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Drowning and near-drowning workshop: an overview

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Drowning, near drowning, pulmonary oedema. decompression illness

Abstract
Drowning is the third most common cause of accidental death in the general population and causes about one fifth of all accidental deaths in children. Alcohol is a frequent association in immersion accidents. Drowning or near drowning is not a diagnosis, the cause for the incident must be found. The ability to predict outcome from near drowning has been difficult, but the presence of spontaneous breathing, or the return of breathing, and the presence of a pulse shortly after rescue are all associated with a good outcome. All near-drowned victims may deteriorate even if they appear 'normal' on hospital admission, and therefore require a period of observation which many consider should be at least 24 hours. Hypothermia as a cerebral protection mechanism in near drowning may play little role in scuba diving. Diving as a cause of drowning has been neglected statistically, and there are only limited data on the interaction between near drowning, compressed gas diving, decompression illness and treatment. Theoretically, near drowning should decrease inert gas elimination because of induced changes in cardiovascular and pulmonary function. There are no data on the management of the near-drowned diver who may require recompression. Theoretically, recompression in the initial management of the near-drowned diver may cause deterioration in the diver's clinical condition. Due to these theoretical risks associated with recompression, the timing of recompression in the near-drowned diver may be important.

Introduction

Drowning is death due to asphyxia while immersed in a liquid medium, whilst near drowning is survival (at least temporarily) from an immersion incident. The classification used in this workshop is listed in Table 1.

Drowning ranks as the twentieth most common cause of death worldwide.[1] Drowning continues to be the third most common cause of accidental death in the general population and is more common in males than females.[1] It accounts for about one fifth of all accidental deaths in children.[2] While statistics for drowning deaths are known, there are no precise data for near-drowning incidents.[1] Some estimates put near-drowning incidents to be 500–600 times more common than drowning.[3]

Alcohol is a frequent association in immersion accidents either in the victim or, in the case of children, in supervising adults. Approximately 40% of childhood drownings occur in private swimming pools. Of note in childhood drowning is that children drown silently. In Australia, drowning is second only to motor vehicle trauma as the most common cause of accidental death.

In the majority of cases, the nature of the liquid medium does not influence the outcome. Drowning or near drowning will be the final common pathway of many problems occurring whilst in the water. One of the difficulties in studying the epidemiology of drowning fatalities has always been the tendency for pathologists to report it as "death by drowning" without any insight into the underlying cause(s). Drowning or near drowning is not a diagnosis, the cause for the incident must be found.

TABLE 1
CLASSIFICATION OF THE IMMERSION INCIDENT

Drowning:	Death due to asphyxia (with or without fluid aspiration) while immersed in a fluid
Near drowning:	Survival from an immersion incident (with or without fluid aspiration)
Secondary drowning:	Delayed onset of pulmonary oedema in a victim who appeared to have recovered from an immersion incident

Australian drowning statistics are collected by the Royal Life Saving Society.[4] In the financial year 1997/8, 326 people drowned, 52 of whom were children under the age of five. These data also show that for every child who drowned, five or six were admitted to hospital as a result of an immersion incident, and of these 5–20% had neurological sequelae. Data are available for each state and

although the numbers may vary, the percentages are approximately the same. Data for South Australia between 1995 and 1998 are shown in Table 2.[4]

TABLE 2
SOUTH AUSTRALIAN DROWNING DATA 1995-1998

TOTAL	**49**
Male	76%
<5 years	14%
15–49 years	53%
At home	20%
Beach	35%
Country area	53%

Outcome

The ability to predict outcome from near drowning has been difficult, but recovery may involve varying degrees of functional disability. There is also a lack of full statistical data providing a ratio of those 'successfully resuscitated' to survivors left with permanent neurological damage.[1]Available data indicate that those factors listed in Table 3 may be associated with a poor outcome. The presence of spontaneous breathing, or the return of breathing, and the presence of a pulse shortly after rescue are all associated with a good outcome. In children, a detected motor response to pain upon arrival in hospital is associated with a good prognosis.[5]

TABLE 3
FACTORS ASSOCIATED WITH A POOR OUTCOME IN IMMERSION INCIDENTS

- Fixed dilated pupils
- Prolonged immersion
- Delay in effective resuscitation
- Absence of spontaneous respiration
- Severe metabolic acidosis – pH <7.1
- Asystole on admission to hospital
- Recovery of a pulse only after arrival at the hospital
- Glasgow Coma Scale (GCS) <5 on admission
- Hyperglycaemia

Kries et al recently published that magnetic resonance spectroscopy was a good predictor of outcome.[6] However, their series contained only 16 patients. Szpilman published a severity classification related to mortality of near-drowned victims based on clinical data from 1831 cases gained at the site.[7] Szpilman's classification has some prognostic merit but relies on the clinical acumen of the rescuers. All but Grade 1 victims require oxygen administration. Grade 2 victims need a later careful clinical assessment. Szpilman's classification is listed in Table 4.

