radionuclide scanning has other limitations. Indium labelling of leucocytes has to be performed under sterile laboratory conditions and is not a simple procedure. Neither gallium nor indium scans can distinguish clearly between inflammatory masses and abscesses, in both of which there is increased accumulation of the agents.

When all these considerations are taken into account a strategy may be formulated for the logical use of imaging techniques. Many workers have suggested that in the presence of localising signs the first investigation should be an ultrasound or a computed tomography scan.[7] My own view is that, whether or not localising signs are present, the first choice should be an ultrasound examination. This is a simple procedure and even in the absence of localising signs a substantial number of abscesses will be detected. Since time on a computed tomography scanner is at a premium computed tomography scans should be reserved for cases where an ultrasound examination is unsuccessful and those in which lesions are suspected in sites which are more difficult to evaluate on ultrasound, such as the retroperitoneum. Usually a firm diagnosis may be arrived at using ultrasound or computed tomography. If, however, there is doubt about the nature of the lesion and a diagnostic aspiration is best avoided a ^{111}In-leucocyte scan should be arranged. This technique should also be used for suspected small collections, particularly when they arise secondary to inflammation of the bowel.

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Regular Review

The management of near drowning

JOHN PEARN

Immersion accidents are not uncommon,[1-4] and are always potentially serious with relatively low survival rates.[5] Unlike many types of accidental or traumatic deaths (poisonings, for example[6]), death rates from drowning have not fallen, and in some countries (the United States[7] and Australia[8]) continue to rise. Doctors may be asked to give medical care to the apparently drowned unexpectedly and at any time—at a local sailing club, a holiday beach across the world, the next door swimming pool,[9,10] the local surgery, or in a hospital intensive care unit.[11-13] The optimum management of the near drowned includes many practical skills—rescue, extraction from the water, resuscitation at rescue site,[14] transport, emergency room management, intensive care treatment, as well as the monitoring of convalescence[15] and making realistic predictions[13,14] about the prognosis in survivors.[16-19]

In near drowning no two cases are the same. Variations in water osmolality (salt versus fresh[14]), questions whether the victim has taken alcohol or drugs[20] or has attempted suicide,[21] non-accidental injury in the case of some child victims,[22,23] immersion hypothermia,[24] the possibility of epilepsy,[25,26] whether resuscitation was given,[27] and whether it was skilled or not,[14,28] may produce an infinitely variable clinical picture. The most important variable of all—the duration of hypoxia—is usually unknown, and mostly unknowable.

The potential of these confounding variables is such that the clinician must have a clear picture of the "hypoxic march," which leads to brain death and then to somatic death (see appendix I).[29] Clinical assessment and the interpretation of clinical signs depend on understanding these pathophysiological events in the victims of immersion.[29]

First aid

First aid for the apparently drowned is standard, though disquieting reports persist that many medical graduates (especially those not in primary care or surgical specialties[30]) feel inadequate in the field when expected to give expired air resuscitation and external cardiac compression. Cardiopulmonary resuscitation uses the same technique whether it is undertaken at the rescue site or in the emergency room (casualty department) while intubation, monitors, defibrillators, ventilators, and other hardware are being coordinated. The airway must be cleared, initial breaths given, the carotid pulse checked, and (if absent) combined external cardiac compression and expired air resuscitation administered.

About fourth fifths of child victims and most adults who do survive will make their first respiratory gasp within five minutes of rescue.[14] Many do so within the first minute after

extraction from the water. The pressure used to inflate the lung of the near drowned victim has to be greater than for someone apnoeic from other (usually non-pulmonary) causes. Lung compliance is dramatically reduced after aspiration of both salt and fresh water, and the fall may be extreme in cases of near drowning where the inhaled fluid contains paint, fertiliser, or sewage.[31] The unconscious (but breathing) victim must be nursed and transported in the coma position, for the airway is particularly likely to be blocked secondarily by regurgitation of large amounts of water and of stomach contents.[32] Amateurish cardiopulmonary resuscitation (or effective cardiopulmonary resuscitation not started within 10 minutes of extraction from the water) is an unfavourable prognostic feature.[13 14]

