The Ineffectiveness of Steroid Therapy for Treatment of Fresh-water Near-drowning

Hugh W. Calderwood, V.M.D.,* Jerome H. Modell, M.D.,† Bruce C. Ruiz‡

The authors evaluated the efficacy of continuous positive-pressure ventilation (CPPV) and methylprednisolone alone and in combination as therapy for near-drowning in 50 dogs that had aspirated distilled water (22 ml/kg or 44 ml/kg). Forty dogs were treated with mechanical ventilation for one hour and 40 for 24 hours. Blood-gas tensions, pH, cardiac output and intrapulmonary shunt (Qh/Qs) were measured frequently for 24 hours. Blood-gas tensions and pH were again measured 48 and 72 hours and seven days later in survivors. Arterial oxygen tension (Pao2) decreased and Qh/Qs increased in all animals following aspiration and before therapy. Forty dogs received methylprednisolone intravenously (30 mg/kg) (20 breathed spontaneously and 20 had CPPV). There was a significant increase in Pao2 and decrease in pulmonary shunt in dogs that were ventilated compared with animals that breathed spontaneously. Treatment with methylprednisolone made no difference in blood gases, pulmonary shunt, or survival rates. Thus, no evidence to support the use of methylprednisolone in the treatment of the pulmonary lesion of fresh-water near-drowning was found. (Key words: Drowning, fresh-water; Hormones, adrenal, methylprednisolone.)

CORTICOSTEROID THERAPY has been advocated for treatment of patients who near-drown.1,2 The rationale for its use has been that since near-drowning is a form of aspiration pneumonitis and corticosteroid therapy has been reported to be beneficial in classic aspiration pneumonitis (gastric acid aspiration), it also was thought to be beneficial in treating patients who nearly drowned.3,4 However, recent controlled studies in animals indicate that methylprednisolone therapy does not alter the course of aspiration pneumonitis caused by hydrochloric acid.3,4 We designed our experiment to determine whether methylprednisolone in pharmacologic doses (30 mg/kg) would influence the survival rate or arterial oxygenation of dogs who nearly drowned in fresh water.

Materials and Methods

Eighty mongrel dogs were studied in three experiments. All dogs were prepared similarly, only the quantities of fluid aspirated and the durations of mechanical ventilation were changed. Each dog was anesthetized with sodium pentobarbital (25 mg/kg, iv), the trachea was intubated with auffed endotracheal tube, and an esophageal thermistor probe was inserted to heart level. A 20-gauge Teflon catheter was inserted percutaneously into the femoral artery and a flow-directed catheter§ was advanced into the pulmonary artery via the external jugular vein. The dogs breathed 100 per cent oxygen via the endotracheal tube for 15 minutes, after which arterial blood was drawn to measure pH, Pco2, and Pao2 with appropriate electrodes.§ The animals then breathed room air for 15 minutes and arterial and mixed-venous blood samples were drawn to evaluate acid-base status and blood-gas tensions. We also determined cardiac output (dye-dilution method), expired CO2 (Pco2), and blood hemoglobin concentration.

§ Swan-Ganz catheters, Edwards Laboratory, Santa Ana, California.
§ IL 113, Instrumentation Laboratories, Lexington, Massachusetts.
Experiments were conducted in three phases.

**PHASE I**

Forty dogs aspirated distilled water (22 ml/kg, 30°C) via the endotracheal tube as described previously. Five minutes after aspiration, arterial blood was drawn to determine pH, PCO₂, and PO₂. The lungs then were drained by gravity. If the dog was apneic, we ventilated it with room air with an Ambu bag until spontaneous breathing returned. Arterial blood was analyzed for pH, PCO₂, and PO₂ 10 minutes after aspiration. The animals breathed 100 per cent oxygen spontaneously for 90 minutes following aspiration, after which they again breathed room air. Twenty-eight minutes after aspiration, cardiac output (CO), PEO₂, hemoglobin concentration, PkCO₂, Paco₂, pH, mixed venous blood oxygen (PVCO₂), carbon dioxide (PVCO₂), and pH (pHv) were determined.

