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Positive End-Expiratory Pressure (PEEP) in Treating Salt Water Near-Drowning

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THE FAVORABLE OUTCOME of a recent case of salt water near-drowning shows the usefulness of treatment with mechanical ventilation plus positive end-expiratory pressure (PEEP).

Report of a Case

On 13 October 1973, a healthy 34-year-old white mechant seaman fell off his small power boat into Puget Sound. His right wrist was lashed to a trailing rope and he was towed for 30 to 45 minutes behind the slowly moving boat. The water temperature was 12°C (53.6°F). After an unsuccessful helicopter rescue attempt, he was retrieved by a large Coast Guard patrol boat. He was transferred to a Fire Department Aid car. At that time he was comatose, but breathing spontaneously; an electrocardiogram showed atrial fibrillation with a ventricular rate of 80. A venous blood sample was taken (results of later analysis were: sodium [Na] 150, potassium [K] 4, chloride [Cl] 107 and carbon dioxide [CO_2] 9 mEq per liter), and he was given 88 mEq of bicarbonate (HCO_3) intravenously and oxygen by face mask.

Forty-five minutes later, at 16:15, the patient was admitted directly to the Harborview Medical Center intensive care unit (ICU). On initial physical examination the patient was talking and combative though unable to answer questions. He was muscular and his body was a dark purple color, especially the chest, neck and face. Auscultation of the chest showed diffuse rales without gallops or murmurs. On electrocardiogram, rapid sinus tachycardia with frequent premature atrial and ventricular beats was noted. Rectal temperature was 35.2°C (95.4°F) and intense shivering prevented auscultation or palpation of blood pressure. On administration of 100 percent oxygen by nonrebreathing mask the patient was tachypneic, hyperpneic and dyspneic with arterial oxygen partial pressure (PaO_2) 146 torr,* arterial carbon dioxide partial pressure ($PaCO_2$) 51 torr, arterial pH (pHa) 7 and HCO_3 12 mEq per liter (see Table 1).

At this time he was given 132 mEq of HCO_3 intravenously and a muscle relaxant to allow intubation and control of shivering and then was mechanically ventilated with a large tidal volume ($V_T = 1{,}000$ ml) and a high minute ventilation ($\dot{V}_E = 20$ liters per minute). Passive rewarming was allowed and an arterial line placed. A chest x-ray film taken at 16:45 showed bilateral pulmonary edema without cardiomegaly. PaO_2 was now 54 torr on 100 percent oxygen. Because of the large alveolar to arterial oxygen tension gradient ($AaDO_2 = 619$ torr), PEEP 15 cm H_2O was added at 18:00 with improvement of PaO_2 to 145 torr. One hour later, due to a fall of PaO_2 to 114 torr and with continual gross, frothy edema fluid bubbling from the endotracheal tube, a Swan-Ganz pulmonary artery catheter was "floated" and revealed a normal pulmonary wedge pressure of 12 mm of mercury and a normal mixed venous oxygen saturation of 75 percent. PEEP was maintained for about six hours during which gross clinical pulmonary edema and rales disappeared. A chest x-ray film taken at 20:00 showed partial clearing of pulmonary edema. During the evening the patient was incontinent of multiple large watery stools and spontaneously diuresed 1,500 ml by 22:30 (urine electrolytes Na 115 mEq per liter, K 16 mEq per liter). At 02:00, 14 October he was extubated and given humified 40 percent oxygen by mask. Six hours later, immediately before discharge from the ICU, PaO_2 was 77 torr on room air and a chest x-ray study showed dramatic, almost complete clearing of the infiltrates. The only other medication given in the ICU was intramuscularly administered low dose penicillin to prevent pneumonia from aspiration of oral pharyngeal organisms.

The patient convalesced four days on the medical wards until 17 October with a continued spontaneous diuresis and weight loss of 5 pounds. Low dose penicillin was continued; no respiratory care was given other than encouragement to cough and

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*One torr is equal to 1 mm of mercury.

TABLE 1.—*Clinical Course in Case of Salt Water Near-Drowning*

Time	*FiO_2*	*PaO_2 (torr)*	*$PaCO_2$ (torr)*	*pHa*	*HCO_3 (mEq per L)*	*PEEP (cmH_2O)*	*Comments*
16:30	1.0	146	51	7.00	12	0	Intubated, given HCO_3 132 mEq
17:15	1.0	54	40	7.42	25	0	$\dot{V}_E$ 20 liters per min on volume ventilator
18:30	1.0	145	27	7.51	21	15	.....
19:20	1.0	114	27	7.48	19	15	Swan-Ganz catheter placed
21:30	1.0	349	27	7.51	21	15	$\dot{V}_E$ 15 liters per min
23:15	0.35	149	31	7.47	22	15	$\dot{V}_E$ 10 liters per min
00:45	0.35	143	29	7.49	22	0	.....
01:25	1.0	364	34	7.44	22	0	Spontaneous ventilation
02:00	0.4	118	31	7.49	23	0	Extubated
08:00	0.21	77	36	7.44	24	0	.....

