Part X: Trauma

Chapter 72: Near-Drowning

T E Oh

Death by drowning claims over 700 lives each year in Britain, 500 in Australia, and 6000 in the USA; and the rate continues to rise in the last 2 countries. The incidence on a global scale is estimated to be 5.6 deaths per 100,000 population. In Queensland, drowning now accounts for most deaths in the under 5 years old, more than road accidents or congenital abnormalities. Alcohol consumption and epilepsy are prominent factors in deaths by drowning. Death may be caused by laryngeal spasm, lung reflexes, and vagal cardiac effects (ie, "immersion") rather than true drowning with aspiration of fluid. Drowning can occur in very shallow water, and the volume of water inhaled by a drowned victim may be relatively small.

Near-drowning may then be defined as survival, at least temporary, following asphyxia while immersed in a liquid medium. Useful figures of near-drowning cases in Australia are unknown.

Pathophysiology

Upon submersion, there is an initial period of voluntary apnea. The "diving reflex" (as induced by cold-water immersion of the face) consisting of apnoea, bradycardia, and intense peripheral vasoconstriction with preferential blood shunting to the heart and brain, occurs in infants and toddlers, and to a lesser extent in man. Initial voluntary apnoea reaches a "breakpoint" (determined by hypercarbic and hypoxic drives) when involuntary inspiration is made. Water then enters the lungs, and at the same time, gasping occurs. Laryngeal spasm may follow in some victims. Airway resistance is increased, reflex pulmonary vasoconstriction occurs, surfactant is diminished, and lung compliance is decreased. Water shifts from alveoli into the circulation. Swallowing, vomiting and aspiration of vomitus is likely. A phase of secondary apnoea follows within seconds of immersion, proceeded by further involuntary gasping and loss of consciousness. Respiratory arrest and cardiac arrhythmia occur several minutes later and precede death.

Hyperventilation before diving increases the risk of death by drowning. The resultant hypocarbia will suppress the central drive to breathe, even in the presence of severe hypoxaemia from the prolonged voluntary breath holding. Consciousness is lost before spontaneous central respiratory efforts resume.

Nature of Inhaled Fluid

In fresh water drowning, water is quickly absorbed into the circulation and may cause haemolysis. Pulmonary surfactant characteristics are altered (denatured) producing widespread atelectasis. Electrolyte changes are usually insignificant and transient. Haemolysis may unusually be significant, which may then produce haemoglobinuria and acute renal failure. Any chlorine and soap in fresh water does not appear to be of any adverse consequence to the lungs.

In sea water drowning, the hypertonic salt water promotes rapid fluxes of water and plasma protein into the alveoli and interstitium, dilutes or washes out surfactant, and disrupts the alveolar-capillary membrane. Both inhaled fresh and sea water produce an inflammatory reaction in the alveolar-capillary membrane, leading to an outpouring of plasma-rich exudate into the alveoli. Inhaled gastric contents may contribute to this reaction.

Lung Injury

Regardless of whether fresh or salt water was the immersion medium, the above changes lead to widespread atelectasis, pulmonary oedema, severe intrapulmonary shunting, gross ventilation:perfusion mismatch, increased pulmonary vasoconstriction, decreased compliance, and marked hypoxaemia. Hypoxaemia and large increases in intrapulmonary shunting can occur with inhalation of as little as 2.5 mL/kg body weight.

Denaturization of surfactant can continue despire successful resuscitation. The term "secondary drowning" has been used to describe pulmonary insufficiency which may develop any time up to 72 hours after the event. This occurs after a period of improvement following resuscitation and is seen in about 5% of survivors. Hyaline membrane formation in small airways and alveoli has been demonstrated at autopsy in patients who have survived from 12-72 hours. Infection and theadult respiratory distress syndrome (ARDS) may follow a near-drowning incident.

Dry Drowning

An estimated 10-20% of drowning are "dry", ie, little or no fluid is found in the lungs at autopsy. It has been suggested that in this group, the initial entry of water into the larynx may produce a (vagal) reflex laryngospasm which persists until asphyxial death supervenes. The laryngospasm is followed immediately by an outpouring of thick mucus, which with bronchospasm, may prevent entry of water when the spasm relaxes shortly before death. "Dry drowning" appears to be more common in adults, and facilitation of such pulmonary reflexes by raised blood alcohol levels has been suggested.

Superimposed Hypothermia

If the environment is cold, cases of drowning may be complicated by acute hypothermia. Cold water impairs motor activity and movement. Even stron swimers with life-jackets drown within minutes if the water is very cold (eg, 4°C). Uncontrolled involuntary hyperventilation occurs in immersion in a cold medium, and consciousness may be impaired in hypothermia. Drowning will result when there is no control of respiration. Hence submersion is not essential for drowning and life-jackets will not always prevent drowning.

