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Renal Function and Cardiovascular Responses during Positive Airway Pressure

José M. Marquez, M.D., Michal E. Douglas, M.D.,† John B. Downs, M.D.,‡ Wen-Hsien Wu, M.D., M.S.,§ Emil L. Mantini, M.D.,¶ Earlene J. Kuck,** Hugh W. Calderwood, V.M.D.††*

The authors determined cardiovascular, renal, and hormonal responses to increased airway pressure during continuous positive-pressure ventilation (CPPV) and continuous positive airway pressure (CPAP). Nine healthy, hydrated laboratory swine had appropriate catheters placed to allow for measurement of intrapleural, aortic, inferior vena caval, and left ventricular end-diastolic pressures; cardiac output; and urinary flow. Samples of arterial blood were analyzed for oxygen and carbon dioxide tensions, *p*H, plasma vasopressin, osmolality, and creatinine and sodium concentrations. Urine was analyzed for osmolality and creatinine and sodium concentrations, and volume was recorded. Intrapleural pressure was subtracted from left ventricular end-diastolic pressure to calculate transmural pressure, a reflection of left ventricular filling pressure. Glomerular filtration rate and urinary free-water and osmolal clearances were also calculated.

Expiratory left ventricular filling pressure was decreased equally by CPAP and CPPV. However, inspiratory left ventricular filling pressure and cardiac output were decreased by CPPV only. Urinary flow and glomerular filtration rate were decreased equally by CPAP and CPPV. Sodium excretion was decreased and plasma vasopressin increased by CPPV, but not by CPAP. Urinary free water and osmolal clearances were not changed by either ventilatory pattern. Although many of the renal-function variables were affected similarly by CPPV and CPAP, these alterations were not influenced solely by cardiac output or vasopressin, because only CPPV depressed cardiac output and increased vasopressin levels. (Key words: Heart: Cardiac output; vascular pressures. Kidney: blood flow; filtration, glomerular; function. Ventilation: continuous positive airway pressure; continuous positive-pressure breathing; positive end-expiratory pressure.)

* Medical Student IV, University of Florida College of Medicine.

† Assistant Professor of Anesthesiology and Surgery, University of Florida.

‡ Associate Professor of Anesthesiology and Surgery, University of Florida.

§ Associate Professor of Anesthesiology, New York University Medical Center; Chief of Anesthesiology, Veterans Administration Hospital, New York, New York.

¶ Fellow, Department of Anesthesiology, University of Florida.

** Assistant in Anesthesiology, University of Florida.

†† Assistant Professor of the College of Veterinary Medicine, and of the Department of Anesthesiology, University of Florida College of Medicine.

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Address reprint requests to Dr. Downs: Department of Anesthesiology, University of Florida College of Medicine, Box J-254, J. Hillis Miller Health Center, Gainesville, Florida 32610.

POSITIVE AIRWAY PRESSURE is frequently used in the treatment of acute pulmonary dysfunction. Numerous investigations of the cardiopulmonary and renal effects of such therapy have been performed in recent years. Controlled mechanical ventilation with positive end-expiratory pressure, called "continuous positive-pressure ventilation" (CPPV), usually increases intrapleural pressure, decreases cardiac filling pressure, and depresses cardiac output.[1,2] It has also been shown to alter intrarenal blood flow and to decrease total renal blood flow, glomerular filtration rate, sodium excretion, and urinary flow (UF).[3] In addition, plasma vasopressin may be increased and the renin–angiotensin system activated, which may contribute to the renal effects of CPPV.[3]

Because spontaneous inspiration decreases intrapleural pressure and therefore may augment thoracic venous return,[2] we suspected that cardiovascular and renal effects might be less pronounced during continuous positive airway pressure (CPAP) than during CPPV. Some investigators have found altered cardiovascular and renal function associated with high levels of CPAP.[4–7] However, we were unable to find any comparison of cardiovascular and renal effects of CPAP and CPPV at expiratory airway pressures of less than 15 torr.

