

ORIGINAL PAPERS

DROWNING SYNDROMES: THE MECHANISM

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Key Words

Accidents, deaths, drowning, incidents, near drowning, salt water aspiration

Abstract

The drowning syndromes should be viewed as a continuum between the aspiration of a relatively small amount of water, causing symptoms and respiratory-based signs, through near-drowning, in which there is loss of consciousness but with survival, to the fatal cases of drowning. The latter rarely involve the gross haemodynamic and biochemical changes seen in some animal experiments.

The behaviour of the victims, animal and human, during the incident is reviewed, as are the experiments conducted on animals, with various types and quantities of aspirate, to model the physiology. These experiments are compared with adult human clinical case series. "Quiet" drownings are described and classified. The clinical features of near drowning are reviewed. Factors which influence survival are noted.

The pathological findings are discussed, with a critical approach to the concept of "dry" drowning, and some postulates on the findings of cranial haemorrhages. Lungs are the primary and dominant organ involved and hypoxia is the major physiological abnormality.

The salt water aspiration syndrome, including its development, clinical and laboratory findings as seen in scuba divers, is also reviewed.

Finally a brief review of the literature specific to scuba drownings is given.

Introduction

Excellent general reviews on this subject have been presented by Donald, Modell, Tabelaing, Neuman and the UHMS Workshop on Drowning at Cancun, Mexico in June 1997.¹⁻⁶ This paper is the basis of one of the presentations at the UHMS Workshop.

Terminology

Drowning refers to the death of an air-breathing animal due to immersion in fluid.

Delayed drowning or *secondary drowning* occurs when the victim appears to recover from the incident, but then proceeds to die.

Near-drowning refers to the loss of consciousness from the incident, which did not lead to death.

The *aspiration syndrome* refers to the effects of aspiration of fluid into the lungs, but without loss of consciousness.

There is a continuum in the severity of symptoms and signs between aspiration, near-drowning and drowning. They can be incorporated together as *the drowning syndromes*, for a greater understanding of each. The continuum needs to be appreciated if a rational approach to the management of near-drowning is to be made.

Post-immersion syndromes refer to the disorders that develop after immersion and subsequent rescue.

Other classifications have been proposed, based on the type and amount of fluid inhaled. These will be referred to later.

Behaviour during drowning

Over the range of animals tested and observed, consciousness is usually lost within 3 minutes of total submersion.

Observation on experimental drownings showed the typical behaviour of *animals* was an immediate struggle for freedom, sometimes with an inhalation.⁷ This was followed by suspension of movement, possible exhalation of a little air and frequent swallowing. Later there was a violent struggle for freedom, followed by convulsive movements and the exhalation of air with spasmodic inspiratory efforts, then death.

Observations of *human* drownings parallel the animal experimentation, involving a panic reaction with violent struggling followed by automatic swimming movements.^{8,9} There may be a period of breath holding and swallowing of large amounts of water. Vomiting may occur, followed by gasping and aspiration of water. Blood stained froth develops in the airways and the patient convulses and then dies.

More recently the observations on children exposed to drown-proof training, as it is euphemistically called, has modified our knowledge of the normal response in human drownings.¹⁰ There is usually a failure of the infant to struggle. The child holds its breath and makes automatic

but ineffectual paddling type movements as the child sinks to the bottom. For many years, drowning was considered a "fight for survival",² but this is now changing.³

When a fully conscious human accidentally falls in the water, he or she fights to survive. In other circumstances drowning may proceed in a quiet and apparently unemotional manner. Examples of *quiet drownings* include:

