sia equipment. Prestretching a new rebreathing bag assures that with inadvertent overinflation the pressure will not exceed 30 to 35 torr. This is similar to prestretching the cuffs of some new disposable endotracheal tubes to lower their pressures.

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The Use of Mechanical Ventilation with Positive End-expiratory Pressure in the Treatment of Near-drowning

R. R. RUTLEDGE, M.D.,* AND R. J. FLOR, M.D.†

The following case of fresh-water near-drowning with aspiration emphasizes the reversibility of the pulmonary injury and illustrates the benefit of mechanical ventilation with positive end-expiratory pressure (MV with PEEP) in treatment.

REPORT OF A CASE

A 16-year-old boy was admitted to our care five hours after near-drowning in chlorinated fresh water with aspiration. The boy's health had been good, and a chest x-ray ten months earlier had disclosed no abnormalities. Submersion was estimated to have lasted 3 minutes. Upon removal from the water, the boy had a pulse but was unconscious and required mouth-to-mouth resuscitation. When respiration returned, it was rapid and shallow, and frothy secretions and cyanosis were present.

Upon arrival in our emergency room two and a half hours later, the patient had pulmonary edema with cyanosis, tachypnea, and tachycardia, but was not hypotensive. Initial arterial blood gases, measured while oxygen was being delivered by face mask, indicated marked hypoxia and desaturation, hypercarbia, and slight acidemia (Pa_{O_2} 25 torr; sat 40 per cent; Pa_{CO_2} 46.5 torr; pH 7.32). An endotracheal tube was introduced and respiration was assisted with an Ambu bag during transport to the intensive care unit, where ventilation was then assisted with a volume ventilator supplying 100 per cent oxygen. A marked alveolar–arterial oxygen gradient (578 torr) persisted in spite of good mechanical ventilation, so controlled MV with PEEP at 10 cm H_2O was instituted three hours later. A rapid and dramatic improvement in the oxygen gradient (384 torr) followed the application of PEEP, and the inspired oxygen concentration had been reduced to 40 per cent one and a half hours later. Several hours later, PEEP was reduced to 5 cm H_2O and maintained there for the next 24 hours while the oxygen concentration was further reduced. Four hours after cessation of PEEP, weaning from artificial ventilation was begun with a T tube; seven hours later, the trachea was extubated. Supplemental oxygen was administered for an additional 14 hours. Chest physiotherapy was continued and assisted hyperinflation was utilized periodically until the patient's discharge on the sixth hospital day. The patient had awakened fully within six hours of admission, and no residual neurologic deficits were present. When he was discharged, his chest was clear to auscultation and x-ray.

Laboratory data of interest include serum K, which was 5.6 mEq/l on admission, at which time the blood was "4+ hemolyzed." Two hours later, serum K was 3.7 mEq/l, serum-free hemoglobin was 24.8 mg/100 ml, and serum osmolarity was 277 mosm/l. Although hemolysis and hyperkalemia were present, urinary discoloration was transient, and no arrhythmia occurred. Serum enzymes were elevated initially and reached a peak approximately 12 hours later. Serum bilirubin was also elevated, and the elevation persisted for three days (maximum 1.8 mg/100 ml). The x-rays (fig. 1) and graph (fig. 2) illustrate the severity

* Instructor.
† Assistant Professor.

Received from the Department of Anesthesiology, University of Miami School of Medicine, Miami, Florida 33152. Accepted for publication July 28, 1972.

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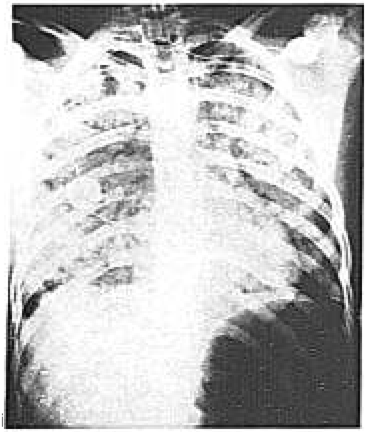

