Modern concepts about drowning

F. Segarra,* M.D. and R. A. Redding, M.D., F.R.C.P.[C], *Pawtucket, R.I.*

Summary: Drowning remains one of the three leading causes of accidental death in the world, yet few textbooks deal with its evaluation and treatment. Because of overemphasis placed upon distinguishing between fresh- and sea-water drowning, the importance of immediate attention to simple and effective resuscitative procedures has been bypassed. We have outlined a continuum of evaluation and treatment in four stages: (1) emergency care at the scene and during transportation, (2) hospital admission resuscitation, (3) intensive care, and (4) return to normal. Four representative cases are presented in order to review pathophysiological concepts in this serious problem.

Résumé: *Concepts modernes concernant la noyade*

Même si la noyade demeure une des trois causes principales de mort accidentelle dans le monde, peu de manuels s'occupent de son évaluation et de son traitement. On a trop insisté sur la distinction à établir entre la noyade en eau douce et en eau salée et on a négligé d'accorder une attention immédiate aux moyens de réanimation simples et efficaces. Nous avons proposé un continuum d'évaluation et de traitement, divisé en quatre étapes: (1) les soins d'urgence sur la scène de la noyade et durant le transport du noyé, (2) les moyens de réanimation lors de l'admission à l'hôpital, (3) les soins intensifs et (4) le retour à la normale. Nous présentons quatre cas typiques, pour passer en revue les concepts physiopathologiques de ce grave problème.

Drowning traditionally has been defined as inundation of the airways with water, preventing gas exchange and resulting in asphyxia. Thus drowning involves rapid development of hypoxia, hypercapnia and respiratory acidosis, with an associated sequence of apnea, hypertension, bradycardia, arrhythmias, hypotension and terminal gasping.

Of the 140,000 or more drowning deaths in the world each year, more than half are of young adults or children. There is estimated to be at least one survival for each death.[1] Considering the increasing popularity of water sports (e.g. swimming, boating, surfing, skin-diving), combined with alcohol indulgence as a contributory cause, drowning has created a major need for improvements in critical-care medicine in many parts of the world. In the United States drowning accounts for over 7000 deaths per year, so that it ranks as one of the three leading causes of accidental death.[2] There is no doubt that this figure can be reduced, but the key to improved survival lies in the first few moments of resuscitation of the drowning victim.

We present four representative cases of drowning dealt with in our hospital in order to illustrate several important aspects of evaluation and treatment.

Case reports

Case 1

A 16-year-old boy was brought to the hospital after submersion for 5 to 15 minutes in a fresh-water pond, 20 minutes away from the hospital. No immediate mouth-to-mouth resuscitation was given when the youngster was pulled from the water and only oxygen-enriched air was given en route to hospital. On admission the patient was unconscious, tachypneic and diaphoretic. Spasticity, clonus and trismus were observed; the pupils were constricted and nonreactive to light. Rales were audible in the bases of both lungs. Although the patient was normotensive, atrial fibrillation was present. Endotracheal intubation was delayed and oxygen administration continued; blood gas values at that time were: pH 7.03, P_{CO_2} 10 mm Hg, and P_{O_2} 113 mm Hg. Intravenous bicarbonate promptly corrected the pH to satisfactory levels, while 50 to 60% inspired oxygen was required to maintain reasonable arterial oxygen levels. Plasma hemoglobin was 120 mg/dl on admission and 11 mg/dl one day later. A chest radiograph on admission was normal, but 2½ hours later diffuse perihilar alveolar infiltrates suggestive of pulmonary edema were observed. Serum electrolytes, blood urea nitrogen and hemoglobin were normal on admission. A leukocytosis with polymorphonuclear preponderance was present throughout his hospital stay.

Five hours after admission the patient's condition deteriorated and he developed decerebrate rigidity and fixed dilated pupils. A fever of 106°F was controlled with refrigerated blanket cooling. In spite of continued therapeutic efforts he died on the ninth hospital day. A postmortem examination showed necrotizing bacterial pneumonitis, multiple acute "stress" ulcers of the gastric mucosa, and extensive cerebral softening and necrosis with edema.