Materials and Methods

Nine healthy, normally hydrated laboratory swine, each weighing 21 ± 0.3 kg (mean ± 1 SEM), were anesthetized with alpha-chloralose (1.0 per cent, iv); their tracheas were intubated orotracheally. Lactated Ringer's solution was infused intravenously at a rate of 4 ml/kg/hr. Animals breathed humidified room air through a system having an expiratory limb connected to a threshold-resistor exhalation valve (J. H. Emerson Co.). A column of water was applied to the exhalation valve to produce expiratory airway pressure of 7 torr during CPAP and CPPV. To obtain CPAP, a continuous flow of fresh gas maintained positive airway pressure throughout the respiratory cycle. During CPPV, animals were paralyzed with continuous intravenous infusions of succinylcholine (0.1 per cent). A mechanical tidal volume of 12 ml/kg was delivered at a ventilatory rate sufficient to maintain arterial P_{CO_2} between 35 and 45 torr.

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We inserted femoral-artery and inferior vena caval catheters and thermistor-tipped, flow-directed pulmonary-artery and suprapubic catheters. A microtransducer-tipped catheter (Millar Instruments, Inc.) was directed into the left ventricle from the femoral artery. A second transducer-tipped catheter was inserted into the right pleural cavity at the fifth intercostal space along the anterior axillary line.[8] Transduced pressure recordings and postmortem examination confirmed correct placement of all catheters.

Mean aortic pressure ($P_{\overline{AO}}$), mean inferior vena caval pressure ($P_{\overline{IVC}}$), intrapleural pressure, and left ventricular end-diastolic pressure (P_{LVED}) were recorded continuously. Heart rate was calculated from a continuously recorded electrocardiogram. Cardiac output was calculated from triplicate measurements using a thermodilution technique with iced 5 per cent dextrose in water as the indicator. Arterial blood was analyzed for gas tensions, *p*H, plasma vasopressin, osmolality, and creatinine and sodium concentrations. Urine collected at 30-min intervals was analyzed for osmolality and sodium and creatinine concentrations, and urinary flow was calculated. All measurements performed on blood and urine samples were done using standard laboratory techniques; results were reported in conventional units. Plasma vasopressin was determined by radioimmunoassay,[9] and results were reported in microunits per milliliter (μU/ml). Calculation of inspiratory and expiratory transmural left ventricular end-diastolic pressure (P_{TLVED}), stroke volume, glomerular filtration rate (GFR), urinary free-water clearance (C_{H_2O}) and osmolal clearance (C_{osm}) were performed using the following equations:

$$P_{TLVED}\ (\text{torr}) = P_{LVED} - \text{intrapleural pressure}$$

$$\text{Stroke volume (ml)} = \text{cardiac output} \div \text{heart rate}$$

$$\text{GFR (ml/min)} = \frac{\text{urinary creatinine} \times \text{UF}}{\text{plasma creatinine} \times 30}$$

$$C_{osm}\ (\text{ml/30 min}) = \text{UF} \times \left(\frac{\text{urinary osmolality}}{\text{plasma osmolality}}\right)$$

$$C_{H_2O}\ (\text{ml/30 min}) = \text{UF} - C_{osm}$$

After a control period of spontaneous ventilation with ambient airway pressure, animals randomly received CPAP or CPPV with 7 torr expiratory airway pressure. Another control period was interposed between CPAP and CPPV. All measurements and calculations were performed at the end of each 30-min period. Results are expressed as means ± SEM. Data obtained during CPAP, CPPV, and control periods were compared using a multiple analysis of variance.

Results

Measured and calculated variables were unchanged during all control periods of spontaneous respiration with ambient airway pressure. Arterial blood *p*H, P_{O_2}, and P_{CO_2} values were also unchanged by CPAP or CPPV.

Both CPAP and CPPV increased end-expiratory intrapleural pressure and P_{LVED} (table 1). End-expiratory P_{TLVED} was decreased equally by CPAP and CPPV. During CPPV, inspiratory intrapleural pressure was increased by each mechanical inspiration, causing inspiratory P_{TLVED} to decrease. However, during CPAP, inspiratory intrapleural pressure decreased and P_{LVED} remained unchanged. Therefore, inspiratory P_{TLVED} was increased during CPAP. Stroke volume was decreased more by CPPV than by CPAP, and tachycardia maintained cardiac output at control levels during CPAP, but not during CPPV.