These 40 dogs were subdivided into four equal groups. Group I received no treatment except initial resuscitation. Group II received methylprednisolone (30 mg/kg, iv) every eight hours for 72 hours; the first dose was given 30 minutes after aspiration. Dogs in Group III were paralyzed with succinylcholine HCl (0.25 mg/kg) after the 28-minute measurements and were ventilated mechanically at a tidal volume of 15 ml/kg with a volume-limited ventilator.** Positive end-expiratory pressure (PEEP) of 10 cm H₂O was employed. The ventilator rate was adjusted to maintain a PaCO₂ of 35 to 40 torr. After 60 minutes of mechanical ventilation, the dogs resumed spontaneous breathing. Group IV was identical to Group III except that the dogs received methylprednisolone (30 mg/kg, iv) every eight hours for 72 hours starting 30 minutes after aspiration.

Paco₂, PCO₂, and pHv were measured 5, 10, 120, and 240 minutes after aspiration while the dogs breathed room air and also 28, 60, and 90 minutes after aspiration while the dogs breathed 100 per cent oxygen. These determinations were repeated in surviving animals 24, 48, 72, and 168 hours later at inspired oxygen concentrations (FIO₂) of 0.21 and 1.0. Hemoglobin concentration, PEO₂, cardiac output, and PO₂ were determined prior to aspiration and then 28, 90, and 240 minutes after aspiration. Qi/Qt was calculated using the modified shunt equation.11 The immobilization period for these animals was limited to 4 hours, after which they were returned to their cages for standard kennel care.

**PHASE II**

Twenty mongrel dogs were studied to evaluate the effects of long-term mechanical ventilation (24 hours) and steroid therapy. They were prepared exactly as the animals in Phase I for the first 28 minutes after aspiration. These dogs were subdivided into four equal groups. The dogs in Groups V and VI were treated exactly as Groups I and II in Phase I except that they breathed room air after 60 minutes and were given sodium pentobarbital as needed to prevent voluntary movement throughout the 24-hour experiment. Dogs in Groups VII and VIII received sodium pentobarbital and pancuronium bromide as needed to produce anesthesia and muscle paralysis for 24 hours. Their lungs were ventilated mechanically for 24 hours at 15 ml/kg tidal volume and 10 cm H₂O PEEP. In addition, Group VIII received steroid therapy. Cardiac output, hemoglobin, PEO₂, blood-gas tensions, and pH were measured every two hours following the four-hour samples.

**PHASE III**

Twenty mongrel dogs were divided into four groups (IX–XII) to study the effects of mechanical ventilation and steroid therapy on massive aspiration of distilled water (44 ml/kg). The same experimental design was used as in the second experiment except that the volume of water aspirated was doubled.

**Emerson Postoperative Ventilator, Model B-PV, J. H. Emerson, Co., Cambridge, Massachusetts**

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11 $Q_i/Qt = \frac{C_{Vo}}{C_{0}} - \frac{C_{0}}{C_{0}}$ where $C_{0}$ is equal to the capillary oxygen content and $C_{0}$ and $C_{0}$ equal the arterial and mixed venous oxygen contents, respectively.
### Table 1. Survival Rates for Dogs after Aspirating Distilled Water

<table>
<thead>
<tr>
<th></th>
<th>Phase I</th>
<th>Phase II</th>
<th>Phase III</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>22 ml/kg, 4 Hours of Anesthesia</td>
<td>22 ml/kg, 4 Hours of Anesthesia</td>
<td>44 ml/kg, 24 Hours of Anesthesia</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>Steroid</td>
<td>CPPV plus Steroid</td>
</tr>
<tr>
<td>Survivors/total, 24 hours</td>
<td>7/10</td>
<td>4/10</td>
<td>6/10</td>
</tr>
<tr>
<td>Survivors/total, one week</td>
<td>6/10</td>
<td>3/10</td>
<td>6/10</td>
</tr>
</tbody>
</table>

### Results

Twenty-four hours after aspiration 60 per cent of the dogs that aspirated 22 ml/kg of distilled water and were anesthetized for only four hours were alive, 45 per cent of the dogs that aspirated this quantity of water and were anesthetized for 24 hours were alive, and only 25 per cent of the dogs subjected to 44 ml/kg aspiration volume and anesthetized for 24 hours were alive. The data for all groups were analyzed by the chi-square method, and there was no significant difference in 24-hour survival rates between those dogs that received steroids and those that did not, or between animals that were mechanically ventilated and those that were not. No week after aspiration, 55 per cent of the dogs that aspirated 22 ml/kg water and were anesthetized for only four hours were still alive. Ten of these 22 animals had received steroids and 13 of the 22 had been ventilated mechanically. Only three dogs (15 per cent) were alive in the groups that aspirated 22 ml/kg of water and were kept anesthetized for 24 hours; all three had been ventilated mechanically, and one had also received steroids. Only two dogs remained alive for 1 week after aspirating 44 ml/kg water. One of these dogs had been ventilated mechanically, one breathed spontaneously, and both received steroids (table 1). Chi-square analysis of data for survivors at one week disclosed no significant difference.