Case 2

A 55-year-old woman drowned in a swimming pool only five minutes away from the hospital. Estimated submersion time was 5 to 15 minutes but mouth-to-mouth resuscitation had been given at the pool-side and en route to hospital. On admission the patient was unconscious, in severe respiratory distress, and suddenly became apneic. She was immediately intubated and artificial ventilation was begun. Blood gases taken at the time of intubation were: pH 7.37, P_{O_2} 38 mm

From the Pulmonary Division, Department of Medicine, The Memorial Hospital, Pawtucket, Rhode Island

*Currently at Lakeville Hospital, Lakeville, Massachusetts

Reprint requests to: Dr. R. A. Redding, Director, Pulmonary Division, The Memorial Hospital, Prospect St., Pawtucket, R.I. 02860

Hg, Pco_2 48 mm Hg. The patient had evidence of severe neurological impairment on admission with tetraparesis, left hyperreflexia and bilateral Babinski's sign. Later a neurologist confirmed the diagnosis of cerebral edema, but also suggested that a cervical cord injury, possibly due to spondylosis and hyperextension, may have been the cause of her drowning. Serum electrolytes and blood urea nitrogen were normal at admission and remained so. A leukocytosis was initially present with a marked shift to the left. A plasma hemoglobin of 128 mg/dl on admission was negligible within 24 hours. An admission chest radiograph showed extensive patchy infiltrates in both lung fields, but repeat films became normal within one week. The patient left the hospital 42 days after admission, nearly fully recovered; the long stay was mainly required for intensive physiotherapy for her muscle weakness and speech therapy for dysarthria.

Case 3

A 14-year-old boy, partially disabled with post-poliomyelitis kyphoscoliosis, apparently developed muscle cramps while swimming in a pond and sank below the surface. He was rescued by a friend within minutes and promptly given artificial respiration and transported to hospital. In the emergency room he was nearly fully conscious, and except for excessive vomiting was asymptomatic. Rapid recovery occurred and no abnormalities were detected in a variety of relevant blood tests. He was discharged three days after admission.

Case 4

A 35-year-old physician was found submerged in his motel swimming pool by a colleague. Mouth-to-mouth ventilation and transportation to hospital were rapidly executed. On admission he was semiconscious, restless, and in moderate respiratory distress. He vomited large quantities of food and alcohol, but showed no evidence of aspiration. Crepitant rales were heard in the right lower lung, but chest radiographs were normal throughout his hospitalization. An electrocardiogram showed a sinus tachycardia with mild ST elevation and T-wave inversion; within hours the ECG became normal. Arterial blood gases showed primarily a metabolic acidosis: pH 7.15, Po_2 125 mm Hg (patient breathing oxygen by nasal cannula) and Pco_2 33 mm Hg. Intravenous bicarbonate promptly corrected the acidosis and hyperventilation. Electrolytes, blood urea nitrogen and plasma hemoglobin were also normal. The initial leukocytosis with a shift to the left was no longer evident after two days. The patient improved rapidly and was discharged four days after admission.

Discussion

Four kinds of water drowning are now recognized:[3] (1) "wet drowning"; (2) "dry drowning", i.e. asphyxia due to glottic spasm without water entry into the lungs; (3) "secondary drowning", i.e. delayed death from drowning, between 15 minutes and several days after the event; and (4) the "immersion syndrome", i.e. sudden death as the result of sudden contact with very cold water, vagal inhibition causing cardiac arrest.[4]