In most parts of the world there is no need to be concerned with rewarming the victim at the stage of first aid as the body temperature is usually in the range of 33-36°C. All victims have a reduced body temperature owing to a combination of submersion and immersion hypothermia.[24] If the temperature is below 28°C, however, ventricular fibrillation and asystole will occur,[33] and attempts to restart the heart by cardiopulmonary resuscitation at the rescue site will be in vain. In these circumstances cardiopulmonary resuscitation must be continued indefinitely until the victim can be rewarmed under controlled conditions in an intensive care unit. Death must never be diagnosed in the field if the victim is cold. Prolonged submersion (for even up to 40[24] or 60 minutes) may be compatible with complete recovery even when complete cardiopulmonary arrest is present at rescue. In such cases submersion hypothermia may have lowered the core temperature below 30°C. Involuntary aspiration of cold water has the same effect as an intravenous injection of cold water.[34] Core chilling spares the brain; brain temperatures fall at up to 1°C a minute during the agonal respirations of drowning animals submerged in fresh water at 20°-22°C.[24]

How long should one persist with cardiopulmonary resuscitation? If the victim has a core temperature above 33°C and has taken neither drus nor alcohol I have yet to see a survivor without brain damage who did not make his or her first respiratory gasp within 40 minutes of rescue.[14] Hence if asystole persists after one hour (and provided that the body is not chilled) persistence in field cardiopulmonary resuscitation is fruitless. Ideally, however, the decision to stop cardiopulmonary resuscitation should be made in a properly equipped emergency room.

Often the first sign of successful cardiopulmonary resuscitation is a convulsive abdominal diaphragmatic heave with a flood of vomitus or swallowed water. Most patients who are going to survive show some signs of returning respiratory activity (usually incoordinate gasping) quickly. Indeed, most (at least in the case of children) become conscious within 20 minutes or so, and many can talk and move by this time. Secondary drowning remains a danger, however, and all immersion victims must be admitted to hospital for observation at least overnight.[35] This deterioration after rescue may occur because of loss of surfactant or its denaturation by the inhaled liquid, or because of damage to alveolar cells from osmotic or anoxic causes.[36] Within minutes after inhalation of small quantities of water (2·5 ml/kg) the intrapulmonary shunt increases from its normal level of perhaps 10% to as much as 75%. Even in victims who are conscious, alert, and clinically normal after near drowning and successful cardiopulmonary resuscitation the shunt takes several days to revert to preimmersion values.[37]

In some circumstances—for example, surfing, or diving into creeks—there may be a substantial probability of spinal injury, and the near drowning may be secondary to this. The possibility of a fracture or dislocation of the cervical spine is a very important aspect of the first aid management of the near drowned.[23 38]

Hospital management

The clinical history is almost always incomplete in the initial stages of the hospital management of the victim of near drowning. The history has to be built up over ensuing hours after admission, and help is required from bystanders, rescuers, police, and ambulance staff. An ideal clinical history will include the documented or estimated time of accident; the type of drowning fluid (salt versus fresh water, for example) and an estimate of its temperature; the degree of water contamination; the estimated duration of immersion or an estimated bracket of time during which the victim could have been submerged; details of the rescue; whether cardiopulmonary resuscitation was attempted and whether it was performed by a trained first aider; whether vomiting occurred after extraction; the time to the first spontaneous gasp after rescue; details of transport to hospital and whether cardiopulmonary resuscitation was maintained; the past health of the victim—epilepsy, asthma; and any other specific features of the immersion incident. Drownings are often the end result of other medical problems,[39 40] and are not always what they seem.

Some 30 years ago a debate (discussed initially in the *BMJ*[41]) centred on the possible differences in the management of the near drowned victim according to whether salt or fresh water had been inhaled. This followed Swann's classic experiments on experimental drowning.[42 43] The work of Modell and colleagues in the 1960s showed that, if the victim survives the submersion, electrolyte changes—if they occurred—were likely to be transient and to revert spontaneously to normal and that the initial management of the critically ill survivor could discount whether salt or fresh water was inhaled or swallowed.[44] Management is governed by clinical assessment and by the results of tests on the victim performed after arrival at hospital and once ventilation and life support (if required) are proceeding (see figure).

