WATER INHALATIONAL ACCIDENTS

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Resuscitation of the apparently drowned is not the prerogative of recent times only, the era of most effective development of cardio-pulmonary resuscitation. It was during the 18th century, particularly with the encouragement of the Royal Humane Society, that methods of resuscitation of the drowned were enthusiastically developed. The means used were often quite ingenious. Still, before the modern practice of expired-air breathing was resurrected, I suppose most members of the audience here can remember, have seen or even have taken part in those rescues which used to be reported in the newspapers as successful only after several hours heroic effort of Holger Nielsen artificial ventilation. My function today is to concentrate particularly on the sequelae to the accidental inhalation of water. That is I am concerned with not the drowned and dead, but rather the near-drowned, post-rescue; and for them, more particularly with intensive therapy for the life-threatening sequelae to the inhalation of water, than with on-the-spot emergency care.

Hospital Circular Letter No 1971/82 (1971)

Firstly, however, I wish to draw the attention of any New Zealand hospital doctors here to the following advice on resuscitation of the drowned sent out to all of our hospitals in 1971. I do not know if any members of the audience here had any hand in drawing up this circular letter; but I cannot see how any person actually doing the work with the critically ill near-drowned could have included so many features then out of date by 5 or 10 years. The errors arise from extrapolation of results from laboratory animals to the human (Table 1) (Miles, 1968). The undue emphasis on "Is it fresh water or is it salt water drowning?" is not warranted. And this opinion is now also being endorsed in the literature on near-drowning (Segarra, et al., 1974). The patient must be managed according to what he actually presents with at examination.

TABLE 1
NEAR-DROWNING

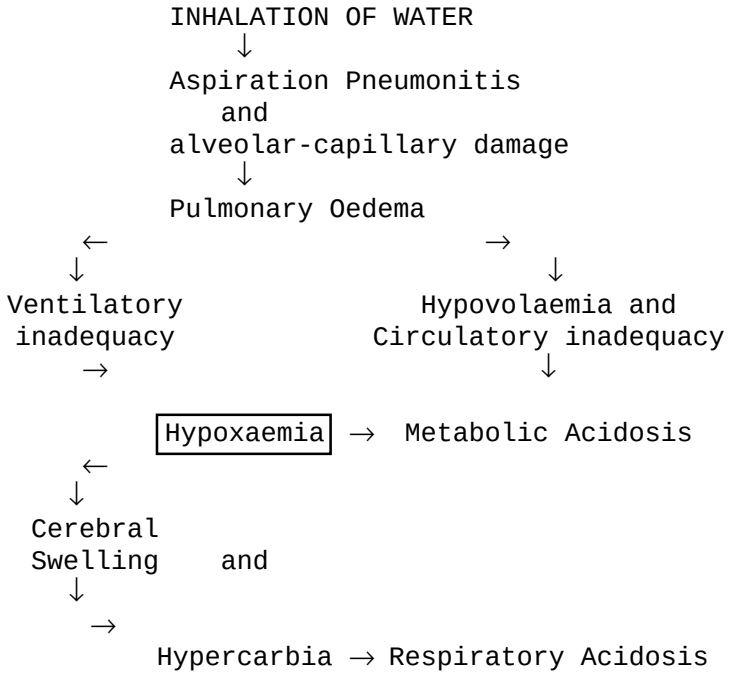
Feature	**in lab animal**	**Human**
Haemodilution and electrolyte disturbances	frequent	infrequent
Pulmonary oedema	often absent	common
Ventricular fibrillation	common	?
Vehicle	lab. dist. water or saline	water with particulate impurities

Miles, 1968. Brit. *Med. Jnl* 1968; III: 597-600

Pathophysiology

Both river fresh water and sea salt water can contain a large amount of particulate matter: diatoms or silt or sand and so on; but the “pure” water of a private pool is also damaging to the alveolo-capillary membranes. The essential lesion in near-drowning is consequent on the inhalation of the water plus its contents. Whether this water is fresh or salt it is so profoundly an irritant that it produces acute pulmonary injury. The essential reaction to the pulmonary injury is exudation of plasma-rich fluid into the alveoli, resulting also in blood volume depletion. We say aspiration pneumonitis is followed by pulmonary oedema and hypoxaemia. This leads to ventilatory and circulatory impairment with hypoxaemia and metabolic acidosis as the fundamental functional disturbances, resulting in respiratory and circulatory failure and perhaps neuro-logical damage(Figure 1).

