

Role of Sinoaortic Baroreceptors in Initiating the Renal Response to Continuous Positive-pressure Ventilation in the Dog

James E. Fewell, Ph.D.,* and Gary C. Bond, Ph.D.†

Experiments were conducted to determine the role of cardiopulmonary receptors with vagal afferents and sinoaortic baroreceptors in initiating the reflex antidiuresis and antinatriuresis during continuous positive-pressure ventilation (CPPV). CPPV was applied to 18 dogs: seven control, five that underwent bilateral vagotomy; and six that underwent sinoaortic baroreceptor denervation. The dogs were anesthetized with pentobarbital, paralyzed with succinylcholine, and their lungs were mechanically ventilated with a volume ventilator. Renal function and systemic hemodynamics were monitored by clearance methods and pressures, respectively. After two 30-min control periods of intermittent positive-pressure ventilation (IPPV), CPPV using 10 cm H₂O positive end-expiratory pressure was applied for two 30-min experimental periods. Three 30-min recovery periods of IPPV followed. In dogs of the control group and vagotomy group, CPPV caused statistically significant decreases from control levels in urinary flow, sodium excretion, and glomerular filtration rate. However, in the dogs of the sinoaortic baroreceptor denervation group, CPPV did not produce any significant change in these variables. Therefore, the results do not support the hypothesis that cardiopulmonary receptors mediate the renal response to CPPV. Rather, the data indicate that the aortic arch and carotid sinus baroreceptors participate in initiating the reflex antidiuresis and antinatriuresis during CPPV. (Key words: Hormones, antidiuretic. Kidney: filtration, glomerular; urine. Receptors: baroreceptors; aortic arch; carotid sinus. Ventilation: continuous positive-pressure breathing; intermittent positive-pressure breathing; positive end-expiratory pressure.)

A REFLEX ANTIDIURESIS frequently occurs during continuous positive-pressure ventilation (CPPV).¹⁻⁴ Although the mechanism producing the antidiuresis remains unknown, Gauer and Henry and their collaborators⁵ believe that the atrial volume receptor mechanism plays an important role. These investigators suggest that the shift in the central blood volume out of the thorax, which occurs during CPPV, results in a decrease in the discharge of atrial volume receptors and a subsequent increase in the release of antidiuretic hormone (ADH). The increase in plasma

ADH is believed by these investigators to be primarily responsible for mediating the antidiuresis.

Others have suggested that changes in renal function during CPPV are predominantly caused by changes in renal hemodynamics rather than by changes primarily related to ADH.^{2,4} Renal hemodynamic changes during CPPV may be caused by alterations in renal sympathetic nerve activity. In support of this is the finding that renal denervation eliminates the renal response to CPPV.⁶

Renal sympathetic nerve activity can be affected by activation of cardiopulmonary receptors⁷ and by changes in the activity of carotid sinus and aortic arch baroreceptors.^{8,9} Two studies have suggested that cardiopulmonary receptors, which have vagal afferents, do not mediate the renal response to CPPV. Baratz, Philbin and Patterson³ demonstrated in dogs that eliminating atrial volume receptor afferents and other cardiopulmonary afferents by bilateral cervical vagotomy fails to abolish or attenuate the increase in plasma ADH and decrease in urinary flow during CPPV. They suggested that the antidiuresis was secondary, not to the atrial volume receptor mechanism, but rather to the decrease in cardiac output observed in their studies during CPPV. Unfortunately, these investigators did not report whether the decrease in cardiac output decreased systemic arterial pressure, which might have directly produced the antidiuresis.^{10,11} Tarak and Chaudhury¹² showed in rats that vagotomy fails to alter the antidiuresis produced during CPPV. However, the antidiuresis could be abolished by preventing the decrease in systemic arterial pressure during CPPV by the infusion of angiotensin. In the studies of Baratz *et al.* and Tarak and Chaudhury, it is suggested that the effects of CPPV on renal function are related, not to changes in atrial volume receptor activity, but perhaps to hemodynamic changes occurring elsewhere in the circulation.

