Management of drowning

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BACKGROUND
In 2004 an estimated 388,000 people died worldwide as a result of drowning. This total, taken from Global Burden of Disease figures, makes drowning the third leading cause of death from unintentional injury (after road traffic accidents and falls). It is a significant underestimate, as it includes only deaths from ‘accidental drowning and submersion’, excluding drowning due to cataclysms (floods), assaults, suicides, and transport accidents. Drowning is a global public health concern which results in significant morbidity and mortality.

DEFINITIONS
Over twenty different definitions relating to drowning have appeared in the medical literature, hindering attempts to implement effective surveillance and management activities. In an effort to allow more accurate comparison of available data on drowning, experts at the World Congress on Drowning, held in Amsterdam in 2002, agreed on the following definition:

Drowning is the process of experiencing respiratory impairment from submersion/immersion in liquid. Drowning outcomes are classified as:
- death
- morbidity or
- no morbidity.

EPIDEMIOLOGY
Table 1 shows the estimated number of deaths attributed to unintentional drowning, in each WHO region in 2004.

Risk factors for drowning include:

Sex
Males are more likely to die or be hospitalised due to drowning than females. Overall the male rate of drowning is more than twice that of females. Studies suggest this is due to increased exposure to water and riskier behaviour such as swimming alone, drinking alcohol before swimming alone and boating.

Age
Children under 5 have the highest drowning mortality rates of any age group worldwide.

Socioeconomic status
Ethnic minority groups have higher rates of drowning mortality rates, possibly due to differences in opportunities to learn to swim.

Occupation
The occupational mortality amongst Alaskan fisherman is 116 per 100,000, with an estimated 90% of deaths due to drowning.

PATHOPHYSIOLOGY
The primary physiological consequences of drowning are prolonged hypoxaemia and the resultant metabolic...
When a drowning victim’s airway lies below a liquid surface, initial breath-holding is inevitably followed by a gasp which draws water into the hypopharynx and triggers laryngospasm. After a period of hypoxaemia, the laryngospasm breaks and there is a further gasp, followed by hyperventilation and aspiration of variable amounts of water. The aspiration of 1-3ml.kg⁻¹ of water results in significantly impaired gas exchange. Injury to other organs arises from the subsequent hypoxia and acidosis. In 10 to 20 percent of patients laryngospasm is maintained until cardiac arrest occurs and, in this situation, no aspiration occurs (previously referred to as dry drowning).

When fresh water is aspirated, the hypotonic solution moves rapidly across the alveolar-capillary membrane. This destroys the surfactant layer and results in alveolar collapse and decreased compliance, with marked ventilation/perfusion (V/Q) mismatching. As much as 75% of blood flow may circulate through hypoventilated lung segments. Aspiration of salt water causes washout of surfactant and exudation of protein rich fluid into the alveoli and pulmonary interstitium. The result is a reduction in compliance, damage to the alveolar-capillary membrane and intrapulmonary shunting. Bronchospasm may occur in both fresh and salt water drowning. There is no difference in outcome between fresh water and salt water drowning; both may result in significant submersion injuries and management is identical.

The release of inflammatory mediators may result in pulmonary hypertension, whilst pulmonary oedema occurs as a result of both negative pressure (following obstruction and laryngospasm) and hypoxic neuronal injury. The destruction of surfactant commonly results in acute respiratory distress syndrome (ARDS). Another frequent complication is ventilator associated lung injury (VALI). In a small number of patients, aspiration of stagnant water, silt, sand, sewage or vomitus may cause bronchial occlusion, pneumonia, abscess formation and inflammatory damage to the alveolar membranes.

Neurological injury is a major determinant of outcome and subsequent quality of life in drowning victims. As well as direct trauma, primary neurological injury occurs due to brain hypoxia and ischaemia. Secondary injury may result from multiple factors including sustained hypoxia, hypotension, acidosis, hyperglycaemia, release of excitatory neurotransmitters, seizures and cerebral oedema.

Autonomic instability is common in both severe hypoxic and severe traumatic brain injury, and may result in tachycardia, hypertension, diaphoresis, agitation and muscle rigidity. This encephalic/hypothalamic storm may present as a syndrome of transient left ventricular hypokinesis, dyskinesis or akinesis, manifesting as ECG changes and raised troponin levels, in the absence of obstructive coronary artery disease or myocarditis. This is also known as Takotsubo cardiomyopathy. Rhabdomyolysis may occur, since there is extensive hypoxic muscle injury and the subsequent myoglobinemia may precipitate acute kidney injury. Electrolyte disturbances may also occur, for example hypernatraemia is seen in children who have ingested large quantities of fresh water.