FIGURE 1



The management of patients rescued from near-drowning can be described in terms of the typical intensive therapy patients. There is both an emergency and a definitive phase each of which has both assessment and treatment aspects. The life-threatening lesions require treatment phase and this may need to be instituted simultaneously with or as assessment is going on.

a. Emergency Management

1. Administering oxygen, should it be available when the victim has been brought ashore.

2. Making patent any obstructed airway. This does not mean wasting time emptying the water out of the patient because if it comes when the patient is tipped head down its source is usually the stomach, not the respiratory tree; but refers to extending the neck to lift the tongue from the posterior pharyngeal wall, or using the fingers to clear the mouth of solid vomitus.

3. Breathing inadequacy must be compensated for. If the patient is apnoeic then mouth-to-mouth expired-air ventilation may be all that is possible. If, for instance, a Life-Support team is available on the beach, then perhaps a self-inflating bag can be used. There is one really essential simple requirement to this manoeuvre, however, and that is visual observation to confirm that effective expansion of the chest does occur.

4. If the circulation is depressed this must be restored. Absent heart beat can be compensated for only by external cardiac massage with a firm surface beneath the patient. Again, a Life-Support unit may be able to supply adrenalin and counter-shock, or life-giving plasma to compensate for the volume depletion which has occurred.

One cardinal rule to resuscitation here is that it should be continued until normothermia is established - because even when the evidence of cardiac activity is absent cases have been reported where the heart has started again as the temperature has risen (Smith, et al., 1973).

b. Definitive Management

Victims recovered from water either are apparently recovered or they require attention.

i. patients apparently recovered should still be observed in a hospital because it has happened before that the onset of what is generally called “delayed drowning” can occur and yet the patient die some hours later following drastic deterioration.]

ii. By contrast patients in the other group are obviously ill. They require careful assessment to determine the degree of functional disturbance. The therapy they then require is available on a progressive scale and its particular level will be indicated from the severity of the lesion. The patient must be treated for what is actually found, and not to theoretical expectations of what can happen because it is “salt water drowning” or, because it is “fresh water drowning”.

Definitive Assessment

This has to be made of respiratory, circulatory, neurological and metabolic function.

1. Respiratory. Especially we look for cyanosis, breathing distress, the appearance of fine foaming, white or perhaps pink, at the nostrils or the mouth. Or chest pain, cough, or occasionally wheezing from bronchospasm. Radiological and blood gas assessment will also be available at the base hospital but careful clinical examination and especially determination of breathing distress is probably as useful as any other manoeuvre and does not involve unnecessary delay before instituting treatment.

2. Circulatory. This assessment can be made on examination of pulse and heart beat, and the adequacy of essential perfusion reflected in the pupils and function of the central nervous system. The typical arrhythmias are tachycardia, sometimes atrial fibrillation, premature beats or gallop rhythm. Hypotension may be present, with pallor and sweating, but occasionally hypertension. The best index of peripheral perfusion is, of course, urine formation which requires an indwelling catheter.

3. Neurological. The patient may show irritability or restlessness proceeding even to frank coma, or convulsions. This will be worsened by hypoxaemia and hypercarbia. Sometimes extensor rigidity or extensor spasms or pisthotonos also indicate central nervous system dysfunction. The secondary return of coma indicates a need for therapeutic intervention to treat brain swelling specifically.

4. Metabolic. Probably pH is the most valuable quantity to measure. In collected data on laboratory findings in cases of secondary drowning (Rivers, et al., 1970). The much vaunted sodium and potassium ion changes could not be predicted for individuals according to whether they were sea water or fresh water near-drowning cases. In our own experience, critically ill people generally seem to have a depression of serum potassium level. Electrolytes may need to be measured repeatedly, initially even every hour or two until balance has been restored. The effects of magnesium aspiration in sea water drowning is not as yet elucidated as far as I am aware. Temperature should be monitored, platelet and white cell counts performed but plasma haemoglobin levels would seem to be a fairly fruitless investigation.

Definitive Treatment

The principles of management of the near-drowned with intensive therapy are well established.

1. Respiratory requirements. These are met in the spontaneously breathing patient by administering enough oxygen to abolish hypoxaemia. This may require even a hyperbaric chamber at 2 atmospheres but if hypoxaemia is severe enough to warrant the latter then generally Intermittent Positive Pressure Ventilation with oxygen is indicated. During transportation this may be provided adequately with a self-inflating bag, but definitively it will require endotracheal intubation and ventilator therapy. Severe hypoxaemia is best treated, of course, with end-expiratory pressure, particularly if pulmonary oedema is present.