Plasma osmolality and sodium and creatinine concentrations were not changed by CPAP and CPPV. Urinary osmolality and creatinine concentrations also remained unchanged; however, urinary sodium concentration decreased during CPPV. Urinary flow and glomerular filtration rate were decreased equally by CPAP and CPPV. Plasma vasopressin was increased by CPPV, but not by CPAP. Neither positive airway pressure pattern altered C_{H_2O} or C_{osm} (table 2).

Discussion

Adverse hemodynamic and renal effects have often been associated with increased airway pressures. Most investigators have evaluated positive expiratory airway pressure applied in conjunction with controlled mechanical ventilation, which has been shown to increase inspiratory airway and intrapleural pressures and to decrease thoracic venous inflow of blood.[1] When blood flow from the inferior and superior vena cava is decreased, cardiac filling and stroke volume may decrease, in spite of increased intraluminal cardiac pressures. In contrast, spontaneous respiration with increased airway pressure has been reported not to decrease cardiac output.[2,10] Presumably, spontaneous inspiration will decrease intrapleural pressure, thereby increasing thoracic venous blood inflow and cardiac output.

An expiratory airway pressure of 7 torr increased expiratory intrapleural pressure equally during CPAP and CPPV. Similarly, end-expiratory P_{TLVED} was depressed equally by CPAP and CPPV. During CPPV, mechanical inspiration increased intrapleural pressure, thereby greatly decreasing venous return and preventing the decreased intrapleural pressure and

TABLE 1. Cardiovascular Responses during Spontaneous Respiration with Ambient Airway Pressure (Control), Continuous Positive-pressure Ventilation (CPPV) with 7 torr PEEP, and Spontaneous Respiration with 7 torr Continuous Positive Airway Pressure (CPAP) (Mean ± 1 SEM, Nine Swine)

	Control	CPPV + 7 torr PEEP	Control	Spontaneous Respiration + 7 torr CPAP
Expiratory P_{pl} (torr)	−3.3 ± 1.0	1.9 ± 1.2*	−2.7 ± 0.9	1.8 ± 1.2*
Expiratory P_{LVED} (torr)	4.0 ± .6	6.6 ± 1.2*	4.6 ± 1.0	7.4 ± 1.4*
Expiratory P_{TLVED} (torr)	7.3 ± .5	4.6 ± 1.3*	7.4 ± 0.8	5.6 ± 1.0*
Inspiratory P_{pl} (torr)	−6.4 ± 1.4	7.0 ± 1.6*†	−4.6 ± 1.6	−6.3 ± 1.5*
Inspiratory P_{LVED} (torr)	2.1 ± 2.0	11.6 ± 0.9*†	1.6 ± 0.8	2.6 ± 1.2
Inspiratory P_{TLVED} (torr)	8.5 ± 1.8	4.9 ± 1.5*†	6.1 ± 1.7	8.9 ± 1.9*
Cardiac output (l/min)	3.0 ± 0.3	2.2 ± 0.2*†	2.9 ± 0.3	2.6 ± 0.2
Heart rate (beats/min)	148 ± 17.8	179 ± 16.9*	139 ± 13.4	167 ± 15.4*
Stroke volume (ml/beats)	22.2 ± 2.3	13.5 ± 1.7*†	21.9 ± 2.3	17.0 ± 2.3*
$P_{\overline{AO}}$ (torr)	127 ± 2.7	120 ± 6.2	125 ± 2.8	124 ± 2.8
$P_{\overline{IVC}}$ (torr)	5.4 ± .9	9.3 ± 1.1*	5.6 ± 0.9	10.5 ± 1.4*

* $P \leq 0.05$ compared with control.
† $P \leq 0.05$ compared with CPAP.