PRE-HOSPITAL CARE

Early resuscitation has been shown to play a significant role in increasing survival. Rescuers may find an individual at any stage of the drowning process and consequently a drowning victim may require anything from simple observation to rapid and continued resuscitation. As with all emergencies, management should be aimed at ensuring adequate Airway, Breathing and Circulation, with cervical spine stabilisation if the patient is unresponsive or there is any possibility of trauma. In the event of cardiac arrest cardiopulmonary resuscitation (CPR) should be commenced in all patients and continued during transfer to hospital, as hypothermia may make the detection of vital signs difficult in the pre-hospital setting. The adage that hypothermic patients are not dead until they are ‘warm and dead’ has good foundation – recovery from prolonged submersion is well documented in children and, although it is less common in adults, there are some remarkable case reports of survival.

Rescue breaths can be given whilst the patient is still in the water however chest compressions are often ineffective due to problems with buoyancy. The patient should be removed from the water at the earliest opportunity, in a supine or foetal position where possible. There is a recognised risk of circulatory collapse during or following rescue from immersion in water. While in the water there is an increase in hydrostatic pressure around the victim’s legs and trunk. This results in increased venous return and pre-load with support of the cardiac output. This increased central volume is detected as relative hypervolaemia by the body and diuresis and natriuresis is triggered, depleting the victim’s intravascular volume. Peripheral vasoconstriction due to the relative cold temperature exacerbates this further. Extraction from the water in the foetal position is said to protect against the circulatory collapse that occurs when this hydrostatic pressure is removed.

Use of the Heimlich manoeuvre to expel water from the lungs has been shown to be ineffective and should not be attempted, as it may cause the patient to vomit and aspirate. Where available, supplemental high-flow oxygen should be given as soon as possible. Ventilation via any method may require higher pressures than expected, due to poor compliance, however, if the pre-hospital team are unable to ventilate the patient, airway obstruction should be suspected. If ventilating by reservoir bag, each breath should be just enough to make the chest wall move in order to prevent excess pressure and minimise iatrogenic lung injury.

Traditionally, rescuers have been advised to begin re-warming as soon as possible, by removing the victim from wet clothing, before wrapping them in blankets and administrating warmed fluid (where facilities allow). However, there is now good evidence that therapeutic cooling improves neurological outcome in out-of-hospital ventricular fibrillation cardiac arrest. Further research is required to determine whether this evidence can be extended to victims of drowning.

DEFINITIVE CARE

Ongoing management should focus on continuing resuscitation, correcting hypoxia and acidosis, and the treatment of concomitant injuries. Some patients – those who give a reliable history of short immersion, without significant injury, change in mental status, respiratory problems or impaired oxygenation – may be safely observed for a period and then discharged.

Airway and respiratory support

Bronchospasm may be provoked by inhalation of water and particulate...
matter, and by cold-induced bronchorrhoea (increased bronchial secretions). This should be treated aggressively to avoid worsening hypoxia. The drug of choice is an inhaled beta-agonist bronchodilator, such as salbutamol. If the patient is sufficiently cooperative (and where it is available) bi-level positive airway pressure (BiPAP) may improve oxygenation. Intubation (using rapid sequence induction) and ventilation are indicated in the following situations:

- severe hypoxia and/or acidosis,
- signs of significant respiratory distress,
- inadequate respiratory effort,
- failure to protect the airway (e.g., low conscious level).

Patients with submersion injuries are at high risk of developing Acute Lung Injury (ALI) and ARDS so protective lung ventilation strategies should be used to minimise iatrogenic damage associated with mechanical ventilation. These include:

- Aim for SaO₂ > 88%, with pH > 7.2. Optimise PEEP.
- Tidal volume < 6ml.kg⁻¹.
  
  (Use ideal body weight:
  Males 50 + [0.91 x (height – 152.4)]cm
  Females 45 + [0.91 x (height – 152.4)]cm)
- Plateau pressure < 30cmH₂O.

