Near-drowning: strategy for survival

Face down, the boy floated in the still water of a backyard swimming pool. But there was no one to notice. A few minutes later, when his mother came back out to see how he was doing, the serenity was shattered by her screams. The father came on the run and raced to the pool to pull the unconscious boy out of the water. What followed were the familiar frantic attempts to get the water out of the boy's lungs and revive him. Then somebody remembered to call the doctor. When he arrived, he was relieved to see that the boy was awake, and as he sent for an ambulance, his calmness seemed to be saying that everything was going to be all right.

But the fact is that many victims of near-drowning never lose consciousness, or they come to almost immediately. This is no guarantee of full recovery, however. Even those who are taken, fully conscious, to a hospital, may take a sudden turn for the worse.

After seeing more than 150 victims of drowning and persons who nearly drowned over the past ten years in Florida, Dr. Jerome H. Modell has concluded that, most lay water-safety manuals notwithstanding, attempting to get rid of inhaled water is not the first thing to do for a victim of near-drowning. Instead, he says, hypoxia and acidosis should get the top priority.

The studies of Dr. Modell and of other experts during the past few years have helped evolve a plan of therapy that begins at the site of the accident and does not end until the patient is discharged from the hospital. The emphasis has been shifted away from fluid and electrolyte disturbances, and the focus is now on restoring adequate ventilation and oxygenation and normal acid-base balance as quickly as possible. The main features, or ABCs, of clinical management, therefore, are:

AIRWAY (or ventilation)

You can't rely on the apparent duration of submersion as a guide to the victim's physiological state. As soon as he is removed from the water, check for breathing movements. If you notice breathing movements and coughing, concentrate on establishing and maintaining adequate ventilation.

If there are no breathing movements, you must take immediate action. Dr. Modell maintains that attempts to drain water from the lungs may waste precious time since the degree of hypoxia increases with the duration of apnea. Hypotonic fluid is absorbed rapidly from the lung; it is useless, therefore, to attempt to drain the lungs of someone who has been rescued from drowning in fresh water. If fluid is drained, it is most likely the result of emptying the stomach. However, after the aspiration of seawater, fluid is drawn into the lungs from the circulation, and by proper positioning of the patient you may be able to promote drainage from the trachea without compromising artificial ventilation. Otherwise, the basic emergency clinical management of freshwater and seawater victims is very nearly the same.

Whoever gets to the victim first should quickly clear the pharynx with his fingers and start exhaled-air resuscitation immediately. As soon as effective artificial ventilation is achieved, check the pulse in the carotid artery. If it is not palpable, you should start closed-chest cardiac massage at once and continue it together with artificial ventilation of the lungs. And as soon as the equipment is available, substitute positive pressure breathing with oxygen for exhaled air. Although mechanical ventilators are able to deliver a higher concentration of oxygen to the victim, these machines are not always readily available, nor are they always in working condition. Also, the equipment available is often too complicated for immediate use by the first-aider. Therefore, the most reliable types of mechanical equipment for resuscitation are the simplest, e.g., the self-inflating bag and the hand-operated oxygen valve.

Because you cannot determine the state of the patient's lungs or evaluate the adequacy of arterial oxygenation at the scene of the accident, all near-drowning victims should be admitted to the hospital, insists Dr. Modell. While transporting the patient, you should continue resuscitative measures as needed. Maximal oxygen concentration—100% oxygen, if possible—should be administered by inhalation, regardless of the apparent clinical condition of the patient.

If still required, both artificial respiration and closed-chest cardiac

continued

This article, which first appeared in the June 1970 issue of EM, *is drawn from two sources. The basic plan of therapy is based on the work of Dr. Jerome H. Modell, professor and chairman of the anesthesiology department at the University of Florida, Gainesville. The illustrative case reports were presented, originally in* Texas Medicine *(Vol. 65, No. 8), by Dr. E. Warner Ahlgren, clinical associate professor of anesthesiology at the University of Texas Southwestern Medical School and physician-director of respiratory therapy at the Children's Medical Center of Dallas, and Dr. Frederick W. Courington, presently a resident in psychiatry at the University of Florida.*

massage must be continued during the trip to the hospital. Massage may be discontinued if an effective palpable spontaneous pulse returns in the carotid artery.

