Drowning

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Key points

Drowning is the second leading cause of unnatural death worldwide after road traffic accidents.

Immediate resuscitation with rescue breaths and relief of hypoxia is fundamental to survival.

Fluid aspiration, hypoxia, hypercarbia, and hypothermia cause multiple organ dysfunction requiring intensive care management.

A protective ventilation strategy should be adopted to manage the acute lung injury/acute respiratory distress syndrome that may follow successful resuscitation after drowning.

Therapeutic hypothermia is recommended in the drowning victim with neurological injury.

After cardiopulmonary arrest from drowning, 75% of the patients die and 30% of survivors have neurological deficits.

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Drowning is the second leading cause of unnatural death after road traffic injuries.[1] Most of these deaths occur in countries with low or middle per capita income. Rates in countries with high per capita income have been decreasing due to socioeconomic changes including urbanization, more indoor leisure activities for children, less use of alcohol around water, and drowning prevention programmes.

Drowning occurs in a predominantly healthy and young population and results in significant morbidity and mortality. The primary event is hypoxia due to aspiration of liquid. Secondary pulmonary and neurological injury after cardiac arrest determines patient survival and subsequent quality of life.

Definitions

Drowning is defined as respiratory impairment due to submersion/immersion in liquid.[2] The liquid/air interface at the entrance of the airway prevents the victim from breathing air.

Four classes of the drowning victim are described (Table 1). Drowning outcomes are classified simply as death, survival with morbidity, and survival with no morbidity. The term near-drowning, previously used to describe survivors of drowning, was abandoned by the World Congress on Drowning in 2002.[2]

Epidemiology

The Office of National Statistics (ONS) registered 195 deaths in England and Wales due to accidental drowning in 2008. This figure excludes drowning deaths secondary to causes such as falls, motor vehicle accidents, deliberate harm, and underlying medical conditions. Drowning deaths given an open verdict by the coroner's court are also omitted as these are registered as deaths of undetermined intent. A more complete picture of all-cause drowning mortality is provided by the National Water Safety Forum. It reported 700 deaths due to drowning within the UK search and rescue area in 2006.

The total incidence of non-fatal drowning is not known in the UK. Data from the Intensive Care National Audit and Research Centre (ICNARC) reveals that for the period 1999–2008, 648 drowning victims required critical care in England, Wales, and Northern Ireland with no trend to increased or decreased incidence with time. Fewer females were admitted than males and higher numbers of admissions were observed during the summer. Risk factors for drowning are listed in Table 2.

Pathophysiology

Pulmonary aspiration, hypoxia, and hypercarbia

Drowning begins when the victim's airway lies below the surface of a liquid and the victim voluntarily holds his breath. Breath-holding may be followed by a period of laryngospasm secondary to the presence of liquid in the oropharynx or larynx. During this period, the victim becomes hypercarbic, hypoxaemic, and acidotic. The hypercarbia stimulates respiration, leading to active respiratory movements. There is no inhalation or exhalation at this stage. The laryngeal spasm eventually abates due to profound hypoxia and the victim inhales liquid.

'Dry drowning' refers to drowning without fluid aspiration, possibly due to profound hypoxia during laryngospasm or vagally mediated cardiac arrest. It was thought to occur in up to 15% of drowning cases, but this is now questioned. A large review of post-mortem findings in drowning showed that death without liquid aspiration rarely occurs.[3] The few victims with dry lungs may have died from natural causes or trauma before airway submersion. The World Congress on Drowning has now abandoned the term dry drowning.

