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The management of near drowning

KD Boffard[a], C Bybee[b], B Sawyer[b] and **E Ferguson[a]**

Introduction

Worldwide, drowning is one of the most common causes of accidental death, especially in children. In the UK, drowning is the third most common cause of accidental death after road accidents and burns.

In principle, two distinct high-risk groups have been identified: children, especially under the age of 4 years (Kibel *et al.*, 1990a; Wintemute, 1990); and boys aged 15–19 years. In the younger age group, immersion injury occurs more commonly in residential swimming pools, while in the lower socio-economic income groups, immersion injury may occur in the bath (Kibel *et al.*, 1990b) or a nappy bucket (Kloeck, 1993). In the older age group, immersion injury may occur in public swimming pools, open waterways or secondary to injury (Kemp and Silbert, 1992). In this group, alcohol misuse is also a factor (Wintemute, 1990).

For every death, six children are admitted to hospital and approximately 20% of these suffer permanent neurological damage (Division for Injury Control, Centre for Environmental Health and Injury Control, Centre for Diseases Control, 1990; Guyer and Ellers, 1990).

Definitions

Immersion event – an event in which a person gets into trouble in the water and drowns, or might have drowned if they were not rescued or had not managed to save themselves.

Immersion injury – any compromise of physical function or mental status resulting from an immersion event, however minor or transient, or secondary to other injury (e.g. head injury, spinal injury, etc.).

Near drowning – non-fatal immersion injury with recovery (however transient) or death that occurs other than at the time of immersion.

Drowning – death by asphyxiation following immersion in a liquid, with death occurring during the time of immersion injury.

Pathophysiology

The foremost problem caused by submersion is the inability to breathe. The adverse effects therefore are related to hypoxia, and the metabolic effects that occur as a consequence, especially metabolic alkalosis or acidosis.

The natural progression of events is fairly typical. The victim, if conscious, may begin struggling on the surface. Owing to exhaustion, panic or inability to remain buoyant (inability to swim), this is followed by intermittent submersion, usually associated with initial breath holding. Large amounts of fluid are swallowed, usually associated with vomiting.

The victim then aspirates small amounts of fluid, which causes laryngospasm, and this in turn may result in complete airway obstruction lasting for up to 2 min. During this period of increasing hypoxia and panic, the victim may continue to swallow fluid into the stomach. Approximately 10–15% of victims proceed to aspirating another aliquot of fluid, which then causes severe laryngospasm, followed by increasing hypoxia, possible convulsions, bradycardia and cessation of cardiac activity. In the remainder (≥ 85%) of victims, laryngospasm relaxes secondary to the hypoxia and unconsciousness, when large amounts of fluid are aspirated (Modell *et al.*, 1968).

At post mortem, victims may be classified as either 'wet' drowning (85–90%) or 'dry' drowning, due to persistence of the laryngospasm, according to the amount of fluid present in the lungs. This distinction is more academic than practical, and does not alter the care of the near-drowned patient. Severe hypoxia and death can occur in the absence of aspiration of large volumes of water.

[a]Johannesburg Hospital Trauma Unit, Johannesburg, South Africa; [b]International SOS, Madeleine Albright Medical Clinic, Port au Prince, Haiti WI.

Address for correspondence: KD Boffard, Johannesburg Hospital Trauma Unit, Private Bag x39, Johannesburg 2000, South Africa.

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Thus, the hypoxia occurs as a result of four mechanisms:

- initial laryngospasm and breath-holding lasting up to 2 min after submersion;
- aspiration of water when laryngospasm subsides with subsequent direct lung injury;
- vomiting and regurgitation with aspiration of swallowed water and gastric contents;
- reflex pulmonary vasoconstriction and subsequent pulmonary hypertension, exacerbating existing hypoxia.

The acute lung injury may be aggravated by contaminants in the water, such as sand, chemicals or bacteria.