British and American authors give an incidence of 80 to 90% for classical wet drowning and only 10 to 20% for dry drowning.[2,3] As illustrated by Case 1, even if initial resuscitative efforts appear to be successful, the patient may still die from secondary drowning.[5] Little attention has been directed towards the causes, mechanisms and management of secondary drowning. The very few standard medical textbooks that mention drowning and its sequelae still place unwarranted emphasis on the type of drowning (i.e. sea-water versus fresh-water) in determining treatment. Innumerable studies in experimental drowning of animals have been reported, but four major differences are apparent between animal drowning in the laboratory and human drowning in natural waters.[5]

First, the degree of hemodilution noted in fresh-water drowning in animal experimentation has seldom been documented in man; a shock-like picture is more often noted in man. Hemolysis and electrolyte disturbances, which frequently occur in drowning animals,[6-10] have not been prominent in man. In a review of 50 cases of near-drowning, Fuller[4] found no hemodilution reflected by changes in hemoglobin, hematocrit or specific gravity of the urine, either in fresh- or sea-water drowning. Similar findings were recently reported by Modell and coworkers.[11]

Second, pulmonary edema, as suggested by chest radiographs in two of the four cases reported here, is common in man but frequently not observed in the laboratory animal.

Third, the incidence of ventricular fibrillation in human drowning is difficult to estimate from available data, but is probably much lower than in animal experiments.[7] The reason might be associated with a dearth of significant electrolyte disturbances in man.[8]

Fourth, accidental human drowning may differ significantly from the laboratory experiment in its environmental setting and context. Unlike laboratory distilled water or saline, aspirated natural water contains impurities that can be toxic to the lung, such as mud, sand, sewage, bacteria, detergents and chlorine. Unlike animal models, human victims can be affected by chronic diseases, previous exposure to sun or cold, excessive exercise, exhaustion, or associated trauma, all of which complicate the picture.

Irregular patchy lung infiltrates of

rather central location is the radiological picture most commonly seen; however, on occasion small nodular densities distributed throughout both lung fields can be observed.[12] The picture of pulmonary edema is usually not attributable to left ventricular failure, but is more likely related to focal changes in permeability across the alveolar-capillary membrane.

Experimentally, equal volumes of sea water and fresh water cause different degrees of hypoxemia and suggest the possibility of more than one pathogenetic factor.[2] Patients frequently show a large alveolar-arterial oxygen difference when breathing 100% oxygen for more than 20 minutes, suggesting that perfusion of non-ventilated alveoli may be one major factor causing hypoxemia in the acute stages of human near-drowning. The absolute right-to-left shunt was found to decrease after 24 to 48 hours; either the shunts had ceased or alveoli were re-expanding by then.[11] Increase in physiological dead space (wasted ventilation) and a decrease in lung compliance have also been observed.[13] It has been postulated that the decreased lung compliance is due to destruction or alteration of lung surfactant, causing an increase in surface tension and alveolar collapse. After 48 hours and improvement of the right-to-left shunt, persistent alveolar-arterial oxygen differences have been observed, possibly attributable to localized areas of alveolar hypoventilation, but also to alterations at the alveolar-capillary membrane with changes in diffusion. Thus the acute hypoxemia has been largely ascribed to perfused but nonventilated alveoli (i.e. an intrapulmonary shunt), whereas persistent low arterial oxygen tension most likely represents perfused but poorly ventilated alveoli, a response of the lung to chemical irritation and aspiration pneumonitis. The occurrence of acute renal dysfunction in near-drowning is not uncommon, with oliguria, proteinuria, and rising blood urea nitrogen and creatinine. The cause is most frequently acute tubular necrosis provoked by a severe degree of hypoxia, hypotension or both. In drowning accompanied by trauma, myoglobinemia may contribute to renal failure.[14] Hemoglobinemia was observed in our cases and occurs commonly; however, rarely will it cause renal impairment. In surviving cases there is usually a spontaneous recovery of renal function within seven to ten days.

The commonly accepted belief that survival from drowning leaves no sequelae is a misconception. One of our patients (Case 2) had severe speech difficulties and tetraparesis for months after her accident. Odahara and Fukuyama[15] recently observed that drowned children may live with permanent severe neurological deficits.

