Immersion, near-drowning and drowning

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“Drowning” is defined as “suffocation by submersion, especially in water”66; it continues to be the third most common cause of accidental death in the general population and, for children in many countries, the second most common cause after road accidents.73 In the USA, 40% of drowning victims are less than 4 yr old.74 In Britain, the Office of Population Census reports that child deaths from this cause continue to be the third most common cause of accidental death after road accidents and burns.

“Near-drowning” is defined as “survival, at least temporarily, after suffocation by submersion in water.”65 We would challenge this definition as most dictionaries define “suffocation” as cessation of breathing leading to unconsciousness or death. However, pulmonary complications may follow aspiration of water without cessation of breathing or loss of consciousness. Thus “near-drowning” should be defined as “survival, at least temporarily, after aspiration of fluid into the lungs.” The importance of the distinction between the two definitions is that aspiration of fluid may lead to later pulmonary complications, even in those without a history of loss of consciousness, and thus care must be exercised in the management of all patients with a history of aspiration. Modell, Graves and Ketover reported68 that in dogs, aspiration of as little as 2.2 ml/kg body weight produced a decrease in PaO₂ to approximately 8 kPa (60 mm Hg) within 3 min. Pulmonary surfactant is altered locally by water in the alveoli resulting in pulmonary shunting via either fluid-filled (salt water) or atelectatic (fresh water) alveoli. Pearn78 stated that within minutes after inhalation of as little as 2.5 ml/kg body weight, the normal intrapulmonary shunt of approximately 10% may increase to as much as 75%. Even victims who are conscious, alert and outwardly clinically normal after a near-drowning incident may take several days to revert to pre-immersion values.78 After aspiration of either fresh or salt water there may be delayed outpouring of fluid into the alveoli, secondary to pulmonary parenchymal damage with transudation of protein rich fluid, so called “secondary drowning”,77 82 with consequent impairment of gas exchange. Orłowski74 cited several cases who appeared normal on assessment in the emergency department, even with normal chest x-rays, but who developed fulminant pulmonary oedema as long as 12 h after the near-drowning incident.

While statistics on the incidence of drowning deaths are available for most nations, there are no precise data for near-drowning incidents, although in the USA they are estimated to be 500–600 times more common than their fatal counterpart.73 In Britain, a comprehensive hospital survey for the years 1988–1989 by Kemp and Sibert73 of 330 drowning and near-drowning incidents in children reported 142 deaths and 188 survivors. One of the great tragedies of drowning or near-drowning is that the victims, frequently young, are generally in good health before the unexpected event which resulted in death or brain damage in a number of survivors. In recent years increased public awareness of the general principles of basic life support and cardiopulmonary resuscitation (CPR) has resulted in many more near-drowning victims arriving in hospital in a state capable of being resuscitated. The many contemporary reports in the literature of apparently hopeless cases being successfully resuscitated, particularly after cold water submersion,11 20–22 38 45 53 54 59 85 86 102 has led to protracted resuscitative efforts by hospital personnel.

Drowning and near-drowning have been the subject of many reviews.15 64 66 74 78 88 While resuscitation and the subsequent respiratory management of the near-drowning patient is now well established and largely non-controversial, the remaining major therapeutic challenge is limitation of brain damage in survivors with its associated consequent human and economic costs. Although the success stories tend to be reported, in the absence of full statistical information it is difficult to obtain an overall picture of the ratio of those who have been resuscitated and left with or without residual brain damage.

The criteria used to identify those with a poor prognosis needs to be refined. Some centres, based on retrospective analysis, have reported their results of various treatment regimens with suggested predictive criteria for recovery without severe brain damage.34 53 68 74 Regrettably, these criteria are often

Key words


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conflicting and thus of little real value other than to act as a reminder that long-term complications occur and caution should be exercised in discussing prognosis with relatives in the early stages. One of the problems appears to be that, although the protective role of hypothermia is now generally recognized, it is not always possible to determine from deep body temperature on arrival at hospital what the cerebral temperature was before severe hypoxia developed.

This review examines the physiological considerations of accidental immersion and discusses the factors which may influence outcome. The recommended management of such victims is also reviewed, with particular emphasis on the role of cardiopulmonary bypass.

**Physiological considerations**

**IMMERSION IN THERMONEUTRAL WATER**

The physiological effects of immersion in thermoneutral water have been reviewed previously by Epstein.24 In sum, changes which occur are largely a consequence of the effects of the external hydrostatic pressure on the resilient body tissues and gas-containing body cavities during head-out immersion. This results in physiological alterations in the cardiovascular and respiratory systems, and in the gastrointestinal tract. The increase in venous return causes a 32–66% increase in cardiac output, increase in right to left shunting as a result of a combination of pulmonary pooling, cephalic movement of the diaphragm and, to a lesser extent, minor compression of the chest wall; overall there is a 65% increase in the work of breathing. The central shift in blood volume is sensed by the body as hypervolaemia and results in diuresis, natriuresis and kaliuresis.25

From a practical viewpoint there is little evidence that these changes cause respiratory or circulatory embarrassment in a healthy individual.61 However, during rescue, after protracted immersion in warm water, or relative shorter immersion in cold water when hypothermia may be present, circulatory collapse and cardiac arrest may occur. This phenomenon, which has been termed “circum-rescue collapse” is considered to be outside the scope of this article and has been reviewed elsewhere.32