In view of the above considerations, we believed that a reevaluation of the role of atrial receptors in initiating the antidiuresis during CPPV was warranted. Furthermore, since arterial hemodynamic changes have been implicated in producing the antidiuresis, we examined the possibility that the carotid sinus and aortic arch baroreceptors may contribute to the renal response.

* Pulmonary Faculty Trainee. Present address; Cardiovascular Research Institute, 1315M, University of California San Francisco, San Francisco, California 94143.

† Associate Professor.

Received from the Departments of Physiology, University of Arkansas for Medical Sciences, Little Rock, Arkansas 72201, and Medical College of Georgia, Augusta, Georgia 30912. Accepted for publication December 13, 1979. Supported by NIH Grant HL 21094. Presented in part at the American Physiological Society Meeting, Hollywood, Florida, 1977.

Address reprint requests to Dr. Bond: Department of Physiology, Medical College of Georgia, Augusta, Georgia 30912.

Methods

Three groups of dogs were subjected to continuous positive-pressure ventilation (CPPV) using 10 cm H₂O positive end-expiratory pressure (PEEP): a group of seven control dogs with intact afferents from the cardiopulmonary region and the sinoaortic baroreceptors; a group of five dogs that had undergone bilateral cervical vagotomy only; and a group of six dogs that had undergone selective denervation of the aortic arch and carotid sinus baroreceptors without affecting the vagal afferents. Bilateral cervical vagotomy was performed on the day of the experiment by cutting the vagus nerves approximately 30 sec after infiltrating the area to be sectioned with lidocaine HCl, 2 per cent. Aortic arch baroreceptor denervation was performed under aseptic conditions several days prior to an experiment according to the technique of Edis and Shepherd.¹³ Carotid baroreceptors were denervated by cutting the sinus nerve, stripping the adventitia from the carotid sinus region, and painting the entire area with phenol, 8 per cent. Effective elimination of the sinoaortic baroreceptors was confirmed by the absence of a reflex bradycardia following the intravenous injection of phenylephrine, 0.3–0.4 mg.¹⁴ The presence of intact cardiopulmonary afferents in the vagus nerve was demonstrated by producing the typical reflex bradycardia and systemic hypotension by the intravenous injection of veratridine, 10–50 µg.¹⁴

For the experiments, all dogs were anesthetized with sodium pentobarbital, 30 mg/kg, injected intravenously. Additional doses of 1 mg/kg were given every 15 min to maintain anesthesia. Each dog was placed supine and its trachea was intubated with a cuffed endotracheal tube. The cuff was inflated to a gas-tight fit and the tube was connected to a volume ventilator (Harvard Apparatus Respiration Pump, Model 607) adjusted to deliver room air at a tidal volume of 15 ml/kg at a frequency of 12 breaths/min. A rigid catheter was placed in line with the endotracheal tube for continuous monitoring of proximal airway pressure. The dog was then paralyzed with succinylcholine, 1 mg/kg. Catheters were inserted into the thoracic aorta via the left femoral artery and the abdominal aorta distal to the renal arteries via the right femoral artery for measurement of pressures. A femoral vein was cannulated for infusion of fluids. A trocar catheter was placed in the pleural space by a small right thoracotomy to measure intrathoracic pressure. A Foley catheter was placed in the bladder to collect urine.

In preliminary experiments, the application of continuous positive-pressure ventilation (CPPV) to sinoaortic baroreceptor-denervated dogs decreased systemic arterial pressure to 75–80 torr. Since the decrease in systemic arterial pressure can produce an

antidiuresis directly by decreasing renal perfusion pressure, we elected to maintain a pressure of 70 torr at the level of the renal arteries throughout the experiment in the sinoaortic baroreceptor-denervated dogs. To accomplish this, a flank incision was made and an adjustable snare was placed around the abdominal aorta approximately 3 cm proximal to the origin of the right renal artery. In order to more fairly compare the renal responses to CPPV among the three groups of dogs, renal perfusion pressure was also maintained at 70 torr in both the control group and the vagotomy group. The pressures in the aorta both proximal and distal to the snare were monitored continuously. The pressure recorded below the snare was considered to represent renal perfusion pressure.

