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Effects of Ventilatory Patterns on Arterial Oxygenation after Near-drowning in Sea Water

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Forty-three dogs were anesthetized and subjected to aspiration of 22 ml/kg of sea water. After 5 minutes, fluid was drained from the lungs by gravity; 33.1 ± 5.9 ml/kg were recovered. Thirty-four dogs were apneic at this time and were treated with intermittent positive-pressure ventilation with a self-inflating bag. Forty-five minutes later, the 40 animals that survived were divided into four equal groups; one group breathed spontaneously and served as a control, the second was treated with IPPV, the third breathed spontaneously against 10 cm H_2O PEEP, and the fourth received IPPV plus PEEP (*i.e.*, CPPV). Arterial oxygen tensions of the animals in both groups with PEEP significantly increased during the 75-minute treatment period. By 48 hours two more dogs had died; however, Pa_{O_2}'s had returned to normal in the 38 that survived, regardless of the mode of treatment. It is concluded that gravity drainage and immediate mechanical ventilation of victims who aspirate large quantities of sea water are important, since 40 of the 43 animals were resuscitated after being submerged for 5–10 minutes. Blood-gas data showed that positive end-expiratory pressure, with or without mechanical ventilation, significantly increased Pa_{O_2} after aspiration of sea water, suggesting that it is indicated in the treatment of sea-water near-drowning victims. Two case reports of human victims of near-drowning in sea water which support the animal studies are presented. (Key words: Complications, aspiration: sea water; Ventilation, positive end-expiratory: aspiration; Ventilation, intermittent positive-pressure: aspiration.)

IT IS WELL KNOWN that pulmonary edema and hypoxemia occur after aspiration of sea water.[1,2] The hypoxemia is largely secondary to true or absolute intrapulmonary shunting.[3] Recently, increasing functional residual capacity (FRC) by combining intermittent positive-pressure ventilation (IPPV) with positive end-expiratory pressure (PEEP), *i.e.*, continuous positive-pressure ventilation (CPPV), has been shown to increase Pa_{O_2} in some patients with severe respiratory insufficiency.[4] Furthermore, Cheney and Martin have shown that this mode of ventilatory support will significantly decrease shunt and increase Pa_{O_2} in animal models of pulmonary edema, so long as PEEP is applied.[5] The above suggest that this treatment also might be beneficial in treating victims of near-drowning. Therefore, the following experiment was designed to determine whether PEEP alone, or in combination with mechanical ventilation, can improve arterial oxygenation during treatment of near-drowning in sea water in dogs.

Methods

Forty-three dogs (33 mongrels and ten beagles) weighing 12.5 ± 2.2 kg were anesthetized with sodium pentobarbitol, 25 mg/kg, intravenously. The trachea of each dog was intubated with a cuffed endotracheal tube, a catheter was placed in the femoral artery, and an esophageal thermistor was inserted. The animals breathed 100 per cent oxygen via a

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nonrebreathing system for 15–20 minutes and arterial blood was analyzed for pH, P_{CO_2} and P_{O_2} with appropriate electrodes. The animals then breathed room air for 15–20 minutes before a second blood sample was drawn for analysis. All values were corrected for body temperature. Following completion of the baseline studies, the animals aspirated 22 ml/kg of sea water¶ through a chamber described previously.[6] At the end of 5 minutes, the animals were placed in a head-down position and fluid was permitted to drain by gravity. If the animal was apneic at this time, resuscitation was attempted by use of IPPV with a self-inflating bag containing air. Pa_{O_2}, Pa_{CO_2}, and pH_a were determined 15 and 30 minutes after aspiration. The dogs then breathed 100 per cent oxygen for 15 minutes and a third postaspiration blood gas sample was drawn. Three dogs died during this 45-minute interval. The 40 animals that survived were divided into four equal groups and treated for the next 75 minutes as follows.

Group I served as the control. Ten dogs breathed 100 per cent oxygen spontaneously for 1 hour through a nonrebreathing system, and then breathed room air for 15 minutes.

Group II received PEEP only. Ten animals breathed spontaneously through a nonrebreathing system. The exhalation valve was connected to 10 cm H_2O positive end-expiratory pressure (PEEP). $F_{IO_2} = 1.0$ for 60 minutes and 0.21 for 15 minutes.