Primary triage has long classified near drowned victims into three groups (recently popularised by Modell and by Conn for prognosis[11 12 33 44-46]): those who are apparently normal when they reach hospital (A for "awake"); those who are conscious but obtunded (B for "blunted" in the Modell-Conn classification); and those who are comatose (C). Those in groups A and B require a chest radiograph, and those in group B may also require baseline biochemical and respiratory function studies. Careful clinical monitoring of these two groups should prevent overinvestigation and polybiochemistry; most will make a successful recovery with good nursing care.

All victims who are comatose (group C) and all others who are not improving clinically hour by hour need a series of laboratory and ancillary investigations for immediate diagnosis, to establish baselines by which improvement may be judged, and against which the rate and severity of any deterioration may be monitored by objective documentation.

The appropriate tests include chest radiographs,[1 47 48] blood gas values,[37 49] arterial pH[19 50] (temperature corrected in hypothermic patients[51 52]), tracheal swabs,[53 54] serum biochemistry[40] and osmolality,[44] blood alcohol concentration,[55]

serum anticonvulsant concentration[25 26] (when appropriate), electrocardiogram (many arrhythmias disappear as rewarming occurs[56]), electroencephalogram, skull and cervical spine radiographs, and a skeletal survey (in the case of infant bathtub immersions[22 23]); all may show characteristic changes. Respiratory function tests should be undertaken as early as is practicable after the victim's clinical state has become stable,[57] computed tomography may be needed in comatose patients not responding, and psychometric assessments should be undertaken in all children and in those adults in whom there is a clinical suspicion of intellectual damage.[16]

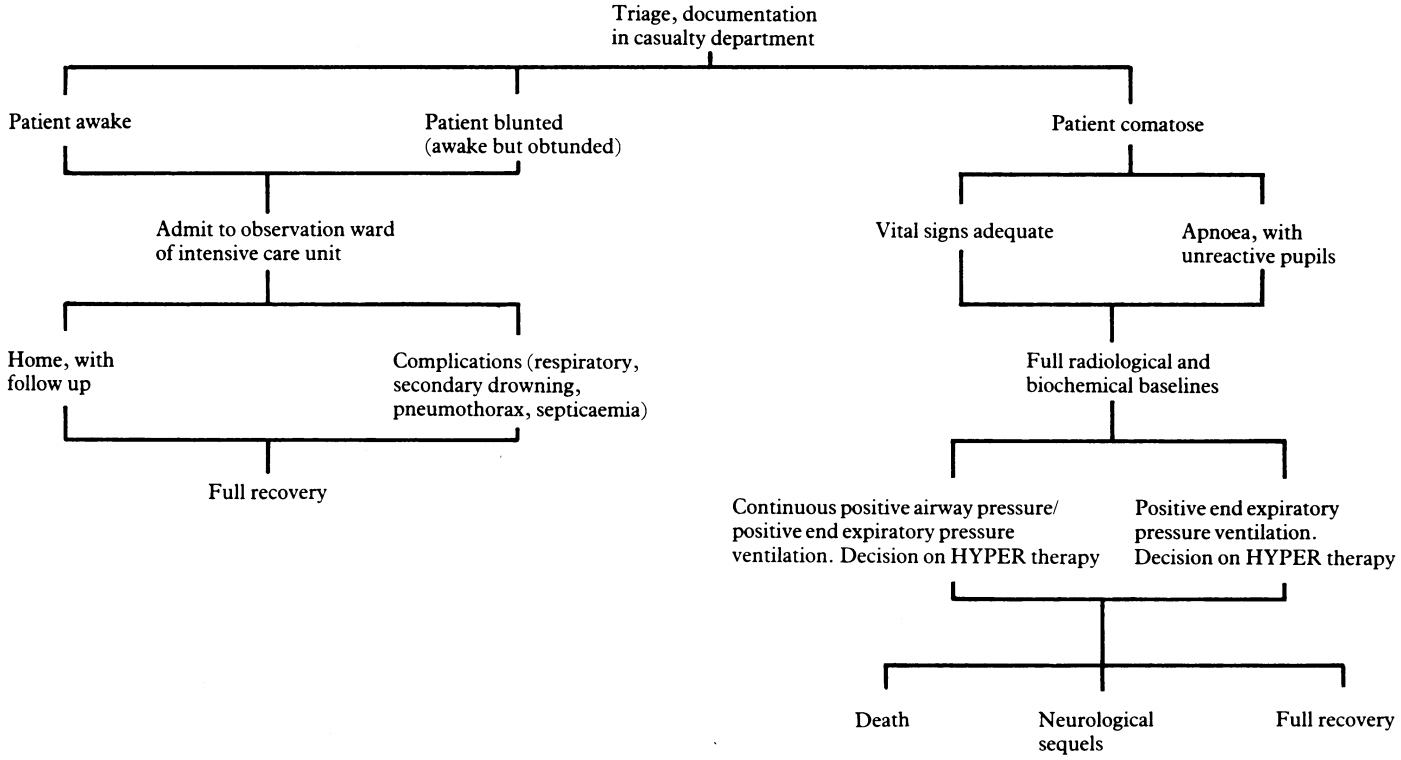
Mechanical ventilation is required when a near drowned survivor cannot maintain his arterial oxygen and carbon dioxide concentrations within normal limits by his own efforts. A Pa_{O_2} of less than 8·0 kPa (60 mm Hg) breathing air together with a Pa_{CO_2} of 7·5 kPa (56 mm Hg) is an indication for mechanical ventilation.[56 58] Most intensive care doctors try to keep the Pa_{O_2} at 13·3-26·6 kPa (100-200 mm Hg) on empirical grounds in the belief that blood to cell diffusion will be facilitated. The ventilator mode should be constant positive airway pressure/positive end expiratory pressure. Almost all near drowned comatose children will require intubation and positive end expiratory pressure, but a few (especially those who are older) will tolerate a mask and benefit from constant positive airway pressure rather than the simple breathing of oxygen enriched air in a face box, face mask, or tent. The use of positive end expiratory pressure has been hailed as one of the most useful therapeutic milestones in the history of medicine.[59] Its use in the near drowned has now been established for well over a decade and is not controversial.[60-64] Weaning to intermittent positive pressure ventilation with zero end expiratory pressure should occur as soon as possible to reduce the risk of pneumothorax[61] and tracheomalacia.