Of the 60 dogs that aspirated 22 ml/kg, 11 required resuscitation with positive-pressure ventilation. When we attempted to drain fluid from their lungs by gravity 5 minutes after aspiration, no fluid could be obtained from 20 dogs. From the remaining 40 animals, 12.8 ± 14.9 ml (or less than 1 ml/kg) were obtained. In the experiment where dogs aspirated 44 ml/kg of distilled water, 10 of the 20 animals required resuscitation with intermittent positive-pressure ventilation. Fluid was drained from 15 of these dogs, but the volume was small (44.2 ± 49.3 ml).

There was a precipitous decrease in arterial oxygen tension \( (P < .001) \) in all animals 5 minutes after aspiration, regardless of the quantity of water aspirated (tables 2, 3, and 4). This arterial hypoxemia was associated with an increase in \( P_{\text{CO}_2} \) and a decrease in \( p\text{H} \). \( P_{\text{CO}_2} \)'s in the dogs that aspirated 22 ml/kg and breathed spontaneously decreased, so that 105 minutes after aspiration they were the same as values found prior to aspiration. Likewise, the \( p\text{H} \) changes that were significant \( (P < .001) \) 5 to 10 minutes after aspiration returned to normal by 105 minutes. Arterial hypoxemia and increased \( Q_i/Q_s \) persisted, however, in the animals that breathed spontaneously, whether or not they received steroids \( (P < .001) \). The dogs that aspirated 22 ml/kg and were ventilated mechanically for 60 minutes had a significant increase in \( P_{\text{CO}_2} \) \( (P < .05) \) and a significant decrease in shunt \( (P < .05) \) (Table 5). This was associated with a decrease in cardiac output that was significant in Group III \( (P < .01) \). Again, there was no difference between the steroid and non-steroid groups. For statistical analysis of \( Q_i/Q_s \) and cardiac output, we combined both groups of a single variable (i.e., both groups that received steroids versus both that did not and, similarly, both groups that were ventilated versus both that were not). There was no difference in \( Q_i/Q_s \) between the groups.
### Table 2. $P_{a,o}$ (Torr) of Dogs that Aspirated Distilled Water (22 ml/kg) and Were Treated with Methylprednisolone (30 mg/kg) and/or Were Mechanically Ventilated for 1 Hour, Mean ± SD (Survivors)

<table>
<thead>
<tr>
<th>Time Post-</th>
<th>$P_{a,o}$ 0.21 Torr</th>
<th>$P_{a,o}$ 0.1 Torr</th>
<th>$P_{a,o}$ 0.01 Torr</th>
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<tbody>
<tr>
<td>Fx, Torr</td>
<td></td>
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<tr>
<td>0</td>
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<tr>
<td>5</td>
<td>0.80 ± 1.0</td>
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<td>0.80 ± 1.0</td>
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<td>30</td>
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<td>60</td>
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<td>90</td>
<td>0.80 ± 1.0</td>
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<td>0.80 ± 1.0</td>
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<td>48</td>
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<td>72</td>
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<td>240</td>
<td>0.80 ± 1.0</td>
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<td>0.80 ± 1.0</td>
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<tr>
<td>Time Post-</td>
<td>( F_{10} = 0.21 )</td>
<td>( F_{10} = 1.0 )</td>
<td>( F_{10} = 0.21 )</td>
</tr>
<tr>
<td>aspiration (PA)</td>
<td>0</td>
<td>5 Min.</td>
<td>10 Min.</td>
</tr>
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<td>-------</td>
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<td>--------</td>
</tr>
<tr>
<td>Group V, control</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>± 16</td>
<td>88</td>
<td>28</td>
<td>35</td>
</tr>
<tr>
<td>± 9</td>
<td>(5)</td>
<td>(5)</td>
<td>(5)</td>
</tr>
<tr>
<td>± 13</td>
<td>120</td>
<td>30</td>
<td>31</td>
</tr>
<tr>
<td>± 13</td>
<td>(5)</td>
<td>(5)</td>
<td>(5)</td>
</tr>
<tr>
<td>Group VI, steroid</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>± 13</td>
<td>91</td>
<td>29</td>
<td>34</td>
</tr>
<tr>
<td>± 13</td>
<td>(5)</td>
<td>(5)</td>
<td>(5)</td>
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<tr>
<td>Group VII, CPPV from</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>30 minutes to 24 hours</td>
<td>93</td>
<td>30</td>
<td>31</td>
</tr>
<tr>
<td>± 14</td>
<td>(5)</td>
<td>(5)</td>
<td>(5)</td>
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<tr>
<td>Group VIII, CPPV from</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 minutes to 24 hours</td>
<td>85</td>
<td>35</td>
<td>29</td>
</tr>
<tr>
<td>± 10</td>
<td>(5)</td>
<td>(5)</td>
<td>(5)</td>
</tr>
</tbody>
</table>