2. There may have been an immense plasma loss and then circulation will be restored only by volume repletion. The stomach may be full of water and this is best aspirated. Once blood volume has been restored maintenance fluid therapy will be required with electrolyte manipulation according to serial bio-chemical testing.

3. Acidosis. pH needs to be brought to a safe level, ie., between 7.20 and 7.25 by the administration of molar bicarbonate. This also means, however, the administration of molar sodium and in the presence of elevated serum sodium this will further increase osmolality.

4. Sequelae of cerebral hypoxaemia are seen rather more frequently, in our practice and I will describe two cases demonstrating management. The principles of a regimen of active intervention for acute brain swelling have been described previously (Trubuhovich and Spence, 1974; Trubuhovich, 1975).

5. Because of the infective nature of aspirated fluid, antibiotic cover on a prophylactic basis is part of our practice and ampicillin would be our antibiotic of first choice. The pneumonitis may or may not be helped by steroids but noting that evidence on this matter seems to be conflicting we would use such drugs.

Case No 1: A 5-year old boy was pulled out of the water at Great Barrier Island, and found to be stuporose and limp, pallid with cyanosis about the lips, and tachypnoeic at 40-50/minute with some grunting. By the time we reached him by Air

Ambulance from Auckland the tachypnoea was settled, his chest sounded “dry” and he seemed in good condition. Nevertheless, we brought him back to Auckland Hospital for overnight stay because of the slight chance of delayed complications developing. In hospital a breathing rate of 35-40/minutes, settled down in a couple of hours to 20/minutes, but his chest X-ray did show a surprising degree of pulmonic change indicative of interstitial oedema, considering the absence of clinical findings. Oedema had all cleared by next morning when he was sent home.

Case No 2: A 15-year old youth had his head held under water for an indeterminate time by the rigging when his yacht capsized. His companion boatsman applied mouth-to-mouth resuscitation in the water while bringing him all the half mile into the shore. There at 1420 hours, he would not tolerate the Minuteman from the ambulance team, but was given an oxygen mask for cyanosis. At the local hospital he was blue on arrival, not completely unconscious but unable to give any details and he pulled his oxygen mask away. A gastric lavage and a haemoglobin estimation were performed and frusemide (kidney stimulant) was given. When the patient’s breathing looked inadequate he was intubated (against his resistance) then despatched to our department still breathing spontaneously but cyanosed. When breathing further deteriorated during ambulance transportation ventilation with an Ambu bag was attempted. On arrival at the Department of Critical Care at 1540 hours, Ambu bag inflations were noticed to produce an air-escaping noise, and the patient was in black cyanosis with cardiac arrest and fixed dilated pupils. The endotracheal tube was in the oesophagus.

A satisfactory circulation was restored after about 10 minutes’ resuscitation during which the patient received adrenalin 1+1+1 mg, molar bicarbonate 300 mmol (and later 50 ml of Tham. 0.3 M), and plasma. The lungs were extremely non-compliant due to interstitial oedema and intra-alveolar fluid which frothed from the respiratory tree. Artificial ventilation with 100% oxygen, and specific treatment for post-hypoxic cerebral swelling, together with dexamethasone and ampicillin and cloxacillin were continued. By 1600 hours, the central venous blood revealed PO2 51 torr, PCO2 97 torr, pH 7.11, base excess 0; Na ion 161, K ion 3.2, Cl ion 112 mmol/l. Arterial lactate later was 5.7 mmol/l. The admitting hospital now reported haemoglobin at 15.0g/100ml. The occasional ectopic beats were treated with lignocaine, and satisfactory circulatory status was maintained by further administration, of plasma, to a total of 1750 ml by 2000 hours, then a further 1000 ml by midnight. Overnight the potassium requirement was 147 mmol to 0900 next morning.

On the day after admission a disconcerting white cell count of 1.0x103/cmm led to steroids being discontinued, and gentamicin added to the antibiotic regimen for gram negative cover. By the fourth day the patient’s stable pulmonary condition had started to worsen with progressive lung stillness, hypoxaemia and eventually hypercarbia, leading to bradycardia and cardiac arrest responsive only initially to resuscitation. The Coroner’s pathologist demonstrated (i) a very extensive haemorrhagic pneumonitis, which he described as characteristic of severe salt water drowning (see JAMA, 11 Sept 1967; 201: 209-211); and (ii) severe cerebral swelling.

The next two cases are presented to demonstrate that the neurological sequelae to near-drowning, hitherto ill-described, can be managed successfully by specific treatment.