TABLE 2. Renal Responses during Spontaneous Ventilation with Ambient Pressure (Control), Continuous Positive-pressure Ventilation (CPPV) with 7 torr PEEP, and Spontaneous Respiration with 7 torr Continuous Positive Airway Pressure (CPAP) (Mean ± 1 SEM, Nine Swine)

	Control	CPPV + 7 torr PEEP	Control	Spontaneous Respiration + 7 torr CPAP
Urinary flow (ml/30 min)	20.8 ± 4.4	13.2 ± 2.8*	21.5 ± 5.1	11.2 ± 2.0*
Glomerular filtration rate (ml/min)	53.6 ± 8.1	36.5 ± 5.5*	66.3 ± 9.0	37.7 ± 6.6*
Urinary sodium (mEq/l)	58.9 ± 17.6	48.2 ± 17.7*	55.9 ± .29	56.1 ± 19.4
Vasopressin (μU/ml)	5.2 ± 1.9	13.6 ± 2.4*	7.9 ± 1.6	10.8 ± 2.5
Free-water clearance (ml/30 min)	−24.6 ± 5.2	−21.2 ± 3.6	−24.6 ± 3.2	−17 ± 2.6
Osmolal clearance (ml/30 min)	45.4 ± 8.1	35.9 ± 4.9	46.2 ± 7.1	32.3 ± 4.1

* $P \leq 0.05$ compared with control.

increased P_{TLVED} that resulted from inspiration during CPAP. In spite of significant increases in heart rate, the decrease in volume secondary to the decreased return of venous blood during inspiration and expiration was sufficient to decrease cardiac output during CPPV. Although return of venous blood was decreased during exhalation, spontaneous inspiration and increased heart rate were able to maintain cardiac output near control levels during CPAP.

Prior investigations have shown alterations in renal function during the application of different levels of positive airway pressure.[3,6,7,11–14] There appears to be general agreement that CPPV decreases urinary flow, glomerular filtration rate, and urinary sodium excretion; increases plasma renin–angiotensin and vasopressin levels; and may alter intrarenal blood flow. Without exception, studies demonstrating alteration in renal function have also demonstrated coincidental decrease in cardiac output. Understandably, this has led investigators to assume a cause-and-effect relationship between increased expiratory airway pressure and the depression of cardiac filling, cardiac output, and renal function.

Decreased urinary flow during application of positive airway pressure has been attributed to several mechanisms. Diminished cardiac output may decrease renal perfusion, glomerular filtration rate, and therefore, urinary flow. However, such a mechanism may be questioned. Qvist *et al.*[1] infused blood into dogs receiving CPPV and returned their cardiac output values almost to pre-CPPV levels. Even though cardiac output was maintained, urinary flow remained depressed. Similarly, in our study cardiac output was decreased only by CPPV, yet we observed urinary flow and glomerular filtration rate to be decreased during both CPAP and CPPV. Such findings suggest that depression of cardiac output alone is not sufficient to explain the decreases in urinary flow and glomerular filtration rate. Rather, other factors must be responsible.

Plasma vasopressin may increase during positive airway pressure and may be responsible for decreased urinary flow.[3,11,13,15] Release of vasopressin may be controlled by several systems.[16] Hypothalamic osmoreceptors, which may sense small decreases in plasma osmolal concentration, may cause an increase in plasma vasopressin of as much as 6 μU/ml in dogs. This level of vasopressin would promote distal tubular reabsorption of water and decrease C_{H_2O} proportionately to the plasma vasopressin concentration. Activation of left atrial volume receptors may increase plasma vasopressin further, thereby increasing renal afferent arteriolar resistance. Such vasoconstriction may cause redistribution of intrarenal blood flow from cortical to juxtamedullary nephrons, thereby decreasing urinary sodium excretion and osmolal clearance. Hypotension may stimulate baroreceptors, causing further increase in plasma vasopressin concentration. Extremely high levels of plasma vasopressin may cause cessation of urinary flow and a poor correlation between vasopressin concentration and C_{H_2O}. Compared with control values, there was no change in serum sodium concentration, serum osmolality, or mean arterial blood pressure, which always exceeded 100 torr during CPPV or CPAP. If data obtained in dogs may be extrapolated, our findings suggest that osmoreceptors and baroreceptors were not responsible for increased release of plasma vasopressin during CPPV. However, left atrial receptors may have been affected differently by CPAP and CPPV. Left atrial volume receptors have been reported to respond to both volume and transmural pressure changes.[16–18] Transmural pressure was decreased more by CPPV than by CPAP. Similarly, plasma vasopressin was increased by CPPV, but not by CPAP, suggesting that a decrease in transmural left atrial pressure may have caused an increase in vasopressin release.