Each experiment consisted of seven consecutive 30-min periods: two 30-min control periods, during which expiratory pressure was atmospheric (IPPV); two 30-min experimental periods, during which expiratory pressure was increased 10 cm H₂O (CPPV); and three 30-min recovery periods, during which expiratory pressure was atmospheric (IPPV). The experimental protocol began when urinary flow had remained stable (± 10 per cent) for two consecutive 30-min periods. CPPV was accomplished by partial static inflation of an occlusive balloon manifold incorporated into the expiratory limb of the ventilator circuit.

Arterial blood-gas values were measured at the midpoint of each 30-min period, and heart rate, systemic arterial pressure, renal perfusion pressure, and pulse pressure were measured at the end of each period. The pressures were transduced (Statham P23Db) and recorded on an Electronics for Medicine DR-8 recorder. Glomerular filtration rate was determined each period from the clearance of inulin. Inulin, 12.5 mg/ml, and succinylcholine, 0.14 mg/ml, in lactated Ringer's solution were infused intravenously at a rate of 0.06 ml/kg min⁻¹. A slight diuresis was produced by the infusion of isosmotic mannitol, 0.03 ml/kg min⁻¹. Urine was collected at the end of each 30-min period and a 10-ml blood sample was drawn at the midpoint of each of these periods. The volume of blood withdrawn was replaced by an equal volume of dextran, 6 per cent, in sodium chloride, 0.9 per cent, in which the erythrocytes from the previous collection period were resuspended. Urinary flow and plasma and urinary concentrations of sodium and inulin¹⁵ were measured and sodium excretion and glomerular filtration rate were calculated. In addition, plasma and urinary osmolality were measured and osmolar clearance and free-water clearance were calculated.

To analyze the data statistically within each experi-

mental group, a one-way analysis of variance for repeated measurements of the same variable was performed.¹⁶ This was followed by Dunnett's multiple-range *t* test to determine which means, at the 0.05 level of significance, were statistically different from the means of the control periods.¹⁷ A one-way analysis of variance was used for comparing the control levels for each measured parameter among the three groups of dogs.¹⁶

Results

Continuous positive-pressure ventilation (CPPV) increased intrathoracic pressure and heart rate in the control dogs (table 1). No significant change in systemic arterial pressure or renal perfusion pressure occurred, but pulse pressure decreased during CPPV. In the vagotomy group, CPPV increased intrathoracic pressure and heart rate and decreased pulse pressure as in the control group. A small, but statistically significant, decrease in systemic arterial pressure occurred, but renal perfusion pressure did not change. In sinoaortic baroreceptor-denervated dogs, CPPV increased intrathoracic pressure but did not change heart rate. Significant decreases in systemic arterial pressure and pulse pressure occurred, but renal perfusion pressure remained constant. Control levels for each of the measured hemodynamic parameters were not statistically different among the three groups of dogs.

Continuous positive-pressure ventilation decreased urinary flow, sodium excretion, and glomerular filtra-

tion rate in the control group and vagotomy group, but did not produce changes in these parameters in the sinoaortic baroreceptor-denervated dogs (fig. 1). CPPV decreased osmolar clearance in the control group and the vagotomy group (table 2). Free-water clearance was increased by CPPV only in the vagotomy group. No significant change in osmolar or free-water clearance occurred in the sinoaortic baroreceptor-denervated dogs. The control values for glomerular filtration rate in the sinoaortic baroreceptor denervation group were significantly lower than the levels in the control and vagotomy groups.

Continuous positive-pressure ventilation produced a significant decrease in *pH* in the sinoaortic baroreceptor-denervated dogs only (table 3). No significant change in *PaO₂* values occurred. Small, but statistically significant, increases in *PaCO₂* values with CPPV were observed following vagotomy or sinoaortic baroreceptor denervation.

Discussion

The results of this study indicate that carotid sinus and aortic arch baroreceptors are primarily responsible for initiating the antidiuresis during CPPV. The results confirm, therefore, the speculation made by several investigators^{3,12,18} that hemodynamic changes on the arterial side of the circulation are involved.