**IMMERSION IN COLD WATER**

The increase in right atrial pressure, resulting from reflex peripheral cold vasoconstriction after immersion in cold water, is additive to the hydrostatic pressure effect described above and enhances urinary output by as much as one-third.33 However, of more importance to the accidental immersion victim are a group of cardiorespiratory reflex responses which can occur on sudden immersion in cold water and last for approximately 2–3 min. These responses, collectively known as the “cold-shock” response, have been reviewed by Tipton.91 They are initiated by a rapid decrease in skin temperature and are probably responsible for the majority of near-drowning incidents and drowning deaths after accidental immersion in open waters in the UK. Respiratory drive is enhanced on immersion in water colder than 25 °C52 and is inversely related to water temperature, reaching a maximum level with immersion in water at 10 °C.96 The response includes an initial “gasp” of 2–3 litre in an adult, followed by uncontrollable hyperventilation which can result in a 10-fold increase in minute ventilation93 which significantly reduces $P_{a\text{CO}_2}$.51 The inspiratory shift in end-expiratory lung volume after cold water immersion can result in tidal breathing within 1 litre of total lung capacity, creating a sensation of dyspnoea. This, coupled with the swim stroke/respiration asynchrony caused by hyperventilation, makes swimming very difficult, and is believed to be one of the major causative factors in the mechanism of cold-water swim failure.31

The maximum breath-hold times of normally clothed individuals are reduced to less than 10 s.92 As a consequence, on immersion in choppy or turbulent water where the airway may be intermittently submerged, there is a significant chance of aspiration during the first few minutes. Breath-holding to facilitate escape from a submerged vehicle may be equally difficult and result in entrapment and drowning, or possibly near-drowning in those who manage to escape or are rescued before a lethal volume of water is aspirated, approximately 22 ml kg$^{-1}$ 98

Thus in healthy young people, who may be competent swimmers but unhabituated to cold, the initial respiratory responses to immersion in cold water may cause short-term incapacitation which could prevent them from relaxing and swimming a short distance to save their lives. In turbulent water, or if escape from a submerged vehicle is necessary, the inability to breath-hold for more than 10–20 s may result in aspiration and near-drowning, or even drowning.

The initial cardiovascular responses to cold immersion include an immediate intense reflex peripheral vasoconstriction, 42–49% increase in heart rate and 59–100% increase in cardiac output,40 with a resulting increase in arterial and venous pressures. These responses significantly increase the workload of the myocardium and, coupled with the concomitant increased catecholamine response,5 16 48 may induce undesirable cardiac arrhythmias.

Additionally, in middle-aged or elderly people with heart disease, hypertension or vascular disease, the initial cardiovascular responses to immersion in cold water may precipitate a cardiovascular accident. It is considered that this mechanism as a cause of incapacitation resulting in drowning is probably underestimated. The eyewitness reports of the sudden cessation of movement in some drowning victims56 is more suggestive of sudden circulatory malfunction. The fact that aspiration may occur subsequent to a cardiovascular incident may mislead some into concluding that drowning was the only problem requiring treatment or the cause of death.

Given the above, it is perhaps less surprising to discover that 60% of the annual open water
immersion deaths in the UK occur within 3 m of a safe refuge, and two-thirds of those who die were regarded as “good swimmers”.44

HYPOTHERMIA

“Hypothermia”, defined as a deep body temperature <35 °C,81 is an inevitable consequence of protracted immersion in cold water and appears to play an important role in the successful outcome of those who have survived prolonged submersion. The mechanism of how deep body temperature is reduced so rapidly in some of these victims is less clear.

The rate of change of core temperature in an immersed body is dependent on the inter-relationship of several physical and physiological factors such as water temperature, relative movement of water adjacent to the skin, body surface area to mass ratio, insulation, peripheral circulation, metabolism or conditions which may affect any of these factors (e.g. injury, intoxication, etc).

With continued body cooling, consciousness becomes progressively impaired until it is lost at a deep body temperature of approximately 30 °C.17 Cerebral blood flow decreases in proportion to the reduction in metabolism,32 while Stone, Donnelly and Frosts89 reported a 6–7% reduction for each decrease in core temperature of 1 °C; cerebral activity is abolished at brain temperatures less than 22 °C.1 However, these data relate to anaesthetized patients in whom shivering was controlled. In victims of accidental immersion in cold water whose airway is being maintained clear of the water by a life jacket, shivering is pronounced with a resulting increased production of carbon dioxide, at least in the early stages, until body temperature decreases to approximately 29 °C. Should ventilation be impaired however, shivering is depressed or even absent while hypercapnia produces cerebral vasodilatation which increases cerebral perfusion.

With further cooling, in spontaneous breathing adults, cardiac arrest from ventricular fibrillation (VF) may occur at a deep body temperature <28 °C, or asystole at approximately 24–26 °C.3 However, with surface cooling, when core temperatures decrease to 35–33 °C, skeletal muscle temperatures are usually at or below 28 °C when neuromuscular performance is significantly impaired, making swimming or other actions necessary for survival extremely difficult.94 Therefore, it is likely that most immersion victims, excepting those wearing a life jacket with a “spray hood”,28 aspirate water as consciousness becomes impaired (i.e. as core temperature decreases to less than 34 °C) and thus well before significant protection from cerebral hypoxia is bestowed.

