Anesthesiology
52:142–148, 1980

Comparison of Ventilatory Patterns in the Treatment of Freshwater Near-drowning in Dogs

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The authors compared controlled mechanical ventilation (CMV) plus positive end-expiratory pressure (PEEP) with continuous positive airway pressure (CPAP) when expiratory pressure was increased in 5-cm H_2O increments to improve Pa_{O_2} and decrease intrapulmonary physiologic shunting ($\dot{Q}_{sp}/\dot{Q}_t$) in 30 dogs that had been near-drowned with fresh water (22 ml/kg). After aspiration, significant arterial hypoxemia and increased $\dot{Q}_{sp}/\dot{Q}_t$ developed in all the animals. When FI_{O_2} was increased from 0.21 to 0.4, a significant decrease in $\dot{Q}_{sp}/\dot{Q}_t$ occurred.

Thirty minutes after aspiration, the dogs were divided into four treatment groups. In dogs that breathed spontaneously with zero end-expiratory pressure (ZEEP), Group I, $\dot{Q}_{sp}/\dot{Q}_t$ did not change after the initial response to an increased FI_{O_2}. Controlled mechanical ventilation (CMV) with ZEEP, Group II, produced a further moderate decrease in $\dot{Q}_{sp}/\dot{Q}_t$. The response to spontaneous ventilation with continuous positive airway pressure (CPAP), Group III, was variable. Four dogs had decreases in $\dot{Q}_{sp}/\dot{Q}_t$ at 15 cm H_2O CPAP to less than 12 per cent of cardiac output, whereas the other six dogs had pulmonary shunting that remained above 40 per cent of cardiac output. All dogs treated with CMV plus PEEP, Group IV, had significant decreases in $\dot{Q}_{sp}/\dot{Q}_t$ at 15 to 20 cm H_2O PEEP. There was a transient increase in pulmonary artery-occluded pressure after aspiration, with a persistent increase in pulmonary vascular resistance. Cardiac output decreased significantly with CMV and with the application of 15 and 20 cm H_2O PEEP and CPAP. It was lowest with the combination of CMV plus PEEP.

This study suggests that PEEP was most effective in reversing $\dot{Q}_{sp}/\dot{Q}_t$ and hypoxemia after freshwater near-drowning. This effect was most consistent in those animals receiving CMV, despite significant decrements in cardiac output. Continuous positive airway pressure alone was variably effective. (Key words: Drowning. Lung: shunting. Ventilation: continuous positive airway pressure; continuous positive-pressure breathing; positive end-expiratory pressure; shunting; zero end-expiratory pressure.)

PREVIOUS STUDIES of dogs have shown that controlled mechanical ventilation (CMV) must be combined with positive end-expiratory pressure (PEEP) to improve arterial blood oxygen tension (Pa_{O_2}) after aspiration of fresh water.[1] Those studies used a fixed level of 10 cm H_2O PEEP. In clinical practice, rather than being set at one level, PEEP is frequently varied to obtain optimal response in Pa_{O_2}, physiologic intrapulmonary shunt ($\dot{Q}_{sp}/\dot{Q}_t$), or compliance. During treatment of human victims of freshwater near-drowning, we noticed that in some patients Pa_{O_2} increased and $\dot{Q}_{sp}/\dot{Q}_t$ decreased during spontaneous ventilation with varied PEEP or continuous positive airway pressure (CPAP), but others needed the addition of CMV to PEEP for optimal improvement (personal observation). In this study we compared CMV plus PEEP with CPAP when expiratory pressure was varied to improve Pa_{O_2} and decrease intrapulmonary physiologic shunting in dogs near-drowned with fresh water.

Methods

Thirty mongrel dogs weighing 21 ± 3 kg (mean ± SD) were anesthetized with sodium pentobarbital, 25 mg/kg, intravenously, and their tracheas were intubated with cuffed endotracheal tubes connected to a constant-volume ventilator (J. H. Emerson Co.) equipped with an air–oxygen blender (bird Corp.). This system allowed us to deliver CMV with zero end-expiratory pressure (ZEEP), CMV with PEEP or continuous positive-pressure ventilation (CPPV), or spontaneous ventilation with either ZEEP or CPAP. Additional 25-mg increments of sodium pentobarbital were given as needed to prevent spontaneous movement. Lactated Ringer's solution at a rate of approximately 12 ml/kg/h was given for maintenance. Femoral-artery and 5-Fr quadruple-lumen thermodilution, flow-directed pulmonary-artery catheters were placed percutaneously. Position of the pulmonary-artery catheter was verified by intravascular pressure tracing. The animals' body temperatures were maintained at 37 ± 1 C with heat lamps when necessary. Femoral arterial blood pressure (BP), mean pulmonary arterial pressure ($\overline{PAP}$). Pulmonary artery-occluded pressure ($\overline{PAOP}$), heart rate (HR), respiratory rate (f), and temperature were monitored. Cardiac output ($\dot{Q}_t$) was calculated by the thermodilution method (IL 601, Instrumentation Laboratories, Inc.), using the mean of three successive determinations during exhalation. The IL 113 electrode system was used to measure pH_a, Pa_{CO_2}, Pa_{O_2}, $pH_{\bar{v}}$, $P\bar{v}_{CO_2}$, and $P\bar{v}_{O_2}$; these values were corrected to body temperature. Hematocrit was measured by the microcapillary method. Right-to-left