Arterial blood P_{CO_2} also may affect release of vasopressin.[19] However, Pa_{CO_2} values were unchanged during all ventilatory periods and therefore could not have accounted for changes in plasma vasopressin. Positive airway pressure may cause increases in renin–angiotensin; these increases have also been shown to promote release of vasopressin.[20] During CPPV the increase in mean airway pressure was greatest, as was the vasopressin level. It has been suggested that intrathoracic venous volume receptors may also stimulate release of vasopressin.[21] Activation of such receptors could explain the difference between plasma vasopressin levels during CPPV and CPAP, because thoracic inflow of blood was decreased more by CPPV. Regardless of etiology, increase in plasma vasopressin concentration could not have been responsible for the changes in renal function we observed, because vasopressin was increased by CPPV alone, whereas urinary flow was decreased equally by CPAP and CPPV.

Data concerning the relationship between plasma vasopressin concentration and renal function of subjects receiving positive airway pressure are confusing. Kumar *et al.*[13] studied patients who received CPPV and found no consistent correlation between increased plasma vasopressin concentration and C_{H_2O} or C_{osm}. We measured plasma vasopressin levels ranging from 5 μU/ml before, to 14 μU/ml during, application of positive airway pressure. Such levels of plasma vasopressin should alter urinary C_{H_2O} and C_{osm}.[16] However, we found that both functions were not altered by CPAP and CPPV. These findings are consistent with those of Kumar *et al.*[13] and suggest that the action of plasma vasopressin on distal tubular reabsorption of water and afferent arterioles may be attenuated by positive airway pressure. Another possible explanation is that an alteration of renal function that is not dependent on vasopressin occurs.

Hall *et al.*[22] demonstrated that alterations in intrarenal hemodynamics may occur during CPPV. Positive airway pressure increased perfusion of juxtamedullary nephrons and decreased perfusion of cortical nephrons. This was also observed by Moore *et al.*[23] to occur in primates. Such redistribution of renal blood flow decreased urinary flow and glomerular filtration rate. Micropuncture studies have shown that juxtamedullary nephrons are more efficient than other nephrons at sodium reabsorption.[24] Thus, a shift in renal blood flow to such areas would account for decreased urinary sodium excretion. Alterations in intrarenal blood flow induced by positive airway pressure may be secondary to increases in neural stimulation, catecholamines, vasopressin, or angiotensin. Renal cortical afferent, but not efferent, arterioles have adrenergic innervation.[3,25,26] Positive airway pressure may increase sympathetic activity or plasma catecholamines, or both.[3] Therefore, with increased stimulation by sympathetics or catecholamines, constriction of afferent arterioles could occur and could alter renal blood flow and its distribution. Circulating levels of vasopressin and angiotensin have been reported to increase during positive-pressure breathing, which may also increase afferent arteriolar tone.[3,11,14,16] Partial occlusion of the thoracic vena cava may increase inferior vena caval and renal vein pressures. This mechanism may also influence intrarenal blood flow and renal tubular function. Kilcoyne and Cannon[27] found that in dogs acute increases in inferior vena caval pressure to 9 torr decreased renal cortical blood flow and urinary sodium excretion and increased juxtamedullary blood flow. Crumb *et al.*[28]

also found glomerular filtration rate and urinary sodium excretion decreased after occlusion of the thoracic inferior vena cava. However, neither group of investigators measured cardiac output, which may have been decreased during partial occlusion of the thoracic vena cava. Both CPAP and CPPV increased thoracic vena caval pressure, which would mimic partial occlusion. Since depression of cardiac output alone could not account for the decreases in urinary flow and glomerular filtration rate that occurred during application of positive airway pressure, it seems reasonable to suggest that renal function of our animals may have been influenced by changes in the hemodynamics of the inferior vena cava.