In spite of the ability of cold water to extract heat from the immersed body, hypothermia per se is unlikely to be a problem within 30 min of head-out immersion for a healthy, clothed adult, even in water as cold as 5 °C. The normal physiological responses to cold reduce heat transfer by mass flow (circulation) to the surface from the deeper tissues and increase shivering heat production by as much as five-fold.30 As a general rule, however, in spite of these responses, during head-out immersion in laboratory conditions, deep body temperature of the average adult wearing outdoor clothing has fallen to 35 °C after approximately 1 h in water at 5 °C, 2 h in water at 10 °C, and 3–6 h in water at 15 °C.94 In open water however, because of the rich supply of blood vessels to the scalp which do not vasoconstrict in the cold, heat loss from the unprotected head may be enhanced by forced convection and evaporation thereby increasing the rate of body cooling considerably. Similarly, if the head is immersed the rate of body cooling is significantly faster.

Because of their greater surface area to mass ratio and reduced subcutaneous fat content, cooling rates of immersed children are also significantly faster than those seen in adults. However, with surface cooling alone, even in children, rates for decrease in body temperature are too slow to cool the brain sufficiently to protect it from the hypoxia encountered in drowning asphyxia. In anaesthetized naked infants, being surface cooled for surgery with ice packs and ice cold water, rectal temperatures decreased by as little as 2.5 °C in the first 10 min of the procedure, and it took 32.5 min for it to reduce to 24–25 °C.70 Thus if cerebral hypothermia is the mechanism through which some children can survive protracted submersion without sustaining lasting hypoxic neurological damage, an alternative method of heat exchange to surface cooling alone must be found.

To date it has been postulated by several authors23 29 33 39 71 78 86 that the “mammalian diving response”, either alone or in combination with hypothermia,33 may be the explanation.

DIVING RESPONSE

The diving response, characterized by apnoea, generalized marked peripheral vasoconstriction and bradycardia18 23 is initiated reflexly by stimulation of the ophthalmic division of the trigeminal nerve on immersion of the face in cold water. It plays a powerful role in oxygen conservation in diving animals, enabling some to remain submerged for periods in excess of 30 min. The response is enhanced by both anxiety and cold water. 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.92 Nevertheless about 15% of people do show a profound reaction23 and this percentage increases with the use of specialist protective clothing, such as immersion “dry” suits,92 which prevent rapid cooling of the cutaneous thermal receptors of the majority of the body, but leave those of the face exposed to the cold stimulus. Tipton, Kelleher and Golden95 have reported that in such circumstances, where the cold-shock and diving responses are stimulated in roughly equal proportions, the resulting fluctuation in the competing parasympathetic and sympathetic chronotropic influences to the heart frequently (in 31 of 36 immersions) caused a variety of cardiac arrhythmias
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The diving response is also reported to be more marked in infants than adults. Gooden, in his excellent review, argued that while the diving response alone is unlikely to explain the reported survival of those cases of protracted submersion in cold water, it does play a contributory role together with hypothermia. He hypothesized that survival from prolonged near-drowning appears to depend on a specific interplay between the diving response and hypothermia, resulting in a protective state of "hypometabolism". Peripheral vasoconstriction, associated with both the diving response and cold, depresses tissue metabolism and thus reduces body oxygen requirements. Gooden postulated that immediately on face immersion the diving response apnoea prevents water aspiration into the lungs. Even if water does enter the larynx he postulated that reflex glottal spasm prevents further penetration into the lungs. The associated bradycardia and selective redistribution of blood conserves oxygen stores thereby protecting the brain from hypoxia during the initial minutes of immersion. Gradually, with surface cooling, hypothermia develops so that by the time oxygen stores are depleted, cerebral metabolism has been reduced to a level which confers some protection from hypoxia.

While to some extent this hypothesis is plausible there are several questions that remain to be answered. First, why is it that in most cases of protracted submersion who survived, sufficient water was aspirated to cause near-drowning? It is possible that aspiration, when it did occur, was a relatively terminal event when the diving response, or laryngospasm, could no longer be maintained because of profound hypoxia. However, from our understanding of surface cooling, discussed above, it is probable that profound hypoxia is present before significant brain cooling has occurred in this circumstance. Second, does the cold-shock response override the diving response in children as it does in adults? The only evidence in the literature relating to children in this context is that of Ramey, Ramey and Hayward who found that children could not hold their breath long enough for a diving response to occur; in their experiment however, the temperature of the water was 29 °C, above that normally associated with the initiation of the most powerful diving response. Skin temperature of children already playing in water is at or in the region of the water temperature and thus, if they become accidentally submerged, should not exhibit a cold-shock response. However, those children who fall into water without prior wetting should display a cold-shock response. Finally, another factor mitigating against the "hypometabolism" hypothesis is the large discrepancy in the rates of cooling reported (varying from 10 °C in 10 min in ice water to 3.5 °C in 6 min in a young adult). Such rates of cooling are not compatible with either surface cooling alone or in combination with a diving response.

Thus while not ruling out entirely the participation of the diving response in the early stages of some of these remarkable survival stories, it is considered that the rapid rates of cerebral cooling encountered may be largely dependent on pulmonary heat exchange after aspiration.