Nonmedical personnel—such as first-aid squads, lifeguards, or simply the person performing the rescue—should be instructed to carry out these basic first-aid procedures for the near-drowning victim, keeping him viable until more definitive treatment can be started at the hospital. These measures—clearing the airway, starting mouth-to-mouth ventilation, and replacing this as soon as possible with 100% oxygen by inhalation—can save precious seconds and probably the patient's life as well.

BREATHING

At the hospital, begin treatment with intensive pulmonary care, Dr. Modell advises. If difficulty is encountered in establishing and maintaining a patent airway, he suggests that a cuffed endotracheal tube be inserted. Emergency tracheostomy is rarely indicated early in therapy, as most patients can be managed by nonoperative methods.

If severe respiratory insufficiency persists, a mechanical ventilator should be connected to the endotracheal tube for ventilatory support. In assessing the condition of the patient, arterial oxygen and carbon dioxide tensions, together with the acid-base status—pH and base excess—are essential and provide your most reliable guide. The amount of oxygen needed in the inspired gas to maintain an acceptable arterial oxygen tension, the pattern of ventilation that should be supplied, and the amount of buffer solution necessary for reestablishing normal acid-base balance can all be determined from the pH, paO_2, and $paCO_2$ values.

Recent studies have shown that mechanical ventilation when combined with positive end expiratory pressure can markedly improve paO_2 in both fresh- and seawater near-drowning victims.

If the patient exhibits obvious pulmonary edema and froth that obstructs the airway, inhalation of an aerosol of an antifoaming agent, such as 20 to 30% ethyl alcohol, is helpful. Racemic epinephrine by aerosol inhalation may also be useful in some cases to reduce bronchospasm secondary to the aspiration of fluid. Since near-drowning with fluid aspiration is a form of aspiration pneumonitis, steroids have been recommended in the past to decrease the inflammatory reaction and antibiotics to prevent secondary infections. Recent experiments, currently in press, do not confirm that prophylactic steroid therapy helps.

If continuous mechanical ventilation is not needed, intermittent positive pressure breathing (IPPB) therapy should be administered at frequent intervals to help reinflate atelectatic alveoli. High humidity therapy may also be indicated in selected patients to aid them in raising secretions.

"Return of consciousness is not synonymous with recovery," notes Dr. Modell. "Delayed death from hypoxia does occur." Therefore, it should be reemphasized that low arterial oxygen tension following the asphyxial episode is a clinical emergency and must be dealt with at once. He does agree that the doctor should also determine if abnormalities in serum electrolytes and blood components are present and correct them, but he insists that first priority must go to dealing with blood-gas and acid-base changes.

CIRCULATION

The length of time the victim was deprived of air varies from case to case. Although knowing the duration of asphyxia can help you make a rough prognosis, you should waste no time deliberating this factor. Recovery has been reported after submersion for up to 22 minutes. Dr. Modell says that if the pulse rate or blood pressure is feeble or absent, closed-chest massage should begin at once, and continue until a palpable, spontaneous pulse returns.

Other investigators maintain that if a spontaneous pulse is still not palpable in the hospital, 1 mg. of epinephrine should be injected into a cardiac ventricle and artificial respiration and closed-chest cardiac massage continued while an EKG examination is done to indicate the presence or absence of ventricular fibrillation. These workers add that as a matter of routine practice, they inject 1 mg. of epinephrine into the hearts of all adults and children as young as 18 months of age. They reduce the dosage proportionately for younger children. Most researchers in this field agree, however, that since metabolic acidosis almost invariably accompanies hypoxia in victims of near-drowning, IV sodium bicarbonate or some other buffer should be used routinely.

And, as soon as possible, the physician should draw arterial blood to perform those tests that will help him determine whether further bicarbonate, oxygen, or support of ventilation is needed. If hypovolemia is present, you may want to give a transfusion with whole blood, blood components, or blood substitutes. Monitoring pulmonary capillary wedge pressure may be helpful in guiding fluid replacement.