Animal studies have highlighted the differences physiologically between fresh-water drowning and seawater drowning. Fresh water is hypotonic, and tends to move across the alveolar–capillary membrane into the intravascular space, leading to haemodilution, hypervolaemia, electrolyte disturbances and haemolysis. Surfactant destruction occurs, producing alveolar instability, atelectasis and decreased compliance, with pronounced ventilation/perfusion (V/Q) mismatch. As much as 75% of the blood flow may circulate through underventilated portions of the lung bed.

Saltwater exerts an osmotic pressure of up to 1000 mOsm/l, and is therefore markedly hypertonic. It causes damage to the alveolar–capillary membrane, with water shifting into the alveolus, associated with large variations in serum electrolyte levels and haemoconcentration. Surfactant washout occurs, with rapid exudation of protein-rich fluid into the alveoli and pulmonary interstitium. Compliance is reduced, direct alveolar–capillary basement membrane damage is seen and shunting occurs. The clinical picture is a transient decrease in intravascular volume. The body redistributes the fluid, resulting in diuresis. Marked hyponatraemia may be seen (Orlowski, 1987).

In human victims, these differences are not as clear. Both salt- and fresh water destroys surfactant and damages the alveolar basement membrane. The end result is non-cardiogenic pulmonary oedema, ventilation–perfusion mismatch and hypoxia.

The mammalian reflex

In humans, typically young children, immersed suddenly in cold water, the mammalian diving reflex may occur and produce apnoea, bradycardia and vasoconstriction of non-essential vascular beds, with shunting of blood to the coronary and cerebral circulation.

Hypothermia

Severe accidental hypothermia with a core body temperature of less than 30°C is associated with marked depression of cerebral blood flow and oxygen requirement, reduced cardiac output and decreased arterial pressure.

Hypothermia is almost always a complication of immersion. When submerged in cold water, the body rapidly loses heat, with water conducting heat 20 times more efficiently than air. Thus, there is rapid heat loss. While hypothermia may itself cause cardiac arrest, it is also protective to the brain through a reduction in metabolic rate, with marked depression of cerebral blood flow and oxygen requirements. Oxygen consumption is reduced by 50% at a core temperature of 30°C (Modell *et al.*, 1968). Victims can appear to be clinically dead because of marked depression of brain and cardiovascular function, and the potential for resuscitation with full neurological recovery is theoretically possible (Schneider, 1992). A number of cases were reported displaying a favourable outcome in victims of cold-water submersion incidents, even after submersion times as long as 40 min (Biggart and Boohn, 1990). It would seem that the common denominator was water at a temperature of less than 10°C. The American Heart Association guidelines make the recommendation that the hypothermic patient who appears dead should not be considered so until a near-normal body temperature is reached (Southwick and Dalglish, 1980; American Heart Association, 1992).

Prevention

Organizations and individuals can play a large part in the management of near drowning and, as with all accidents, the primary means of reducing morbidity and mortality is by prevention (Pearn and Nixon, 1977; Carey, 1993). Prevention should be considered under the following headings.

Passive

Measures designed to prevent immersion in the first place:

- swimming pool fences;
- swimming pool nets;

- protection or draining of garden ponds;
- lockable bucket lids;
- lockable (childproof) doors to toilets and areas containing water.

Active
Measures designed via the victim or first responders:

- supervision of infants and toddlers;
- lifeguards;
- education of parents and children;
- public awareness;
- basic cardiopulmonary resuscitation (CPR) education.

Management

Prehospital phase
Resuscitation by the re-establishment of ventilation is the single most important step towards achieving a favourable outcome. This should be started as soon as the victim is reached ('rescue breathing') (American Heart Association, 1994) and should not be delayed until the victim is removed from the water.

The airway should be opened. It should be established that no spontaneous breathing is present. There may be a need to clean the airway of debris, but not of aspirated water. Once the victim has been removed from the water, conventional airway management with adjuncts can be accomplished in the victim of near drowning (American Heart Association, 1994). Generally, only a modest amount of water is aspirated by victims, irrespective of whether it is seawater or fresh water (Modell and Davis, 1969). Manoeuvres to empty the lungs of water are not necessary. CPR or chest compressions cannot be managed effectively in water.