PULMONARY AND GASTRIC COOLING

After submersion, the victim may attempt to breathe for as long as possible before neurogenic afferents from the respiratory muscles or cutaneous thermoreceptors build to an intolerable level, forcing the individual to take a breath. It has been shown that voluntary breath-holding can be extended by movements of the respiratory muscles, and swallowing, against a closed glottis. This may be the explanation for the large amounts of water reported to have been found in the stomach of some drowning victims but not in others (i.e. ingestion of water is only likely to be found in those who attempted to extend voluntary breath-holding). Depending on the volume and temperature of the water swallowed, heat exchange between the blood and gastric wall could contribute significantly to body cooling, especially in children. When continued breath-holding is impossible, aspiration of water into the airways and alveoli occurs with inspiration and possibly reflex coughing, provided reflex laryngeal spasm does not supervene.

Regardless of the method of entry of cold water into the "core" of the body, be it lungs, stomach, or both, it precipitates a decrease in deep body temperature. The magnitude of this decrease is directly proportional to the volume of water aspirated, its temperature differential with the body and state of the circulation. However, to protect the brain from hypoxic damage it would be necessary to cool the brain very rapidly, at least 7 °C in 10 min. This doubles cerebral hypoxia survival time but even colder brain temperatures are required for longer survival periods.

Gooden argues that, to explain some of the remarkable survival stories reported, such a mechanism of cooling lacks quantitative evidence. On simple thermal physical principles of "methods of mixtures", he points out that "a (normal) person would need to ingest an amount of ice-cold water equivalent to 20% of body mass in order to reduce the average body temperature to 30 °C. Thus a child weighing 20 kg would need to ingest approximately 4 litre of ice-cold water". Such volumes of aspirated water are not compatible with resuscitation nor are they usually encountered in near-drowning victims. Harris stated that it is unusual to aspirate more than 200 ml but it is unclear how this estimation has been determined, particularly as it has been shown experimentally that aspirated fresh water is quickly absorbed and redistributed within the body.

Gooden’s calculation, however, appears to be based on the requirement for the entire body mass to be cooled to 30 °C. Using the same simple methods of mixtures, the volume of ice water necessary to cool the blood volume of a 20-kg child (approximately 1.6 litre) to 30 °C would be approximately 300 ml. It is accepted that this is a gross over simplification as the actual amount required to be
aspirated to cool the brain would need to be in excess of this because of the dynamic heat exchange that would occur with the perivascular tissues and perfused organs; in addition, pulmonary heat exchange is reduced because of pulmonary shunting. Nevertheless, given that a large proportion of the cardiac output in a child in such circumstances (i.e. with generalized peripheral vasoconstriction) is diverted to the cerebral circulation, it is conceivable that selective cooling of the brain occurs some time before the general body mass equilibrates. Thus aspiration of cold water may play a critical role in the production of cerebral hypothermia and brain survival in the submerged child or young adult of relatively small body mass.

At first sight the volume of water required to be aspirated would appear to be in conflict with Modell’s conclusion that volumes in excess of 22 ml kg\(^{-1}\) are usually incompatible with life. This would allow a maximum aspiration of 440 ml for a child with a body mass of 20 kg. While this is more than the theoretical 300 ml to cool the blood, as stated above, larger volumes would in reality be required to negate dynamic heat exchange with the perivascular tissues and perfused organs. However, during the drowning process, aspiration of water is unlikely to occur in one single bolus but is more likely to be a dynamic process with flushing of small volumes of cold water in and out of the lung. Fainer, Martin and Ivy\(^{26}\) demonstrated in dogs that rapid violent respiratory movement continued for about 70 s after submersion. It is proposed that with each tidal volume of water aspirated, heat is exchanged but the total volume retained in the lungs when respiration ceases is significantly less than the total volume of water which has entered and exited the lungs up to that time.

This hypothesis is supported by a recent animal experiment by Conn and colleagues\(^{16}\) which showed that aspiration of cold water produces extremely rapid blood cooling; in lightly anaesthetized dogs, the mean carotid artery temperature decreased by approximately 8.0 °C in the first 2 min of drowning, but only by 0.8 °C in a control head-out group of similarly shaved dogs; rectal temperature lagged “considerably behind”. Regrettably the experimental method did not make it possible to estimate how much fluid had been aspirated in those 2 min. When the animals were killed after 10 min of submersion, the volumes aspirated, estimated from post-mortem weight changes, were considerably in excess of the accepted lethal level of 22 ml kg\(^{-1}\).\(^{1,68}\) Incidentally, it is quite difficult to find the origin of the value of 22 ml kg\(^{-1}\) quoted frequently by Modell and others; in the original report by Modell and Moya,\(^{69}\) the value appears to be 20 ml lb\(^{-1}\), with death caused by VF rather than irreversible pulmonary damage.