- 1 Hyperventilation with breathhold diving. Craig reported eight cases of hyperventilation before breathhold diving, which resulted in loss of consciousness due to the development of hypoxia.¹¹ This developed before the blood carbon dioxide levels rose sufficiently to require surfacing to breathe. In these cases loss of consciousness occurred without any obvious warning and the underwater swimmer then aspirated and drowned.
- 2 Uncontrollable hyperventilation in cold water, hypothermia and/or cardiac arrhythmias, leading to loss of function and drowning have been well described by Keatinge and others.¹²⁻¹⁴
- 3 Drugs and alcohol increase the incidence of drowning by impairing judgment, increasing risk taking, vomiting, heat loss and reducing the physical and emotional capacity in the struggle to survive.^{3,15-17} It is likely that nitrogen narcosis may have a similar effect in divers.
- 4 Diving equipment problems may produce hypoxia. These include the dilution hypoxic effects with mixed gas breathing, ascent hypoxia and carbon monoxide toxicity by the blockage of oxygen metabolism. They are all likely to cause loss of consciousness without excess carbon dioxide accumulation, associated dyspnoea or distress.¹⁷
- 5 Salt water aspiration. In animals 2.2 ml of fresh water per kg body weight drops the PaO₂ to approximately 60 mm Hg within three minutes, or to 40 mm Hg with sea water.^{18,19} A similar situation was observed clinically in the salt water aspiration syndrome of divers²⁰ Exercise may exacerbate this situation.
- 6 Other causes of unconsciousness leading to drowning have been described e.g. some marine animal envenomations, coincidental medical illnesses, cerebral arterial gas embolism (CAGE) etc. These may suppress the psychological stress component of the drowning.

Animal experiments

In the 1930s many animal experiments were conducted, both in Europe and North America, demonstrating that if an animal was immersed and drowned

in water containing chemical traces or dyes, these would spread through the tracheo-bronchial tree to the alveoli surfaces.^{7,21,22} In the case of fresh water, this was also absorbed into the blood stream.²¹ A consistent fall in arterial oxygen content was observed, followed by a rise in arterial carbon dioxide and sometimes ventricular fibrillation.^{23,24}

Swann and his colleagues from Texas, in a series of accurate but misleading experiments,^{25,26} flooded animals' lungs with fresh or salt water and demonstrated significant differences between the two, due to osmotic pressures. In both cases, flooding of the lungs produced a reduction in PaO₂ and pH, with a rise in the PaCO₂. This was attributed to airway obstruction.

Because fresh water is osmotically much weaker than blood, it moved into the bloodstream and produced haemodilution, reducing the concentration of most of the blood contents including proteins, sodium, chloride, etc. The subsequent reduction in the osmotic pressure of the blood resulted in haemolysis and liberation of both haemoglobin and potassium, with various metabolic and renal complications. Deaths were often cardiac in nature and due to ventricular fibrillation.

However, when the animals' lungs were flooded with sea water, which has a higher osmotic concentration than blood, water was drawn from the bloodstream into the lungs producing pulmonary oedema and haemoconcentration. This caused an increase in the haematocrit, blood proteins and electrolytes.

For many years physicians attempted to correct these presumed electrolyte, metabolic and cardiac abnormalities in human drownings, but their cases did not seem to conform to this animal model. Moreover, earlier workers had shown that in dogs that drowned there was still large volumes of air in the lungs,⁷ as there is in humans. The Texan model had water-filled lungs.

Colebatch and Halmagyi, working in Australia in 1961, produced an animal model of drowning, more relevant as regards clinical management of patients, by aspiration of only 1-3 ml/kg body weight.²⁷⁻³² By using these smaller volumes they demonstrated the dominance of arterial hypoxia, largely independent of the amount of fluid inhaled. Pulmonary hypertension, vagal inhibition and reduced compliance were also noted. Sea water aspiration was usually associated with significant pulmonary oedema but the fresh water was often absorbed from the lungs within 2 to 3 minutes. Lung surfactant is affected differently by fresh water, which appears to destroy the surfactant, and salt water, which dilutes and washes it out.³³

Subsequent animal experiments, using intermediate volumes of aspirate, by Modell and others, verified that shunting of blood, the perfusion of blood through non-

ventilated areas of lung, was the predominant factor causing persistent arterial hypoxaemia.^{18,19,33}

Human series

Clinical series, described by Fuller in 1963,^{34,35} Griffin in 1966³⁶ and Modell et al. in 1976,⁴ illustrated the considerable differences between near-drowning humans and animals with flooded lungs. Human cases did, however, reflect the animal experiments in which smaller amounts of aspirate were administered. Significant electrolyte disturbances and cardiac arrhythmias were not frequent.

The initial symptoms were respiratory, then came the effects of hypoxia causing subsequent pulmonary and cerebral damage. Aspiration of vomitus, following the ingestion of sea water, was frequent in both the early and the resuscitation phases.