Group III received respirator therapy only (IPPV). Each of the ten animals was paralyzed with succinylcholine and its ventilation controlled with a volume-limited respirator (Emerson Post-Operative Ventilator) with a tidal volume of 15 ml/kg. The rate was adjusted to maintain Pa_{CO_2} between 35 and 45 torr. $F_{IO_2} = 1.0$ for 60 minutes and 0.21 for 15 minutes. After 75 minutes of mechanical ventilation, the dogs were permitted to breathe spontaneously.

Group IV received respirator therapy and PEEP (*i.e.*, CPPV). These ten dogs were treated like Group III, except that the exhalation port of the ventilator was connected to a system which produced 10 cm H_2O PEEP.

¶ Obtained from the Atlantic Ocean, 5 miles east of Jacksonville, Florida. Electrolyte concentrations were: sodium 484 mEq/l, potassium 10.2 mEq/l, chloride 578 mEq/l, magnesium 109 mEq/l, and calcium 21.2 mEq/l.

Additional blood gas tensions and pH values were determined at intervals over the next 3 hours at both $F_{IO_2} = 0.21$ and $F_{IO_2} = 1.0$. The arterial catheter then was removed and the incision closed. All surviving animals were placed in an oxygen tent containing approximately 40 per cent oxygen for at least 24 hours or until a Pa_{O_2} above 70 torr was maintained during breathing of room air. At 24, 48, 72, and 168 hours blood samples were obtained from the femoral artery while the dogs breathed room air and analyzed for pH, P_{CO_2} and P_{O_2}. The temperature of each animal was taken and its head was inserted into a polyethylene bag into which 100 per cent oxygen flowed at a rate of 25–35 l/min. Holes made in the bag permitted the excess oxygen and carbon dioxide to escape freely. After 15–20 minutes another sample of arterial blood was obtained and analyzed.

Results

Of the 43 animals subjected to aspiration of 22 ml/kg of sea water, 42 were still under water 5 minutes later. We drained 33.1 ± 5.9 ml/kg of fluid from the 43 dogs by gravity. Nine dogs made ventilatory efforts, but only five appeared to move any air at this time. The remaining 34 dogs were apneic. When intermittent positive-pressure ventilation with a self-inflating bag containing air was applied, 33 of the latter dogs regained spontaneous ventilation; however, two died within 27 minutes. One of the survivors required closed-chest cardiac massage in addition to IPPV. All dogs still alive 45 minutes after aspiration, when the different modes of treatment were initiated, had severe arterial hypoxemia (tables 1 and 2). Hypercarbia and acidosis also were observed after aspiration (tables 3 and 4).

During the 75-minute experimental treatment period, Pa_{O_2}'s increased slightly in the control animals. Pa_{O_2}'s of the animals ventilated mechanically without PEEP were not significantly different from these values (tables 1 and 2). The animals treated with mechanical ventilation plus PEEP (CPPV) had the highest mean Pa_{O_2}'s during the treatment period (fig. 1). Within 15 minutes of initiation of treatment,

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TABLE 1. Arterial Oxygen Tension (torr) before and after Aspiration of 22 ml/kg Sea Water at F_{IO_2} = 1.0 (Mean ± SD)

	Preaspiration		Hours after Aspiration									
			Treatment				Posttreatment					
	0	¼ Hour	1	1¼	1½	1¾	3¼	4¼	24	48	72	168
Group I, spontaneous ventilation	574 ±47	115* ± 98	125* ±116	136* ±105	159* ±117	177* ±132	216* ±185	270* ±197	477 ±154	477 ±154	492 ±100	518 ±66
Group II, spontaneous ventilation and 10 cm H_2O PEEP	562 ±67	182* ±110	491† ±126	473† ±140	510† ± 80	521† ± 71	310* ±197	333* ±188	521 ± 57	526 ± 31	553 ± 42	543 ±48
Group III, IPPV (15 ml/kg) tidal volume	561 ±42	164* ±118	161* ± 85	181* ±113	200* ±132	205* ±123	294* ±178	284* ±190	410* ±147	464 ±146	487 ±102	517 ±29
Group IV, CPPV (15 ml/kg) and 10 cm H_2O PEEP	551 ±31	141* ±121	555† ± 77	552† ± 86	534† ± 90	537† ± 87	356* ±191	387 ±188	496 ±110	518 ± 64	522 ± 81	536 ±46

* Compared with zero time for the group, $P < 0.01$.
† Significantly higher than Group I value, $P < 0.001$.