Treatment

Fluid and electrolyte changes have been emphasized in the past, directing treatment mainly towards the nature of the immersion, sea- versus freshwater, rather than towards the individual's immediate problems. A practical approach to the management of any near-drowning victim divides treatment into four stages.[16]

Stage 1. On-the-scene emergency care

It should be emphasized that this stage is most important in determining survival. As in any resuscitation procedure the ABC's of emergency care are utilized: (A) establishing and maintaining an *A*irway; (B) providing mouth-to-mouth ventilation if the patient is not *B*reathing or is breathing inadequately; and (C) applying external *C*ardiac massage if no pulse or heart beat is felt or heard. Supplemental oxygen should be quickly available and used as an adjuvant rather than as a substitute for adequate ventilation.

Emergency care continues through the rapid and safe transportation of the patient to the hospital. The use of helicopters promptly brought to the accident scene, bearing a specially trained team including a physician and pertinent resuscitation equipment, has been recently proposed by French investigators, and their preliminary reports are quite encouraging.[17,18]

Stage 2. Emergency room

Continuity of care is absolutely necessary when the patient enters the hospital. In addition to what has been done en route, insertion of an endotracheal and a nasogastric tube, and measurement of arterial blood gases should be considered priority items. The use of a ventilator should be determined by initial evaluation of the patient's minute ventilation, tidal volume, respiratory rate and colour of the skin. While awaiting the blood gas results the use of oxygen-enriched air is probably safe, and periodic hyperinflation or even constant positive-pressure breathing (CPPB) could counteract collapsed alveoli in selected cases. It should be noted that pressure-preset ventilators may require high pressure settings in order to adequately ventilate low compliance lungs, and that ruptured alveoli and pneumothorax may be anticipated. Intravenous bicarbonate should be administered to correct the metabolic acidosis which almost invariably is present. While establishing an intravenous route, determinations of electrolytes, blood type, plasma hemoglobin, and leukocyte count and dif-

ferential should be done. Catheterization may be necessary to monitor hourly urinary output particularly if shock is present. Lastly, appropriate radiography and an electrocardiogram may be needed.

Stage 3. Intensive care unit

The nearly-drowned patient will require frequent scrutiny of vital signs, cardiovascular status, ventilatory function and mental status, which usually are obtained only in an intensive care unit with the aid of flow charts.

Frequent measurement of blood gases will be helpful in adjusting ventilation, assessing and correcting acidosis, and adjusting inspired oxygen concentrations to maintain a normal arterial oxygen tension between 60 and 100 mm Hg. The use of plasma expanders such as low molecular weight dextran can be useful in combating shock. If attention is paid to balancing circulating blood volume with appropriate fluid replacement based on central venous pressure measurements and hourly urinary output, vasopressor therapy will rarely be needed. Bronchospasm can be relieved by small doses of inhaled β-adrenergic drugs (isoproterenol) or by intravenous aminophylline. To minimize the bronchiolar-alveolar response to polluted water or aspirated gastric contents, very high doses of intravenous corticosteroids given during the first few days have been proved useful. For example, the equivalent of 0.5 g of hydrocortisone sodium succinate every six hours intravenously is considered sufficient. Bacterial infection can supersede the acute irritant action of inhaled material and should be anticipated by obtaining daily cultures. Since infections are frequently caused by gram-negative bacteria, appropriate broad-spectrum antibiotics may be withheld for one or two days to permit cultures to be obtained. Diuretics have been frequently used to treat the associated pulmonary edema, but are generally of little benefit since the edema is usually inflammatory rather than a result of left ventricular failure, and diuresis can aggravate or precipitate hypovolemia rather than correct the pulmonary problem. Although it has been postulated that heparin may prevent pulmonary capillary thrombosis,[12] its general use is not warranted because the risk of bleeding by far exceeds its potential benefit. Cardiac arrhythmias are not uncommonly encountered in nearly drowned patients, making ECG monitoring and use of pertinent antiarrhythmia drugs necessary in such cases.