Case No 3: A 16-year old Maori boy described as mentally retarded, was brought from the bottom of the Onehunga swimming pool at 1400 hours, cold and pulseless. After mouth-to-mouth resuscitation and oxygen in the ambulance, he was making only spontaneous breathing gasps by 1415 hours, but in a few minutes was hyperventilating, and had a strong pulse but was still cyanosed. He was in opisthotonus, with intermittent convulsive restlessness, unrelieved at the nearest hospital by diazepam

10 mg intravenous, then morphine 10 mg at 1430 hours. His chest X-ray indicated oedematous lungs so he was given frusemide 40 + 40 mg. The pupils were noted to be large and reacting sluggishly. Though breathing from an MC oxygen mask he seemed cyanosed and arterial blood gas analysis showed a PaO2 of 57 torr pH 7.23 and base deficit 13.5mmol/l. When I saw the patient there at 1520 hours he needed three orderlies and two sisters to hold him down from his "convulsive" restlessness; he was struggling to hyperventilate and was quite inaccessible but extremely irritable. His pupils were markedly dilated and reacting sluggishly. Administration of d-tubo curarine 45 + 15 mg intravenously enabled a little hand-ventilation then endotracheal intubation within a minute. Diazepam 20 mg provided sedation. Following hyperventilation with 100% oxygen the patient then became rapidly pink throughout. He was catheterised, and an intravenous line inserted for plasma administration when his systolic blood pressure dropped to 80 mm Hg. The heart rate was noted to be fluctuating inbursts from 80-120/min. He was then transferred by ambulance to Auckland Hospital on hand-ventilation with 100% oxygen. His problems were considered to be posthypoxic cerebral oedema and pulmonary oedema subsequent to fresh water near drowning.

In our department the patient was treated for acute brain swelling with hyperventilation with 100% oxygen after curarisation and full sedation; strict haemodynamic control, which required 3 units of plasma over the first 2 hours for a slight hypotensive tendency; hypothermia to 34°C, from 38.5°C on admission; and dexamethasone. Pulmonary oedema was treated by end-expiratory pressure of 8 cm wpg added to artificial ventilation. After about 30 minutes PaO_2 was 450 torr. PaCO2 44, pH 7.36, base deficit 0. The only initial problem at management was maintaining an adequate serum potassium level as it tended to be about 3.0 mmol/l. With the appropriate treatment he made a very rapid recovery from his pulmonary and cerebral oedemas and was satisfactory for transfer back to the other hospital on the fifth morning. His mental retardation was not apparently worsened by this episode.

Case 4: An 11 months infant was found at 1830 hours in a home swimming pool pallid and apnoeic but spontaneous breathing commenced only after 7 minutes of mouth-to-mouth resuscitation. When admitted via the Accident and Emergency Department of Princess Mary Hospital after 3 hours he was unconscious, barely responding to painful stimuli, but pupils were reacting to light. Management consisted of oxygen, antibiotics, intravenous fluids (but no dexamethasone). Blood pressure was 150/100 mm Hg, heart rate 140/minute and breathing rate 40/minute. The infant was initially inert with noisy breathing but later became responsive to painful stimuli, was irritable, restless and crying. At 0230 hours generalised fitting occurred for about 90 seconds and half an hour later the patient was given diazepam 2 mg and dexamethasone. He slept more deeply after this and gave a slight response to painful stimulation but following two further fits breathing was shallow although the child was said to rouse to painful stimuli "easily enough". After further fitting at 1000 hours next morning the patient was referred to our department, and when first seen was comatosed and generally flaccid with focal convulsions; cyanosed and vasoconstricted; and markedly hyponatraemic (Na ion 113 mmol/l at 0900 hours). Systolic blood pressure was 180 mmHg, heart rate 125 per minute, temperature 37.5°C. The infant was sedated, curarised, intubated and received constant hyperventilation with oxygen, anticonvulsants and dexamethasone were given, and 14 ml of molar bicarbonate solution raised the serum sodium level to 120 mmol/l by 1230 hours. The hyponatraemia further slowly improved over the next 2 days. Opiates were required occasionally for some haemodynamic instability.

On the morning after transfer an electroencephalogram was reported as very severely abnormal "even by Department of Critical Care standards". By the 9th day the patient seemed lighter neurologically though his cardiovascular system was still irregular.

A first trial of decurarisation that day was abandoned because of bizarre and persistent abnormal spontaneous movements but these were of a lesser degree 3 days later so decurarisation was proceeded with and the patient finally satisfactorily extubated on the 14th day after transfer. Consciousness rapidly improved but peripheral tone was markedly depressed, probably because of the large quantities of diazepam and sedating drugs he had required. He was transferred back to the paediatric ward and when seen two weeks later before leaving hospital was described by his mother as being perfectly normal and having acquired new skills and new words.