The theory of water flushing in and out of the lung rather than absorption of water as the primary cause of the rapid cooling of blood on submersion is supported further by data from Conn and colleagues.\(^{16}\) The marked decrease in carotid artery blood temperature in dogs (average weight 9.2 kg) was 8.5 °C after 2 min in fresh water at 4 °C and 7.5 °C in salt water; while over the remaining 8 min of the experiment, mean carotid temperature decreased by another 2.4–10.9 °C in fresh water and by 3.1–10.6 °C in salt water. Body mass increased by 1.5 kg (fresh) and 0.6 kg (salt) in the 10 min. Although Conn and colleagues\(^{16}\) reported that all animals were tachypnoeic on immersion, the study did not state when respiration ceased. In an earlier experiment by Fainer, Martin and Ivy,\(^{26}\) using 160 dogs, it was reported that violent respiratory movement lasted approximately 71 s and thereafter respiratory movement was limited to the occasional agonal movement. Thus it would be fair to assume that, in the experiment by Conn and colleagues,\(^{16}\) most of the aspiration had taken place during the 2-min period showing the greatest decrease in carotid temperature. Furthermore, if one assumes no, or little, absorption in the salt water group in this time, it suggests that in the equivalent fresh water group, 7.5 °C of the 8.5 °C decrease in blood temperature in the initial 2 min was caused by flushing and 1.0 °C by absorption of approximately 900 ml of fresh water.

**AMBIENT COOLING**

Increasingly, hypothermia has been recognized as a favourable prognostic indicator. However, the rate of onset of hypothermia is clearly critical; to be of benefit it must occur rapidly before profound hypoxia develops.

Exposure to cold ambient conditions during resuscitative procedures, while awaiting the arrival of the ambulance, facilitates continued cooling if adequate insulation is not provided. Furthermore, thermoregulation in the hypoxic individual is likely to be severely compromised. This, combined with high evaporative heat loss from wet clothing and increased conductive heat loss to the ground beneath the supine patient, cause deep body temperature to continue to decrease. By the time the victim arrives at hospital, body temperature, in a patient with intact circulation, may have decreased significantly. Unless such a decrease occurs quickly, and before hypoxia becomes severe, little useful cerebral protection is provided.

Thus cooling which occurs during rescue and transport to hospital is of little benefit and may render deep body temperature on arrival in hospital an unreliable prognostic indicator.

**Rescue and treatment**

**RESCUE**

Advice on rescue lies more correctly with rescue organizations but a few important principles are offered for consideration:

- **Landing a casualty should never be delayed to enable attempts at in-water resuscitation.**
- **Where possible, casualties wearing life jackets whose airway is clear of the water should be recovered in a horizontal attitude.**\(^{32}\)
The possibility of cervical spine trauma should be considered in unconscious victims rescued from shallow water and appropriate measures and techniques used.37

TREATMENT

Out-of-hospital management

It should be presumed that all near-drowning victims rescued from UK open waters are hypoxic, acidotic and hypothermic.

The primary aims of treatment of the near-drowned are listed below in order of priority:

1. Effective immediate relief of hypoxia. The speed of achieving this has the greatest influence on outcome.73 87
2. Restoration of cardiovascular stability.87
4. Speedy evacuation to hospital.

Airway patency and adequate ventilation should be achieved rapidly. There is a significant risk of regurgitation and aspiration of gastric contents during resuscitation.74 Tracheal intubation, if indicated, should be carried out at the earliest opportunity.87 High fractional inspired oxygen \(\left(\text{Fi}_\text{O}_2\right)\) should be delivered using a self-inflating bag and reservoir; if available, a positive end-expiratory pressure (PEEP) valve set to 5 cm H\(_2\)O should also be used. Application of cricoid pressure during EAR, or bag and mask ventilation, reduce the risk of regurgitation and aspiration.74

The use of the Heimlich manoeuvre in near-drowning has been considered by the Emergency Cardiac Care Committee of the American Heart Association and is not recommended.83 There is insufficient evidence to support the claims that it is effective in removing aspirated liquid. Additionally, the manoeuvre itself may delay early intubation, aggravate visceral or spinal injury, cause confusion among rescuers with yet another procedure to follow,83 induce vomiting74 and increase the risk of precipitating an undesirable cardiac arrhythmia in the hypothermic casualty rescued from cold water.

In the hypothermic near-drowning casualty, because of the marked cold-induced peripheral vasoconstriction, characteristically low volume and profound bradycardia, peripheral pulses are notoriously difficult to feel. Accordingly, the carotid artery should be palpated gently for at least 1 min.37 74

Extreme caution must be taken not to precipitate VF by rough handling.75 VF responds poorly to defibrillation at core temperatures less than 28 °C; at this temperature, Purkinje tissue loses its conduction advantage over normal ventricular muscle fibres. Nevertheless, it may be worth attempting, especially in children, as some successes have been reported.88 100 However, even when sinus rhythm is achieved at these temperatures it tends to be unstable and revert easily to VF. If attempts at defibrillation at less than 28 °C are not immediately successful they should be discontinued until cardiac temperature increases to greater than 29 °C.

The casualty should be insulated against further heat loss and transported to the nearest emergency department as rapidly as gentle handling allows.88 Any life-support measures instituted at the scene should be continued during transportation.74 Some authors advocate immediate transfer of all severely hypothermic and clinically lifeless patients to the nearest facility with cardiopulmonary bypass facilities.11 60

Hospital management

Near-drowning is an emergency: even those victims who appear normal on arrival at hospital can deteriorate rapidly.74 An accurate, rapid initial assessment of the victim is essential. A suggested algorithm for subsequent management, based on the classification by Simcock of near-drowning victims,87 is shown in Figure 1.

Survival figures for victims of near-drowning are very encouraging for those with a minimal reduction in the Glasgow coma score (GCS) but much less favourable for those with low scores.34 74 Factors

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**Figure 1** A suggested algorithm for management of near-drowning, based on the classification by Simcock of near-drowning victims.87 ALS = Advanced life support.
which have been associated with favourable outcome are listed in table 1, but none, either individually or in combination, has been shown to have absolute predictive significance. Therefore, full resuscitative efforts should be attempted in all near-drowning patients arriving in emergency departments.