Metabolic acidosis is an almost constant finding in near-drowning victims. Because this derangement is so constant, it is now recommended that every patient receive IV sodium bicarbonate, at a dosage of about 1 mEq./kg. of body weight, in the emergency room, even before any lab studies are done. If the patient is unconscious or stuporous, you should give 2 ampuls of IV sodium bicarbonate, each ampul containing 44.6 mEq. of bicarbonate in 50 cc. of water, as quickly as possible. This treatment doesn't usually correct the metabolic acidosis totally, but it does improve the pa-

Dos and don'ts in near-drowning

ON THE SCENE

Do

1. Establish and maintain airway (A).
2. Institute mouth-to-mouth breathing (B).
3. Begin external cardiac compression if necessary to maintain circulation (C).
4. Summon emergency vehicle for transportation to a hospital.
5. Administer 100% oxygen as soon as possible (via positive pressure ventilation if necessary).

Do not

1. Attempt to drain water from the victim unless it can be done without compromising ventilation.
2. Slacken resuscitative efforts for even a few seconds.

IN THE EMERGENCY ROOM

Do

1. Maintain airway (A).
2. Maintain breathing (B).
3. Maintain circulation (C). (Defibrillate if necessary.)
4. Initiate definitive care:
   a. Give sodium bicarbonate, 1 mEq. per estimated kg. body weight, intravenously, immediately.
   b. Give 100% oxygen.
   c. Give intravenous lactated Ringer's solution—10 ml. per kg. body weight for the first hour.
   d. Insert Foley catheter into bladder and connect it to metered output container.
   e. Order laboratory studies:
      Arterial blood gases
      Serum electrolytes
      Hemoglobin and hematocrit
5. Take portable chest x-ray.
6. Transfer to intensive care facility.

Do not

1. Inflate stomach during resuscitation.
2. Hesitate to administer the bicarbonate and oxygen before getting any laboratory studies.

IN THE HOSPITAL

Do

1. Reevaluate airway and insert nasotracheal or orotracheal tube if necessary (A).
2. Reevaluate breathing; administer a high oxygen concentration and, if necessary, based on clinical status and arterial blood gases, begin mechanical ventilation and positive end expiratory pressure (B).
3. Reevaluate circulation; monitor electrocardiogram; treat for shock; watch for fibrillation (C).
4. Correct acid-base disturbance in accordance with arterial blood-gas values: administer (weight in kg. × 0.25 × base excess) mEq. $NaHCO_3$ intravenously immediately for full correction.
5. Adjust electrolytes according to serum electrolyte values.
6. Adjust fluid intake to maintain high urine output.
7. Repeat laboratory studies every four to six hours.

Do not

1. Slacken high oxygen administration unless paO_2 confirms it is no longer necessary.
2. Slacken acid-base monitoring and necessary respiratory and nonrespiratory support.

Chart developed by Drs. Frederick W. Courington and E. Warner Ahlgren. Reproduced through the courtesy of *Texas Medicine*.

tient's condition and normally suffices until the arterial blood-gas values become available. Additional doses of sodium bicarbonate may be required during therapy.

How you handle these four cardinal derangements (airway, breathing, circulation, and acidosis) is essentially an extension of the ABCs of emergency resuscitation. Sodium bicarbonate and oxygen are merely additional measures that must be provided on the way to the hospital or, at least, as soon as the patient arrives there.

Another constant feature in these situations, according to Dr. Modell, is profound hypoxia, which he believes is the primary cause of death in many cases. Since nearly all hypoxic patients vomit, aspiration from near-drowning hypoxia often occurs and causes confusion as to just what the chest x-ray does, in fact, show. Immersion pneumonitis, often unrecognized as such, is a patchy, perihilar, not peripheral, infiltrate of the lungs. The aspirated vomitus associated with hypoxia can easily mask the x-ray and make diagnosis difficult. This infiltration may persist for days. On the other hand, severe hypoxia may be present even without marked x-ray changes.