TABLE 2. Arterial Oxygen Tension (torr) before and after Aspiration of 22 ml/kg Sea Water at F_{IO_2} = 0.21 (Mean ± SD)

	Preaspiration	Hours after Aspiration									
				Treatment	Posttreatment						
	0	¼	½	2	2½	3	4	24	48	72	168
Group I, spontaneous ventilation	93 ±14	41* ±18	40* ± 7	52* ±15	52* ±13	54* ±16	55* ±16	85 ±17	92 ±18	96 ±14	97 ±7
Group II, spontaneous ventilation and 10 cm H_2O PEEP	98 ±10	32* ± 8	41* ± 8	81*† ± 7	65* ±18	60* ±21	70* ±23	95 ±14	96 ±11	101 ± 9	98 ±8
Group III, IPPV (15 ml/kg) tidal volume	94 ±15	37* ±14	42* ±11	59* ±19	55* ±23	53* ±26	63* ±25	79 ±21	83 ±25	90 ±16	89 ±9
Group IV, CPPV (15 ml/kg) and 10 cm H_2O PEEP	89 ±11	37* ± 9	44* ± 8	88† ±14	68* ±20	67* ±19	65* ±16	92 ±10	93 ± 6	97 ± 9	99 ±8

* Compared with zero time for the group, $P < 0.01$.
† Significantly higher than Group I value, $P < 0.001$.

TABLE 3. Arterial pH (Mean ± SD) before and after Aspiration of 22 ml/kg Sea Water at F_{IO_2} = 0.21

	Preaspiration	Hours after Aspiration											
					Treatment			Posttreatment					
	0	¼	½	¾*	1*	1½*	2	3	4	24	48	72	168
Group I, spontaneous ventilation	7.46 ±0.09	7.22‡ ±0.05	7.25‡ ±0.05	7.24‡ ±0.08	7.30‡ ±0.05	7.35‡ ±0.05	7.41 ±0.04	7.43 ±0.05	7.45 ±0.05	7.36‡ ±0.05	7.42 ±0.04	7.42 ±0.02	7.42 ±0.04
Group II, spontaneous ventilation and 10 cm H_2O PEEP	7.40 ±0.05	7.20‡ ±0.06	7.19‡ ±0.06	7.18‡ ±0.08	7.18‡ ±0.10	7.22‡ ±0.04	7.27‡ ±0.04	7.39 ±0.05	7.39 ±0.06	7.38 ±0.04	7.41 ±0.04	7.42 ±0.04	7.42 ±0.03
Group III, IPPV, (15 ml/kg) tidal volume	7.37 ±0.14	7.21† ±0.13	7.26† ±0.06	7.29 ±0.09	7.29 ±0.09	7.35 ±0.06	7.39 ±0.06	7.43 ±0.08	7.41 ±0.03	7.38 ±0.16	7.36 ±0.06	7.38 ±0.05	7.42 ±0.03
Group IV, CPPV, (15 ml/kg) and 10 cm H_2O PEEP	7.42 ±0.12	7.21‡ ±0.06	7.25‡ ±0.05	7.27‡ ±0.08	7.26‡ ±0.11	7.30‡ ±0.06	7.32† ±0.04	7.40 ±0.09	7.44 ±0.08	7.38 ±0.04	7.43 ±0.03	7.42 ±0.03	7.41 ±0.04

* F_{IO_2} = 1.0.
† Compared with zero time for the group, $P < 0.05$.
‡ Compared with zero time for the group, $P < 0.01$.

