spleen is calculated to be only about 10% of the estimated blood volume[36] and since it has been shown that the renal vascular response to hemorrhage in conscious dogs is identical before and after splenectomy,[24] splenectomies were not undertaken in our animals.

One disadvantage of a direct measuring flow probe technique is that changes in intraorgan flow distribution cannot be seen. Data obtained with radioactive microspheres or radioactive washout techniques indicate that shifts in intrarenal blood flow can occur with hemorrhage. It is felt by some that blood flow is shifted away from the cortical to the medullary areas of the kidney with hemorrhage.[23] There are also data, however, that show that hemorrhage decreases medullary blood flow to a greater extent than it does cortical blood flow.[37] Studies also exist showing that halothane itself produces a shift of blood flow from the outer to the inner cortex.[10] While shifts in intraorgan blood flow may be of significance in injury to the kidneys resulting from hypoperfusion, it has not been shown, in the case of anesthetic agents, that such shifts are of any consequence in producing renal damage as long as total perfusion is unimpaired. Thus, it is our opinion that flow probe devices continue to be the technique of choice for studying regional hemodynamics. The technique permits chronically instrumented conscious animal preparations and gives a continuous picture of the organ blood flow pattern. These advantages in most circumstances outweigh the ability to see intraorgan flow shifts at least in the kidney.

In summary, halothane does not decrease renal blood flow but rather decreases renal vascular resistance and maintains renal blood flow stable in the face of a diminished perfusion pressure, *i.e.*, autoregulation is preserved. Furthermore, acute, moderately severe hemorrhage in a conscious dog does not decrease renal blood flow. Rather, renal vasodilation occurs and this decrease in renal vascular resistance maintains renal blood flow. Lastly, when halothane is administered to hypovolemic dogs who have

been hemorrhaged approximately one-third of their blood volume, renal perfusion is preserved.

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