Hypothermia occurs in some of these cases and may serve to protect the hypoxic cerebrum by reducing oxygen consumption and carbon dioxide production. However, the patient's body temperature usually increases spontaneously and external warming is not generally necessary. Many patients actually develop a fever during the course of recovery.

How well do the ABCs of resuscitation work in practice? Extremely well, according to case reports presented by Drs. Frederick W. Courington and E. Warner Ahlgren. The cases involve children who nearly drowned in fresh water—by far the most common victim and circumstance of near-drowning—and illustrate the various aspects of therapy, from pool-side resuscitation, to im-

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mediate emergency room measures, to inpatient care.

Case 1. A three-and-a-half-year-old boy weighing 15 kg. was retrieved from the bottom of a swimming pool. The length of time he was submerged was unknown. After being revived at the side of the pool, he was brought to a community hospital, where oxygen, Ringer's solution, and 15 mEq. of IV sodium bicarbonate were administered. He was then transferred to Children's Medical Center.

At 7:30 p.m., upon admission to the intensive care unit, the patient had loud, grunting respiration with intercostal retraction and prolonged expiration. Semiconscious and hyperreflexic, the child had periods of tremor of the extremities, alternating with stiffness and pronation of the feet. When his name was called, he gazed at the speaker but said nothing. Despite his unusual respiration, no pathologic auscultatory signs were apparent. Ventilation appeared to be adequate.

As long as the boy was inhaling 100% oxygen, the arterial blood-gas values were pH, 7.35; pCO_2, 47 mm. Hg; pO_2, 318 mm. Hg; and delta base (or base deficit), O mEq./liter. Serum sodium was 135 mEq./liter; chloride, 100 mEq./liter; and potassium, 3.4 mEq./liter. Hemoglobin was 11.7 gm.%; hematocrit, 33%; and leukocytes, 6700/cu. mm.

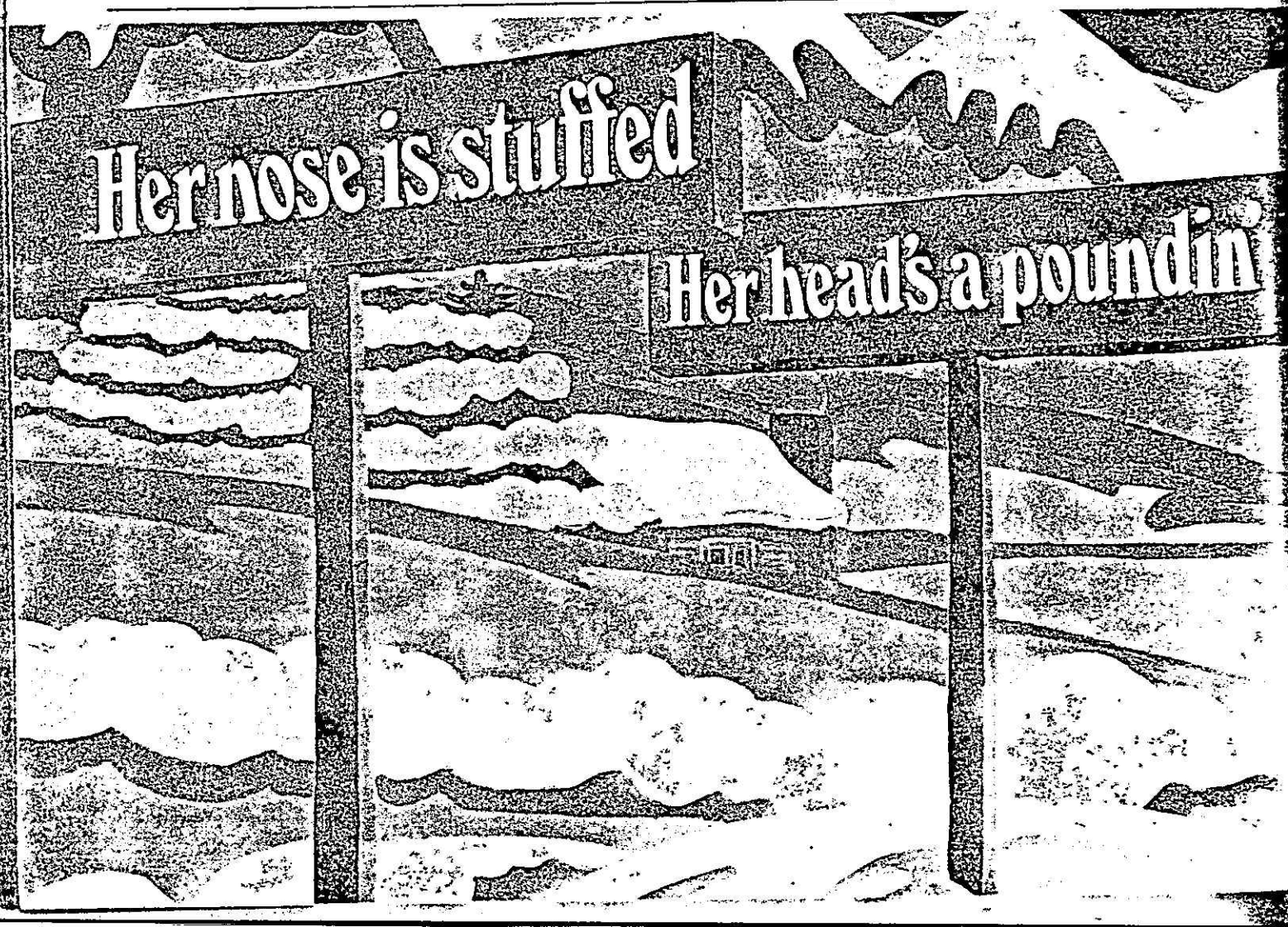
Urinalysis revealed a "moderate" level of free hemoglobin. An AP chest x-ray showed bilateral parenchymal infiltration—more evident on the right side—in the central lung fields. The child was started on ampicillin to deal with a possible superimposed pneumonia.