TABLE 4. Arterial Carbon Dioxide Tension (torr) before and after Aspiration of 22 ml/kg Sea Water at F_{IO_2} = 0.21 (Mean ± SD)

	Preaspiration	Hours after Aspiration											
					Treatment			Posttreatment					
	0	¼	½	¾*	1*	1½*	2	3	4	24	28	72	168
Group I, spontaneous ventilation	33 ± 9	47‡ ± 8	44‡ ± 7	49‡ ± 7	42† ± 8	41† ± 6	37 ± 6	34 ±5	33 ±7	38 ±5	36 ±6	37 ±1	38 ±2
Group II, spontaneous ventilation and 10 cm H_2O PEEP	40 ± 8	53‡ ±10	53‡ ± 9	53† ±13	55‡ ±13	50‡ ± 6	47 ±13	35 ±4	36 ±4	37 ±5	35 ±4	37 ±3	36 ±5
Group III, IPPV (15 ml/kg) tidal volume	41 ±12	49 ±11	43 ±10	48 ± 9	45 ±16	40 ± 6	36 ± 5	33 ±9	35 ±5	37 ±3	39 ±5	37 ±5	36 ±6
Group IV, CPPV (15 ml/kg) and 10 cm H_2O PEEP	35 ±12	47† ± 8	41 ± 6	44 ± 9	44 ±13	41 ± 6	40 ± 4	34 ±6	30 ±6	38 ±4	35 ±2	36 ±3	36 ±3

* F_{IO_2} = 1.0.
† Compared with zero time for the group, $P < 0.05$.
‡ Compared with zero time for the group, $P < 0.01$.

their Pa_{O_2}'s increased from 141 ± 121 torr to 555 ± 77 torr at $F_{I_{O_2}}$ = 1.0. At the completion of treatment, mean Pa_{O_2} at $F_{I_{O_2}}$ = 0.21 was 88 ± 14 torr, compared with a preaspiration value of 89 ± 11 torr (table 2). Pa_{O_2}'s at all times tested during treatment were significantly higher in this group than in either the control group or animals ventilated without PEEP ($P < 0.001$). In the dogs that breathed spontaneously with PEEP, Pa_{O_2}'s also increased significantly, and by the end of the treatment period mean Pa_{O_2} at $F_{I_{O_2}}$ = 1.0 was 521 ± 71 torr. Mean Pa_{O_2} while breathing room air at this time was 81 ± 7 torr, compared with a preaspiration value of 98 ± 10 torr. These values are higher than those in either the control group or the animals ventilated without PEEP ($P < 0.001$). There was no significant difference between the values for animals breathing spontaneously with PEEP and those being ventilated mechanically with PEEP at any time.

When treatment with PEEP or CPPV was discontinued 120 minutes following aspiration, Pa_{O_2} decreased. Mean Pa_{O_2}'s in these groups remained above that of the control group for the remainder of the experiment; however, they were not significantly different (tables 1 and 2). Within 24 hours of near-drowning, Pa_{O_2}'s of all but four animals returned to values similar to those before near-drowning, and after a week all survivors had Pa_{O_2}'s of at least 75 torr while breathing room air. Pa_{CO_2}'s and pH_a's also returned to normal in all groups. During the week that the animals were observed, two more dogs died.

Discussion

Previously, we demonstrated that when dogs aspirated 11 ml/kg of sea water and no attempt was made to drain the fluid and/or to resuscitate the animal, 80 per cent succumbed within approximately 10 minutes.[3] In contrast, during the present study, of the 43 animals that aspirated twice that volume of sea water and were treated with gravity drainage and IPPV, 40 survived for at least 24 hours. The procedure of draining fluid from the lungs and initiating IPPV employed with these animals occurred between 5 and 10 minutes of the onset of aspiration. The fact that these animals remained under water for at least 5 minutes, and were then resuscitated from the acute hypoxia, hypercarbia, and acidosis that occurred, is indeed encouraging. This leads one to question how long an interval between onset of immersion and resuscitation is possible if survival is to occur. It also lends greater credibility to reports in the literature of patients who have been successfully resuscitated 10 minutes,[7] 17 minutes,[8] and even 22 minutes[9] after onset of immersion.