* \( P < .05 \) compared with 10-minute values at \( F_{10} = 0.21 \).
<table>
<thead>
<tr>
<th>Time Post-aspiration (PA)</th>
<th>( F_{10} = 0.21 )</th>
<th>( F_{10} = 0.1 )</th>
<th>( F_{10} = 0.21 )</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5 Min.</td>
<td>10 Min.</td>
<td>20 Min.</td>
</tr>
<tr>
<td>Group IA, control</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>104 ± 0</td>
<td>30 ± 10</td>
<td>38 ± 8</td>
</tr>
<tr>
<td>Group IA, steroid</td>
<td>87 ± 12</td>
<td>24 ± 7</td>
<td>33 ± 4</td>
</tr>
<tr>
<td>Group IA, CPPV from 30 minutes to 24 hours PA</td>
<td>85 ± 5</td>
<td>26 ± 6</td>
<td>34 ± 12</td>
</tr>
<tr>
<td>Group IA, CPPV from 30 minutes to 24 hours PA plus steroid</td>
<td>83 ± 4</td>
<td>29 ± 16</td>
<td>37 ± 3</td>
</tr>
</tbody>
</table>

* \( P < .05 \) compared with 10-minute values at \( F_{10} = 0.21 \).

\( ^{1}P < .02 \) compared with 28-minute values at \( F_{10} = 1.0 \).
TABLE 5. Percentage Changes in Cardiac Output from Baseline Values (at 0 Time) and \( \dot{Q}_v/\dot{Q}_a \) for Dogs That Aspirated Distilled Water (22 ml/kg) and Received Methylprednisolone Therapy and/or Mechanical Ventilation for an Hour, Mean ± SD (Sample Number)

