The Management of Near Drowning

KD Boffard, C Bybee, B Sawyer, E Ferguson

Introduction
World-wide, drowning is one of the most common causes of accidental death, especially in children. In the U.K. drowning is the third most common cause of accidental death after road accidents and burns(1).

In principle, two distinct high-risk groups have been identified: children especially under the age of 4 years,(2,3) 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(4), or a nappy bucket(5). In the older age group, immersion injury may occur in public swimming pools, open waterways, or secondary to injury(6). In this group, alcohol misuse is also a factor(2).

For every death, six children are hospitalised, and approximately 20% of these suffer permanent neurological damage(7, 8). The tragedy is that the victims are almost invariably young and in good health prior to the incident.

Drowning and near drowning have been the subject of several reviews (9,10,11,12,13). Resuscitation and management of the initial immersion incident has been well described and is well established, the understanding of the pathophysiology and limitation of neurological damage is still the subject of discussion.

The criteria used to identify those patients with a poor prognosis still have to be further refined. Some criteria are conflicting, and there are large “grey” areas. Caution must be exercised in discussion of the prognosis with the relatives at the early stages of management. One of the problems appears to be that, although the protective role of hypothermia is now generally recognised, it is not always possible to determine from the core body temperature on arrival at the hospital, what the cerebral temperature was at the time that the hypoxia first developed(13) (i.e. the brain may have suffered hypoxic damage prior to the hypothermia).

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. Due 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 two minutes. During this period of increasing hypoxia and panic, the victim may continue to swallow fluid into the stomach. 10 to 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 of victims(±85%), laryngospasm relaxes secondary to the hypoxia and unconsciousness, when large amounts of fluid are aspirated(10).

At post mortem, the 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.

Thus the hypoxia occurs as a result of four mechanisms:

- Initial laryngospasm and breath-holding lasting up to 2 minutes 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 intra-vascular space, leading to haemodilution, hypervolaemia, electrolyte disturbances and haemolysis. Surfactant destruction occurs, producing alveolar instability, atelectasis and decreased compliance, with pronounced 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 interstitial. 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.

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

The most significant changes in cold water immersion are cardiovascular, and have been documented as the “cold-shock” response. They are initiated by a rapid decrease in skin temperature. Respiratory drive is enhanced on immersion in water at less than 25°C. It reaches a maximum at 10°C. The response includes an initial 2-3 litre “gasp”, followed by an uncontrollable hyperventilation, which can result in a tenfold increase in minute ventilation. This hyperventilation will wash out CO₂, with resulting alkalosis, and shift of the oxygen dissociation curve to the left (both from alkalosis and hypothermia). The end result is poorer oxygen delivery. The inspiratory shift in end-expiratory lung volume after cold water immersion can result in tidal breathing at near lung capacity, creating a sensation of dyspnoea. This makes swimming difficult and is believed to be one of the causative factors in the mechanism of cold water swim failure.

The cardiovascular response to cold-water immersion includes:

- Immediate intense reflex vasoconstriction
- 42-49% increase in heart rate
- 59-100% increase in cardiac output
- Arterial and venous pressures increase
- Myocardial workload increases

**Hypothermia**

Hypothermia is normally defined as the state of a core body temperature of less than 35°C.

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. Consciousness becomes progressively impaired. With further cooling in spontaneously breathing adults, cardiac arrest from ventricular fibrillation may occur at a body temperature less than 28°C, with asystole at less than 24°C-26°C.

Hypothermia is almost always a complication of immersion. When submerged in cold water, the body rapidly loses heat, with water conducting heat twenty times more efficiently than air. There is thus 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. However, it is likely that most victims become hypoxic associated with laryngospasm and aspi rate as the level of consciousness becomes impaired as core temperature falls below 35°C, and therefore well before the protection provided by the hypothermia occurs.

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. A number of cases have been reported displaying a favourable outcome in victims of cold water submersion incidents, even after submersion times as long as 40 minutes.
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 that appears dead should not be considered so until a near normal body temperature is reached(24,25).

The Mammalian Diving 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(26).