The abilities of the kidneys to respond to CPPV should have been similar in the dogs of the three experimental groups irrespective of the lower glomerular filtration rate in the sinoaortic baroreceptor de-

TABLE 1. Effects of Continuous Positive-pressure Ventilation on Hemodynamics in Dogs of the Control Group (n = 7), Vagotomy Group (n = 5), and Sinoaortic Baroreceptor Denervation Group (n = 6)

	Control 1	Control 2	CPPV 1	CPPV 2	Recovery 1	Recovery 2	Recovery 3
Control							
ITP (torr)	-3.6 ± .5	-3.3 ± .5	0.7 ± .3*	0.1 ± .3*	-2.5 ± .2	-2.8 ± .2	-3.0 ± .3
HR (beats/min)	129 ± 16	120 ± 15	140 ± 17*	143 ± 16*	136 ± 18	136 ± 18	134 ± 19
MAP (torr)	157 ± 7	162 ± 11	161 ± 14	147 ± 14	148 ± 10	148 ± 11	140 ± 7
PP (torr)	34 ± 2	32 ± 1	27 ± 3*	26 ± 3*	34 ± 3	33 ± 2	31 ± 2
RPP (torr)	71 ± 1	71 ± 1	72 ± 2	72 ± 2	72 ± 1	71 ± 1	71 ± 1
Bilateral vagotomy							
ITP (torr)	-2.4 ± .3	-2.4 ± .3	0.9 ± .5*	1.4 ± .5*	-1.8 ± .5	-1.9 ± .5	-2.1 ± .5
HR (beats/min)	150 ± 7	150 ± 7	166 ± 6*	160 ± 6	156 ± 6	155 ± 6	162 ± 5
MAP (torr)	161 ± 10	163 ± 10	150 ± 10*	155 ± 10	160 ± 10	157 ± 9	152 ± 9
PP (torr)	29 ± 3	28 ± 3	23 ± 2*	24 ± 2*	27 ± 2	27 ± 2	26 ± 2
RPP (torr)	71 ± 1	70 ± 1	70 ± 1	70 ± 1	71 ± 1	71 ± 1	70 ± 1
Sinoaortic denervation							
ITP (torr)	-2.5 ± .4	-2.5 ± .4	0.4 ± .8*	-0.3 ± .7*	-2.8 ± .4	-2.4 ± .4	-2.9 ± .4
HR (beats/min)	139 ± 14	138 ± 14	139 ± 14	135 ± 14	132 ± 14	132 ± 14	133 ± 14
MAP (torr)	139 ± 10	133 ± 10	96 ± 9*	98 ± 9*	117 ± 6*	113 ± 4*	108 ± 6*
PP (torr)	31 ± 2	30 ± 2	25 ± 2*	26 ± 2*	29 ± 3	30 ± 3	30 ± 2
RPP (torr)	70 ± 1	71 ± 1	70 ± 1	68 ± 2	72 ± 2	69 ± 2	69 ± 1

Values represent means ± SEM. ITP = intrathoracic pressure; HR = heart rate; MAP = mean arterial pressure; PP = pulse pressure; RPP = renal perfusion pressure. Asterisk indicates a

significant difference, at the .05 level, from the mean of the control level.