The American Heart Association (1994) recommends that a period of 30–45 s should be taken to establish pulselessness before CPR is commenced, in order not to confuse it with bradycardia. However, when in doubt, effective CPR should be started as soon as possible and continued throughout transport, or until a measurable cardiac output is present. Warmed oxygen in high concentration must be given.

Where there is any suspicion of cervical spine injury (such as the individual having dived into shallow water), the victim's neck should be supported in a neutral position.

Intermittent positive pressure ventilation (IPPV) with 100% oxygen should be commenced. This can be by bag–valve–mask but, because of the risk of aspiration from a stomach full of swallowed water, the establishment of a definitive airway is critical, and endotracheal intubation should take place as soon as possible. Artificial ventilation should be initiated, especially where pulmonary oedema is present. Constant positive airway pressure (CPAP) or positive end expiratory pressure (PEEP) with a pressure of 10 cmH_2O is helpful to reduce alveolar collapse in the presence of reduced quantities of surfactant.

Approximately 75% of paediatric victims of immersion injury make their first gasp within 5 min of rescue.

Where possible the following parameters should be sought and recorded:

- core temperature of the victim, using a low-reading thermometer;
- time and duration of immersion;
- time resuscitation started;
- time to first response to resuscitation;
- temperature (and type) of the water, and nature of any contamination.

Hospital phase
Resuscitation should continue, with endotracheal intubation, adequate oxygenation and adequate ventilation. CPR should continue if required. Any patient who has a history of apnoea, cyanosis or has respiratory changes should be observed for 24 h in hospital as late onset pulmonary oedema and cerebral oedema, can occur. If not already passed, a nasogastric tube should be placed to empty the stomach of residual water.

Baseline investigations include:

- fill blood count and platelets;
- coagulation profile (clotting time, fibrin degradation products and *d*-dimers);
- urea and electrolytes;
- creatinine;
- blood glucose;
- arterial blood gases;
- serum lactate level;
- blood cultures, if indicated;
- chest X-ray;
- cervical spine X-ray, if indicated;
- toxicology (especially alcohol), if indicated;
- skeletal survey if non-accidental injury is suspected in toddlers (consider the possibility of child abuse in all domestic drowning and near drowning).

Respiratory system
Adequate oxygenation is essential. Endotracheal intubation remains preferable, as it allows good bronchial toilet. Close monitoring of the arterial blood gases, and the serum lactate level should be maintained. Simple oxygen therapy is adequate if the arterial parhal pressure of oxygen (PaO_2) is greater than 80 mmHg (10.5 kPa), with an FiO_2 of less than 0.5 (inhaled oxygen concentration of <50%). IPPV, if required, should be maintained with a PEEP of 5–10 cmH_2O.

With artificial ventilation, there is a high risk of barotrauma to normal areas of the lung, resulting from hyperinflation, and tidal volumes should be maintained at no more than 6 ml/kg. Ideally, the plateau pressure should not exceed 25 mmHg. A recent report describes the use of artificial surfactant (McBrien *et al.*, 1993) as a possible aid to therapy.

Central nervous system
The cerebral insult, as a result of hypoxia, is the single most significant consequence of near drowning, and the majority of long-term sequelae such as convulsions and neurological complications stem directly from this insult. Every effort should be made to prevent secondary brain damage from further anoxia, metabolic abnormalities such as alkalosis or acidosis, or hypotension.

In spite of profound cerebral hypoxia, the outcome of cold-water immersion is better than expected. This may be due to the protective effects of the hypothermia.

Studies evaluating the use of treatment regimens such as barbiturates have not shown any difference in outcome (Bohn *et al.*, 1986).

Cardiovascular system
Children are more likely to have fluid and electrolyte imbalances than adults. Hypovolaemia and hypervolaemia should be corrected. A central venous monitoring line for monitoring changes in the venous pressure, or even monitoring pulmonary artery wedge pressures, may be useful. Inotropic support may be required.

Renal system
Renal impairment may occur, secondary to hypoxia or hypotension, and should be dealt with accordingly.