Cardiovascular Effects

The cardiovascular system of most near-drowned victims shows remarkable stability. A wide variety of ECG changes have been reported, but early reports of ventricular fibrillation secondary to fresh water aspiration were probably overemphasized. Blood pressure changes seem to be secondary to the state of oxygenation. Blood volume changes secondary to fresh or salt water aspiration are rarely significant to be life-threatening. Consequently, changes in haemoglobin and haematocrit are usually not marked.

Management

The basic pathophysiological problems of fresh and salt water drownings are similar, ie, hypoxaemia, pulmonary oedema, metabolic acidosis and circulatory dysfunction. Initial management of the critically ill survivor is thus similar, discounting whether fresh or salt water was the immersion medium. Therapy is directed towards restoring adequate oxygenation and circulation, correcting acid-base imbalance, and cerebral resuscitation and protection. Many regimens such as HYPER which aggressively treats over *hy*dration, *p*yrexia, *e*xcitability, and *r*igid motor posturing use empirical and controversial treatment methods discussed below.

1. Immediate First Aid Treatment

Cardiopulmonary resuscitation is initiated. Lung drainage procedures are controversial. There is a likelihood of inducing vomiting, since over half the immersion victims vomit during resuscitation. Portable oxygen-powered suckers are inadequate for aspiration of vomitus. Mouth-to-mouth ventilation with external cardiac massage where indicated, should be instituted immediately. Oesophageal obturators are useful in experienced hands, but the mask and bag resuscitators are generally unsuitable outside the hospital environment. Oxygen should be given and the victim kept warm while en route to hospital. The possibility of spinal injury, especially in diving or surfing accidents, must be remembered during resuscitation.

2. Hospital and Intensive Care Treatment

It is important to know the time and place of the immersion, the immersion medium and its temperature and degree of contamination, the resuscitation details including duration of apnoea or asystole, the level of consciousness at the time, whether head or neck injuries were sustained, and the past health of the victim (eg, whether an epileptic, asthmatic, or alcoholic).

(a) *Restoring Ventilation and Oxygenation*

Oxygen is given by a semi-rigid mask if the patient is breathing spontaneously. Bronchospasm if present, is relieved by aminophylline and beta-2 adrenergic agents. Comatose patients are intubated. Mechanical ventilation is instituted in patients with severe hypoxaemia and pulmonary oedema. Positive end expiratory pressure (PEEP) improves pulmonary oedema as well as ventilation:perfusion mismatch. The level of PEEP varies with the clinical situation. Intermittent mandatory ventilation (IMV) with PEEP may be used to control $PaCO_2$ if the patient is able to tolerate the ventilation pattern. If the patient is able to spontaneously maintain a normal $PaCO_2$ without too much effort, continuous positive airway pressure (CPAP) may be used instead of controlled ventilation. (See Chapter 22, Mechanical Ventilatory Support.) Awake patients seldom require endotracheal intubation. CPAP, by means of a tight-fitting CPAP facemask, is applied to these patients. Treatment of ARDS is described in Chapter 25, Adult Respiratory Distress Syndrome.

(b) ***Restoring Circulation***

Low cardiac output is corrected by positive inotropic agents (eg, adrenaline, dopamine or dobutamine infusion) and fluid replacement. Isotonic fluids are usually all that is required, but plasma and blood may be needed if haemolysis is severe. Fluid replacement is guided by central venous pressure and pulmonary capillary wedge pressure measurements in case of shock. The regimen of fluid restriction with IV frusemide, aimed at lowering raised intracranial pressure in near-drowned vistims is controversial.

Arrhythmias from acidosis, hypoxia, hypothermia and electrolyte abnormalities are treated conventionally. (See Chapter 7, Cardiac Arrhythmias.)

(c) ***Correcting Acidosis***

Intravenous sodium bicarbonate (50-100 mmol) is given if the metabolic acidosis is significant (eg, pH < 7.0).

(d) ***Rewarming***

Core temperature must be kept above 28°C, as temperature below that may give rise to spontaneous ventricular fibrillation and coma may be anticipated at temperatures below 30°C. Induced hypothermia for brain protection in near-drowning victims (eg, using surface ice packs) is controversial and its effectiveness unknown. Normothermia should probably be maintained. However, it is pointless to rewarm the immersed victim rapidly is his temperature is above 30°C. Rewarming can be accomplished over about 6 hours by warmed intravenous fluids, humidification of inspired gases, and heated blankets. Hot baths are difficult to carry out in practice. More aggressive forms of treatment include warm peritoneal lavage and cardiopulmonary bypass but are rarely indicated.