Hypothermia

Hypothermia is classified as mild (32–35°C), moderate (30–32°C), or severe (<30°C). Submersion in icy water leads to rapid hypothermia and may provide some protection

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Table 1 Classification of drowning victims at scene[10]

Class 1	No evidence of inhalation of water
Class 2	Evidence of inhalation of water and adequate ventilation
Class 3	Evidence of inhalation of water and inadequate ventilation
Class 4	Absent ventilation and circulation

Table 2 Drowning risk factors[5]

Age	Incidence peak in toddler age group due to lapses in supervision
	Incidence peak in adolescents due to risk-taking behaviour
Sex	Males>females
	Due to more risk-taking behaviour among males
Occupation/leisure activities	Fishermen
	Equipment failure in scuba diving
Environmental	Access to water
	Rural areas
	Warm weather countries
	Floods
Impaired judgement	Alcohol
	Drugs
	Hypothermia
Medical conditions	Cardiac
	Myocardial infarction, arrhythmia, long QT syndrome
	Neurological
	Seizure, syncope, stroke
Trauma	
Foul play	Child abuse
	Suicide
	Attempted murder

against hypoxia, especially in young children. However, in most drowning cases, adequate protective hypothermia is unlikely to occur before hypoxia ensues.[4]

The key events of pulmonary aspiration, hypoxia, hypercarbia, and hypothermia result in multiorgan dysfunction or failure.

Pulmonary injury

Fluid aspiration during drowning, whether fresh or salt water, initiates an acute lung injury (ALI). Fresh water aspiration washes out surfactant resulting in alveolar collapse and atelectasis. The hypotonic fluid also exerts a direct toxic effect on alveolar and vascular endothelial cells, leading to interstitial and alveolar oedema. Salt water aspiration produces acute alveolar oedema due to the generation of an osmotic gradient across the alveolar membrane. In all types of drowning, bronchospasm occurs as a result of fluid introduction into the airways. Acute emphysema may develop due to alveolar rupture secondary to fluctuations in airway pressure with ventilation against a closed glottis. Inhaled toxins including chlorine, pollutants, and particulate material may also contribute to pulmonary dysfunction.

This pulmonary aspiration leads to ventilation/perfusion mismatch, shunt, and reduced lung compliance. Clinically, this presents as hypoxaemia associated with a clinical picture of ALI/acute respiratory distress syndrome (ARDS) secondary to direct pulmonary injury.

Risk of infective pulmonary complications is increased by aspiration of contaminated liquid or gastric contents. Microorganisms present in water include a variety of bacteria, fungi, algae, and protozoa.[5] Aerobic Gram-negative bacteria including *Pseudomonas* and *Aeromonas* species can cause fulminant pneumonia within hours of drowning, whereas fungal infection, for example, *Pseudallescheria boydii* may take weeks or months to present clinically.

Cardiovascular

Cardiovascular dysfunction occurs secondary to hypoxia, acid–base disturbances, catecholamine stress, and hypothermia. The diving reflex might be associated with some myocardial protection when present. It is characterized by apnoea, vasoconstriction of non-vital capillary beds and bradycardia in response to cold-water stimulus of the ophthalmic division of the trigeminal nerve. Blood flow is redistributed to the heart and brain and myocardial oxygen consumption falls in response to the bradycardia. It can be marked in infants, but its significance in adults is questionable.

Hypoxia and hypothermia trigger massive catecholamine release. Intense vasoconstriction occurs and peripheral pulses may be hard to detect. High catecholamine levels, acid–base disturbance, and hypothermia lead to rhythm disturbances, cardiac failure, and ultimately cardiac arrest. Later, a systemic inflammatory response syndrome develops secondary to release of proinflammatory mediators.

Central nervous system

The neurological injury seen in drowning is global and secondary to hypoxia. It leads to cerebral oedema and cell death. Factors determining the degree of neurological injury include water temperature, submersion time, presence of the 'diving reflex', and coexisting cardiovascular and neurological disease.[6]

Electrolytes, blood volume, and haematology

Significant electrolyte changes secondary to aspiration of large fluid volumes is rare. An exception is drowning in extremely electrolyte-rich liquids. Life-threatening hypercalcaemia and hypermagnesaemia has been reported in Dead Sea drowning victims.[3]

Profound lactic acidosis is frequently seen in drowning victims due to cellular hypoxia. This is demonstrated by the observation that restoration of oxygen delivery can reverse even severe metabolic derangements.[7]

Haemolysis with consequent renal injury and disseminated intravascular coagulation may occur. However, it requires aspiration of large fluid volumes, so it is rarely seen in victims who survive drowning.