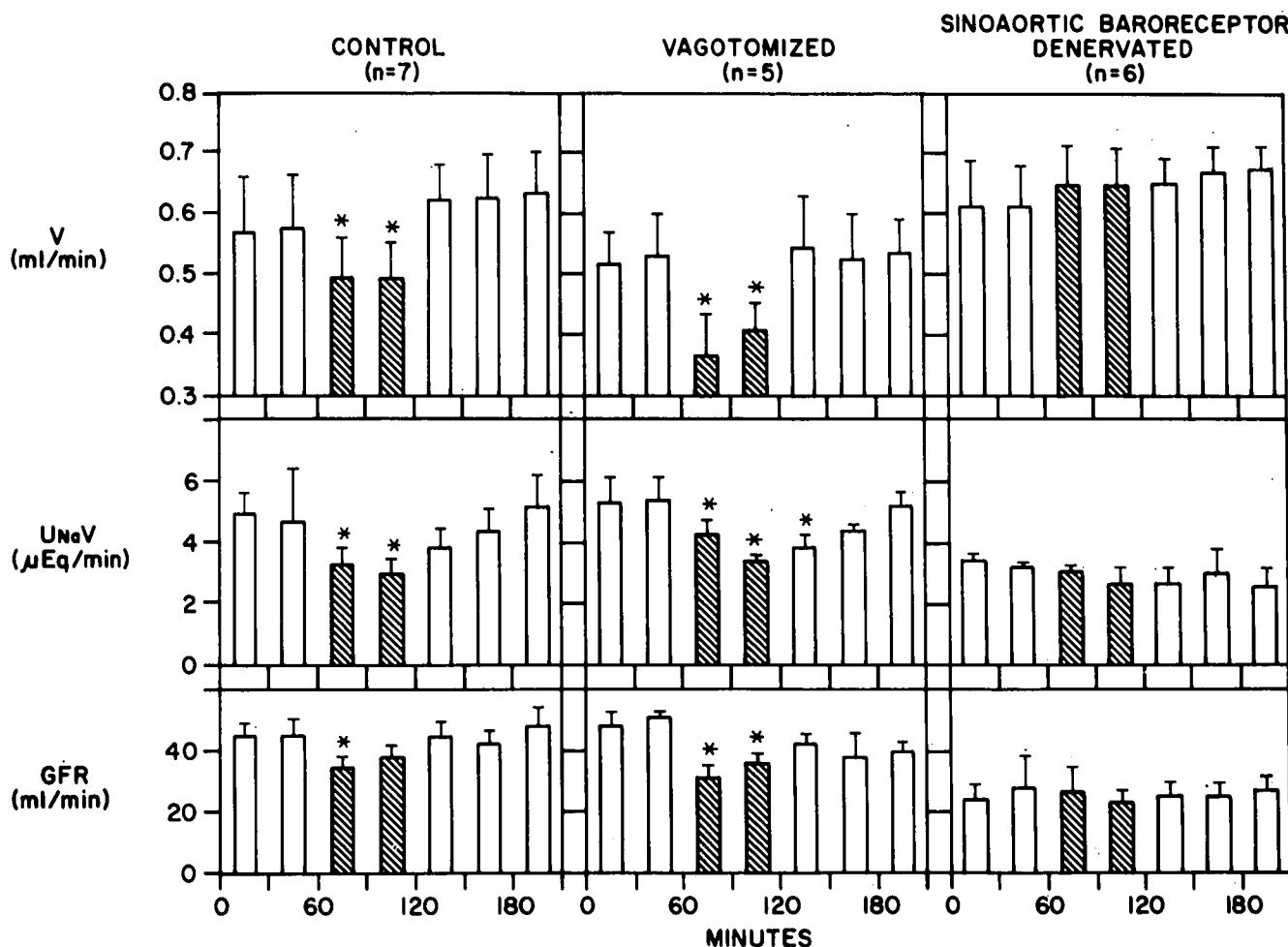


FIG. 1. Effects of continuous positive-pressure ventilation on renal function in the dogs of the control group ($n = 7$), the vagotomy group ($n = 5$), and the sinoaortic baroreceptor denervation group ($n = 6$). The crosshatched bars represent periods of PEEP. Values represent means \pm SEM. V = urine flow; $U_{Na}V$ = sodium excretion; GFR = glomerular filtration rate. Asterisk indicates a significant difference from the mean of the control level at the .05 level of significance.

nervation group. Control levels of all other measured hemodynamic and renal parameters, including sodium excretion, were not significantly different when the three groups were compared. Since sodium excretion was similar and glomerular filtration rate was reduced, the fractional reabsorption of sodium was lower during the control periods in the sinoaortic baroreceptor denervation group than in the control and vagotomy groups. This indicates that dogs in the sinoaortic baroreceptor denervation group had a greater potential for reducing their sodium excretion during CPPV than dogs in both control and vagotomy groups. Furthermore, two dogs in the sinoaortic baroreceptor denervation group had control glomerular filtration rates similar to those of the dogs in the control group, and CPPV did not decrease urinary flow, sodium excretion, or glomerular filtration rate.