All near-drowned victims may deteriorate, even if they appear 'normal' on hospital admission. Some authors believe they should be observed for six hours, but others consider it more prudent to observe them for 24 hours.[1]

Drowning and scuba diving

Diving as a cause of drowning has been neglected statistically. Rarely does the drowning literature refer to diving as an important cause of drowning or near drowning. The Divers Alert Network (DAN) reported in 1997 that 63% of diving fatalities drowned.[3]

There are also limited data on the interaction between near drowning, compressed gas diving, decompression illness (DCI) and treatment (the author found one article in a Medline search). Whether near drowning delays inert gas elimination, thus predisposing a diver to DCI is unknown. Theoretically, near drowning should decrease inert gas elimination because of induced changes of cardiovascular and pulmonary function. Near drowning causes a reduction in cardiac output,[8] an increase in areas of low ventilation-perfusion ratios (shunt) in the lungs, and pulmonary hypertension.[9]

Experimentally, pulmonary hypertension causes venous sludging in the epidural plexus, promoting bubbling and venous infarction of the spinal cord.[10] However, experimental data on spinal cord DCI ignore the negative physiological effects of neurogenic paralysis on blood pressure and cardiac output, which will decrease spinal cord perfusion and increase any vascular stasis thus promoting

TABLE 4
SZPILMAN'S CLASSIFICATION[7]

GRADE	CLINICAL SIGNS	MORTALITY
Grade 1	Normal pulmonary auscultation with coughing	0%
Grade 2	Abnormal pulmonary auscultation with rales in some pulmonary field	0.6%
Grade 3	Acute pulmonary oedema without hypotension	5.2%
Grade 4	Acute pulmonary oedema with hypotension	19.4%
Grade 5	Isolated respiratory arrest	44%
Grade 6	Cardiopulmonary arrest	93%

further bubbling. Local changes in tissue perfusion (due to local effects of hypoxia, acidosis, and vasodilation) and a decreased cardiac output will also reduce tissue perfusion, and may cause prolonged tissue supersaturation, thus increasing the risk of DCI.

The pathophysiology of cerebral injury in near drowning is one of global hypoxia. Disruption of the blood-brain barrier results in cerebral oedema accompanied by a rise in intracranial pressure (ICP). Large increases in ICP are infrequent in near drowning and tend to appear after the initial resuscitation and in victims with some evidence of neuronal dysfunction. A rapid increase in ICP is an indicator of the severity of the neuronal injury and is a result of the brain injury rather than the cause of it.[5] Levels of ICP >20 mmHg and a cerebral perfusion pressure <60 mmHg are associated with a poor outcome despite aggressive therapy.[5] However, a normal ICP is not a guarantee of normal outcome. The impact of these changes on DCI is unknown.

Hypothermia and near drowning

Near drowning and the potential protective mechanisms of hypothermia have been the subject of many reviews. An excellent review concerning this topic was published by Golden et al.[1] A hypothermic decrease in cerebral metabolism has been considered as an explanation for survival following prolonged immersion, especially in children. Profound hypothermia is protective because it reduces oxygen consumption.

However, cardiovascular and respiratory responses due to cold water immersion (severe bradycardia or catecholamine excess causing ventricular arrthymias, cold water stimulation of breathing and a decrease in breath-hold time) would most likely be fatal before the victim had time to become hypothermic.[11] In addition, studies by Sterba et al have shown that sudden immersion in cold water will increase metabolic rate.[12]

The so-called 'protective mechanism' of hypothermia, therefore, may be incorrect and misleading. Hypothermia noted in a victim who has drowned in warm water is probably an indication of cerebral death, but aggressive resuscitation should not be abandoned because of this assumption. In addition, spearfishermen, snorkellers and compressed gas divers wear protective suits that delay the onset of hypothermia (hence these swimmers and divers rarely become profoundly cold) and this would prevent the suggested protective mechanism of hypothermia.

Management

Treatment is directed towards both the primary cause and near drowning. Early institution of continuous positive airway pressure (CPAP) or mechanical ventilation (IPPV), and positive end expiratory pressure (PEEP) are often associated with a rapid correction of hypoxia.[13] PEEP will not alter the underlying pulmonary injury, but will enable oxygenation during recovery. Initially, high-inspired oxygen concentrations may be needed. Animal data have shown a return of a normal A-a gradient and P_aO_2 within 10 minutes of resuscitation using 5 cms PEEP when ventilated with an FiO_2 of 100%.[13]

Steroids,[14] diuretics and prophylactic antibiotics are not indicated in the clinical management of near drowning.[1] Antibiotics, however, may be needed if the near drowning occurred in polluted waters. The appropriate antibiotic will be determined by sputum and/or blood cultures. Animal data have failed to show any benefit from exogenous surfactant, although there are anecdotal case reports of its successful use in a nine-year-old, near-drowned victim.[15] Evidence of trauma (cervical spine injuries must never be overlooked) and sinus and ear barotrauma should be looked for. These may act as a nidus of infection later.