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Received from the Department of Anesthesiology, University of Florida College of Medicine, Gainesville, Florida 32610. Accepted for publication July 20, 1979. Presented in part at the annual meeting of the American Society of Anesthesiologists, Chicago, Illinois, October 1978. Supported in part from the Edwin Bailey fund for Research in Drowning.

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TABLE 1. Intrapulmonary Physiologic Shunt Fraction ($\dot{Q}_{sp}/\dot{Q}_t$) (Mean ± SD) Pre- and Post-Freshwater Aspiration and During Spontaneous Ventilation (Group I), CMV (Group II), CPAP (Group III), or CMV with PEEP (Group IV): Statistically Significant Differences ($P < 0.05$) are Marked and Defined

Minutes after Aspiration	$F_{I_{O_2}}$	Group I Spontaneous with ZEEP	Group II CMV with ZEEP	PEEP or CPAP (cm H_2O) →	Group III Spontaneous with CPAP	Group IV CMV with PEEP
0	.21	0.10 ± 0.04	0.12 ± 0.06	0	0.10 ± 0.05	0.11 ± 0.06
15	.21	0.72 ± 0.12*	0.78 ± 0.08*	0	0.69 ± 0.12*	0.75 ± 0.08*
30	.21	0.68 ± 0.08*	0.64 ± 0.16*	0	0.67 ± 0.10*	0.67 ± 0.11*
45	.40	0.60 ± 0.16*	0.53 ± 0.14*	0	0.59 ± 0.13*§	0.48 ± 0.10*
60	.40	0.61 ± 0.15*	0.42 ± 0.11*	5	0.46 ± 0.13*§†	0.29 ± 0.12*†‡
75	.40	0.58 ± 0.15*	0.44 ± 0.05*§	10	0.35 ± 0.16*†‡	0.22 ± 0.13*†‡
90	.40	0.55 ± 0.16*	0.41 ± 0.12*§	15	0.28 ± 0.18*†‡	0.19 ± 0.17†‡
105	.40	0.61 ± 0.18*	0.42 ± 0.09*§	20	0.29 ± 0.19*§†‡	0.13 ± 0.10†‡
120	.40	0.62 ± 0.13*	0.38 ± 0.07*‡	0	0.42 ± 0.18*†‡	0.40 ± 0.13*‡

CMV

* $P < 0.05$ when compared with values at zero time period for the same group.
† $P < 0.05$ when compared with values at the 45-minute time period for the same group.
‡ $P < 0.05$ when compared with Group I at the same time period.
§ $P < 0.05$ when comparing Group II or Group III with Group IV at the same time period.

intrapulmonary physiologic shunt fraction ($\dot{Q}_{sp}/\dot{Q}_t$) was calculated with the computer program of Ruiz *et al.*,[2] and oxygen availability was calculated as $Ca_{O_2} \times \dot{Q}_t \times 10$. Pulmonary vascular resistance (PVR) was calculated as

$$PVR = \frac{\overline{PAP} - \overline{PAOP}}{\dot{Q}_t} \times 79.98$$

Control data were obtained while the animals spontaneously breathed room air. To qualify for this study, $Pa_{O_2} \geq 70$ torr and $Pa_{CO_2} \leq 45$ torr were required of all dogs immediately after induction of anesthesia. All dogs aspirated distilled water, 22 ml/kg, at time zero via a gravity-flow device described previously.[3] After aspiration, the animals were allowed to breathe room air spontaneously at ZEEP, and all measurements and calculations were made 15 and 30 min later. Next, the fraction of inspired oxygen ($F_{I_{O_2}}$) was increased to 0.4. Fifteen dogs, chosen at random, were paralyzed with a continuous infusion of succinylcholine hydrochloride and CMV was instituted with a tidal volume (V_T) of 15 ml/kg and f 10/min. The remaining 15 animals continued to breathe spontaneously. After 15 min had elapsed, data were obtained.