Previously, we have shown that although sea water washes out some material with surface activity, it does not alter the surface tension characteristics of the pulmonary surfactant that remains behind.[10] In this regard, aspiration of sea water differs from aspiration of fresh water, which significantly elevates the minimum surface tension values of pulmonary surfactant seen on maximum film compression.[10] This suggests that the pathophysiologic process causing hypoxemia after aspiration of sea water is different than that caused by fresh water near-drowning. The primary problem after aspiration of sea water is that of fluid-filled, but perfused, alveoli, accounting for the large absolute or true intrapulmonary shunt.[3]

The dramatic increase in arterial oxygen tension in the animals that had positive end-expiratory pressure applied to their airways, with or without mechanical ventilation, suggests that increased ventilation to the perfused areas occurred. This, in turn, decreased the magnitude of the intrapulmonary shunt. A similar increase in Pa_{O_2} occurred in an earlier study after aspiration of fresh water, when mechanical ventilation was combined with PEEP.[11] However, it did not occur when PEEP was used in spontaneously breathing dogs. We attribute the difference between the results observed in spontaneously breathing animals with PEEP in the two studies to the fact that the animals that aspirated fresh water suffered a change in the surface tension properties of pulmonary surfactant, while those that aspirated sea water did not.[10] It seems reasonable that if the surface tension properties of pulmonary surfactant were normal, merely placing positive pressure on end exhalation would tend to prevent airway closure at end-expiration and promote

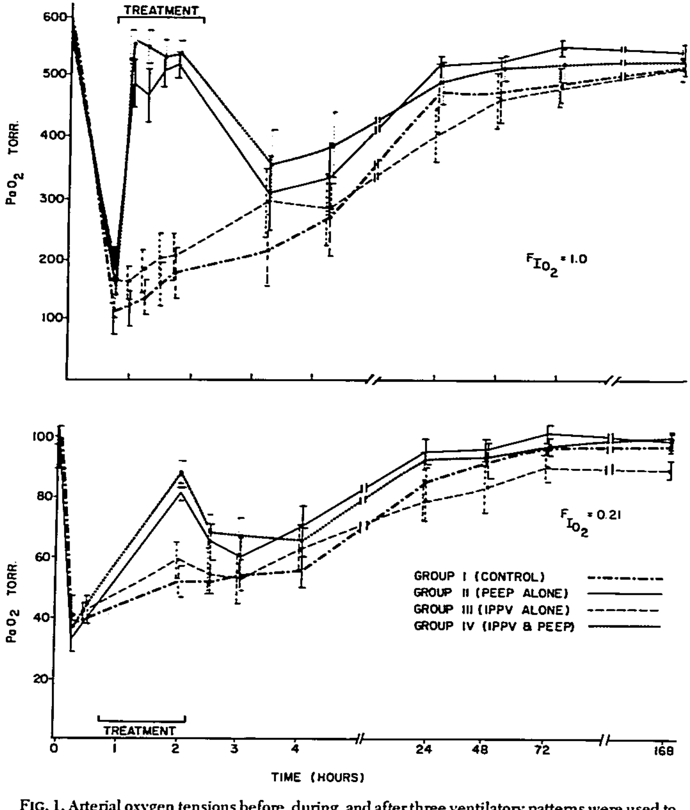

FIG. 1. Arterial oxygen tensions before, during, and after three ventilatory patterns were used to treat near-drowning in sea water. Values for these groups are compared with those for nontreated dogs. All values represent means ± SD.

maintenance of a greater functional residual capacity. On the other hand, if the surfactant were altered or destroyed, the alveoli would tend to collapse and require greater opening pressures than are usually obtained in the anesthetized, spontaneously breathing dog. In this case, if the alveolus were opened forcibly by the peak end-inspiratory pressure achieved during mechanical ventilation, it would stand a better chance of remaining open with PEEP. However, if the alveoli were partially opened with each breath with intermittent positive-pressure ventilation, but PEEP was not applied, the alveoli, being deficient in normal surfactant properties, would tend to recollapse.