Management adjuncts

Rewarming
Hypothermia is common after immersion injury. Rewarming must take place with intensive monitoring. Patients who have spontaneous respiratory effort and whose hearts are beating, no matter how severe the bradycardia, should not receive unnecessary resuscitation procedures. The hypothermic heart is very irritable and fibrillates easily. Patients with a core temperature of <29.5°C are at high risk for ventricular arrythmias, and should be rewarmed as rapidly as possible. Recent studies have not shown any increase in ventricular arrythmias with rapid rewarming.

A hypothermic heart is resistant to both electrical and pharmacological cardioversion, especially if the core temperature is <29.5°C, and CPR should be continued if necessary.

If the core temperature is greater than 29.5°C and fibrillation is present, one attempt at electrical cardioversion should be made. If this is ineffective, intravenous bretylium may be helpful.

Patients with a core temperature of between 29.5 and 32°C, and who are haemodynamically stable, may be rewarmed more slowly. However, active core rewarming is still generally required.

Patients with a core temperature of >32°C can generally be rewarmed using external rewarming.

Potential methods of warming include the following.

External
- removal of wet or cold clothing and drying the patient;
- infrared (radiant) heat;
- electrical heating blankets;
- warm air heating blankets.

In the presence of hypothermia 'space blankets' are ineffective as there is minimal intrinsic body heat to reflect.

Internal
- Heated, humidified respiratory gases to 42°C;
- warmed i.v. fluids to 37°C;
- gastric lavage with warmed fluids (usually saline at 42°C);
- continuous bladder lavage with water at 42°C;
- peritoneal lavage with potassium-free dialysate at 42°C (use 20 ml/kg every 15 min);
- intrapleural lavage;

- extracorporeal rewarming has been successfully used (Bolte *et al.*, 1988; Norberg *et al.*, 1992).

It is recommended that resuscitation should not be abandoned while the core temperature is subnormal as it may be difficult to distinguish between cerebroprotective hypothermia and hypothermia resulting from brain-stem death.

Inotropes
The inotrope of choice for cardiovascular support after near drowning is adrenaline. Adrenaline is a natural catecholamine with both α- and β-adrenergic activity (Otto and Yakaitis, 1984). Adrenaline's α-adrenergic effect (although not its β-adrenergic effect) makes ventricular fibrillation more susceptible to direct countershock. The primary beneficial effect of adrenaline in cardiac arrest is peripheral vasoconstriction, which leads to a favourable redistribution of blood flow from peripheral to central circulation during CPR, with improved coronary and cerebral perfusion pressure (Michael *et al.*, 1984; Koehler *et al.*, 1985). The normal dose of adrenaline is a 1–3 mg bolus, with an initial maintenance infusion based on a dose of 0.03 μg/kg/min. This is then titrated according to the needs of the patient.

Antibiotics
Aspiration problems are primarily chemical in nature, and bacterial contamination does not play a primary role in the morbidity and mortality associated with immersion injury. Antibiotic therapy should be withheld as prophylaxis and used therapeutically, based on proven cultures or contamination. If possible, a sample of the immersion medium should be obtained for culture.

Temperature
After rewarming, pyrexia is common and needs to be differentiated from pyrexia due to systemic infection, particularly if the temperature develops after 24 h. In the absence of blood cultures, a broad-spectrum intravenous antibiotic, effective against Gram-negative organisms, should be used. Amoxycillin/clavulanate, or a broad-spectrum cephalosporin, can be used.

Bronchodilators
Cold-induced bronchorrhoea or irritation of the bronchial tree by inhaled water or particulate material may produce cough and bronchospasm. These may worsen hypoxia, and should be aggressively treated using an inhaled β-agonist bronchodilator (Shepherd, 2000).

Psychosocial support
The effect on the victim's family can be devastating, with feelings of guilt playing a very large role. Separation and divorce occur frequently. Long-term care of children disabled as a result of the near drowning can also place additional strains on relationships. In-house and outside agencies should be utilized by staff to assist wherever possible.