The management of fluid balance remains controversial; One regimen of fluid deprivation and the administration of frusemide (1 mg/kg) is aimed at lowering raised intracranial pressure, but whether this is beneficial (as Canadian reports suggest[46]) remains unknown. Some workers believe that the use of plasma expanders may be required if a satisfactory circulating volume cannot be maintained,[63] especially if hypovolaemia is unmasked by positive end expiratory pressure.[65]

Substantial volumes of water may be absorbed from the stomach, and to prevent this a stomach tube must be passed—with the trachea guarded by a cuffed tube[66]—for aspiration of stomach contents causes serious complications.[59 67]

The role of barbiturate rescue remains controversial. Initial very good results (brain sparing after severe hypoxia) in man[68 69] and in studies on primates[70 71] led to clinical studies of patients having open heart surgery and with head injuries[72] and to its use in near drowning.[11 33 46 66] Both pentobarbitone and phenobarbitone (in the latter case 25-50 mg/kg/day for up to four days) have been used in paralysed and ventilated victims of immersion, but the results remain uninterpretable in the scientific sense.[14 73]

Induced hypothermia is currently used in many centres; it is achieved by placing ice packs over the femoral vessels in the inguinal region and with intravenous barbiturates and chlorpromazine. Its use is controversial, and whether it is



Algorithm for management of near drowning. After Pearn,[14] published by permission of W B Saunders Co.

effective also remains unknown.[14 45 65] Conn, who popularised its use for all comatose immersion victims, now believes that normothermia should be maintained; if the intracranial pressure cannot be kept normal by other therapeutic measures then the core temperature may be reduced to 30°±1°C, on the grounds that both the cerebral oxygen requirements and intracranial pressure are reduced, but the core temperature must be kept above the level at which spontaneous ventricular fibrillation may occur—28°C.[1]