The two groups were then divided into two subgroups. Each of the four resultant groups received one of four treatment modalities. Five dogs were allowed to continue breathing spontaneously at ZEEP (Group I). Another five dogs were ventilated by CMV and ZEEP (V_T = 15 ml/kg; f = 10/min) (Group II). Ten dogs were allowed to breathe spontaneously and CPAP was increased by 5 cm H_2O every 15 min until 20 cm H_2O was reached (Group III). Ten dogs received CMV (V_T = 15 ml/kg; f = 10/min) and PEEP was increased by 5 cm H_2O every 15 min until 20 cm H_2O was reached (Group IV). Data were obtained 15 min after establishment of the new conditions. Finally, end-expiratory pressure was decreased abruptly from 20 cm H_2O to zero in Groups III and IV and data were obtained after 15 min.

The data were analyzed by use of Student *t* tests for paired and unpaired data to determine statistical significances of differences between time periods in each group and also among groups at equivalent time periods. $P < 0.05$ was regarded as significant.

Results

Significant increase in $\dot{Q}_{sp}/\dot{Q}_t$ developed after aspiration in all the animals (table 1). When $F_{I_{O_2}}$ was increased from 0.21 to 0.40 in the 14 animals¶ that breathed spontaneously after near-drowning (Groups I and III), $\dot{Q}_{sp}/\dot{Q}_t$ decreased an average of 8 per cent of cardiac output within 15 min. The 15 dogs that had their ventilation controlled during breathing of 40 per cent oxygen (Groups II and IV) had an average decrease of shunting of 15 per cent within 15 min. The difference between the decreases in shunting of the dogs that breathed spontaneously and those treated by CMV was significant.

During the 75-min treatment period (from 30 to 105 min after aspiration), $\dot{Q}_{sp}/\dot{Q}_t$ did not change significantly in the five animals that breathed spontaneously with ZEEP (Group I) or in the five animals that had CMV with ZEEP (Group II) (table 1).

The ten animals that were treated with CPAP and spontaneous ventilation (Group III) had variable responses. The mean $\dot{Q}_{sp}/\dot{Q}_t$ decreased with each 5-cm

¶ Blood was not analyzed for one animal at this time because of technical difficulties.