Prognosis

Analysis of paediatric immersion injuries to identify factors predicting outcome is difficult. This is because there is a wide variation of circumstances in each immersion event. New developments in identifying prognostic factors include testing brain-stem auditory-evoked responses and cerebral blood flow measurements. Reduced cerebral blood flow taken with an elevated blood sugar level has been shown to have increased predictability of a poor outcome (Ashwal *et al.*, 1990).

There are no absolute predictors of outcome. Prognosis is linked to the duration of the hypoxic insult and to the core temperature. In addition, other prognostic indicators have been utilized:

- submersion time – a submersion time of longer than 9 min is associated with a poor prognosis;
- time to first gasp – if the first gasp occurs within 1–3 min after the start of CPR, the prognosis is good;
- CPR time – CPR longer than 25 min is associated with a poor prognosis;
- rectal temperature – if the rectal temperature is less than 33°C on arrival, the chances of survival are increased;
- persisting coma – indicates a poor prognosis (see below);
- arterial blood pH – if the pH is less than 7.0, despite treatment, this carries a poor prognosis;
- serum lactate – if the serum lactate level is >5.0 mg/dl, this carries a poor prognosis;
- arterial blood PaO_2 – if the arterial oxygen partial pressure persists at less than 60 mmHg (8 kPa), despite treatment, the prognosis is poor.

Orlowski (1987) has described a prognostic scoring system, based on risk factors:

- age less than 3 years;
- estimated submersion time greater than 5 min;
- no attempt at resuscitation for more than 10 min after rescue;
- patient in coma on admission to the resuscitation room;
- arterial blood gas pH <7.1.

Score <2 risk factors/5 – >90% chance of recovery;
Score >3 risk factors/5 – < 5% chance of recovery.

The Glasgow Coma Scale is also fairly accurate in predicting outcome and worldwide most workers agree with Orlowski's findings (Oakes *et al.*, 1982; Nagel *et al.*, 1990; Quan *et al.*, 1990; Quan and Kinder, 1992).

Conn *et al.* (1980) described a simple neurological classification for the victims of immersion injury using three main categories:

- Category A – Awake;
- Category B – Blunted;
- Category C – Comatose.

Those children in Category A initially, and at assessment 5–8 h later, have a greater than 95% chance of recovery with little or no neurological impairment.

Those children who present or remain in Category C have a less than 5% chance of good neurological recovery, and will be either brain dead or suffer moderate to severe neurological impairment (Modell *et al.*, 1980).

Conclusion

Bystander CPR and availability of advanced life support (ALS) in the form of a rapid response by emergency medical services (EMS), with consequent reduction in hypoxic time, has led to an increase in the number of near-drowning victims who are successfully resuscitated and reach the emergency departments of hospitals. Unfortunately, many of these victims die later or are left with severe neurological impairment. This has prompted a continued search for criteria to predict the outcome of the near-drowned victim. Various scoring systems have been described.

Aggressive prehospital management by ALS providers and decision making with insight does make an impact on the outcome of the near-drowned victim. However, prevention remains the single most important method of decreasing the incidence, morbidity and mortality of immersion injury.

References

American Heart Association. 1992. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. *J Am Med Assoc* **268**: 2244–47.

American Heart Association. 1994. *Advanced cardiac life support*, 10–12.

Ashwal S, Schneider S, Tomasi L, Thompson J. 1990. Prognostic implications of hyperglycaemia and reduced cerebral blood flow in childhood near drowning. *Neurology* **40**: 820–23.

Biggart MJ, Boohn DJ. 1990. Effects of hypothermia and cardiac arrest on near drowning. *J Paediatr* **117**: 179–83.

Bohn DJ, Biggar WD, Smith CR, Conn AW, Barker GA. 1986. Influence of hypothermia, barbiturate therapy and intracranial pressure monitoring on morbidity and mortality after near drowning. *Crit Care Med* **14**: 529–33.

Bolte RG, Black PG, Bowers RS, Thorne K, Corneli HM. 1988. The use of extracorporeal rewarming in a child submerged for 66 minutes. *J Am Med Assoc* **260**: 377–79.

Carey VF. 1993. Childhood drownings: who is responsible? *Br Med J* **307**: 1086–87.