Investigations reveal: Hypoxaemia; a variable arterial CO₂; acidosis; the effects of reduced pulmonary compliance; patchy and variable consolidation in lung X-rays; and a polymorphonuclear leucocytosis. Others changes depend on other organ involvement.

Excellent reports of series of paediatric drownings have been published¹⁰ and much work on the relevance of hypothermia to drowning has been reported. Neither will be dealt with here.

Survival from near-drowning

In human drownings, deterioration after initial resuscitation is frequently recorded and this influences management. The likely causes for delayed deaths include extensive and progressive lung damage, cerebral hypoxia, secondary infections (usually of the lungs), renal failure and iatrogenic factors.

Factors which negatively influence survival have been well documented by Modell.³

- 1 Prolonged immersion
- 2 Delay in effective cardio-pulmonary resuscitation
- 3 Severe metabolic acidosis (pH <7.1)
- 4 Asystole on admission to hospital
- 5 Fixed dilated pupils
- 6 A low Glasgow coma score (<5).

Nevertheless, none of these predictors is infallible and survival with normal cerebral function has been reported with all of the above factors. Even a flat EEG may be reversed.

Cases have been reported that have been submerged for between 15 and 45 minutes,³⁷⁻⁴³ and have survived without neurological sequelae. There have been many other cases that have not been reported. Two such cases with which I have been involved were submerged for 15-20 minutes. Such cases have been used to encourage rescuers to persevere with resuscitation efforts.

The explanations offered for survival after such prolonged submersion are :

- 1 Hypothermia is protective and develops very rapidly with aspiration of water.^{10,12,41-43} In swimmers and divers hypothermia may well develop before the incident.
- 2 The "diving reflex" is a possible, but contentious, explanation.^{10,43} Within seconds of submersion the diving reflex (reflex inhibition of the respiratory centre in the medulla) may be triggered by sensory stimulation of the trigeminal nerve. This results in a bradycardia and shunting of the blood to the brain and coronary circulations. It is independent of baroreceptor or chemoreceptor inputs. The diving reflex is more intense in the frightened or startled animal, compared with those which dive or submerge voluntarily, but it is not known whether this is applicable to humans. Typically water temperatures above 20° do not inhibit the diving reflex, but progressively lower temperatures augment it.
- 3 Respiratory gas exchange in the lungs can continue after submersion. With or without the effects of laryngospasm, often some litres of air remain within the lungs, allowing for exchange of oxygen and carbon dioxide. Whether fluid enters the lungs in an unconscious victim will depend on many factors, including the orientation of the body. The nose and mouth being below the lungs will reduce the chances of fluid replacing air in the lungs. Increased pressure (depth) might increase the availability of oxygen uptake through Henry's Law. In a comatose state, with low oxygen utilisation and the effects of hypothermia and the diving reflex, a retained respiratory gas volume might add considerably to the survival time, although this is not often mentioned in the drowning literature.
- 4 Gas exchange between the pulmonary blood and the aspirated fluid might have a marginal effect on prolonging life.

Despite the fact that spectacular and successful rescue can be achieved after prolonged submersion, it is more frequent that this is not so, many victims lose consciousness and die after only a few minutes of submersion.

Pathology^{10,44-46}

Drowning is the commonest cause of recreational scuba deaths, but is usually a secondary effect, with the primary cause leading to loss of consciousness.¹⁷ Other accidents (dysbaric, medical illnesses, trauma etc.), occurring whilst immersed or submerged, are likely to result in the secondary complication of drowning, with all its pathological sequelae. Drowning often complicates the interpretation of the diving accident and produces a combined pathology.

Various aspects of drowning pathology need to be addressed.

“Dry drowning”

Many references have been made to the possibility of drowning without any aspiration of fluid. It was stated by Cot in 1931 that 10% of victims of drowning do not aspirate water.⁴⁷ They died from acute asphyxia while submerged, this is now attributed to reflex laryngospasm.