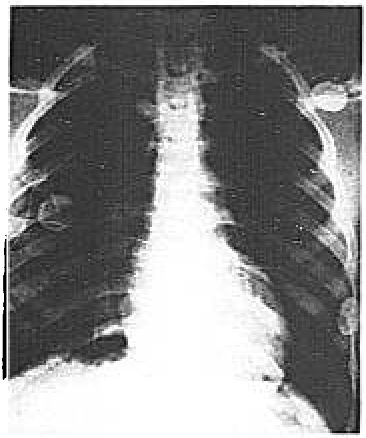

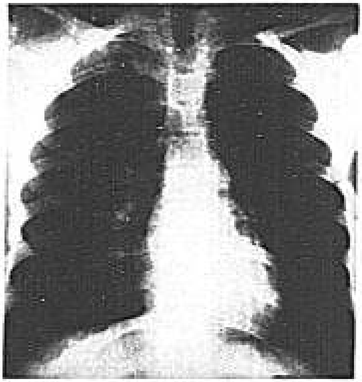

FIG. 1. Chest x-rays. *Above, left,* six hours after aspiration and prior to the application of PEEP; *below, left,* eight hours after institution of PEEP; *above,* at the time of discharge.

of the pulmonary injury and the efficacy of MV with PEEP.

Discussion

A delay of four hours from the time of fresh-water aspiration until the first blood sample was obtained may have allowed redistribution and excretion of potassium and free hemoglobin such that the elevated values found in other cases of fresh-water drowning may have been missed in this case.[1] The elevated bilirubin attests to a great degree of hemolysis. Of particular note was the absence of marked acidosis in spite of severe, prolonged arterial desaturation prior to admission. Such absence of acidosis may be explained by a greatly increased cardiac output which compensates for diminished arterial oxygen content.

The inactivation of surfactant and injury to alveolar cells by aspirated fluids, particularly fresh water, have been documented.[2] However, there has been no major advocation of the use of PEEP for treatment of near-drowning.[3] It would appear from this case that such therapy may be of great value. If the pathophysiology is indeed the result of alveolar collapse, early re-expansion should allow better oxygen transfer, thereby allowing lower alveolar oxygen concentrations and lessening the chance of "oxygen toxicity." In addition, PEEP can clear intra-alveolar fluid and perhaps conserve surfactant by interrupting wash-

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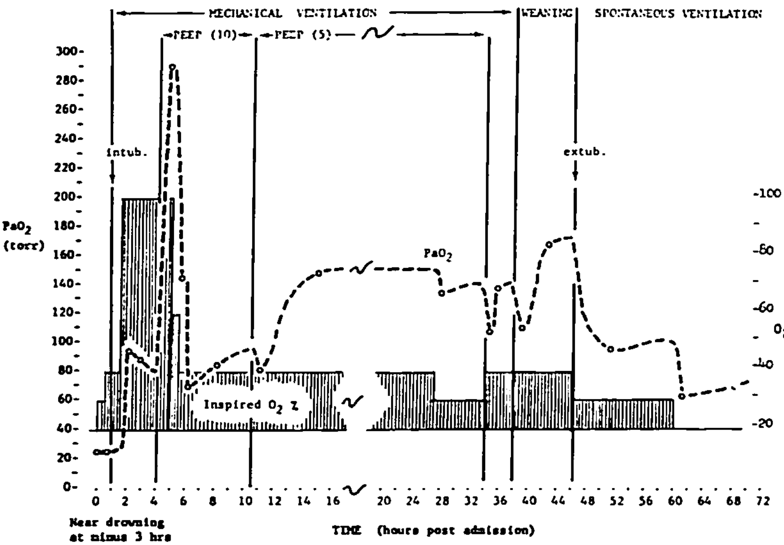

FIG. 2. Variation of arterial oxygen tension (dashed line) as inspired oxygen concentration (bar graph) and mode of ventilation (dividing lines) were changed.

out. Reducing atelectasis will also lessen the chance of superimposed infection. Corticosteroids can be utilized, as in this case, to reduce pulmonary tissue reactivity and to lessen possible cerebral edema resulting from antecedent hypoxia.[4] Antibiotics need not be used unless infection is evident.

In summary, a near-drowning victim treated by means of MV with PEEP and corticosteroids showed rapid improvement and recovery. It is suggested that PEEP be utilized early to stabilize injured alveoli deprived of surfactant, thus averting later complications.

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