Patients with no evidence of aspiration

Victims of near-drowning who appear normal in the emergency department can deteriorate unexpectedly. All should be admitted to a general ward for a period of observation, generally accepted to be a minimum of 6 h. Other authors, however, consider it prudent to admit the victim for 24 h because of the risk of late onset pulmonary and cerebral oedema. It is recommended that the investigations listed in table 2 are considered. Hypothermic patients who are conscious and cooperative can be rewarmed in a bath of warm water (40 °C) until rectal temperature has increased to 36 °C. Arterial blood-gas values, obtained while breathing air, and core temperature must be normal before discharge. Signs of aspiration, which may become manifest later, include cough, tachypnoea, retrosternal discomfort, audible crackles on auscultation of the chest, abnormal arterial oxygen tension with the patient breathing air, and fever. Infection after immersion is common; usually this is confined to the chest, although there are reports of intracerebral abscess and septicaemia. However, the benefits of prophylactic antibiotic therapy have been questioned. It is generally agreed that antibiotic therapy should be withheld until bacteriological studies, or clinical examination, indicate active infection. Patients should be informed of the potential seriousness of any pyrexial illness which develops within a few days of discharge. They should be advised to attend either their general practitioner or return to the A&E department in such an event.

Patients with evidence of aspiration but without ventilatory compromise

These patients may develop pulmonary oedema and deteriorate precipitously. Accordingly, they should be admitted to a high dependency (HDU) or intensive care unit (ICU). Monitoring should include continuous pulse oximetry and frequent arterial blood-gas analysis. The onset of pulmonary oedema is usually within a few hours of aspiration of water, either fresh or salt, and can be rapidly progressive. Continuous positive airways pressure (CPAP) support provided by a mask or nasal cannula (in infants who are obligate, nasal breathers), should be offered in the first instance. The aim is to achieve an arterial oxygen partial pressure (PaO₂) of more than 8 kPa with an FIO₂ less than 0.5. If this is unsuccessful the facility for tracheal intubation and intermittent positive pressure ventilation (IPPV) with PEEP must be available immediately.

Patients with ventilatory compromise or hypothermia, or both

Patients who have clinical or investigative evidence of inadequate ventilation must undergo tracheal intubation and ventilatory support with PEEP. This should be maintained at 8–10 kPa or at 10–12 kPa in those with ischaemic heart disease. Ideally, FIO₂ should be less than 0.5 and PEEP provided at the lowest pressure consistent with minimizing intrapulmonary shunt and achieving these aims.

All victims of near-drowning who are hypothermic should be rewarmed in an ICU with continuous monitoring. Those who are cardiovascularly stable may be warmed slowly but actively, at a controlled rate of approximately 1 °C h⁻¹. This can be achieved using warmed humidified inspired gas, warmed i.v. fluids, warming mattress/blanket or wrapping exposed areas in dry woollen blankets. It is advisable to consider rapid re-warming for any patient whose core temperature is less than 28 °C because of the perilous state of the myocardium and its propensity for ventricular arrhythmias below this temperature. Patients who have an unstable cardiovascular system or are in cardiopulmonary arrest should be warmed more aggressively with consideration given to extracorporeal rewarming (ECR) using haemofiltration or cardiopulmonary bypass (CPB).

Methods used to re-warm rapidly without ECR include bladder irrigation, gastric lavage, oesophageal heat interchanger, pericardial lavage, peritoneal dialysis thoracotomy and pleural lavage. The easiest way to accomplish this in most hospitals is peritoneal dialysis. The dialysate

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**Table 1** Favourable prognostic indicators for near-drowning

- Children 3 yr +
- Female
- Water temperature < 10 °C
- Duration of submersion < 5 min
- No aspiration
- Time to effective basic life support < 10 min
- Rapid return of spontaneous cardiac output
- Spontaneous cardiac output on arrival in emergency department
- Core temperature < 35 °C
- Minimum blood pH > 7.1
- Blood glucose < 11.2 mmol litre⁻¹
- GCS: no coma on admission
- Pupillary responses: present

**Table 2** Suggested investigations for near-drowning patients

- Arterial blood gas analysis
- Biochemistry:
  - Electrolytes
  - Glucose
  - Urea and creatinine
  - Total creatinine kinase
- Coagulation studies
- Haematology
- Blood and tracheal aspirate for aerobic and anaerobic culture
- Chest radiograph
- Electrocardiogram
- Rectal temperature measured at least 15 cm from the anal sphincter
- Drugs screen for overdose of
  - Alcohol
  - Tricyclics
  - Benzodiazepines
  - Paracetamol
  - Aspirin, etc
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(2 litre of potassium free) is warmed to 54 °C so that it enters the peritoneal cavity at 43.5 °C, and hourly cycles are preferred. The advantages of ECR are rapid restoration of normothermia up to 10.7 °C h⁻¹, reduction of high blood viscosity found in hypothermia and, most importantly, when bypass is used, there is re-institution of perfusion regardless of cardiac rhythm with avoidance of "rewarming shock". An additional benefit is the ability to rapidly off-load ultrafiltrate in the presence of overwhelming pulmonary oedema; Norberg and colleagues removed 2300 ml in 1 h to facilitate withdrawal from CPB.