Virtually every review of drowning over the rest of this century refers, without question, to this belief and the incidence is often extended to 20%. “Dry drowning” conflicts with the animal work of the 1930s, but was given support by Pearn in a fascinating philosophical review of pathophysiology, in 1985.¹⁰ Pearn, who is an eminent paediatrician, admitted that it is not so frequent in children’s drowning.

In 1993 a recent review by Modell reiterated the concept.³ Three references were given. Two refer to autopsy findings,^{16,44} the third to one of Modell’s own papers,⁴ however perusal of that paper reveals little supporting evidence. He stated that by the time 81 near-drowning/aspiration victims had reached hospital, 10 had a PaO₂ of 80 mm Hg or greater. No information was available as to whether those cases were fresh or salt water victims, but the majority were in fresh water.

As it has been stated earlier, fresh water is absorbed very rapidly from the lungs, and therefore autopsy findings, and indeed the PaO₂, cannot really be used to imply (let alone prove) the absence of an aspirant. Especially is this so when these investigations are performed some time after the event. So, there is good reason to question the literature on “dry drowning”.

Reference to the anaesthetic literature is also informative. Miller’s textbook of Anaesthesia,⁴⁸ with its associated references,⁴⁹⁻⁵¹ define and describe laryngospasm as the exaggerated and prolonged response of a protective glottic closure reflex. There is no airflow and the true vocal cords cannot be seen. The development of hypoxia and hypercarbia remove the effect. Thus

laryngospasm eventually ceases spontaneously as hypoxia and hypercarbia develop. This implies that laryngospasm will not, by itself, be continued until death. The glottic closure will relax prior to death, allowing the passage of gases and fluids into the lungs.

In the absence of more definite information, it would probably be sensible to presume that all near-drowning or drowning victims have aspirated and base one’s first aid and management on this presumption. “Dry drowning” could well be an artefact of fluid absorption from the lungs, or death from other causes. I have never witnessed it in divers who drowned in salt water.

Autopsy observations^{10,44-46}

The theory and the practice of “drowning” autopsies are surprisingly contentious for such a common disorder. Frequently there are coincidental signs of immersion, marine animal injury or resuscitation damage. The stomach may contain swallowed fluid.

Autopsy examination of 118 consecutive drowning cases suggested that 85% aspirate 22 ml of fluid, or less, per kg body weight.⁴⁵ It was therefore considered unlikely that drowning victims die acutely of electrolyte imbalance and/or ventricular fibrillation. Death is more likely to be secondary to asphyxia and hypoxia.

The respiratory findings are congested, voluminous lungs and, in the airways; frothy, haemorrhagic sputum (especially in salt water cases), vomitus, foreign bodies and particulate matter. Respiratory infections, abscesses etc. are not infrequent if death is delayed. Otherwise there is little typical to describe macroscopically.

The pulmonary changes at autopsy reflect not only the pathology of drowning, but the effects of resuscitation and the changes in the lung fluids between the time of rescue and the time of death. Many such factors may influence the final macroscopic result.

Histological changes may demonstrate toxic effects both of chemicals and the specific aspirate. The surfactant changes, including denaturation, can progress even after apparent clinical improvement. The usual epithelial and endothelial changes, with detachment of the basilar membrane and cellular disruption have been described.¹⁰ Usually death is due to progressive, or irreversible, pulmonary damage associated with the drowning per se and there are obvious reasons for this. They include progressive surfactant damage despite rescue, pneumonitis from the aspirate or vomitus, infections etc. Pulmonary oxygen toxicity, associated with resuscitation attempts, may also be present.

The major effects on the neurological system are those of hypoxic brain damage and subsequent cerebral oedema with raised intracranial pressure. Delayed drowning deaths follow damage to lungs, brain or kidneys.

Neither the Gettler chloride tests nor the specific gravity of serum can be relied upon to establish a diagnosis of death by drowning.

Identification and comparison of environmental and systemic diatoms has been recommended, but is complex and infrequently performed. Also it does not prove drowning, merely aspiration of water while the circulation is still functional.

Conventional pathology teaching claimed that mastoid and middle ear haemorrhages were indicative of drowning. As we explained in 1976 these haemorrhages are the sequel of barotrauma, not drowning.⁴⁶ The ready acceptance of this explanation by diving pathologists took us by surprise and I have decided to extend the concept a little further.