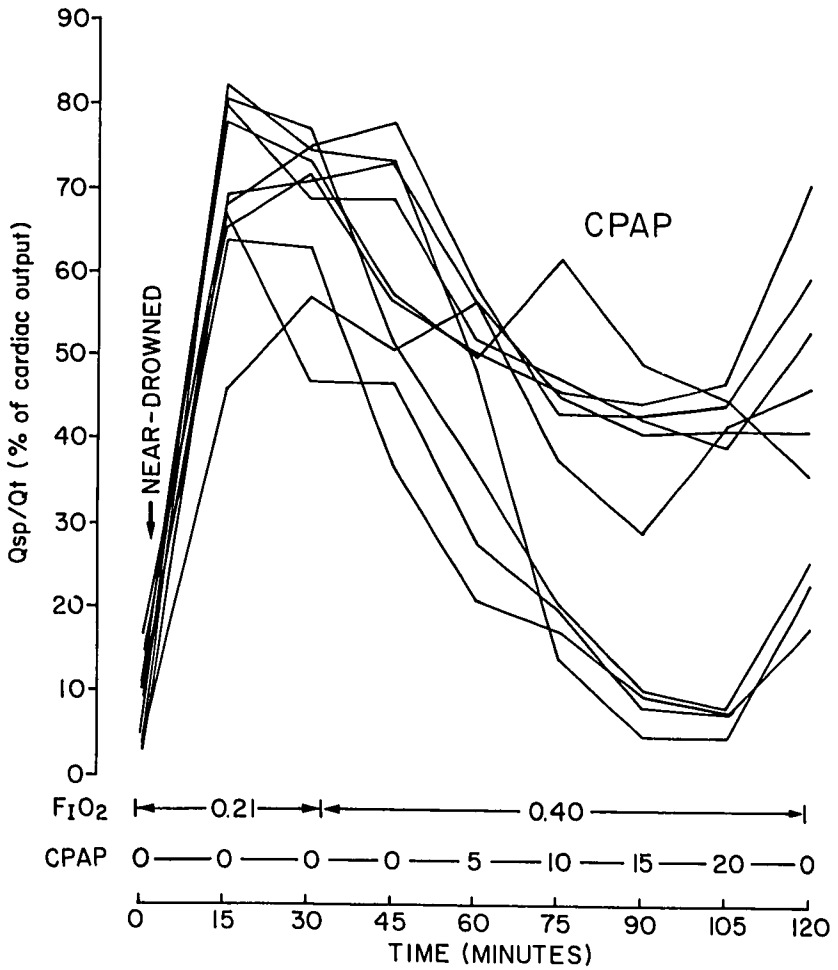

FIG. 1. Individual variations in intrapulmonary physiologic shunt fraction ($\dot{Q}_{sp}/\dot{Q}_t$) of each animal after freshwater aspiration and treatment with CPAP, which was varied from 0 to 20 cm H_2O. The $F_{I_{O_2}}$ was changed from 0.21 to 0.40 30 min after aspiration in all animals.

H_2O increment of CPAP from 0 to 15 cm H_2O CPAP (table 1). At each level of CPAP, the $\dot{Q}_{sp}/\dot{Q}_t$ was less than at ZEEP. The $\dot{Q}_{sp}/\dot{Q}_t$ also was less at 15 and 20 cm H_2O CPAP than at 5 cm H_2O CPAP. When examining the responses of the individual animals in this group to increasing levels of CPAP, we saw two distinctly different patterns. Four of the ten animals had marked decreases in $\dot{Q}_{sp}/\dot{Q}_t$ with 15 cm H_2O CPAP to only 5 to 10 per cent of cardiac output. The remaining six animals had $\dot{Q}_{sp}/\dot{Q}_t$ greater than 40 per cent of their cardiac outputs at 20 cm H_2O CPAP (fig. 1).

In the ten animals that had PEEP applied while CMV was being administered (Group IV), $\dot{Q}_{sp}/\dot{Q}_t$ decreased with each incremental increase in PEEP. The $\dot{Q}_{sp}/\dot{Q}_t$ was less at all levels of PEEP than with ZEEP. The range of $\dot{Q}_{sp}/\dot{Q}_t$ in the individual dogs at 20 cm H_2O PEEP with CMV was 4 to 32 per cent of cardiac output; seven of the ten animals had shunting of 12 per cent or less at that time.

All of the animals experienced acidemia after aspiration, but the treatment groups could not be differentiated from controls for either pH_a or Pa_{CO_2} (table 2). Changes in Pa_{O_2} followed an inverse relationship to intrapulmonary physiologic shunting (table 2).

The $\overline{PAOP}$ in the 30 dogs increased significantly from 6 ± 4 torr before near-drowning to 12 ± 7 torr 15 min after aspiration. At 30 min, it had decreased to 8 ± 6 torr, and by 45 min, it was no longer significantly increased. The $\overline{PAOP}$ increased when PEEP was applied in Groups III and IV; however, since intrapleural pressure was not measured, we were not able to calculate the effective transmural filling pressure (fig. 2).

Peripheral vascular resistance increased significantly from 183 ± 82 to 259 ± 123 dynes/sec/cm^{-5}, 15 min after the 30 dogs aspirated water. The values were still significantly increased 30 min and 45 min after aspiration, and for the control animals (Group I), they remained so at all time periods studied throughout the experiment (fig. 3). The two groups treated with end-expiratory pressure also had significantly increased PVR even when mean Pa_{O_2} had returned to at least 99 torr.

Fifteen minutes after aspiration, cardiac output decreased from 4.4 ± 0.9 to 3.4 ± 1.0 l/min in the 30

TABLE 2. Arterial Blood P_{O_2}, P_{CO_2}, and *p*H Pre- and Post-Freshwater Aspiration and During Spontaneous Ventilation (Group I), CMV (Group II), CPAP (Group III), or CMV with PEEP (Group IV) (Mean ± SD)