Those in cardiopulmonary arrest

Full resuscitation must be attempted in accordance with the Resuscitation Council (UK) guidelines and an attempt made to re-warm the casualty to 33 °C. This may cause manpower problems when prolonged external chest compressions (ECC) are required. Successful manual massage maintained for a period of 4.5 h has been demonstrated, while Ireland and colleagues reported success after 5.5 h using a “Thumper” cardiopulmonary resuscitator system (Michigan Instruments Inc., Grand Rapids, MI, USA) mechanical device. The advantage of a mechanical device is to ensure consistent effective compression during rewarming thus maintaining an adequate circulation to meet metabolic demands and facilitate regular emptying of the heart during ECR. Jones and Swan also recommend the use of such devices in prolonged resuscitation.

Cardiopulmonary bypass

Rewarming by institution of emergency CPB has been used with dramatic effect in severely hypothermic casualties who were in full arrest, many after submersion in cold water. The most notable of these was the neurologically intact survival of a 2.5-yr-old girl who was trapped under ice for 66 min. The majority of reports describe partial bypass from the femoral artery to femoral vein, and Seldinger technique to apply percutaneous CPB via the femoral veins, with a portable pump and paediatric oxygenator. CPB provides fast rewarming of the core, maintains tissue perfusion and oxygenation while assisting the cold and inefficient heart to regain normal electromechanical function.

The advocates of median sternotomy claim that exposure and cannulation of the aorta and right atrium is faster than dissection and cannulation of the femoral vessels in the pulseless patient, and that flow rates are faster. There may also be advantage in having access to the heart for massage, defibrillation and decompressive venting of a cold and dilating ventricle. A survey of members of the American Association of Thoracic Surgeons and the Southern Thoracic Surgical Association in the early 1980s produced evidence of 174 cases of hypothermia rewarmed with CPB with a survival rate of 45%. A review of the literature in 1994 analysed data from 68 hypothermic patients undergoing CPB. In reports describing attempted resuscitation of two or more cases the survival rate was 50%; if case reports and singletons were included the rate improved to 60%. VF was associated with greater survival than asystole, but the difference (73% vs 52%) was not significant. Walpoth and colleagues also noted that documented VF had a favourable outcome with CPB in hypothermia. Eighty percent of survivors returned to their previous level of function. The recommendations of that review were that CPB resuscitation is advised for hypothermic patients in arrest and for those with core temperatures less than 25 °C, irrespective of rhythm; patients in a stable rhythm of 25–28 °C can be treated with CPB or conventional warming methods. The maxim “nobody is dead until warm and dead” should always be borne in mind.

Cessation of resuscitative efforts

Hyperkalaemia in severely hypothermic patients is usually indicative of asphyxial cardiac arrest before significant cooling occurred. A potassium concentration in excess of 10 mmol litre⁻¹ is not compatible with successful resuscitation and is advocated for use in the triage of avalanche victims. Hypoxic encephalopathy is probably the most debilitating of the consequences of near-drowning. The majority of flaccid, comatose victims admitted to the ICU suffer either severe cerebral injury or cerebral death. Early prediction of outcome requires accurate determination of the nature and extent of neurological injury; it is also fundamental for patient management. In children, there are four distinct outcomes: full recovery, neurologically impaired survival, persistent vegetative state and death. Cerebral resuscitative efforts to reverse the changes of hypoxic damage and reperfusion injury have rarely been successful. Poor outcomes are especially likely when ICP > 20 and CPP < 60 mm Hg, despite therapy.

Prognosis

One USA source cited by Orlowski estimated that one-third of all near-drowning survivors were moderately to severely brain damaged. The prognosis for children in the UK appears to be similar: of 188 hospital resuscitations of near-drowning patients reported by Kemp and Sibert, 33 had fixed dilated pupils on admission to hospital; of these 13 died, 10 had severe residual neurological defects (spastic quadriplegia) and 10 recovered fully. A recent report from Seattle found an unfavourable outcome in patients who were comatose on admission: of 72 admissions there were 38 deaths, 14 in a...
vegetative state, three with dependent neurological impairment, six with mild neurological impairment and 11 discharged normal. The Australian experience was similar: 31 admissions with cardiac arrest of whom 17 died and six survived with severe spastic quadriplegia. This could be related to the warmer waters found in the Brisbane area. This view is supported by the Canadian experience where prolonged in-hospital resuscitation and aggressive treatment of near-drowning victims who initially had absent vital signs and were not hypothermic (<33°C), resulted either in eventual death or an increase in the number of survivors with a persistent vegetative state.

A prognosis score for paediatric near-drowning victims has been derived by Orlowski. It is based on five indicators: age younger than 3 yr, maximum submersion estimated longer than 5 min, post-rescue delay in resuscitation for greater than 10 min, coma at time of admission to hospital and initial pH less than or equal to 7.1.

Neurological classification of 101 paediatric drowning and near-drowning victims based on the level of consciousness, as assessed by GCS, has been used for prognostic purposes. GCS <5 predicted a high-risk group of patients with 80% mortality or permanent neurological sequelae. In general, 2–6 h after the accident, patients who remain decorticate, decerebrate or flaccid fair poorly; patients who are improving at this time, but remain unresponsive, have a 50% chance of doing well, while those who are showing definite signs of improvement (alert, stuporous or obtunded but responding to painful stimuli) generally do well and most have a normal or near normal outcome. A recent article by Kreis and colleagues suggested that the most accurate method of predicting neurological outcome is by magnetic resonance spectroscopy (MRS): their spectroscopic prognosis index distinguished between good (n = 5) and poor (n = 11) outcome, with one false negative after a borderline MRS result, and no false positive results (100% specificity).