Autopsies on drowning cases who have submerged while still alive, although unconscious, may show other cranial haemorrhages which are sometimes interpreted as a cause of the accident. Meningeal haemorrhages, both dural and arachnoid, are frequently observed. These are usually not very extensive and are quite different to the brain haemorrhages of arterial gas embolism or decompression sickness. They are probably derived from the haemorrhages of descent sinus barotrauma, which ruptured into the cranial cavity when the enclosed and compressed gas expanded as the body surfaced.

Salt water aspiration syndrome

A common diving illness in the Royal Australian Navy (RAN) in the 1960s was the salt water aspiration syndrome.^{17,20} It is called "salt water fever" by the Australian abalone divers. Its importance lay not only in the light it shed on near drowning cases, but also because it was often confused with other diving or infectious diseases.

This condition, which is due to the aspiration of small amounts of salt water during diving, may occur because of inexperience, during buddy-breathing training or due to a faulty regulator. At that time RAN regulators did not have purge valves. Novices were trained in buddy-breathing during their first dive, in the open ocean. This frequently led to aspiration of sea water. In other cases the aspiration occurred on the surface, after the diver had removed his regulator.

Experienced divers often developed it after a fast towed search, while abalone divers often used inadequate surface supply equipment and "leaky regulators". Their term

"salt water fever" indicated that they were well aware of the cause.

Other water users to present with a similar disorder, but possibly not as frequently, include snorkellers, surfers and helicopter rescuees.

A prospective survey was carried out on 30 consecutive cases who presented for treatment, their symptomatology was documented and investigations performed. Subsequently, "volunteers" were encouraged to aspirate sea water through doctored demand valves. The clinical and laboratory manifestations they developed were consistent with those in the clinical series. The following observations were made on the clinical cases.

IMMEDIATE SYMPTOMS

On specific interrogation a history of aspiration was given in 27 (90%). Often this was not causally associated by the novice diver with the subsequent events. Over 90% noted an immediate post-dive cough, with or without sputum. It was usually suppressed during the dive. Only in the more serious cases was the sputum bloodstained, frothy and copious.

SUBSEQUENT SYMPTOMS

The wide range of symptoms included rigors, tremors or shivering; anorexia, nausea or vomiting; hot or cold sensations; dyspnoea; cough; sputum; headaches; malaise; and generalised aches. Table 1 gives the incidence of each symptom

TABLE 1
SYMPTOMS OF SALT WATER ASPIRATION

Symptoms	%
Rigors, tremors or shivering	87
Anorexia, nausea or vomiting	80
Hot or cold sensations	77
Dyspnoea	73
Cough	67
Sputum	67
Headaches	67
Malaise	53
Generalised aches	33

RESPIRATORY SYMPTOMS

There was often a period of 1-2 hours before dyspnoea, cough, sputum and retrosternal discomfort on inspiration developed. In the mild cases, respiratory symptoms persisted for only an hour or so while in the more severe cases they continued for days. The respiratory rate

roughly paralleled the degree of dyspnoea. Respiratory stimulants appeared to aggravate the dyspnoea and tachypnoea.

Physical examination of the chest in about half the cases revealed crepitations or occasional rhonchi, either generalised or local. Rarely, they were high pitched and similar to that of obstructive airways disease. Signs usually disappeared within the first 24 hours.

Administration of 100% oxygen was reliably effective in relieving respiratory symptoms and removing cyanosis when this was present.

X-ray of the chest revealed areas of patchy consolidation or a definite increase in respiratory markings in about half the cases. These usually cleared within 24 hours, but remained longer in severely affected cases. X-rays taken after the incident and repeated within a few hours sometimes showed a variation of the site of the radiological abnormality.

Expiratory spirometry performed repeatedly over the first six hours showed an average drop of 0.7 litres in both FEV₁ and FVC measurements. These usually reverted to baseline levels soon after this time, although the changes could persist in a lesser form for up to 24 hours. Even those patients who had no respiratory symptoms demonstrated a reduction in lung volumes. Arterial blood gases, when performed, revealed oxygen tensions of 40-75 mm Hg with low or normal carbon dioxide tensions.