	Minutes after Aspiration								
	0 $F_{I_{O_2}}$ .21	15 $F_{I_{O_2}}$ .21	30 $F_{I_{O_2}}$ .21	45 $F_{I_{O_2}}$ .40	60 $F_{I_{O_2}}$ .40	75 $F_{I_{O_2}}$ .40	90 $F_{I_{O_2}}$ .40	105 $F_{I_{O_2}}$ .40	120 $F_{I_{O_2}}$ .40
Group I, Spontaneous with ZEEP									
P_{O_2} (torr)	90 ± 7	29 ± 5*†	34 ± 5*†	50 ± 9*	51 ± 7*	48 ± 12*	47 ± 10*	46 ± 12*	45 ± 11*
P_{CO_2} (torr)	33 ± 2	49 ± 3*	45 ± 6*	46 ± 9*	42 ± 7*	38 ± 4*	37 ± 6	39 ± 9	39 ± 10
*p*H	7.39 ± .03	7.25 ± .03*	7.24 ± .06*	7.23 ± .07*	7.26 ± .06*	7.26 ± .05*	7.31 ± .08*	7.32 ± .10	7.27 ± .12*
Group II, CMV with ZEEP									
P_{O_2}	86 ± 6	25 ± 5*†	30 ± 5*†	49 ± 10*	53 ± 6*§	59 ± 7*§	62 ± 11*‡§	57 ± 7*§	60 ± 13*
P_{CO_2}	35 ± 8	50 ± 6*	51 ± 10*	50 ± 15*	44 ± 8	41 ± 7	43 ± 8	40 ± 7	41 ± 6
*p*H	7.41 ± .04	7.22 ± .03*	7.18 ± .07*	7.16 ± .11*	7.26 ± .13*	7.22 ± .14*	7.24 ± .13*	7.26 ± .13*	7.27 ± .12*
PEEP or CPAP (cm H_2O) ↓	0	0	0	0	5	10	15	20	0
Group III, Spontaneous with CPAP									
P_{O_2}	94 ± 18	38 ± 6*†	35 ± 7*†	52 ± 14*	60 ± 17*	78 ± 29†‡	99 ± 52†‡	101 ± 57†‡	65 ± 24*
P_{CO_2}	33 ± 5	45 ± 8*	43 ± 8*	46 ± 11*	45 ± 10*	44 ± 9*	44 ± 9*	44 ± 9*	35 ± 11†
*p*H	7.40 ± .04	7.26 ± .08*	7.28 ± .11*	7.23 ± .06*	7.28 ± .06*	7.29 ± .05*†	7.25 ± .06*	7.20 ± .06*‡	7.25 ± .07*
Group IV, CMV with PEEP									
P_{O_2}	85 ± 12	30 ± 3*†	33 ± 6*†	56 ± 10*	83 ± 27†‡	103 ± 40†‡	125 ± 43†‡	121 ± 38†‡	61 ± 12*‡
P_{CO_2}	38 ± 14	42 ± 7	44 ± 9	47 ± 7	42 ± 9	41 ± 11	45 ± 11	51 ± 10*	43 ± 6
*p*H	7.40 ± .02	7.25 ± .11*	7.29 ± .09*	7.23 ± .06*	7.28 ± .06*	7.29 ± .05*‡	7.25 ± .06*	7.20 ± .06*‡	7.25 ± .07*

* $P < 0.05$ when compared with values at zero time period for the same group.
† $P < 0.05$ when compared with values at the 45-minute time period for the same group.
‡ $P < 0.05$ when compared with Group I at the same time period.
§ $P < 0.05$ when comparing Group II or Group III with Group IV at the same time period.