The magnitude of the diving response found in humans is qualitatively similar to that found in diving mammals, but quantitatively less marked, with the cold-shock response predominating in the majority of normally clothed adults(27). The diving response is also reported to be more marked in infants than in adults(28). However, it seems unlikely that the diving response alone accounts for the survival of some victims, a combination of hypothermia and the diving response may play a role.

Cooling of the victim may be a combination of “external” cooling from skin contact with the cold water, and “internal” cooling as a result predominantly of swallowing large amounts of cold water. Thus swallowing and aspiration of cold water may play a significant part in cerebral cooling and brain survival in the submerged child.

Prevention
Organisations 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(29,30). 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 involving the victim or first responders
  - Supervision of infants and toddlers
  - Lifeguards
  - Education of parents and children
  - Public awareness
  - Basic CPR education

Management
**Pre-Hospital Phase**
Resuscitation by the reestablishment of ventilation and relief of hypoxia is the single most important step towards achieving a favourable outcome(11, 31). This should be started as soon as the victim is reached (“Rescue breathing”) (32), and should not be delayed until the victim is removed from the water.

The primary aims therefore are:

- Scene safety, and safety of the rescuer.
- Establishing a clear airway
- Minimising hypoxia
- Establishing adequate circulation
- Minimising further heat loss
- Early transfer to definitive care.

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.

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 removed from the water, usual airway management with adjuncts can be accomplished in the victim of near drowning(33). Generally, only a modest amount of water is aspirated by victims, irrespective of whether it is seawater or freshwater(11). Maneuvers to empty the lungs of water are not necessary. CPR or chest compressions cannot be managed effectively in water.

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 cm. H₂O 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 minutes of rescue.

In hypothermia, with a vasoconstricted patient, it may be extremely difficult to feel pulses, even when normal. The American Heart Association recommends that a period of 30–45 seconds should be taken to establish pulselessness before CPR is commenced, in order not to confuse it with bradycardia(32). 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.
Wet or cold clothing must be removed as soon as possible, and rewarming commenced if feasible. Reflective “space blankets” may be helpful in minimising further heat loss.

The use of the Heimlich manoeuvre is not recommended (34). It is unlikely that it will clear the airways of much liquid, but is highly likely that it will induce vomiting with the potential for further aspiration, and may further delay the onset of Advanced Life Support. In addition, cardiac arrhythmias may result if the victim is severely hypothermic.

Where possible the following parameters should be sought and recorded:

- Core temperature of the victim, using a subnormal type thermometer
- Time and duration of immersion
- Time resuscitation started
- Time of 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. A full primary and secondary survey are required. It must be assumed that the victim, especially if not an infant, drowned as a result of other injuries (for example secondary to a head injury of cervical spine injury), and specific effort must be made to seek these and exclude them. Medical causes, such as infarct must also be ruled out.

Orlowski (14) cited several cases that appeared normal on assessment in the emergency department, even with normal chest X-rays, who developed fulminant pulmonary oedema as long as 12 hours after the immersion incident. While it has been recommended that the period of observation should be at least 6 hours (35), any patient who has a history of apnoea, cyanosis, or has respiratory changes, should be observed for 24 hours 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:

- Arterial blood gases
- Coagulation profile
- Clotting times
- Fibrin degradation products
- d-dimers
- Cultures
  - Blood cultures if indicated
- Tracheal aspirate
- Electrocardiography (12 lead ECG)
- Electrolyte screen
  - Urea and electrolytes
  - Creatinine
  - Blood glucose
  - Creatinine kinase
- Serum lactate level
- Haematology
- Blood count
- Platelets
- Radiography
  - Cervical spine X-Ray if indicated
  - Chest X-ray
  - Skeletal survey if non-accidental injury is suspected in toddlers
  - (Consider the possibility of child abuse in all domestic drowning and near drowning).
- Toxicology (especially blood alcohol) if indicated

**Respiratory System**

Adequate oxygenation is essential. Endotracheal intubation remains preferable, since 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 PaO₂ is greater than 80 mm Hg (10.5 kPa), with an FiO₂ of less than 0.5 (inhaled oxygen concentration of <50%). IPPV if required, should be maintained with a PEEP of 5-10 cm. H₂O. A balance should be achieved to try to attain the lowest pressure possible to minimise shunting, yet high enough to maintain alveolar patency in the presence of reduced surfactant. Pulmonary alveolar recruitment should be used early if the PaO₂ starts falling, and in some cases, prone ventilation may be required.