Table 3 Initial assessment of drowning victims[2,5]

History	
Victim information	Age, sex
	Medical history, allergies, drug history
	Precipitating events—trauma, alcohol, drugs
Scene information	Time of incident, submersion time
	Witnessed?
	Water type, temperature, contaminants
Pre-hospital care	Initial ABC and GCS
	CPR—time started, any delay
Examination	Respiratory distress—tachypnoea, cyanosis, wheeze, crepitations
	Circulatory insufficiency—pulse, BP, capillary refill
	Neurological status—GCS, pupils
	Core body temperature
	Secondary survey
Investigations	Capillary blood glucose
	Arterial blood gases
	Venous blood—urea, creatinine, electrolytes, CK, full blood count
	Toxicological assays for drugs and alcohol
	12-lead ECG
	Chest X-ray
	Trauma imaging—cervical spine imaging, CT head
	Microbiology—sputum/tracheal aspirates

Renal

Acute kidney injury can occur in drowning victims. Several aetiologies are implicated including myoglobinuria from muscle injury, lactic acidosis, hypoxaemia, and hypoperfusion.

Clinical presentation

The degree of physiological derangement experienced is determined by the timing of rescue and ranges from no evidence of harm to cardiopulmonary arrest. Initial assessment of the victim is summarized in Table 3. Patients with a Glasgow Coma Score of 15, lack of clinical signs of respiratory distress, and normal room air oxygen saturations can be safely discharged home 4–6 h after emergency department presentation.[8]

Pre-hospital management

Immediate resuscitation

Hypoxia is the major cause of death in drowning victims and the aim of immediate care is to restore adequate oxygen delivery to tissues. Cervical spine injury is rare in drowning and attempts at immobilization should not delay removal of a patient from the water. In the unconscious patient, the airway should be opened and a pulse check performed. Detection of a pulse may be difficult due to profound vasoconstriction. If in doubt, basic life support should be started following Resuscitation Council guidelines. In cardiopulmonary arrest due to drowning, five rescue breaths must be delivered immediately. Resuscitation is then continued using a ratio of 30 chest compressions to two breaths as for other causes of cardiopulmonary arrest. In the apnoeic patient with a palpable pulse, only mouth-to-mouth ventilation is performed. Once more trained individuals and equipment are available, advanced life support should be commenced. The usual rhythm in these cases is pulseless electrical activity; ventricular fibrillation is rare.

Basic life support plays a key role in survival. Studies have shown that the only drowning victims with cardiorespiratory arrest who survived were those who received immediate cardiopulmonary resuscitation (CPR).[4]

Table 4 Rewarming methods

Passive	Warm environment >30°C (rate 0.5–1°C h^{-1})
	Remove wet clothing
	Insulating cover
Active, external	Conduction methods, e.g. warmed pads
	Convection methods (rate 2–3°C h^{-1}), e.g. forced air warming blanket
	Radiant methods, e.g. radiant heater
	Secondary decrease in core temperature may occur due to peripheral vasodilatation
Active, internal	Humidified warm inspired gases (rate 0.5–1.5°C h^{-1})
	Warmed i.v. fluids
	Body cavity lavage (rate 2–3°C h^{-1}), e.g. bladder, peritoneal, gastric
	Intravascular thermal regulation system (rate 1–1.5°C h^{-1})
	Extracorporeal methods: Haemodialysis (rate up to 5°C h^{-1})
	Cardiopulmonary bypass (rate up to 10°C h^{-1})

Hospital management

Resuscitation and rewarming

Resuscitation should continue following Resuscitation Council guidelines. There are no reliable predictors for successful resuscitation and there are case reports of survivors with extreme initial physiological derangement. Therefore, resuscitation should be attempted in all drowning victims.