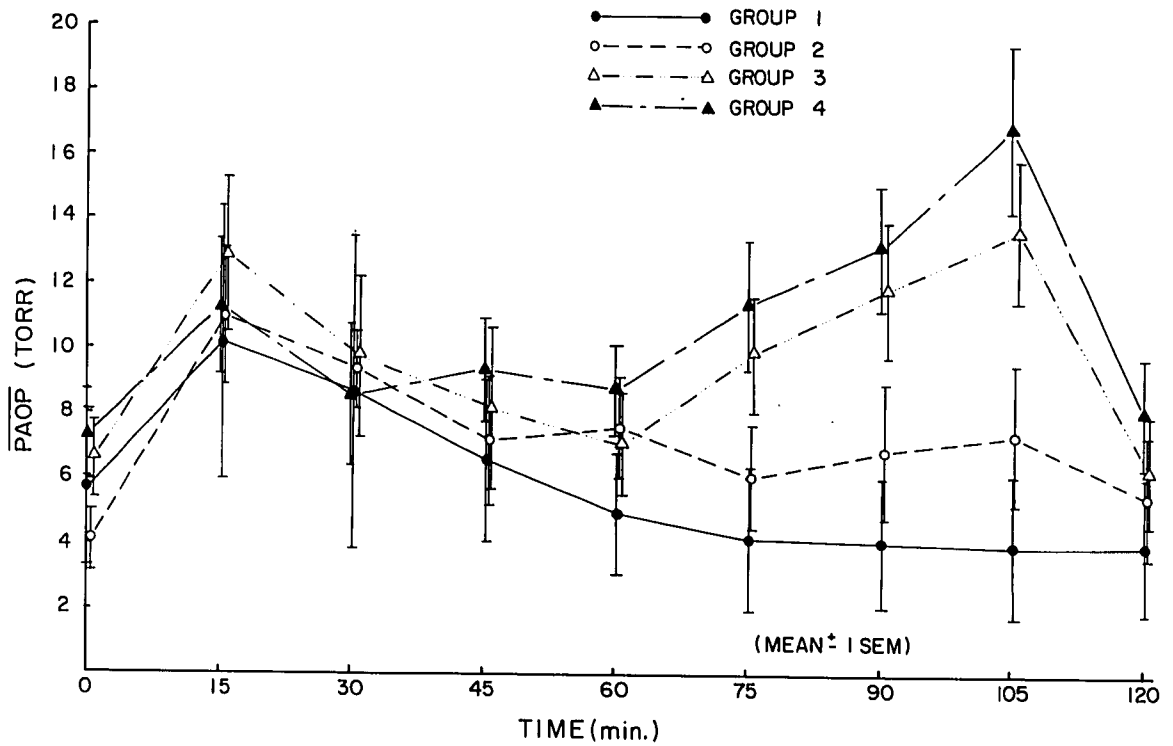

FIG. 2. Pulmonary arterial occlusion pressure ($\overline{PAOP}$) (mean ± 1 SE) before and after near-drowning in animals permitted to breathe spontaneously at ZEEP (Group I), with CMV (Group II), with CPAP (Group III), and with CPPV (Group IV). All groups had significant increases in $\overline{PAOP}$ 15 min after aspiration. $\overline{PAOP}$ then returned to pre-aspiration levels, but became significantly increased again at 75, 90, and 105 min in Groups III and IV.

dogs. However, 30 min after aspiration, cardiac output values for the dogs did not differ from those measured prior to near-drowning. Treatment with CMV alone decreased cardiac output significantly (table 3). Application of 5 and 10 cm H_2O CPAP did not decrease cardiac output in spontaneously breathing dogs, nor did 5 or 10 cm H_2O PEEP further decrease cardiac output in animals receiving CMV. However, application of 15 and 20 cm H_2O PEEP with both spontaneous and controlled ventilation did significantly decrease cardiac output. Cardiac output was depressed more when CMV was being given simultaneously with 15 and 20 cm H_2O PEEP than with spontaneous ventilation and CPAP. When the relationship between cardiac output and $\dot{Q}_{sp}/\dot{Q}_t$ in the animals receiving positive end-expiratory pressure was subjected to regression analysis, there was a significant correlation in the dogs breathing spontaneously ($n = 58$; $r = 0.55$; $P < 0.001$), but not in those whose ventilation was controlled mechanically ($n = 60$; $r = 0.19$; $P > 0.2$).

Fifteen minutes after PEEP was abruptly decreased from 20 cm H_2O to zero in Groups III and IV, there were significant increases in $\dot{Q}_{sp}/\dot{Q}_t$ and cardiac output and a decrease in Pa_{O_2} values (tables 1, 2, and 3).

Oxygen availability decreased significantly after near-drowning and remained depressed in all four groups. When comparing groups, we saw no difference among treated and untreated animals at any time period except during CMV with 20 cm H_2O PEEP.

Discussion

All dogs that aspirated distilled water had severe arterial hypoxemia and intrapulmonary physiologic shunting. Spontaneous breathing of 40 per cent oxygen for 15 min decreased $\dot{Q}_{sp}/\dot{Q}_t$, which suggests that, although $\dot{Q}_{sp}/\dot{Q}_t$ was caused largely by perfused areas of lung that had no ventilation, a fraction of the $\dot{Q}_{sp}/\dot{Q}_t$ can be categorized as relative shunting, or areas of low but finite ventilation-to-perfusion ratio.[4] A further decrease in $\dot{Q}_{sp}/\dot{Q}_t$ occurred when CMV was applied while the dogs breathed 40 per cent oxygen. This suggests that CMV either further recruits alveoli to participate in gas exchange or decreases cardiac output, either of which produces a secondary decrease in $\dot{Q}_{sp}/\dot{Q}_t$. However, we could not demonstrate a significant correlation between cardiac output and $\dot{Q}_{sp}/\dot{Q}_t$ in animals whose ventilation was controlled, thus suggesting that recruitment occurred.