If a decision is made not to induce hypothermia certainly there is no point in rewarming the victim rapidly if the temperature is above 30°C. Rewarming should be allowed to progress over the six to eight hours after rescue. Many near drownings occur in tropical and temperate waters and a victim's temperature may fall further in air conditioned intensive care units unless it is monitored. The implications of hypothermia after immersion may be difficult to interpret,[14] for it may be due to either of two opposing influences: hypothermia may result from chilling from cold water with the implication of brain sparing, or it may be a measure of long immersion time and a decompensated cardiovascular system, in which case it signals a poor prognosis.[74]

Much of the stimulus for new empirical regimens for managing the near drowned—for example, the HYPER approach[1 11 12 33 46 66]—came from a wish to control raised intracranial pressure. Any hypoxic insult causes a period of persistent hypoperfusion owing to increased cerebral vascular resistance.[75] The effect of this may undoubtedly summate with any rise in intracranial pressure. After hypoxia the blood-brain barrier breaks down and both fluid and macromolecules pass into parenchyma[76]; functional, if not anatomical, dissolution of vascular and cell wall integrity leads to intracellular and extracellular increases in osmotic pressure,[77] and oedema develops. In survivors the speed with which cerebral oedema develops remains uncertain, but it is not a problem in the first 24 hours after rescue.[19 78 79]

Raised intracranial pressure may be controlled by treatment with both short acting and long acting barbiturates, lignocaine,[80] by gentle nursing techniques with the reduction of handling and painful stimuli to a minimum, by hyperventilation,[65] by hypothermia (with and without chlorpromazine), and probably by dexamethasone[14] (0·1 mg/kg every eight hours), although steroids may not be as effective in postanoxic cerebral oedema as they undoubtedly are in cerebral neoplasms. In the past five years some units have monitored intracranial pressure by an epidural or intraventricular pressure transducer, but the technique remains controversial and we do not know whether it improves survival.

This new, aggressive empirical approach to the management of near drowned victims is controversial because it has a real iatrogenic cost. Hypothermia,[65] monitoring of intracranial pressure,[18] and treatment with barbiturates[73] may all cause serious complications. Furthermore, we still do not know whether treatment of a raised intracranial pressure makes any difference to prognosis in the near drowned.[18 81 82] Clinicians have to treat near drowned victims in these uncertain times; I believe the correct approach is to maintain a healthy scientific objectivity and take part in controlled multicentre trials. The near drowned victim has everything to gain from inclusion in such studies. In desperate cases (patients who are apnoeic, requiring cardiovascular support, and with fixed dilated pupils several hours after rescue) there is everything to be gained by being part of a multicentre series with the therapeutic variables of hypothermia, barbiturate rescue, and monitoring of intracranial pressure all controlled in scientific fashion.

Complications in the period after rescue may include hyperthermia,[24] seizures, gastrointestinal bleeding,[14] spasms, mania with combative flavour,[14] multiorgan failure,[11] septicaemia (often due to exotic marine[83] or sewage[54] organisms), tracheal necrosis,[14] pneumothorax, secondary drowning,[14 35 64] pneumonia,[47 48] and necrotising pneumonitis.[14 31] Some at least of these should be preventable with prophylatic antibiotics[14] and intravenous cimetidine (in the comatose).

Giving a prognosis

The management of immersion victims includes giving a prognosis to parents and relatives. If the victim has made a first respiratory gasp within 30 minutes of rescue and is contintuing to improve (as shown by vital signs, level of consciousness, and so on) over succeeding hours the prognosis is good. If the victim is a child and the first gasp was made within 20 minutes of extraction from the water the prognosis is excellent.[16 17] About half of all children who are pulled apparently lifeless from fresh water will survive,[5 84 85] and over two thirds will do so who are rescued from the surf.[86] Of all children who are rescued and who survive, only about 3% will exist in a vegetative state.[16] A further 2% develop chronic hypertonic quadriplegia and other neurological complications such as extrapyramidal signs and peripheral neuropathies.[14] Of all child survivors who appear to function normally, about one third have wide subscale discrepancies on formal psychometric testing (minimal cerebral dysfunction). Growth and hearing are not affected by this type of acute hypoxic insult. Sequential recovery is possible over the ensuing four to six months in those severely affected.[15 87]