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>25</th>
<th>90</th>
<th>240</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group I, control</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \dot{Q}_v/\dot{Q}_a ) (per cent)</td>
<td>4.3</td>
<td>47.9*</td>
<td>43.3</td>
<td>35.4</td>
</tr>
<tr>
<td>± 3.2</td>
<td>± 15.1</td>
<td>± 15.5</td>
<td>± 11.4</td>
<td></td>
</tr>
<tr>
<td>(10)</td>
<td>(10)</td>
<td>(10)</td>
<td>(8)</td>
<td></td>
</tr>
<tr>
<td>Cardiac output (Δ per cent)</td>
<td>-</td>
<td>-0.8</td>
<td>-11.3</td>
<td>-11.8</td>
</tr>
<tr>
<td>± 34.7</td>
<td>± 24.8</td>
<td>± 30.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(10)</td>
<td>(10)</td>
<td>(8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Group II, steroid</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \dot{Q}_v/\dot{Q}_a ) (per cent)</td>
<td>5.4</td>
<td>46.1*</td>
<td>47.2</td>
<td>47.9</td>
</tr>
<tr>
<td>± 2.7</td>
<td>± 13.5</td>
<td>± 12.1</td>
<td>± 22.4</td>
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<tr>
<td>(10)</td>
<td>(10)</td>
<td>(10)</td>
<td>(7)</td>
<td></td>
</tr>
<tr>
<td>Cardiac output (Δ per cent)</td>
<td>-</td>
<td>-13.8</td>
<td>1.3</td>
<td>-18.3</td>
</tr>
<tr>
<td>± 37.7</td>
<td>± 46.2</td>
<td>± 39.2</td>
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<tr>
<td>(10)</td>
<td>(10)</td>
<td>(8)</td>
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</tr>
<tr>
<td><strong>Group III, CPPV from 30 to 90 minutes postaspiration</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>( \dot{Q}_v/\dot{Q}_a ) (per cent)</td>
<td>7.8</td>
<td>48.1*</td>
<td>25.81§</td>
<td>38.5</td>
</tr>
<tr>
<td>± 6.1</td>
<td>± 13.7</td>
<td>± 11.8</td>
<td>± 8.0</td>
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<tr>
<td>(10)</td>
<td>(10)</td>
<td>(10)</td>
<td>(8)</td>
<td></td>
</tr>
<tr>
<td>Cardiac output (Δ per cent)</td>
<td>-</td>
<td>-23.2†</td>
<td>-50.01§</td>
<td>-27.01</td>
</tr>
<tr>
<td>± 19.2</td>
<td>± 15.8</td>
<td>± 25.8</td>
<td></td>
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<tr>
<td>(10)</td>
<td>(10)</td>
<td>(10)</td>
<td>(8)</td>
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<tr>
<td><strong>Group IV, CPPV from 30 to 90 minutes postaspiration plus steroid</strong></td>
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</tr>
<tr>
<td>( \dot{Q}_v/\dot{Q}_a ) (per cent)</td>
<td>7.2</td>
<td>38.5*</td>
<td>28.1</td>
<td>31.7</td>
</tr>
<tr>
<td>± 6.8</td>
<td>± 16.5</td>
<td>± 15.1</td>
<td>± 7.9</td>
<td></td>
</tr>
<tr>
<td>(10)</td>
<td>(10)</td>
<td>(10)</td>
<td>(8)</td>
<td></td>
</tr>
<tr>
<td>Cardiac output (Δ per cent)</td>
<td>-</td>
<td>-7.0</td>
<td>-13.7</td>
<td>20.0</td>
</tr>
<tr>
<td>± 40.7</td>
<td>± 36.2</td>
<td>± 97.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(9)</td>
<td>(9)</td>
<td>(9)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* \( P < .001 \) when 28-minute values are compared with zero-time values.
† \( P < .05 \) when 28-minute values are compared with zero-time values.
§ Group III was significantly different from the other groups at 90 minutes (\( P < .01 \)).
\( P < .05 \) compared with postaspiration values (28 minutes).

that received steroids and those that did not, but there was improvement in \( \dot{Q}_v/\dot{Q}_a \) at 90 minutes in those groups mechanically ventilated (\( P < .001 \)). Cardiac output was significantly different at 90 minutes for both steroid (\( P < .05 \)) and mechanical ventilation (\( P < .02 \)) parameters, due primarily to the large decrease in cardiac output in Group III. Once mechanical ventilatory support was withdrawn, \( P_{aO_2} \)'s decreased but were still...
significantly higher than values 10 minutes after aspiration and from those of the animals that were permitted to breathe spontaneously (P < .05). Over the next week the survivors slowly recovered normal PaO₂'s in all groups.

The dogs that aspirated 22 ml/kg and were treated for 24 hours had a similar transient increase in PaO₂ and a similar decrease in pH. There was an increase in shunt, which was not changed with steroid therapy alone, but those animals that received ventilatory therapy had significantly higher PaO₂'s during the 24 hours of mechanical ventilation than when they breathed spontaneously after aspiration (P < .05) (table 3). The dogs that aspirated 44 ml/kg showed similar results (table 4). The Ā/Q̄ and CO followed the same trends as in Phase I, but because of the small size of the subgroups (n = 5) and technical errors that reduced data points for some time periods (n = 2), we did not have sufficient measurements of Ā/Q̄ and CO for statistical analysis in Phases II and III.