Stage 4. Return towards normal

This stage is much less dramatic, in that decision-making is much less urgent; however, this phase is equally important to the patient's eventual recovery. Predisposing conditions that contributed to the drowning accident or resulted from it now require evaluation and treatment. For example, treatment of trauma, including fractures of long bones or spine, may be necessary. In the meantime, weaning from the ventilator, closure of the tracheostomy, and treatment of frequent nosocomial pulmonary or renal infections must be instituted. Neurological sequelae may require many months of arduous effort by both the patient and members of the physiotherapy department. The patient in Case 2 required speech therapy and active exercise rehabilitation for several months, even after discharge from hospital.

In summary, a continuum of emergency care commencing at the accident scene and leading to assessment and therapy for the primary event and subsequent associated complications, is vital if improved survival and successful rehabilitation of the drowning victim is to be accomplished.

References

1. BIRCH AC: Drowning. *Practitioner* 206: 644, 1971
2. MODELL JH: *Pathophysiology and Treatment of Drowning and Near-Drowning.* Springfield Ill, Thomas, 1971
3. RIVERS JF: Drowning: its clinical sequelae and management. *Br Med J* II: 157, 1970
4. FULLER RH: Drowning and the post-immersion syndrome: a clinicopathological study. *Milit Med* 128: 22, 1968
5. MILES S: Drowning. *Br Med J* III: 597, 1968
6. GREEN DG: Drowning (chap 47), in *Handbook of Physiology,* American Physiological Society, Baltimore, Williams and Wilkins, 1965, section 3, vol II, pp 1195-1204
7. MODELL JH, MOYA F: Effects of volume of aspirated fluid during chlorinated fresh water drowning. *Anesthesiology* 27: 662, 1966
8. SWAN HG, SPAFFORD NR: Body salt and water changes during fresh and sea water drowning. *Tex Rep Biol Med* 9: 356, 1951
9. MODELL JH, WEIBLEY BS, RUIZ BC, et al: Serum electrolyte concentrations after fresh water aspiration. *Anesthesiology* 30: 421, 1969
10. REDDING JS, VOIGT GC, SOFAR P: Treatment of sea-water aspiration. *J Appl Physiol* 15: 1113, 1960
11. MODELL JH, DAVIS JH, GIAMMARA ST, et al: Blood gases and electrolyte changes in human near-drowning victims. *JAMA* 203: 337, 1968
12. S'JONGERS JJ, VOGELAERE P, SEGERS A, et al: Physiopathologie et traitement de la noyade. *Brux Med* 50: 295, 1970
13. HALMOGY DPJ, COLEBATCH HJH: Ventilation and circulation after fluid aspiration. *J Appl Physiol* 16: 35, 1961
14. GRAUSZ H, AMEND WJC JR, EARLEY LE: Acute renal failure complicating submersion in sea-water. *JAMA* 217: 207, 1971
15. ODAHARA M, FUKUYAMA S: Four cases of post-drowning disease. *Jap Pediatr J* 73: 262, 1970
16. Guidelines to management of some respiratory problems (editorial). *Clin Anesth* 3: 198, 1968
17. DUPOUX J, HAGLUND P, CASTAING R: Peut-on améliorer le secours aux noyés des plages océanes? Résultats de l'expérience de secours médical héliporté aux noyés, réalisée par le Centre de Réanimation Respiratoire de Bordeaux pendant l'été 1969. *Bord Med* 3: 1639, 1970
18. DUPOUX J, SURVEILLE J, FAVAREL-GARRIGUE JC, et al: L'emploi de l'hélicoptère pour l'évacuation des blessés de la route, et des noyés est-il justifié? Relation d'une expérience de secours aux blessés de la route, pratiquée dans le Sud-Ouest pendant l'été 1967. *Rev Corps Sante Armees* 9: 735, 1968