It is generally agreed that hypothermia is neurologically protective, especially in children, although Kruus and co-workers concluded that a core temperature of less than 30°C is indicative of prolonged immersion and therefore of more severe brain hypoxia. In reports where rapid reversal of the hypoxic state was achieved, in combination with ICU support, often with ECR and occasionally with control of intracranial pressure (ICP), there was complete or near-complete return of neurological function with time.

Many studies assessing predictive factors have analysed data from near-drownings in warmer waters than are usually found in the UK. Therefore, it may be inappropriate to assess immersion victims using the same criteria, as the presence of hypothermia may offer some cerebral protection.

Complications

In addition to the complications already described (pulmonary oedema, infection, hypothermic cardiac arrhythmia, hypoxic encephalopathy and cerebral oedema), disseminated intravascular coagulation (DIC) has been described in near-drowning, as has rhabdomyolysis.

Rhabdomyolysis complicated by acute renal failure has been reported in two normothermic casualties of near-drowning in cold (14°C and 17°C) surf. One victim who was retrieved in cardio-pulmonary arrest and was alert, lucid and apparently normal 1 h later, had a plasma creatinine concentration of 2106 μmol litre⁻¹ 3 h after immersion. He required support with peritoneal dialysis for 11 days and forced saline diuresis to avert oliguria. The second patient discharged 2 h after the event, represented 24 h later complaining of weakness, myalgia and dark urine. His serum creatinine concentration was 359 μmol litre⁻¹, and forced saline/frusemide diuresis avoided the need for dialysis. Excessive or sustained muscle exercise and heat stress are well recognized causes of rhabdomyolysis; cold stress is a significantly less common trigger. However, in view of the potential consequence of acute renal failure, it would seem prudent to measure total creatinine kinase activity in addition to serum electrolytes and simple urinalysis in survivors of near-drowning.

Peripheral neurological sequelae may be manifestations of traumatic or even "non-freezing cold injury" to peripheral nerves rather than hypoxic damage from near-drowning. One should also be mindful of the near-drowning patient having a predisposing injury or medical condition, while the possibility of there being a drug-related condition should also be borne in mind.

Other treatment modalities

Artificial surfactant (Exosurf) has been used successfully in the management of a 9-yr-old victim of near-drowning with a rectal temperature of 30.3°C who was acidotic (pH 6.95). Despite circulatory and ventilatory support (FiO₂ 1, minute volume 230 ml kg⁻¹, PEEP 5 cm H₂O, peak ventilator airway pressure 40 cm H₂O) acidosis persisted and there was severe and progressive respiratory distress (pH 6.87, PaO₂ 7.6 kPa, PaCO₂ 12.4 kPa, base excess −17.5 mmol litre⁻¹). Four hours after admission artificial surfactant was instilled slowly via the tracheal tube. Within 5 min there was an improvement in gas exchange (PaO₂ 11.5 kPa, PaCO₂ 9.5 kPa) and a decrease in peak pressure to 30 cm H₂O, with no change in ventilator settings. His trachea was extubated 3 days later and he was discharged neurologically intact.

Animal models of near-drowning have failed to demonstrate any improvement in pulmonary function after the use of exogenous surfactant, although damage to the alveolar capillary membranes is reduced. This disappointing finding may be a result of using volumes of surfactant which were too large and instilled too soon after injury, causing documented severe hypoxia (i.e. a second major insult). The use of smaller volumes, instilled or nebulized at different times, with additional PEEP, have not been reported.
Immersion, near-drowning and drowning

A study on the effect of corticosteroid therapy on survival and blood-gas exchange in dogs after asphyxia of fresh water drowning demonstrated no beneficial effect. Their value in the management of pulmonary injury has not been proved and probably should not be used.

Conclusions

The rapid onset of cerebral hypothermia would appear to be the essential mechanism by which some submerged victims, in particular children, appear to be able to withstand protracted periods of asphyxia without suffering irreversible neurological damage. For this protective mechanism to be effective, brain cooling must be established rapidly, before severe hypoxia occurs. Such rates of cooling are unlikely to be achieved through surface cooling alone, or in combination with the diving reflex. It is postulated that rapid selective brain cooling is achieved through pulmonary heat exchange by repeated flushing of the lungs with cold water during the drowning process before cardiorespiratory arrest supervenes.

Successful treatment depends on speedy recovery and the immediate institution of CPR if indicated. All near-drowning victims, even those who appear to be ostensibly clinically well, should be transferred to hospital for pulmonary screening. Although some successful resuscitations of victims in cardiac arrest who are flaccid with fixed dilated pupils have been reported, the great majority would appear to have a poor prognosis. The prognosis is better for those with profound hypothermia but rewarming may be difficult in the absence of a good circulation. Accordingly, consideration should be given to transferring all profoundly hypothermic patients who are in cardiac arrest to a centre where cardiopulmonary bypass facilities are available, sooner rather than later.

References

Immersion, near-drowning and drowning