It is possible that the reduced glomerular filtration rate during the control periods in the sinoaortic baroreceptor

receptor denervation group was related to a slightly increased level of renal sympathetic nervous activity. However, it is likely that further increases in activity could have occurred during CPPV to decrease glomerular filtration rate. Study and Shipley¹⁹ have shown that increased renal sympathetic nervous activity can decrease urinary flow and glomerular filtration rate to almost zero in pentobarbital-anesthetized dogs having glomerular filtration rates similar to those of the dogs in our sinoaortic baroreceptor denervation group. Thus, we believe that the reduced glomerular filtration rate during the control periods in the sinoaortic baroreceptor-denervated dogs did not significantly alter their ability to respond to CPPV.

That vagotomy did not affect the renal response to CPPV provides evidence that cardiopulmonary receptors with vagal afferents do not initiate the antidiuresis. Our findings, therefore, disprove the hypothesis of Gauer *et al.*⁵ that the antidiuresis associated

TABLE 2. Effects of Continuous Positive-pressure Ventilation on Osmolar Clearance and Free-water Clearance in Dogs of the Control Group (n = 7), Vagotomy Group (n = 5), and Sinoaortic Baroreceptor Denervation Group (n = 6)

	Control 1	Control 2	CPPV 1	CPPV 2	Recovery 1	Recovery 2	Recovery 3
C_{osm} (ml/min)							
Control	1.44 ± .13	1.42 ± .12	1.07 ± .14*	1.05 ± .18*	1.53 ± .23	1.43 ± .23	1.54 ± .19
Bilateral vagotomy	1.17 ± .13	1.15 ± .11	0.73 ± .05*	0.76 ± .05*	1.00 ± .07	1.02 ± .08	1.07 ± .10
Sinoaortic baroreceptor denervation	1.14 ± .16	1.06 ± .16	1.09 ± .15	1.17 ± .16	1.25 ± .15	1.28 ± .15	1.34 ± .16
C_{H_2O} (ml/min)							
Control	-.91 ± .11	-.82 ± .14	-.58 ± .10	-.56 ± .13	-.89 ± .24	-.80 ± .22	-.91 ± .18
Bilateral vagotomy	-.67 ± .18	-.62 ± .16	-.37 ± .08*	-.36 ± .09*	-.46 ± .11	-.49 ± .11	-.53 ± .13
Sinoaortic baroreceptor denervation	-.52 ± .14	-.44 ± .12	-.44 ± .12	-.53 ± .12	-.53 ± .12	-.57 ± .12	-.65 ± .15

Values represent means ± SEM. C_{osm} = osmolar clearance; C_{H_2O} = free-water clearance. Asterisk indicates a significant difference, at the .05 level, from the mean of the control level.

TABLE 3. Effects of Continuous Positive-pressure Ventilation on Arterial Blood Gases and pH in Dogs of the Control Group (n = 7), Vagotomy Group (n = 5), and Sinoaortic Baroreceptor Denervation Group (n = 6)

	Control	CPPV	Recovery
pH			
Control	7.37 ± 0.01	7.33 ± 0.02	7.34 ± 0.02
Bilateral vagotomy	7.38 ± 0.01	7.34 ± 0.02	7.36 ± 0.02
Sinoaortic baroreceptor denervation	7.42 ± 0.01	7.33 ± 0.02*	7.38 ± 0.03
P_{aO_2} (torr)			
Control	88 ± 2	93 ± 4	85 ± 4
Bilateral vagotomy	88 ± 2	90 ± 5	89 ± 5
Sinoaortic baroreceptor denervation	90 ± 4	89 ± 2	88 ± 3
P_{aCO_2} (torr)			
Control	35 ± 1	36 ± 1	35 ± 1
Bilateral vagotomy	35 ± 1	38 ± 1*	36.0 ± 1
Sinoaortic baroreceptor denervation	35 ± 0.5	40 ± 1*	35.0 ± 1

Values represent means ± SEM. An asterisk indicates a significant difference, at the .05 level, from the mean of the control level.

with increased expiratory pressure is initiated by atrial receptors. However, our findings are similar to the results reported by Baratz *et al.*³ In our study, arterial pressure decreased significantly during CPPV in the vagotomy group. However, we eliminated the possible direct effect of changes in arterial pressure by maintaining renal perfusion pressure constant. Therefore, we have shown that the antidiuresis during CPPV is reflex in nature and independent of cardiopulmonary receptors with vagal afferents.