(e) ***Cerebral Protection***

Attempts at brain resuscitation and protection are probably important. (See Chapter 41, Cerebral Protection for full discussion.) Treatment protocols include intracranial pressure monitoring, hyperventilation (to maintain a $PaCO_2$ of approximately 30 mmHg or 4 kPa), lowering raised body temperature, maintaining adequate oxygenation and circulation, and controlling intracranial hypertension, hypertension, hyperglycaemia and fits. Steroids and barbiturate therapy are controversial - their benefits remain unproven and they should probably not be given.

(f) ***Other Treatment***

In general, prophylactic antibiotics are not useful. A broad spectrum antibiotic (eg, amoxicillin or cephalosporin) is indicated if there are signs of infection. It may be necessary to prescribe against Gram-negative and anaerobic bowel organisms (eg, with gentamicin and metronidazole). The site of immersion may have some influence on the type of inhaled organism and thus antibiotics. A nasogastric tube should be inserted to decompress the stomach and drain possible large volumes of water.

Investigations and Monitoring

1. Cardiovascular Monitoring

This includes ECG, arterial and central venous pressure, and (if indicated) a Swan-Ganz catheter for pulmonary artery pressure.

2. Oxygenation

Monitoring of arterial blood gases, saturation (by pulse oximetry) and lung shunting (eg, alverolar-arterial oxygen gradient) will indicate progress and guide therapy.

3. Body Temperature

4. Serum Biochemistry

Theoretically, serum electrolyte levels fall in fresh water drowning and rise in salt water drowning. However, gross changes are rarely seen in human victims because a very large water volume would need to be aspirated to produce persistent changes in serum concentrations. Serum osmolality estimation on admission may be useful.

5. Haematological

Haemoconcentration may disguise the presence of anaemia. Tests for haemolysis are:

(a) Free haemoglobin in urine.

(b) Free haemoglobin in plasma.

(c) Decreased serum haptoglobin. (Free plasma haemoglobin combines with serum haptoglobins and the resultant complex is taken up by the liver).

(d) Increased serum methaemoglobin. (Free plasma haemoglobin divides into globin and haem. The haem moiety is oxidized into methaem, which combines with serum albumin.)

6. Radiology and Imaging

A chest X-ray may show infiltrates and pulmonary oedema. Patients with normal X-rays on admission generally survive with therapy. Skull and cervical X-rays are required if the possibility of spinal injury exists. Head CT scans are indicated for comatose patients. Where there is suspicion of child abuse (eg, bath tub immersion), consideration is given to a skeletal survey examination.

7. Neurological

Evoked brain potential tests and electroencephalograms may be useful additional investigations to head CT scans. Psychometric assessments are recommended in survivors with suspected intellectual damage.

8. Drug Assays

Blood alcohol estimations and those of serum levels of anti-convulsant and sedative drugs may be indicated on admission.

9. Microbiological

Cultures of aspirated water, tracheal swabs and sputum may be indicated in severely polluted water immersion.

Complications

Complications of near-drowning after rescue which may be seen in the ICU include secondary drowning (see above), fits, hyperpyrexia, pneumonia, septicaemia, gastrointestinal bleeding, ARDS, and multi-organ failure.

Prognosis

In near-drowning without aspiration of water, complete recovery usually results if resuscitation is commenced early. With aspiration, the outlook is less predictable. While the severity of hypoxaemia and metabolic acidosis frequently correlates with the extent of pulmonary injury, blood gases on admission should not be used as prognostic factors of survival. Surf immersion, cold water, short immersion times, and skilled administration of cardiopulmonary resuscitation are favourable factors. Triage classification following successful resuscitation have been reported to be prognostically useful:

A = awake patients (ie, conscious and alert) and
B = blunted patients (ie, obtunded but rousable and responds purposefully to painful stimuli) had a better than 90% chance of recovery without neurological deficit.
C = comatose patients (ie, unrousable, with abnormal respiration and no purposeful response to pain stimuli) had a 34% mortality, and a fifth of survivors had neurological impairment.

Time of first respiratory efforts after rescue is believed to be important. Prognosis is good if the first gasp is within 30 min of rescue and there is continuing clinical improvement, especially in children. The presence of fixed dilated pupils on admission to ICU, and an arterial pH below 7.0 are bad prognostic sign.

Of all children rescued lifeless, about 1/2 from fresh water and over 2/3 from surf drownings will survive. About 3% of all child survivors will exist in the vegetative stage, and 2% have major chronic neurological problems. A third of apparent normal child survivors show minimal cerebral dysfunction on psychometric testing, but sequential recovery is possible. Resuscitation and treatment should not be abandoned early, especially in young victims, since survival after immersion for minutes has been reported, particularly in cold water immersion.