Summary

An account of near-drowning has been presented to emphasise the dysfunctional sequelae of hypoxaemia, hypovolaemia and acidosis - rather than rapid fluid shifts, electrolyte changes and ventricular fibrillation. Specific treatment can be applied to treat brain swelling subsequent to hypoxaemic cerebral insult.

References

Hospital Circular Letter. Treatment of Drowning. Hospital Circular Letter No *1971/82*.. Department of Health, Wellington; 19 May 1971.

Miles, S. Drowning. Brit M*ed Jnl* 1968; 3: 597-600.

Rivers JF, Orr G, Lee HA. Drowning - Its Clinical Sequelae and Management. Br*it Med Jnl* 1970, 2: 157-161.

Segarra F, Redding RA. (1974), Modern Concepts about Drowning. CMA Journal 1974, 110: 1057-1062.

Safar Peter (ed). Cardia-Pulmonar*y Resuscitation*. Norway, World Federation of Societies of Anaesthesiologists, 1968.

Smith EH., Pickering D. Near-drowning in Osford Children. Arc*h Dis Child*. 1973; 48: 655.

Trubuhovich RV, Spence M. *Scientific Abstracts, 1st World Congress of Intensive Care*. Glasgow: Bell and Bain, 1974: 85-86.

Trubuhovich RV. Conservative Management of Acute Brain Swelling. *Proc Univ of Otago Med School Centenary*. Dunedin: John McIndoi, 1975: 31.

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FOETAL DECOMPRESSION SICKNESS?

Tests on sheep have shown, by inference, that while it is probably safe for a pregnant woman to make a shallow dive it may not be safe for her baby if she makes deeper ones even though they are within the no-decompression limits of the US Navy tables. Experiments at the Texas A and M University's Sea Grant facility, under the supervision of physiologist William Fife, have involved monitoring of the umbilical arteries by implanted sensors when pregnant sheep were "dived" in a chamber to 30 metres for 25 minutes. Although such a dive profile does not require a human to make decompression stops on ascent (though careful people might include a stop), sheep foetuses were shown to have massive bubble flow of a degree likely to be fatal had not the sheep been recompressed promptly and then brought up at a slower rate. This work is to be reported in detail at a later date in Undersea Biomedical Research, but in the interim a warning is reasonable to women who propose to dive in even the earliest stages of pregnancy lest their babies, like those of the sheep, are more susceptible to bubble production than are they themselves. There are, however, no known instances of such damage occurring.

ADDENDUM: UPDATING FOR 1978

Emphases in management altered from the above regime which would be applied today to patients with post-ischaemic/hypoxic encephalopathy, eg. after a water accident, are:

1. Early loading of the patient with barbiturate to confer “cerebral protection”.

   The agent used for this is preferably thiopentone, to 30mg/kg in an hour.

   This may require circulatory support with vasopressor agents. See the Brain Resuscitation Symposium issue in *Critical Care Medicine* to be published in July, 1978, especially the paper by Breivik et al. which details some very interesting cases of resuscitation from near-drowning. See also the issue “Management of Acute Intracranial Disasters” in *International Anesthesiology Clinics*, Volume 16 to be published in early 1979 (Editor: RV Trubuhovich) for the general background.

2. Closer attention to management of serum osmolality in the presence of cerebral oedema.

   (a) A hyper-osmolar state is managed by the administration of near-isotonic saline to reduce serum osmolality gently at the rate of 10(-20) mmol/day.

   (b) A hypo-osmolar state is managed by fluid restriction, and possibly the use of mannitol 20% (0.5-1.0 g/kg) or even frusemide.

Severe disturbances of osmolality and inappropriate management of them can cause fatal deterioration in cerebral oedema.

RN Trubuhovich

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FOURTH ANNUAL CONFERENCE on the CLINICAL APPLICATION OF HYPERBARIC OXYGEN

The Fourth Annual Conference on the Clinical Application of Hyperbaric Oxygen is scheduled for Thursday through Saturday, 7-9 June 1979.

In addition to Plenary Sessions dealing with clinical applications of hyperbaric oxygen, nursing and technical applications, and original communications, a Plenary Session devoted to “What’s New in Diving Medicine” is added this year.

The first call for Abstracts (200 words or less) on original papers in hyperbaric medicine is made. The deadline for receipt of Abstracts is 15 January 1979. Abstracts should be sent to:

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