Previous investigators have shown that CPPV using 10 cm H₂O positive end-expiratory pressure produces greater decreases in urinary flow than occurred in our experiments.^{3,4} We observed in the control group and the vagotomy group average decreases in urinary flow of 20 and 29 per cent, respectively. Baratz *et al.*³ reported a decrease in urine flow of 48 per cent in control dogs, and a decrease of 58 per cent in vagotomized dogs, while Hall *et al.*⁴ found a decrease of 40 per cent in control dogs. The quantitative difference in the urinary flow responses in our experiments and those of others could be related to the lower renal

perfusion pressure in our experiments. It was necessary to maintain renal perfusion pressure at 70 torr, since the application of CPPV to sinoaortic baroreceptor-denervated dogs produces large decreases in systemic arterial pressure that could directly affect renal function. Nevertheless, since the renal responses to CPPV in both the control and the vagotomy group were qualitatively similar to the renal responses reported by other investigators,^{3,4} we believe that the decreased renal perfusion pressure does not significantly affect the interpretation of our results.

The small increase in P_{aCO_2} during CPPV in the dogs of the vagotomy and sinoaortic baroreceptor denervation groups probably results from increased dead-space ventilation, as both anatomic and alveolar dead space are increased during CPPV.^{20,21} A reduction in tidal volume, resulting from an increased compressed gas volume in the ventilator circuit, may also play a role.²² A rise in P_{aCO_2} of the magnitude observed in our experiments would not significantly influence the renal response to CPPV.³

Although the purpose of our study was to determine

the receptors that initiate the reflex antidiuresis during CPPV, it is of interest to speculate on the suggestion by Gauer *et al.*⁵ that antidiuretic hormone mediates, at least in part, the renal response. Baratz *et al.*³ have demonstrated that plasma ADH levels increase during CPPV in dogs both before and after bilateral cervical vagotomy. Based on our results, we would suggest that the rise in ADH that Baratz *et al.* observed after vagotomy might be produced by alterations in sinoaortic baroreceptor activity.^{23,24} Consequently, we would anticipate that in the sinoaortic baroreceptor denervation group a rise in hormone levels would be absent or at least attenuated with CPPV. However, even if ADH levels increase during CPPV, available evidence indicates that the rise has little influence in mediating the antidiuresis.² The small changes in free-water clearance that occurred in our experiments were in a direction opposite to that expected from the renal action of increased ADH levels. It is likely that free-water clearance changed because of a decrease in osmolar clearance associated with the alteration in glomerular filtration rate²⁵ or a neurogenically mediated increase in renal tubular sodium reabsorption.²⁶ More important in ruling out a role for ADH is our recent finding that denervation of renal sympathetic nerves effectively eliminates the antidiuresis.⁶

Based on our studies, we propose the following mechanism to explain the changes in renal function during CPPV with a PEEP of 10 cm H₂O. CPPV decreases cardiac output and produces a transient decrease in systemic arterial pressure and a sustained decrease in pulse pressure, which, in turn, decreases carotid sinus and aortic arch baroreceptor activity. Integration of these neural signals from the arterial baroreceptors by the central nervous system results in increased renal sympathetic nervous activity, and this increases renal vascular resistance and decreases glomerular filtration rate. The decreases in urinary flow and sodium excretion appear to result from the decreased glomerular filtration rate.