An alternative explanation for the increased Pa_{O_2} observed in the dogs that aspirated sea water and were treated with either CPPV or PEEP could be that a diminution of oxygen consumption and a significant change in cardiac output occurred. Neither oxygen consumption nor cardiac output was measured in our studies. However, if this were the dominant mechanism for the improvement in arterial oxygenation, we would expect identical results after aspiration of fresh water; yet PEEP did not improve Pa_{O_2} in spontaneously breathing animals after aspiration of fresh water.[11]

The arterial oxygen tensions of most animals had returned to near normal by 24 hours, regardless of treatment, and Pa_{O_2}'s by 48 hours were not significantly different from preaspiration values in any group. This could be compared with our earlier fresh-water study, where significant hypoxemia was still present after 48 hours, and it took 3 to 7 days for the animals to regain normal Pa_{O_2}'s.[11] This, again, can be explained by the fact that, with normal surfactant activity in the animals that aspirated sea water, a much more rapid return to normal $\dot{V}_A/\dot{Q}$ would be expected, whereas after aspiration of fresh water regeneration of normal surfactant would be necessary before one would expect Pa_{O_2} to stabilize.

In conclusion, this study gives additional, albeit indirect, evidence for our earlier observations that one of the major differences between the effects on the lung of aspiration of fresh water and aspiration of sea water is that the surface tension properties of pulmonary surfactant are altered with fresh water but not with sea water. We also demonstrated that gravity drainage and immediate mechanical ventilation of the victim who aspirates large quantities of sea water are important, since they significantly increase the survival rate. The blood-gas data obtained in this experiment suggest that positive end-expiratory pressure, with or without mechanical ventilation, is indicated in the treatment of sea-water near-drowning victims to improve arterial oxygenation. One would expect that if this therapy were continued for the first 24 hours after aspiration, the decrease in Pa_{O_2} that occurred between the time therapy was discontinued and the time the 24-hour blood samples were obtained could be avoided.

While this manuscript was being written, we had the opportunity to apply the above-described principles in treating two patients. The following case reports illustrate that PEEP can cause a dramatic increase in Pa_{O_2} in patients who have aspirated sea water. Early use of PEEP resulted in complete resolution of the pulmonary damage in these two patients within 48 hours.

Report of Two Cases

Patient 1. A 27-year-old Caucasian man was wading in approximately 3 feet of sea water, and dove head-first onto a sandbar. He recalls lying on the ocean floor, unable to move his extremities, and holding his breath for as long as he can remember. An observer estimates that he was submerged face-down for approximately 4 minutes before he was carried, apneic and pulseless, to the beach. Mouth-to-mouth resuscitation and external cardiac massage were performed for approximately 2 minutes. Spontaneous respirations and pulses then returned. The patient was transported by ambulance and was conscious upon arrival at the hospital at 5:05 P.M.; however, he was in severe respiratory distress. Respiratory rate was 40/min, with marked intercostal retractions and cyanosis. The pulse was rapid and thready, and systolic blood pressure was 68 torr. Cyanosis persisted, in spite of administration of 100 per cent oxygen by mask, and analysis of arterial blood revealed severe hypoxemia with mixed respiratory and metabolic acidosis (Pa_{O_2} 51 torr, Pa_{CO_2} 48 torr, pH_a 7.27). There was some improvement in the respiratory status of the patient following placement of a nasotracheal tube, intermittent positive-pressure ventilation (IPPV**), and administration of sodium bicarbonate, 7.5 g iv, (Pa_{O_2} 104 torr, Pa_{CO_2} 27 torr, pH_a 7.44 at $F_{I_{O_2}} = 0.6$). A roentgenogram of the chest, taken prior to insertion of the endotracheal tube, showed bilateral infiltrates, which decreased following initiation of IPPV. Physical examination and roentgenograms of the cervical spine confirmed that the patient had a spinal cord compression at the level of C5–C6 secondary to a compression fracture of the fifth cervical vertebra. Following intravenous infusion of an unknown amount of 5 per cent dextrose in 0.50 physiologic saline solution blood pressure increased to 96 torr systolic and pulse rate decreased to 92 beats/min. The ventilator was changed to a Bird Mark VII and the patient was transferred to the Gainesville Veterans Administration Hospital at 8 P.M.

Upon admission to that hospital blood urea nitrogen was 20 mg/100 ml, sodium 140 mEq/l, potassium 3.6 mEq/l, and chloride 100 mEq/l. The leukocyte count was 5.4×10^3 /cu mm, and hematocrit was 43.5 per cent, with no free hemoglobin present.