**Discussion**

Previous studies have shown that methylprednisolone therapy does not alter the responses of dogs to aspiration of HCl at pH 1.0 and pH 1.8.3,4 In those experiments, continuous positive-pressure ventilation (CPPV) did improve arterial oxygenation and survival. These findings agree with those in our previous study of different ventilatory patterns in treatment of fresh-water near-drowning.6 The present experiment shows that methylprednisolone did not improve survival or arterial oxygenation after aspiration of distilled water. CPPV, however, increased arterial blood oxygenation and decreased Ā/Q̄. The dogs that were ventilated mechanically after aspiration had the greatest increase in PaO₂, despite a decrease in cardiac output. In man, it has been shown that PEEP should be titrated to obtain an optimum level (the greatest improvement in PaO₂ with the least interference in cardiac output).7 This technique was not used in the present experiment since we desired to have a standard animal model with a fixed PEEP. It is interesting to speculate whether, had PEEP been titrated to the specific needs of each dog and fluid balance meticulously monitored to maintain cardiac output in the face of an increased intrapleural pressure, the shunt would have decreased further and PaO₂ increased further. One would expect that this would occur.

Anticipating the question whether a beneficial effect of steroids would be seen immediately or whether it would be necessary to study the animals for a longer period, we embarked on the 24-hour experiments. Steroids did not influence the survival rate or PaO₂ either in the dogs permitted to breathe spontaneously or in those ventilated mechanically. Even though PaO₂ improved, the survival rates in the dogs that had aspirated distilled water (22 ml/kg) and were ventilated for 24 hours were lower than those that had short-term ventilation. The mortality rate in the control group for this portion of the experiment was high. The dogs in the short-term ventilation part of the experiment were awake and moving about in their cages the evening of the experiment. The animals in the second phase of the experiment were kept anesthetized and secured supine for this time. The 24 hours of intravenous-barbiturate anesthesia and the continuous dorsal recumbency may have contributed to mortality, since all other factors were the same in the two groups. Since patients are not customarily anesthetized during long-term ventilatory therapy, it is not possible to transfer the data to man and speculate whether a routine of 24 hours of ventilatory support would be beneficial or detrimental to human victims who nearly drown.

One could question whether the insult in the first experiment was sufficiently severe to permit detection of a beneficial effect of corticosteroid therapy. However, when 44 ml/kg of water were aspirated, we still did not see a beneficial effect of steroids, whether or not the animals were mechanically ventilated. The overall mortality rate of 75 per cent at 24 hours in this group is not different from the 80 per cent mortality rate we reported in previous studies with this volume of water.5,8

We found that the dogs treated with methylprednisolone were hypotensive approximately 2 or 3 minutes after receiving the drug. Blood pressures returned to pre-drug
levels in approximately 3 to 5 minutes. Some animals that were given the drug later in the course of therapy were also ataxic for a short time, or vomited. Thus, we questioned whether the dose of steroids employed in our experiment was too large. However, for treatment of patients with severe pulmonary insult or "shock lung," pharmacologic doses of methylprednisolone (30 mg/kg) have been advocated.9

We found no evidence to support the use of methylprednisolone therapy in treating the pulmonary lesion of fresh-water near-drowning. Therapy should be individualized, with minute-to-minute monitoring of both respiratory and cardiovascular systems so that optimum breathing patterns, adequate circulating blood volume, and a reasonable cardiac output can be maintained.

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Detailed data for Pco2 and pH values of all groups can be ordered from ASIS/NAPS, Microfiche Publications, 305 East 46th Street, New York, New York 10017.

References

Oxygen Therapy

FACE MASK AND Fio2. The authors studied tracheal oxygen concentrations in patients receiving supplemental oxygen via a face mask. Inspired gas was sampled by means of a catheter. Fio2 may be affected by factors other than flow rate, such as respiratory rate, pattern of breathing, and the design and fit of the mask. When low flows were used, considerable patient variation was observed. As might be expected, when the flow rate was greater than 15 l/min this variability was reduced. Turbulence in and around the face mask resulted in dilution with room air. The use of a shield to prevent turbulence resulted in Fio2's closer to those predicted. It was felt that humidification was also improved by this change. The authors suggest that this alteration might be clinically useful. (Wexler HR, Aberman A, Scott AA, et al: Measurement of Intratracheal Oxygen Concentrations during Face Mask Administration of Oxygen: A Modification for Improved Control. Canad Anaesth Soc J 22: 417–435, 1975.)