Conn AW, Montes JE, Banker GA, Edmonds JF. 1980. Cerebral salvage in near drowning following neurological classification by triage. *Can Anaesth Soc J* **27**: 201–10.

Division of Injury Control, Centre for Environmental Health and Injury Control, Centre for Diseases Control. 1990. Childhood injuries in the United States. *Am J Dis Child* **144**: 627–46.

Guyer B, Ellers B. 1990. Childhood injury in the United States: mortality, morbidity and cost. *Am J Dis Child* **144**: 649–52.

Kemp A, Silbert JR. 1992. Drowning and near drowning in children in the United Kingdom: lessons for prevention. *Br Med J* **304**: 1143–46.

Kibel SM, Joubert G, Bradshaw D. 1990a. Injury related mortality in South African children 1981–1985. *S Afr Med J* **78**: 398–403.

Kibel SM, Nagel FO, Myers J, Cywes S. 1990b. Childhood near drowning – a 12 year retrospective review. *S Afr Med J* **78**: 418–21.

Kloeck W. 1993. Infant drownings in nappy buckets [Letter]. *S Afr Med J* **83**: 131.

Koehler RC, Michael JR, Guerci AD *et al*. 1985. Beneficial effects of epinephrine infusion on cerebral and myocardial blood flows during CPR. *Ann Emerg Med* **14**: 744–49.

McBrien M, Katumba JJ, Muktar A. 1993. Artificial surfactant in the treatment of near drowning. *Lancet* **342**: 1485–86.

Michael JR, Guerci AD, Koehler RC *et al*. 1984. Mechanisms by which epinephrine augments cerebral and myocardial perfusion during cardiopulmonary resuscitation in dogs. *Circulation* **69**: 822–35.

Modell JH, Davis JH. 1969. Electrolyte changes in human drowning victims. *Anaesthesiology* **30**: 414–20.

Modell JH, Davis JH, Giammona ST *et al*. 1968. Blood gas and electrolyte changes in human near-drowning. *J Am Med Assoc* **203**: 99–105.

Modell JH, Graves SA, Kuck EJ. 1980. Near drowning: Correlation of level of consciousness and survival. *Can Anaesth Soc J* **27**: 211–15.

Nagel FO, Kibel SM, Beatty DW. 1990. Childhood near drowning factors associated with poor outcome. *S Afr Med J* **78**: 422–25.

Noberg WJ, Agnew RF, Brunsvold R, Sivanna P, Browdie DA, Fisher D. 1992. Successful resuscitation of a cold water immersion victim with the use of cardiopulmonary bypass. *Crit Care Med* **20**: 1355–57.

Oakes DD, Sherck JP, Maloney JR, Charters C. 1982. Prognosis and management of victims of near drowning. *J Trauma* **22**: 544–49.

Orlowski JP. 1987. Drowning, near-drowning and ice water submersion. *Pediatr Clin N Am* **35**: 75–92.

Otto CW, Yakaitis RW. 1984. The role of epinephrine in CPR: A reappraisal. *Ann Emerg Med* **13**(pt 2): 840–43.

Pearn J, Nixon J. 1977. Prevention of childhood drowning accidents. *Med J Aust* **1**: 616–18.

Quan L, Kinder D. 1992. Paediatric submersions: Pre-hospital predictors of outcome. *Paediatrics* **90**: 909–13.

Quan L, Wentz KR, Gore IJ, Copass MK. 1990. Outcome and predictors of outcome in paediatric submersion victims receiving pre-hospital care in King County, Washington. *Paediatrics* **86**: 586–93.

Schneider SM. 1992. Hypothermia: from recognition to rewarming. *Emerg Med Rep* **13**: 1–20.

Shepherd S. 2000. Submersion injury, near drowning. http://www.emedicine.com/emerg/topic744.htm.

Southwick FS, Dalglish PL Jr. 1980. Recovery after prolonged asystolic cardiac arrest in profound hypothermia: A case report and literature review. *J Am Med Assoc* **243**: 1250–53.

Wintemute GJ. 1990. Childhood drowning and near drowning in the United States. *Am J Dis Child* **144**: 663–69.