References

1. Ueda H, Neclerio M, Leather RP, et al: Effects of positive end expiratory pressure ventilation on renal function. *Surg Forum* 23:209-211, 1972
2. Kumar A, Pontoppidan H, Baratz RA, et al: Inappropriate response to increased plasma ADH during mechanical ventilation in acute respiratory failure. *ANESTHESIOLOGY* 40:215-221, 1974
3. Baratz RA, Philbin DM, Patterson RW: Plasma antidiuretic hormone and urinary output during continuous positive-pressure breathing in dogs. *ANESTHESIOLOGY* 34:510-513, 1971
4. Hall SV, Johnson EE, Hedley-Whyte J: Renal hemodynamics and function with continuous positive-pressure ventilation in dogs. *ANESTHESIOLOGY* 41:452-461, 1974
5. Gauer OH, Henry JP, Sicker HO: Cardiac receptors and fluid volume control. *Prog Cardiovasc Dis* 4:1-26, 1961
6. Fewell JE, Bond GC: Renal denervation eliminates the renal response to continuous positive pressure ventilation. *Proc Soc Exp Biol Med* 161:574-578, 1979
7. Karim F, Kidd C, Malpus CM, et al: The effects of stimulation of the left atrial receptors on sympathetic efferent nerve activity. *J Physiol* 227:243-260, 1972
8. Katz MA, Shear L: Effect of renal nerves on renal hemodynamics. I. Direct stimulation and carotid occlusion. *Nephron* 14:246-256.
9. Kezdi P, Geller E: Baroreceptor control of postganglionic sympathetic nerve discharge. *Am J Physiol* 214:427-435, 1968
10. Abe Y, Dixon F, McNay JL: Dissociation between autoregulation of renal blood flow and glomerular filtration rate. *Am J Physiol* 219:986-993, 1970
11. Shipley RE, Study RS: Changes in renal blood flow, extraction of inulin, glomerular filtration rate, tissue pressure and urine flow with acute alterations of renal artery blood pressure. *Am J Physiol* 167:676-688, 1951
12. Tarak TK, Chaudhury RR: The mechanism of positive pressure respiration induced antidiuresis in rats. *Clin Sci* 20:407-415, 1965
13. Edis AJ, Shepherd JT: Selective denervation of aortic arch baroreceptors and chemoreceptors in dogs. *J Appl Physiol* 30:294-296, 1971
14. Goetz KL, Bond GC: Reflex diuresis during tachycardia in the dog; evaluation of the role of the atrial and sinoaortic receptors. *Circ Res* 22:434-441, 1973
15. Heyrovsky A: A new method for the determination of inulin in plasma and urine. *Clin Chim Acta* 1:470-474, 1956
16. Winer BJ: *Statistical Principles in Experimental Design*. New York, McGraw-Hill, 1971, pp 261-273
17. Steele RGD, Torrie JH: *Principles and Procedures of Statistics*. New York, McGraw-Hill, 1960, pp 111-112
18. Goetz KL, Bond GC, Bloxham DD: Atrial receptors and renal function. *Physiol Rev* 55:157-205, 1975
19. Study RS, Shipley RE: Comparison of direct with indirect renal blood flow, extraction of inulin and diodrast, before and during acute renal nerve stimulation. *Am J Physiol* 163:442-453, 1950
20. West JB: *Pulmonary Pathophysiology—The Essentials*. Baltimore, Williams and Wilkins, 1977, p 193
21. Dueck R, Wagner PD, West JB: Effects of positive end-expiratory pressure on gas exchange in dogs with normal and edematous lungs. *ANESTHESIOLOGY* 47:359-366, 1977
22. West JB: *Pulmonary Pathophysiology—The Essentials*. Baltimore, Williams and Wilkins, 1977, p 186
23. Bond GC, Trank JW: Plasma antidiuretic hormone concentration after bilateral aortic nerve section. *Am J Physiol* 222:595-598, 1972
24. Share L, Levy MN: Cardiovascular receptors and blood titer of antidiuretic hormone. *Am J Physiol* 203:425-428, 1962
25. Orloff J, Wagner HW, Davidson DG: The effect of variations in solute excretion and vasopressin dosage on the excretion of water in the dog. *J Clin Invest* 37:458-464, 1958
26. Dibona GF: Neurogenic regulation of renal tubular sodium reabsorption. *Am J Physiol* 233:F73-F81, 1977