GENERALISED SYMPTOMS

Patients complained of being feverish, some with rigors, in most cases. Rigors were usually some hours after the aspiration of sea water. Malaise was the next most prominent feature. Headaches and generalised aches through the limbs, abdomen, back and chest were important in some cases, but usually not dominant. Anorexia was common and unexpected in this group of healthy young men.

In some there was an impairment of consciousness, including a transitory mild confusion (three cases), syncope with loss of consciousness on standing (two cases).

The feverish symptoms were interesting and are also seen in near-drowning cases. Shivering, similar in some cases to a rigor, and in some cases to generalised fasciculation, was a characteristic feature in the colder months. It was precipitated or aggravated by exposure to cold, exercise or breathing 10% oxygen (a research procedure, not recommended clinically). It was relieved by administration of 100% oxygen. It occurred especially in those exposed to cold because of duration and depth of dive, clothing worn, and environmental conditions during the dive and subsequently. The association of shivering with hypoxia and cold has been described by others.⁵² The

shivering occurs concurrently with the pyrexia, which also takes an hour or two to develop.

Pyrexia was able to be verified in half the cases, up to 40°C (mean 38.1°C, SD 0.6), and the pulse rate was elevated (mean 102 per minute, SD 21), over the first six hours.

Some patients realised that relief from these symptoms could be obtained by either hot water baths or showers, or lying still in a very warm bed.

These systemic signs and symptoms also usually reverted to normal within six hours, and rarely persisted beyond 24 hours, unless the case was of considerable severity.

Haemoglobin, haematocrit, ESR and electrolytes remained normal. The white cell count was usually normal, although a mild leucocytosis (not in excess of 20,000 per cu mm) was noted in a few cases, with a moderate polymorphonuclear increase and a shift to the left.

Lactic dehydrogenase estimations revealed a mild rise in some cases. X-ray and lung volume changes are described above.

A subsequent investigation into the causes of recreational scuba diving deaths^{53,54} revealed that water aspiration was part of the sequence leading to death in 37% of the cases, often a consequence of equipment or technique problems. In these cases "leaking regulators" were often observed and commented on by the victim beforehand or demonstrated during the diving equipment investigation. Although there was often a fault in the actual regulator, with a failure of valve seating, the degree of leaking was frequently demonstrated to increase with the volume of air being required (e.g. with exertion, swimming against currents, panic etc.) and/or with a diminished line pressure to the second stage. Salt water aspiration often formed a vicious circle with panic and exhaustion.

Hypoxia from salt water aspiration, as could be expected, aggravated the problems of fatigue and exhaustion, and was a precursor to loss of consciousness (with or without dyspnoea) in both near drowning and drowning cases.

Discussion

There is no distinct division, in the initial presentation, between aspiration, near drowning and drowning. Aspiration syndromes merge with near drowning, often the intensity of symptoms and the degree of consciousness depend on circumstances, the activity of the victim and whether oxygen is being administered.

Near drowning cases sometimes die hours, or even days, later. They are then re-classified as delayed or secondary drowning. Some of the apparently drowned victims, by virtue of adequate CPR and enthusiastic intensive care management, surprisingly recover without sequelae.

In a prelude to the 1997 UHMS Workshop on Drowning and Diving, the Chairman made the following statement: "As you know, the drowning literature ignores diving whilst the diving literature ignores drowning."⁵⁵

It is paradoxical that drowning, which causes more than 80 times the number of deaths in recreational divers than either decompression sickness or contaminated air, does not rate more than a paragraph or two in some diving medical texts. Nevertheless, of the major seminal reviews presented on this subject, many have been by diving physicians.^{1,5,6}

In reviewing the literature on drowning, the only papers that I could find that specifically relate any of the drowning syndromes to scuba diving, was my own one on the salt water aspiration syndrome,²⁰ and one with an anecdotal review followed by a case report.⁵⁶

One can only assume that it is too common a disorder to excite much academic interest.

In general, apart from the treatment of the near drowned, which is frequently reviewed, very little critical thought or assessment has been directed towards the literature that is available and most reviews are merely a rehash of previous presentations. Even more uncommon is the addressing of the problem of drowning syndromes with scuba, either death or accidents.⁵⁷

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**ROYAL AUSTRALIAN NAVY
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