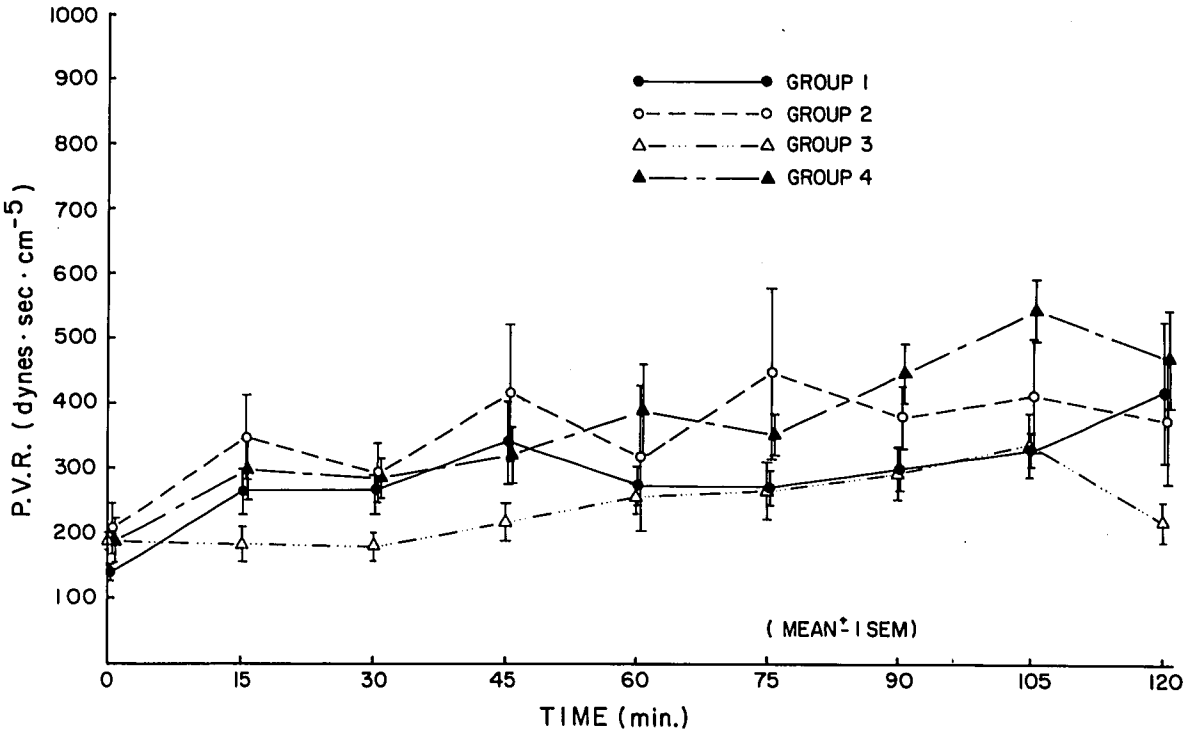

FIG. 3. Pulmonary vascular resistance (PVR) (mean ± 1 SE) before and after near-drowning in animals permitted to breathe spontaneously at ZEEP (Group I), with CMV (Group II), with CPAP (Group III), and with CPPV (Group IV). The PVR was significantly increased at at least four time periods after aspiration in all groups.

We found that CPAP significantly increased Pa_{O_2} and decreased $\dot{Q}_{sp}/\dot{Q}_t$ in some animals but not others. However, every animal treated with CMV and PEEP had a significant improvement in arterial oxygenation and decrease in shunting. It has been shown that freshwater aspiration alters the surface tension properties of pulmonary surfactant and thus promotes alveolar instability and collapse.[5] It may be that some of our animals needed a greater transpulmonary pressure than they could spontaneously generate to open alveoli. Therefore, CPAP at the levels used was unable to maintain a sufficient functional residual capacity (FRC) to better match $\dot{V}_A/\dot{Q}$. By producing higher peak-inflation pressure and thereby, increased transpulmonary pressure, CMV may have effectively opened alveoli in these animals' lungs and allowed PEEP to maintain a more normal FRC.