** MA-1 Ventilator; Puritan-Bennett Company, Kansas City, Mo.

Shortly after arrival in the Emergency Room, during spontaneous breathing of 100 per cent O_2, Pa_{O_2} was 61 torr. The endotracheal tube was connected to an Emerson Post-Operative IMV†† Ventilator set to deliver 12 breaths/min and 10 torr positive end-expiratory pressure (PEEP). Analysis of arterial blood showed a marked improvement in oxygenation, but metabolic acidosis persisted (Pa_{O_2} 160 torr, Pa_{CO_2} 22 torr, pH_a 7.25, at $F_{I_{O_2}}$ = 0.36). The patient was given sodium bicarbonate, 7.5 g, and the rate of the ventilator was decreased from 12 to 8 breaths/min. Soon the patient began to breathe spontaneously between the intermittent mandatory ventilation (IMV), yet Pa_{O_2} was 454 torr with $F_{I_{O_2}}$ = 1.0 (Pa_{CO_2} 39 torr, pH_a 7.37) and Pa_{O_2} was 132 torr with $F_{I_{O_2}}$ = 0.30 (Pa_{CO_2} 38 torr, pH_a 7.37). The ventilator IMV rate and $F_{I_{O_2}}$ were gradually decreased over the ensuing 6 hours with no significant deterioration in arterial blood-gas values. Sixteen hours after aspiration of salt water, mechanical ventilation was discontinued, and the patient breathed spontaneously with 10 torr PEEP without difficulty (Pa_{O_2} 97 torr, Pa_{CO_2} 42 torr, pH_a 7.40 at $F_{I_{O_2}}$ = 0.30). Arterial blood-gas analysis following a gradual decrease in PEEP to 2 torr revealed no deterioration, and the trachea was extubated. Analysis of arterial blood 15 minutes later (20 hours after aspiration) showed Pa_{O_2} 72 torr, Pa_{CO_2} 35 torr, and pH_a 7.44 while the patient breathed room air spontaneously. Pa_{O_2} continued to increase to 87 torr 45 hours after aspiration and to 92 torr at 70 hours.

In addition to the above therapy, the patient was given 4 mg dexamethasone 8 hours after near-drowning. An aspirate of the endotracheal tube was cultured and revealed no growth. No antibiotic therapy was instituted. Despite the complete resolution of his pulmonary damage, the patient remains quadriplegic at this time.

Patient 2. A 33-year-old Caucasian man was found floating face-down in a salt-water tidal pool. He had been drinking heavily and had been severely beaten before falling into the water. Upon examination in the emergency room of the local hospital, he breathed spontaneously but was deeply cyanotic and responded only to pain. He had numerous bruises about his face and body, as well as a fractured left seventh rib. Blood pressure and pulse rate were normal, but rales were audible throughout both lung fields. The trachea was intubated and the patient was permitted to breathe spontaneously through a Briggs T-tube adapter through which 60 per cent O_2 was delivered. Arterial blood drawn under these conditions had a P_{O_2} of 68 torr, P_{CO_2} 40 torr, and pH 7.45. The hematocrit of venous blood was 43 vol per cent; serum electrolyte concentrations were sodium 144 mEq/l, potassium 3.5 mEq/l, and chloride 106 mEq/l.

†† Intermittent mandatory ventilation (IMV) mechanically hyperinflates the patient's lungs at a preset rate, but allows spontaneous ventilation to occur between mechanical hyperinflations.[12]

The patient was transferred via ambulance approximately 60 miles to the William A. Shands Teaching Hospital. During transit he became apneic and was treated by intermittent positive-pressure ventilation with a self-inflating AMBU bag containing an oxygen reservoir to permit administration of 100 per cent O_2. This ventilatory support was continued en route and upon arrival at the emergency room of the hospital. During ventilation with the self-inflating bag and 100 per cent O_2, arterial pH was 7.36, Pa_{CO_2} 33 torr, and Pa_{O_2} 72 torr. Then, the endotracheal tube was connected to a Bird Mark VI-14 ventilator at an IMV rate of 12/min and 6 cm H_2O PEEP was added. Pa_{O_2} increased to 348 torr, pH_a was 7.38, and Pa_{CO_2} was 30 torr. Intrapulmonary shunt was calculated at 21.5 per cent, and arterial-venous oxygen content difference was 3.96 vol per cent.