Next morning, the patient awoke, alert and talking, but when he tried to walk, he showed ataxia. His lungs remained clear to auscultation and showed considerable clearing of the infiltrate when x-rayed. With the patient breathing 50% oxygen, arterial blood-gas values were pH, 7.33 pCO_2, 32.5 mm. Hg; pO_2, 170 mm. Hg; and base deficit, 5 mEq./liter. The value for hemoglobin was 12.4 gm.%, and the hematocrit, 37%.

The ataxia persisted, however; repeated x-rays of the skull proved to be within normal limits. Psychological tests reportedly showed "none of the usual signs suggestive of organic brain dysfunction." At this point, oxygen was discontinued.

By the third hospital day, the patient appeared alert and active. An EEG was interpreted to be "within the range of normal variation for age." The patient was then discharged home; except for the chest x-ray, he seemed normal. A year later, the x-ray was normal.

This patient did well, Drs. Courington and Ahlgren believe, because



he received sodium bicarbonate intravenously and uninterrupted high concentrations of oxygen by mask early in his treatment. When he arrived at Children's Medical Center, he had no metabolic acidosis and only a mild respiratory acidosis. There was only slight hemodilution and an insignificant degree of hemolysis. His electrolyte values were within normal limits. The chest x-ray was typical for immersion pneumonitis in its initial stages, and rapid but incomplete clearance on the second day was still incomplete when the boy was discharged on the fourth hospital day.

Looking back over this case, Drs. Courington and Ahlgren think that antibiotic treatment for possible aspiration pneumonitis was probably unnecessary. In any event, they attribute the child's rapid recovery primarily to proper, early treatment in the community hospital.

Case 2. A 13.7-kg., two-and-a-half-year-old boy was found floating face down in a swimming pool at 5:30 p.m. by his grandmother, who said she had left him no more than ten minutes before. Although the exact timing cannot be determined, he was immersed probably less than five minutes. Upon removal from the pool, he cried and vomited, apparently without aspirating. Taken to a community hospital by ambulance, he was treated there briefly and then was sent to Children's Medical Center. The youngster received oxygen from the time that he was picked up by the ambulance.