The increase in $\overline{PAOP}$ seen immediately after as-

TABLE 3. Cardiac Output (Mean ± SD) Pre- and Post-Freshwater Aspiration and during Spontaneous Ventilation (Group I), CMV (Group II), CPAP (Group III), or CMV with PEEP (Group IV): Statistically Significant Differences ($P < 0.05$) are Marked and Defined

Minutes after Aspiration	$F_{I_{O_2}}$	Cardiac Output (l/min) Group I Spontaneous with ZEEP	Cardiac Output (l/min) Group II CMV with ZEEP	PEEP or CPAP (cm H_2O) →	Cardiac Output (l/min) Group III Spontaneous with CPAP	Cardiac Output (l/min) Group IV CMV with PEEP
0	.21	4.0 ± 0.5	4.4 ± 1.0	0	4.3 ± 0.9	4.6 ± 1.2
15	.21	3.4 ± 0.8	2.9 ± 0.6*	0	3.7 ± 1.1	3.5 ± 1.2*
30	.21	3.4 ± 0.8	4.1 ± 1.2	0	4.0 ± 1.4	3.9 ± 1.0†
45	.40	3.3 ± 1.2	2.5 ± 1.4*	0	3.7 ± 1.1	2.8 ± 0.9*
60	.40	3.2 ± 0.6*	2.6 ± 1.1*	5	3.5 ± 0.9	2.6 ± 0.9*
75	.40	3.2 ± 0.6*	2.4 ± 1.0*	10	3.2 ± 1.1*	2.5 ± 0.7*
90	.40	3.1 ± 0.5*	2.3 ± 0.3*‡	15	2.7 ± 1.0*†§	1.9 ± 0.5*†‡
105	.40	2.9 ± 0.3*	2.1 ± 0.5*‡§	20	2.4 ± 0.7*†§	1.6 ± 0.3*†‡
120	.40	3.0 ± 1.1	2.5 ± 0.9*	0	3.7 ± 1.3§	2.4 ± 0.8*

CMV ↓ (Group II) CMV ↓ (Group IV)

* $P < 0.05$ when compared with values at zero time period for the same group.

† $P < 0.05$ when compared with values at the 45-minute time period for the same group.

‡ $P < 0.05$ when compared with Group I at the same time period.

§ $P < 0.05$ when comparing Group II and Group III with Group IV at the same time period.

piration was probably due to a temporary hypervolemia from rapid absorption of the distilled water. The return of $\overline{PAOP}$ to normal within 30 to 45 min is consistent with results of previous studies that have shown that the hypervolemia seen with aspiration of this quantity of fluid is transient.[6] An increase in PVR, presumably in response to hypoxia, occurred immediately after aspiration. Although this may have contributed to the decrease in cardiac output that was observed, the fact that cardiac output had increased by 30 min, while PVR was still 67 dynes/sec/cm^{-5} above normal and $\overline{PAOP}$ was 2 torr above normal, suggests the heart was not capable of handling the acute increase in blood volume that resulted from water aspiration, but recovered once this fluid was redistributed and no longer caused a significant increase in $\overline{PAOP}$.

The results of this study differed from those of a previous study of the effect of PEEP therapy on near-drowned dogs breathing spontaneously reported by this laboratory.[1] In that experiment, we did not exceed 10 cm H_2O PEEP. In the current study, eight of ten dogs had less $\dot{Q}_{sp}/\dot{Q}_t$ at 15 cm H_2O CPAP than at 10 cm H_2O CPAP, which emphasizes the importance of individualizing positive airway pressure. Also, in the previous study, inspiratory pressures were permitted to decrease below ambient whereas in the present study, CPAP was used to minimize decreases in inspiratory pressure. In clinical practice, we have observed a decrease in $\dot{Q}_{sp}/\dot{Q}_t$ and an increase in compliance when some patients are converted from PEEP to CPAP while breathing spontaneously even though the transpulmonary pressure is decreased. Perhaps this difference in pressure pattern may have helped keep lung units functional during all portions of the respiratory cycle in the dogs that had significantly decreased $\dot{Q}_{sp}/\dot{Q}_t$ when CPAP was applied during spontaneous ventilation.

In the present study, we made no attempt to augment effective circulating blood volume or cardiac output when PEEP was increased. Cardiac output was most severely affected in those animals with CMV and PEEP administered simultaneously. Thus, even though Pa_{O_2} was increased and $\dot{Q}_{sp}/\dot{Q}_t$ decreased compared with untreated animals, oxygen availability was not improved. This finding prevents us from making any definitive recommendations as to the direct transfer of these animal studies to human application. We conclude that PEEP is the single most important variable in reversing the arterial hypoxemia in freshwater near-drowning. An adequate response can be produced by means of spontaneous ventilation and CPAP in some instances, but in others, it may be necessary to combine mechanical inflation of the lung with PEEP to improve arterial oxygenation significantly. When treating an individual patient, we must consider the effects of therapy on both cardiac output and $\dot{Q}_{sp}/\dot{Q}_t$. If a significant decrease in cardiac output occurs, it may be necessary to augment cardiac output to ensure more adequate oxygen delivery.

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