FiO_2 = inspired oxygen fractional concentration
PaO_2 = arterial oxygen partial pressure
$PaCO_2$ = arterial carbon dioxide partial pressure
pHa = arterial pH
HCO_3 = bicarbonate
PEEP = positive end-expiratory pressure
$\dot{V}_E$ = minute ventilation

deep breathe periodically. On a follow-up clinic visit on 24 October 1973, the patient was doing well without complaints. Chest x-ray studies showed minimal perihilar interstitial markings.

Discussion

The major problems following near-drowning with aspiration are hypoxemia and metabolic acidosis.[1,2] The mechanisms of arterial hypoxemia include reflex airway closure[3] and chemical pneumonitis from aspiration of vomitus, algae, mud and so forth.[2] Probably more important, following fresh water aspiration there is damage to surfactant with alveolar collapse and pulmonary shunting.[2] Pulmonary shunting in sea water aspiration comes from flooding of alveoli with a protein rich exudate drawn from the plasma by the hypertonic salt water.[2]

Recent experimental work suggests that PEEP with mechanical ventilation is the optimal pattern of respiration following fresh water aspiration.[4] Equally, in sea water aspiration, PEEP with or without mechanical ventilation dramatically reverses intrapulmonary shunting.[5] Several case reports also suggest its usefulness following both fresh and salt water aspiration.[5-7]

PEEP during mechanical ventilation is the application of positive airway pressure during expiration such that airway pressure does not fall to atmospheric at end expiration. PEEP may be generated by connecting the exhalation port of the ventilator to underwater seal drainage or to a special pressure sensitive valve. (Although a narrowed expiratory orifice will also generate PEEP,[8] creation of dangerously high airway pressures during coughing and straining precludes this method.[9]) Curarization during PEEP is not necessary as there are no significant cardiopulmonary differences between spontaneously triggered mechanical ventilation and controlled mechanical ventilation.[10] There appears to be no increased incidence of barotrauma when PEEP is used as an adjuvant to mechanical ventilation.[11]

Arterial oxygen tension, functional residual capacity and static lung compliance increase[12] and pulmonary shunting decreases[13] with increasing levels of PEEP. Cardiac output and systemic blood pressure may increase or decrease.[12,14] Improvement in oxygenation by PEEP in acute hypoxemic respiratory failure has been ascribed to recruitment of gas exchange airspaces and prevention of terminal airway closure.[12] Careful monitoring is essential to balance the improved gas exchange versus the potential reduction in cardiac output.

A previous large series of salt water near-drownings showed low mortality and morbidity, but the therapeutic management (which included diuretics, HCO_3, intermittent positive-pressure breathing (IPPB), supplemental oxygen and mechanical ventilation only for coexisting respiratory acidosis) allowed hypoxemia and acidosis to persist many hours after admission to the hospital.[1] The aggressive therapy in this patient rapidly terminated severe noncardiogenic pulmonary edema and reduced to a minimum the risks of hypoxemia and acidosis.

As in many other causes of pulmonary edema, PEEP should be considered in the management of salt water near-drowning with aspiration.

Summary

A case of salt water near-drowning is described. Recent experimental work suggests that PEEP is

the optimal pattern of breathing following such an insult. Hypoxemia and pulmonary edema in the patient were quickly alleviated by mechanical ventilation with PEEP.

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Successful Treatment of Non-Meningitic Listerial Brain Abscess without Operation

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THE MOST COMMON MANIFESTATION of Listeria monocytogenes infection in the central nervous system is purulent meningitis or meningoencephalitis.[1] Focal or disseminated suppuration in the brain without meningitis is decidedly rare and only six cases have been recorded in the English literature.[2-7] We report another case of non-meningitic listeriosis with both cerebral and pulmonary abscesses. This case is also unusual in that the patient had a prolonged, relapsing illness and recovered completely without surgical intervention.

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Report of a Case (Chart 1)

A 35-year-old man with polycystic kidneys, chronic renal failure and a cadaveric renal transplant, presented in February 1971 with bilateral diffuse pulmonary infiltrates. Open lung biopsy showed acute, necrotizing bronchopneumonia. Cultures and special stains for aerobic and anaerobic bacteria, fungi, Pneumocystis carinii, and viral inclusion bodies were negative. The patient was nevertheless treated with a trial of pentamidine, penicillin, and cloxacillin. Within two and a half weeks, there was complete radiographic resolution of the pulmonary infiltrates, and he was maintained on prednisone 10 mg and azathioprine 50 mg daily.

In May 1971 he was readmitted to hospital with fever, chills, mental obtundation, expressive aphasia and right facial paresis. Meningeal signs were absent. Consolidation of the apical-posterior segment of the left upper lung was noted. There were no cardiac murmurs or evidence for peripheral embolization. Laboratory findings included hemoglobin of 8.4 grams per 100 ml, hematocrit 23.5 percent, leukocyte count 6,300 per cu mm with 16 percent neutrophils, 83 percent lymphocytes, and 1 percent monocytes, and platelet count