**Advanced respiratory techniques**

It may be possible to remove plugs of foreign material and vomitus using bronchoscopy, and bronchioalveolar lavage can be of use in obtaining samples for culture in cases of aspiration pneumonia. Surfactant therapy has been used in some drowning victims and has been shown to improve ventilation, oxygenation and fluid leak. The use of Extracorporeal Membrane Oxygenation (ECMO), in patients who remain hypoxic despite aggressive mechanical ventilation, has achieved dramatic effects in both adults and children.

**Cardiovascular support**

In both salt water and fresh water drowning, intravascular depletion is common due to intracompartmental fluid shifts and pulmonary oedema (as well as diuresis). Fluid resuscitation using warmed isotonic crystalloids (20ml.kg⁻¹) or colloids may be indicated. Vasoactive drugs may also be required, in particular in the presence of other traumatic disease processes such as neurogenic shock and blunt myocardial injury, or where there is underlying cardiac disease. Cardiac arrhythmias, including ventricular tachycardia, ventricular fibrillation, bradycardia and asystole can occur, often due to hypothermia rather than electrolyte imbalance, and should be treated according to standard international resuscitation guidelines.

**Temperature**

Temperature management in patients following drowning is a topic of ongoing research and clinical interest. Traditional studies have supported vigorous rewarming of hypothermic patients to normothermia via a number of different modalities (warmed fluids, warmed inspired air, bladder, peritoneal and pleural lavage). More recent literature suggests that mild therapeutic hypothermia is effective in improving neurological outcome and mortality rates in out-of-hospital VF cardiac arrest. Several case reports in drowning victims, who have made a full neurological recovery following coma and cardiac arrest, suggest that therapeutic hypothermia may confer neuroprotection in this setting; however its role is yet to be established by clinical trials.

At the World Congress on Drowning a consensus on temperature management was reached, based on the available evidence:

“The highest priority is restoration of spontaneous circulation, subsequent to this continuous monitoring of core and/or brain (tympanic) temperatures is mandatory in the ED and intensive care unit and to the extent possible in the prehospital setting. Drowning victims with restoration of adequate spontaneous circulation, who remain comatose, should not be actively warmed to temperature values above 32-34°C. If core temperature exceeds 34°C, hypothermia should be achieved as soon as possible and sustained for 12 to 24 hours…”

The patient’s cardiovascular status will dictate the method of rewarming, and so the rate at which they are rewarmed. Those who are haemodynamically unstable, or in cardiac arrest, require rapid rewarming. Cardiopulmonary bypass (CPB) techniques or veno-veno haemodialysis can achieve a temperature increase of 5-10°C.h⁻¹ and ECMO is also highly effective. However, where such facilities are not available, then traditional techniques should be employed – meta-analysis has demonstrated the efficacy of pleural lavage when CPB is not available, or transfer to a tertiary centre not possible or would require unacceptable transfer times.

**Other considerations**

Appropriate treatment of hypoglycaemia, electrolyte imbalances and seizures should be initiated where necessary. The use of corticosteroids have been shown to be of no long term benefit and therefore should not be given unless otherwise indicated. Antibiotic prophylaxis also has no proven benefit and is not recommended unless the patient was submersed in grossly contaminated water. Tetanus immunisation status should be checked and a booster, or course of treatment, should be given if necessary. Associated injuries should be identified early, as these may complicate further management.

**PROGNOSIS**

Drowning is a frequent accident, associated with high morbidity and mortality. There is no validated clinical scoring system to predict survival and long term neurological recovery in drowning victims. Some factors have been shown to adversely affect survival, including prolonged submersion, delay in the initiation of effective CPR, asystole on arrival at hospital, fixed dilated pupils, a low Glasgow Coma Score, severe metabolic acidosis (pH < 7.1), Prompt resuscitation and aggressive treatment are therefore crucial to optimal survival. In cases where it is clear that submersion has been very prolonged, or there is evidence of fatal concomitant injury, life may be pronounced extinct. In all other cases, advanced life support should be implemented and continued until a full evaluation can be made as to the futility or otherwise of continued resuscitation.
SUMMARY

Early resuscitation plays a vital role increasing survival and should follow the same Airway, Breathing, Circulation approach to management as all medical emergencies. Further management should focus on correcting hypoxia and acidosis, making full use of protective lung ventilation strategies and advanced respiratory techniques, where available. Attention should also be paid to concomitant injuries.

REFERENCES