At 6:45 p.m., about one hour and 15 minutes after being found in the pool, the boy was admitted to Children's awake and responding but disoriented. He was slightly spastic and had hyperreflexia, but no other pathologic signs were visible. Although breathing rapidly (40 per minute), with slight subcostal retraction when inhaling, he was in no respiratory distress. Blood for lab studies was drawn, an IV infusion was begun, and the child was admitted to the intensive care unit, where he was placed in an open-top tent with mist and oxygen delivered from a pair of jet nebulizers.

Arterial blood-gas values at 7:15 p.m., with the patient breathing 85% oxygen, were: pH, 7.19; pCO_2, 57 mm. Hg; pO_2, 220 mm. Hg; and base deficit, 7 mEq./liter. For his mild metabolic acidosis, he was given IV sodium bicarbonate, 30 mEq.; first, 20 mEq. injected rapidly and then 10 mEq. mixed with the IV infusion. Serum electrolyte values were: sodium, 127 mEq./liter; chloride, 94 mEq./liter; and potassium, 3.9 mEq./liter. The hemoglobin value was 11.9 gm.%; hematocrit, 35%; and leukocytes, 14,650/cu. mm., with a normal differential.

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An AP chest x-ray, taken upon admission—about an hour and a half after his near-drowning—showed patchy perihilar infiltrates and clear peripheral lung fields.

Another arterial blood-gas study at 9 p.m., with the boy breathing 85% oxygen, showed pH, 7.34; pCO_2, 52 mm. Hg; pO_2, 350 mm. Hg; and base excess, 0.5 mEq./liter. The patient seemed more alert and relaxed at this time.

By the following morning he had improved sufficiently to be moved from the intensive care unit to a regular hospital room. The AP chest x-ray was repeated and showed significant clearing.

On the morning of his third hospital day, the child was breathing room air, and his arterial blood-gas values were pH, 7.33; pCO_2, 40 mm. Hg; pO_2, 84 mm. Hg; and base deficit, 4.5 mEq./liter. The hemoglobin value was 13.1 gm.% and the hematocrit, 36%. As for serum electrolyte values, they were now sodium, 134 mEq./liter; chloride, 104 mEq./liter; and potassium, 4.8 mEq./liter. Now clinically well, the patient was discharged from the hospital.

This patient received no bicarbonate therapy prior to his transfer to Children's Medical Center, although he was given oxygen previously. In this case, the more traditional approach of correcting acidosis *after* determining arterial blood-gas values was adopted, instead of the preferred practice of giving bicarbonate early. The patient showed mild metabolic and respiratory acidosis and a large intrapulmonary shunt, as evidenced by an arterial oxygen tension of only 220 mm. Hg while he was breathing 85% oxygen. The sodium and chloride values suggested some dilution from inhaled water. The chest x-ray revealed the typical immersion pneumonitis; subsequent films, however, showed significant clearing.

Case 3. This patient, a boy a little more than two years old, was the worst off of the three young victims of near-drowning. He was found floating face down in a swimming pool at 2:30 p.m. by his mother and an intensive care unit nurse who happened to be visiting. As soon as they pulled the child out of the water, the nurse gave mouth-to-mouth resuscitation and directed the mother to apply external cardiac compression. These measures promptly revived him. Before transfer to the Children's Medical Center, he was taken to a community hospital, where IPPB with oxygen was begun and IV fluids were administered by cutdown.

When he arrived at Children's Medical Center at 5:30 p.m., the boy was comatose and rigid, and making no effort to breathe. His skin was cold and clammy, pulse was feeble and slow—60 per minute—and the rectal temperature was 30° C. He responded only to deep pain stimulation. His pupils were dilated and barely reactive to light; there were no deep-tendon reflexes.

On auscultation, he had rales and rhonchi in both lungs—perhaps because he had had bronchitis for four days before falling into the pool. Arterial blood-gas values, with the patient breathing 40% oxygen, were: pH, 7.05; pCO_2, 42 mm. Hg; pO_2, 20 mm. Hg; and base deficit, 18 mEq./liter. The hemoglobin value was 13.1 gm.%; hematocrit, 39%; leukocytes, 21,300/cu. mm., with 87% lymphocytes. Lab data obtained from the community hospital where the child was first taken disclosed the following values: sodium, 129 mEq./liter; chloride, 100 mEq./liter; potassium, 3.4 mEq./liter; bicarbonate, 11.7 mEq./liter; and BUN, 18 mg./100 cc.