Child victims who are still apnoeic with fixed dilated pupils when they reach the intensive care unit (usually one to two hours after hypoxia) do not have such a good prognosis. The further survival of this group of selected cases may be as low as 50%.[88] In a retrospective analysis of Conn's Toronto series of those not treated with hypothermia or barbiturate rescue the further mortality was one third, with one quarter of the original group showing serious permanent neurological sequelae.[46] The presence of dilated pupils in the intensive care unit is a bad sign as regards survival and neurological salvage.[13 46 89 90] The individual weighting of clinical prognostic indicators has not yet been undertaken scientifically, and the subject is topical (see appendix II).[1 14 18 65 73] I believe the best prognostic indicator is the time to the first gasp.[14]

Prevention

The management of near drowning includes taking steps to see that the accident does not recur[91]—as it does in some 5-8% of child victims. Drownings occur in high risk but definable groups,[8 92 93] their causes are understood,[23] and potential part remedies are straightforward—by education,[94] ergonomic improvements,[95] and legislation.

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Appendices

APPENDIX I

Hypoxic march in the pathophysiology of drowning

Sequence	Pathophysiology	Notes
(1) Involuntary submersion	Voluntary apnoea, tachycardia, hypertension, hypoxia, hypercarbia, acidosis	Diving reflex[96-98] occurs in infants and toddlers[14] (blood shunting, bradycardia) Voluntary apnoea is biphasic[99]
(2) "Breakpoint" reached, involuntary inspiration	Arterial hypoxaemia, tachycardia, tissue hypoxia, tissue acidosis	Breakpoint determined by both hypercarbic and hypoxic drives,[50 100] which are synergistic
(3) Water enters lungs	Increased peripheral airway resistance[101]	Laryngeal spasm occurs in 10-15%[49 102]
	Reflex pulmonary vessel vasoconstriction[98]	Pulmonary hypertension and shunts develop[103]
	Decreased lung compliance	"Stiff lung syndrome" leads to resuscitation difficulties[104]
	Fluid shifts occur across alveolar membrane	Water flux occurs in direction of alveolus to blood in both saltwater and freshwater drownings
	Surfactant diminished	By loss (salt water) or denaturation (fresh water)[102]
(4) Decompensation	Gasping occurs with further inhalation[105]	Froth and foam production in airways. Water flux continues
	Swallowing occurs with secondary emesis[106]	
	Secondary apnoea	
	Consciousness lost	
(5) Neuronal dysfunction	Electroencephalogram becomes flat	Different neurones show selective vulnerability
	Blood-brain barrier breaks down	
(6) Cardiac dysfunction	Bradycardia, arrhythmias, asystole[107]	Temperature modified
(7) Brain death[50]		Confounding influence of drugs, alcohol,[20] and hypothermia[24]
(8) Somatic death		Occurs within 1-60 minutes after submersion (median 3-10 minutes) dependent on age, water temperature, and degree of tissue hypoxia

APPENDIX II

Ten prognostic guides in early phase of management of the near drowned

Factor	Notes
(1) Site of drowning	Freshwater risks worst; surf best
(2) Water temperature	Cold water best
(3) Immersion time	Usually unknowable[16]; estimate by "bracket" method
(4) Time to first spontaneous respiratory gasp	Within 15-30 minutes after rescue <10% of survivors have mental retardation or spastic quadriplegia Within 60-120 minutes or later 50-80% of survivors show serious neurological sequelae
(5) Whether cardiopulmonary resuscitation administered, and whether given by trained operator	Some 30% of potential fatalities saved by skilled resuscitation at site of rescue
(6) Presence of coma on arrival in intensive care unit	Usually 1-2 hours pass after submersion before victim is in an intensive care unit, even in best retrieval systems
(7) Presence of fixed dilated pupils on arrival in intensive care unit	
(8) Intensive care therapy, especially positive end expiratory pressure ventilation	Role of barbiturate rescue, hypothermia, and control of intracranial pressure uncertain
(9) First measured arterial pH	Usually not undertaken in practice before 1-3 hours after rescue. Arterial pH below 7·0 is bad prognostic sign
(10) First measured arterial oxygen tension	Usually not measured before 1-3 hours after rescue. Pa_{O_2} below 8·0 kPa (60 mm Hg) in air is bad sign

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