Management of the near-drowned recompressed diver

Resuscitation may initially retard inert gas elimination in a patient with compromised cardiovascular and respiratory systems. IPPV and PEEP are associated with a decrease in cardiac output and blood pressure due to impaired venous return, decreased ventricular filling, increased pulmonary vascular resistance, and altered configuration and compliance of the right and left ventricles, even in patients without significant pulmonary pathology.[16,17]

There are no data on the management of the near-drowned diver who may require recompression (no reference was found by the author using a Medline search). Recompression in the initial management of the near-drowned diver may cause deterioration in the diver's clinical condition. Recompression and hyperbaric oxygen therapy (HBOT) may depress inert gas elimination because of worsening pulmonary oedema when applied to a patient with decreased left ventricular function. Recompression and HBOT, by increasing systemic vascular resistance and afterload, may further decrease left ventricular contractility, cause centralisation of blood volume and an imbalance between right and left heart function that worsens pulmonary oedema in these patients.[18]

There may also be an increased risk of pulmonary barotrauma in a patient receiving IPPV and PEEP. Recompression may also increase the work of breathing in a spontaneously breathing patient in respiratory distress because of the increase in gas density with increased pressure. An increase in the work of breathing may promote carbon dioxide retention and precipitate oxygen toxicity.

Due to these theoretical risks associated with recompression, the timing of recompression in the near-drowned diver may be important. More data are needed. A summary of the management of near drowning is presented in Table 5.

TABLE 5
SUMMARY OF THE MANAGEMENT OF NEAR DROWNING

- Rescue and remove from danger
- Begin CPR as soon as possible
- Transfer to hospital
- Determine primary cause*
- Advanced resuscitation: CPAP, intubate, ventilate, high inspired oxygen, PEEP, assess circulation
- Biochemical analysis including arterial blood gas, serum electrolytes (including magnesium* and CPK*), full blood count, bleeding and clotting profile
- CXR, cervical spine* films if needed
- Look for evidence of ear and sinus barotrauma (these may become a nidus for infection later)*
- Nasogastric tube
- Bladder catheterisation
- Monitor temperature
- Invasive haemodynamic monitoring as required (Swan Ganz etc)
- Inotropes and fluids as required
- Treat primary cause
- Watch for fluid depletion (osmotic-induced diarrhoea due to swallowed sea water)*
- Treat complications as they become evident
- Consider recompression in any compressed gas (scuba) diver*

*Often forgotten

TABLE 6
CAUSES OF PULMONARY OEDEMA POST IMMERSION

Diving (*breathhold and scuba)

Near drowning*
Aspiration (fluid and gastric contents)*
Marine envenomation*
Inhalation of contaminants (carbon monoxide, oil, bacteria from equipment)
Intra-alveolar haemorrhage (on anticoagulants)*
Neurogenic pulmonary oedema (hypoxia)*
Negative pressure pulmonary oedema (laryngospasm)*
Pulmonary oedema of immersion
Pulmonary oedema and strenuous swimming*
Pulmonary decompression illness ('chokes')
Underwater blast*
Pulmonary barotrauma of descent (BH)
Oxygen toxicity

Non diving

Myocardial infarction
Acute allergy
Chest infection
Trauma

Pulmonary oedema post immersion

The prevalence of pulmonary oedema (without evidence of aspiration) during scuba diving is unknown.[19] One series quotes a rate of about two per cent of divers.[20] There are numerous diving and non-diving causes of pulmonary oedema and these are listed in Table 6. The potential impact of near drowning and pulmonary oedema on inert gas kinetics has been modelled by Doolette and Acott.[21]

A return to diving following near drowning

A return to diving following a near-drowning episode has not been discussed in the medical or diving literature. Medical suitability to return to diving would depend on the presence of neurological and respiratory sequelae and the cause of the near-drowning episode. Normal respiratory function may take up to 16 weeks to return in non smokers who have near drowned.[22]

Summary

The 2001 SPUMS workshop on drowning and near drowning in divers addressed the following issues:

The epidemiology of drowning in compressed air divers (Australian and New Zealand data)[23,24]

Whether or not there is an increased risk of decompression illness with near drowning (using a probabilistic model of inert gas kinetics)[21]

The pathophysiology of near drowning[25]

The differential diagnosis of pulmonary oedema following compressed air diving, including the salt water aspiration syndrome and immersion pulmonary oedema[26,27]

The physiological effects of IPPV and PEEP in a hyperbaric environment[28]

The use of lignocaine as adjunctive therapy in dysbaric disease[29]

A diving physician's personal view of why divers drown[30]

Near drowning and the unresponsive diver; rescue training for recreational scuba divers[31]

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