A roentgenogram of the chest taken on admission to the hospital showed bilateral fluffy infiltrates suggestive of pulmonary edema and a fractured seventh rib. Two teeth in the digestive tract were also noted.

PEEP was increased to 10 cm H_2O, which raised Pa_{O_2} to 459 torr on an $F_{I_{O_2}}$ of 1.0. An increase of PEEP to 12 cm H_2O produced no further improvement. $F_{I_{O_2}}$ was then gradually decreased to 0.3 and the IMV rate decreased to 8/min. PEEP was maintained at 10 cm H_2O overnight. Under these conditions Pa_{O_2} remained in the range of 86 to 110 torr. A roentgenogram of the chest taken six hours after admission to Shands Teaching Hospital showed marked clearing of the infiltrates. The only therapy the patient received, other than intensive pulmonary care described above, was intravenous administration of fluids. He received no antibiotic or steroid.

Since the patient had a rubber endotracheal tube with a high-pressure cuff in place, we elected to exchange it for a tube with a low-pressure cuff that had been implant-tested. Some difficulty was encountered in reintubation and the patient had to be ventilated with a self-inflating bag and mask without PEEP for approximately 15 minutes. Despite apparently adequate ventilation, pink frothy fluid was noted exuding from the larynx, and copious amounts of this fluid were suctioned after reintubation. A roentgenogram of the chest taken immediately after reintubation showed the reappearance of bilateral pulmonary edema; however, after reinstitution of PEEP at 8 cm H_2O, Pa_{O_2} rose to 439 torr at $F_{I_{O_2}}$ = 1.0. Roentgenograms of the chest again showed marked clearing.

The patient's condition remained stable, and $F_{I_{O_2}}$ was decreased. The IMV rate was progressively decreased to 2/min and the patient was maintained on 8 cm H_2O PEEP until the following day. PEEP was then reduced by 1 cm H_2O/hr with no deterioration of Pa_{O_2}. Immediately prior to extubation at PEEP 2 cm H_2O and $F_{I_{O_2}}$ 1.0, Pa_{O_2} was 530 torr. The trachea was extubated. The lungs remained clear, and Pa_{O_2} was 82 torr during breathing of room air. The patient recovered uneventfully.

These case histories illustrate that prompt respiratory therapy with PEEP can readily reverse the arterial hypoxemia resulting from aspiration of sea water. The transition from controlled to spontaneous ventilation should be accomplished gradually by employing IMV. The length of time necessary to maintain PEEP varies somewhat from patient to patient. In our first patient, pulmonary stability was achieved after 20 hours, but approximately 48 hours were required before our second patient could be weaned successfully from PEEP. The courses of our patients support the findings in our animal studies demonstrating the effectiveness of positive end-expiratory pressure in treatment of near-drowning in sea water.

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Central Nervous System

HYPERTENSION, CEREBRAL BLOOD FLOW, AND CAROTID ENDARTERECTOMY Internal carotid-artery pressure (PICA) and regional cerebral blood flow (rCBF) were measured in 22 patients undergoing carotid endarterectomy. Hypertension (range of mean arterial pressures 111–147 torr) was induced by intravenous injection of angiotensin amide while the carotid artery was clamped; this caused an increase in PICA in patients with good collateral circulation and initially high stump pressures but had little effect in those with initially low stump pressures. Regional CBF, measured by 16 collimated counters after intra-arterial ^{133}Xe injection, changed in proportion to PICA in normocapnic patients (PaCO_2 = 40 torr) but in hypocapnic patients (PaCO_2 = 29 torr) there was little change in rCBF unless PICA fell below 70 torr. There was no evidence that hypocapnia was beneficial. Moderate hypertension may improve rCBF in normocapnic patients with adequate collateral circulation. *(Boysen, G., Engell, H., and Henriksen, H.: The Effect of Induced Hypertension on Internal Carotid Artery Pressure and Regional Cerebral Blood Flow during Temporary Carotid Clamping for Endarterectomy. Neurology 22:1133–1144, 1972.)*