Treatment consisted of nasotracheal intubation, mechanical ventilation with 100% oxygen, and warming by a circulating water mattress. The child, who weighed 13.6 kg., also received 25 mEq. of sodium bicarbonate and 200 mg. of hydrocortisone sodium succinate (*Solu-Cortef*, Upjohn) intravenously.

By 6:45 p.m., an hour and a quarter after admission, his body temperature had risen from 30° C. to 35° C. He was now breathing spontaneously and opening his eyes, but he still failed to respond to stimulation. Arterial blood-gas studies were repeated at 7:30 p.m., when his rectal temperature was 37.5° C. and he was breathing 100% oxygen. The findings were: pH, 7.28; pCO_2, 42 mm. Hg; pO_2, 42 mm. Hg; and base deficit, 7 mEq./liter. The child was then given 22 mEq. of sodium bicarbonate intravenously.

An AP chest x-ray showed scattered pneumonitis, which was denser in the left upper lobe. (The previous chest x-ray, taken at the community hospital, had been normal.) At 9 p.m., the patient's pupils were found to be reactive and in addition he was responding to the voice.

By the next morning, the boy was alert and responsive; his respiratory rate, however, was 70 per minute. At 9 a.m., he was still breathing 100% oxygen, and his arterial pH was 7.40; pCO_2, 30 mm. Hg; pO_2, 149 mm. Hg; and base deficit, 5 mEq./liter. Another AP chest x-ray was made and it now showed some clearing. The boy was given 78 mEq. of sodium bicarbonate intravenously. He was suctioned well, given chest physiotherapy, and briefly hyperinflated. The nasotracheal tube was then removed. Next, the child was placed in an atmosphere of 60% oxygen. At 8 p.m., he was still breathing rapidly. However, his lung fields were clear to auscultation, and he was coughing well.

On the third hospital day, while breathing room air, the patient's arterial blood-gas values were: pH, 7.41; pCO_2, 41 mm. Hg; pO_2, 56 mm. Hg; and base excess, 1 mEq./liter. His lung fields continued to clear.

On the fourth day, he was discharged, apparently well. Residual tubular breath sounds, scattered squeaks, wheezes, and rhonchi were

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the only adverse sign still evident as he left for home.

The most fortunate thing that happened to this severely ill child was the fact that he was discovered in the pool by a nurse who was trained in cardiopulmonary resuscitation. And, working with the mother, she succeeded in reviving him.

At the community hospital, the child had an intravenous cutdown done and was ventilated via a mechanical respirator, which delivered 40% oxygen. He was given no sodium bicarbonate, however, although he did get isoproterenol because of bradycardia. Since he had signs of cerebral edema, steroids were started to help reduce inflammation and swelling.

Initial treatment at the Children's Medical Center was aimed at correcting the hypothermia, establishing optimal ventilation, and correcting the severe hypoxia and metabolic acidosis. Although the chest x-ray taken at the community hospital was clear, the one taken at the medical center revealed patchy pneumonitis. Because the neurological signs probably represented the effects of cerebral edema, the hypothermia was beneficial.

With warming and improved peripheral circulation, which caused flushing of the anaerobic metabolites from previously poorly perfused skeletal muscles, the child required further bicarbonate administration to correct the metabolic acidosis. A large intrapulmonary shunt persisted throughout most of his stay in the hospital. Despite a pO_2 of 56 mm. Hg while breathing room air, he was well enough to go home on the fourth hospital day.

As the clinical management of these cases has demonstrated, the emphasis in the care of the near-drowning victim has shifted away from stress on electrolyte changes. Whether at the site of rescue, in the ER, or in the intensive care unit, the advice of the experts now is to concentrate on airway, breathing, circulation—the ABCs of resuscitation—and on acidosis. □

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