During resuscitation, attempts should be made to raise the body temperature of hypothermic patients. A number of rewarming methods exist (Table 4). Passive rewarming is appropriate for mild hypothermia, but moderate and severe hypothermia will require active external and active internal rewarming, respectively. When return of cardiac output is achieved in the unconscious patient, it is recommended that rewarming is not continued to normothermia, but to 32–34°C.[5]

Case reports exist of remarkable survival after prolonged submersion and protracted resuscitation, especially in children. The decision to cease resuscitation in drowning victims is complex. Factors associated with prolonged asphyxia include immersion >10 min, delay in commencement of CPR (>10 min), and CPR duration >25 min.[2] Consideration of these factors and application of clinical judgement should inform decision-making in this difficult area.

Ventilation

Management of ARDS in drowning victims must follow a protective lung ventilation strategy with low tidal volumes (6 ml kg^{-1}

ideal body weight), plateau pressure below 30 cm H_2O, and with PEEP and $F_{I_{O_2}}$ titrated to $P_{a_{O_2}}$. Caution should be exercised with regard to the use of permissive hypercapnoea if neurological injury is a possibility.

The use of extracorporeal membrane oxygenation, surfactant therapy, inhaled nitric oxide, and inhaled prostacyclin in drowning victims with ARDS has been described.[9] The use of these therapies should be considered in lung failure resistant to mechanical ventilation.

Corticosteroids are ineffective in treating the pulmonary damage associated with drowning and should not be used.[3] Antibiotics should be given if there is evidence of infection. Prophylactic antibiotics are of unproven benefit, but should be considered in the case of a victim being submerged in grossly contaminated water.

Cardiovascular

Fluid resuscitation is required in victims of drowning due to hypovolaemic shock secondary to extravasation of fluid from pulmonary and systemic capillaries. Pharmacological treatment of persistent hypotension and myocardial dysfunction should then be guided by the use of invasive haemodynamic monitoring.

Neuroprotection

Although little evidence exists for the efficacy of neuroresuscitative measures in drowning, the 2002 World Congress on Drowning made a number of recommendations based on evidence for interventions in hypoxic brain injury from other causes.[5] Key among these is the recommendation that after restoration of spontaneous circulation in cardiac arrest due to drowning, patients who remain comatose should only be actively warmed to 32–34°C. This mild hypothermia should be maintained for 12–24 h and hyperthermia should be prevented during the recovery period. Although there is an evidence base for the use of therapeutic hypothermia after out-of-hospital cardiac arrest, there are no studies to date which assess the intervention in cardiac arrest due to drowning.

Other neuroprotective measures, not specific to drowning, recommended include avoidance of hypoxaemia, maintenance of low normocapnia, maintenance of adequate mean arterial pressure, nursing with 30° head-up tilt, glucose control (target 5–10 mmol $litre^{-1}$), and prompt treatment of seizures.

Other considerations

Supportive care may be required to manage dysfunction of other organ systems. Associated traumatic injuries and underlying medical conditions should be sought in all patients and managed appropriately. In the paediatric patient, child protection issues should be considered.

Outcome

In a series of 448 cases of drowning in Cornwall (A. Simcock, personal communication), out of 64 patients defined as Class 4, there were 15 survivors (23.4%). Twelve of these survivors had a favourable neurological outcome and the other three were lost to follow-up. Three deaths (12%) were reported in 25 Class 3 victims and one death in 189 Class 2 victims. Other large case series in adults and children have reported similar death rates and a 30% incidence of neurological deficit in survivors of cardiopulmonary arrest due to drowning.[5] It is too early to assess the impact of therapeutic hypothermia on the rate of neurological impairment in survivors of drowning.

Conflict of interest

None declared.

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Please see multiple choice questions 9–12.