With artificial ventilation, there is a high risk of barotrauma to normal areas of the lung, consequent to hyperinflation, and tidal volumes should be maintained at no more than 6ml/Kg. Ideally, the plateau pressure should not exceed 25 mm. Hg. A recent report describes the use of artificial surfactant (36) 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 the 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 in 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 (37).

**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. Rhabdomyolysis has been described, and can be complicated by acute renal failure.

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 <29.5ºC 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 <29.5ºC, and CPR should be continued if necessary.

If the core temperature is greater than 29.5ºC, 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 29.5ºC. and 32ºC., and who are haemodynamically stable may be rewarmed more slowly. However, active core rewarming is still generally required. Patients with a core temperature of >32ºC., can generally be rewarmed using external rewarming.

Methods of rewarming include:

- **External**
  - Remove wet or cold clothing and dry the patient
  - Infrared (radiant) heat
  - Electrical heating blankets
  - Warm air heating blankets

In the presence of hypothermia, “space blankets” are ineffective, since there is minimal intrinsic body heat to reflect.

**Internal**

- Heated, humidified respiratory gases to 42ºC.
- Warmed i.v. fluids to 37ºC.
- Gastric lavage with warmed fluids (usually saline at 42ºC.)
- Continuous bladder lavage with water at 42ºC.
- Peritoneal lavage with potassium-free dialsate at 42ºC. Use 20 ml/Kg every 15 mins.
- Intrapleural lavage

- Haemofiltration
- Extracorporeal rewarming has been successfully used (38,39,40)

The advantages of extracorporeal warming include rapid warming time (up to 10ºC per hour), reduction in blood viscosity, and reinstitution of perfusion, regardless of cardiac rhythm (41).

It is recommended that resuscitation should not be abandoned while the core temperature is sub-normal, since 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 (42). Adrenaline’s α-adrenergic effect, (though 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 (43,44). The normal dose of adrenaline is a 1-3 mg. bolus, with an initial maintenance infusion based on a dose of 0.03µg / 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 a pyrexia due to systemic infection, particularly if the temperature develops after 24 hours. 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 beta-agonist bronchodilator (45).
Management of Near Drowning

Psychosocial support
The effect on the victim’s family can be devastating, with guilt feelings playing a very large role. Separation and divorce occur frequently. Long term care of disabled children consequent on the near drowning also places additional strains on relationships. In-house and outside agencies should be utilised 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, magnetic resonance spectroscopy(46) 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(47).

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 utilised:

• Submersion time
  A submersion time of longer than 9 minutes is associated with a poor prognosis.

• Time to first gasp
  If the first gasp occurs within 1-3 minutes after the start of CPR, the prognosis is good.

• CPR time
  CPR longer than 25 minutes is associated with a poor prognosis.

• Rectal temperature
  If the rectal temperature is less than 33ºC. 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 PaO2
  If the arterial oxygen partial pressure persists as less than 60 mm Hg. (8.0 kPa) despite treatment, the prognosis is poor.

Orlowski(14) has described a prognostic scoring system, based on risk factors (table 2).

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;2 risk factors/5</td>
<td>&gt;90% chance of recovery</td>
</tr>
<tr>
<td>&gt;3 risk factors/5</td>
<td>&lt; 5% chance of recovery</td>
</tr>
</tbody>
</table>

The Glasgow Coma Scale is also fairly accurate in predicting outcome, and worldwide most workers agree with Orlowski's findings(48,49,50,51).

Conn(52) 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 hours later have a greater than 95% chance of recovery with no or little 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(33).

Conclusion
Bystander CPR and availability of Advanced Life Support (ALS) in the form of 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 pre-hospital 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
The Management of Near Drowning

KD Boffard, C Bybee, Brad Sawyer and E Ferguson

*J R Army Med Corps* 2001 147: 135-141
doi: 10.1136/jramc-147-02-05

Updated information and services can be found at:
http://jramc.bmj.com/content/147/2/135.citation

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/