In addition to pressure changes in the inferior vena cava, blood flow patterns to and from the kidney may have contributed to the altered renal function observed during CPAP and CPPV. Vena caval blood flow is influenced by the ventilatory pattern.[2] During spontaneous ventilation, intrapleural pressure decreases, thereby augmenting blood flow from the inferior vena cava to the heart. In addition, blood ejected into the aorta may be similarly affected by changes in intrapleural pressure. During expiration, intrapleural pressure is more positive, and, therefore, thoracic inflow of blood and filling of the aorta are decreased. Thus, arterial blood flow to and venous flow from the kidneys may reflect pulsatile patterns dependent upon respiration. During spontaneous inspiration, venous blood flows away from the kidneys, and arterial flow is augmented. During CPPV, with the abolishment of spontaneous ventilation, this may not be the case. Mechanical inspiration increases, rather than decreases, intrapleural pressure. Thus, augmentation of venous and arterial flow would be diminished by CPPV. Although we did not measure inspiratory aortic and inferior vena caval pressures, we can appreciate the change in blood flow induced by the respiratory pattern by examining the transmural inspiratory mean aortic ($P_{\overline{TAO}}$) and inferior vena caval ($P_{\overline{TIVC}}$) pressures. Subtracting inspiratory intrapleural pressure from mean aortic and inferior vena caval pressures (table 1), we obtained the following values (mean ± SEM):

	CPPV	CPAP
Inspiratory $P_{\overline{TAO}}$ (torr)	113.0 ± 1.3	130.3 ± 1.7
Inspiratory $P_{\overline{TIVC}}$ (torr)	2.3 ± 1.2	16.8 ± 1.4

During CPPV, inspiratory transmural aortic and inferior vena caval pressures were decreased, as were P_{TLVED} and stroke volume (table 1). During CPAP, inspiratory transmural aortic, inferior vena caval, and left ventricular end-diastolic pressures were greater, although stroke volume was decreased. During expiration, CPAP and CPPV decreased transmural pressures equally, which accounted for the decreased stroke volume occurring during CPAP. Mean inferior vena caval pressure was increased and stroke volume decreased by both CPAP and CPPV, which may account for similar depressions in urinary flow and glomerular filtration rate by the two airway pressure patterns. During CPPV, inspiratory venous and arterial blood flows were decreased, which may have depressed renal function further, accounting for the increased sodium reabsorption and plasma vasopressin levels during CPPV.

Succinylcholine may also affect renal function. Renal blood flow and glomerular filtration rate decrease transiently in dog and man after a bolus injection of succinylcholine. However, renal function consistently returns to normal within minutes.[29,30] During CPPV, we administered succinylcholine as a continuous infusion at a rate just sufficient to keep animals from making spontaneous respiratory efforts. Since indices of renal function in both control groups before and after CPPV were not statistically significantly different, it seems likely that succinylcholine did not affect renal function during CPPV.

Although the mechanisms are speculative, it is clear that positive airway pressure consistently altered renal function. The cardiovascular effects of positive airway pressure depend upon a complex interaction among airway pressure and intravascular blood volume, contractile state of the heart, lung and thoracic compliance, and ventilatory pattern. Therefore, it should not be surprising to find renal function similarly affected. We found urinary flow and glomerular filtration rate to be depressed by both CPPV and CPAP. Only CPPV caused an increase in plasma vasopressin and a decrease in urinary sodium concentration.These changes did not occur during CPAP, and may have been related to maintenance of cardiac output, transmural cardiac pressures, and preservation of pulsatile blood flow to and from the kidney during spontaneous inspiration. Although our animals had normal cardiopulmonary function, these findings may be applicable to patients with increased right-to-left intrapulmonary shunting of blood and decreased compliance of the lung. Airway pressure patterns, even in the presence of altered pulmonary mechanics, would still create the changes in transmural pressures and blood flow that we observed during inspiration. Airway pressure and ventilatory patterns are frequently applied to establish optimal cardiopulmonary function without consideration of their renal effects. Therefore, further studies designed to examine renal function of patients with al-

tered cardiopulmonary function during CPAP and CPPV are indicated.

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