

decompression sickness, air embolism, nitrogen narcosis or contaminated air poisoning!

The only part of the survey which gave me some glimmer of hope was the finding that 78.3% considered that more training in diving medicine was needed.

For years I have been making excuses for my MM colleagues, when talking to diving instructors. I stress that they are very busy people, that diving medicine is a negligible part of their training and that it is unfair to expect more expertise.

Well, no longer. By condoning MM medicals, I am not doing a service to my conscientious medical colleagues, to my fellow diving instructors, or to the innocents who undertake diver training with a trust in the professionalism of their advisers.

I am an aviation medical examiner and the skill I need in that capacity is infinitesimal compared to a diving medical examination. Yet, for aviation medicals I have to be a qualified aviation medical examiner. That means I must have some form of training, or at least have demonstrated a knowledge in that field.

Why should an untrained and unqualified physician (untrained and unqualified in this speciality) be permitted to do diving medicals when he has no concept of the physics or physiology of the diving environment or of the demands of this environment, or the equipment the candidate must use to sustain life? Why do they presume such knowledge? I would not be so presumptuous to advise on a gynaecological condition, or do a dermal biopsy, even if I did need the money.

In Australia there is adequate training available. We have a four week course at the Navy base especially designed to introduce physicians to diving medicine. Yet many physicians will reject this training, but perform the medical examinations. There must be a reason. The obvious ones are as follows:-

1. Financial reward.
2. The examiner does not recognise his limitations.
3. Lack of knowledge.

The phone calls that I receive suggests a combination of all three, and the results of the survey lend support to this proposition.

The commercial factor could certainly be significant in Australia. Our Department of Health, although it does not admit to this, allows general medical practitioners to claim a rebate for the medicals performed on divers. It does not permit this practice among specialists in this field, thereby encouraging a minimal and inadequate standard of medical examination.

I think there is a conspiracy between the MM medico and the MM dive instructor (and me until very recently) to pretend that the diving candidate is being well served. I feel that the dive instructors are often more hypocritical than the physicians. At least in many cases, the physicians really do not have the knowledge to start with. Most diving instructors are trained and knowledgeable, and are aware of the problems and complications associated with many of the medical diseases.

Some of the instructor organisations have produced their own MM medical forms which allow any doctor to

pretend a knowledge, simply by ticking a few boxes. Perusal of these forms usually reveals that they would be barely adequate even for snorkellers, let alone scuba divers.

In my opinion a good and conscientious instructor will not only advise the candidate to have a comprehensive diving medical, but he will insist upon it. The easier option is to let any doctor tick the form and thereby exonerate him legally for training unfit divers.

The chances of a candidate being failed on a MM medical or having any diving restrictions imposed, is minimal. Thus, more profit and less hassle to the instructor.

I am certainly not blameless. I have known of this practice for many years. Perhaps I was not prepared to look at it squarely until I read the results of the Queensland survey. Nevertheless, the information was there, with almost every phone call from a prospective diving medical examiner.

I do not know what is going to happen in other countries, but in Australia it is about time we put our house in order.

I do not think this article will endear me to many of my colleagues, either dive instructors or doctors. Nevertheless, it had to be said, and perhaps even a few of the young kids coming through may benefit from the mud that flies afterwards.

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Readers can find the names and addresses of those members of SPUMS who have done at least the introductory course at the RAN School of Underwater Medicine, or an equivalent course elsewhere, on pages 33 and 34 of this issue.

## IMMERSION HYPOXIA

### PULMONARY AND CEREBRAL CHANGES IN THE SUBMERGED VICTIM

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Underwater accidents can happen at any time, but with the appropriate training and skills possessed by the experienced diver, fatalities should be rare. Management of the near-drowned demands a mix of two themes, the adherence to a rescue and resuscitation drill, and an appreciation that no two incidents will be the same. In the latter instance, the variables of water temperature, degree of hypothermia, hypoxic time, age, and background health status all differ in a particular patient in a particular accident.

Management of the near-drowned is optimal if the medical attendant is familiar with the pathophysiology of drowning,<sup>1</sup> and with the possible events in the lungs and brain of the near-drowned<sup>2</sup> during cardiopulmonary resuscitation (CPR).

#### Inhaled Water

In "wet" drownings (perhaps 90 per cent of fatalities) death results from inhalation of water into the alveoli.

Water is a mixture of inorganic substances, gases, dissolved organic matter, and particulate matter, including bacteria, fungi, diatoms, zooplankton, and phytoplankton.

Sea water contains a mixture of salts, of which total concentration varies from place to place but of which proportions remain constant. Salinity is defined as the total mass of solids (in grams) in solution in one kilogram of water and is expressed as parts per thousand (ppt) or gm/kg. In oceanic sea water, a typical salt-ion profile is as follows: sodium 10.5 gm/kg; magnesium 1.3 gm/kg; calcium 0.4 gm/kg; chloride 18.9 gm/kg; and sulfate 2.6 gm/kg. Oceanic sea water thus typically contains 34.48 gm/kg of dissolved salts, of which 29.54 gm/kg is sodium chloride. Sea water contains 2.9 per cent sodium chloride compared with 0.87 per cent sodium chloride for human plasma.

Fresh water contains variable amounts of organic material, dissolved salts, and free and nascent gases. Fresh water in the lungs causes much greater loss of pulmonary surfactant than occurs with salt water near-drowning. In experimental studies using dogs, the test subjects survive total immersion marginally longer if the drowning medium is salt water (or even normal saline) than if it is fresh water, but the reasons are unclear.

One of the complications of drowning is that there are several pathways which may lead to brain death, which is the potential end result. The first link in the chain leading to death may be cerebral hypoxia, carbon dioxide narcosis, laryngeal spasm, vagal cardiac inhibition, or ventricular fibrillation. In a pedantic sense, some investigators define "true drowning" as death due to the aspiration of fluid into the air passages; and death due to laryngeal spasm or vagal cardiac effects might be more correctly termed "death from submersion" rather than drowning.

Irrespective of the osmolality of the inhaled water, it is now known that relatively large volumes of water (either salt, or fresh) pass across the alveolar-capillary interface. The Dutch scientist, den Otter showed that the pulmonary vein runs almost water-clear in the few minutes after water is inhaled into the lungs of experimental mammals.<sup>3</sup>

In diving accidents, it may be that only small amounts of water inhaled, which set up major problems in the lungs, initiating a chain of events which lead on to potential disaster. As little as 1 ml/kg of fresh water instilled into the trachea causes gross physiologic responses in the lungs. In studies using a simulated aspiration of 11 ml/kg of fresh water or less, gross lung changes result. However, the resulting increase in blood volume is not greater than the capacity of the heart and kidney to compensate for this potential fluid overload. The heart compensates by using the basic reflexes described by Starling's law, the Bainbridge reflex and the Anrep effect.

Although CPR drills are the same, in "dry drowning" (perhaps 10-20 per cent of cases) the lungs are probably more elastic (less reduction of compliance), at least in the first minutes of a successful rescue. There is now good experimental evidence (from the Canadian wildlife authorities) to support the concept of "dry drowning".<sup>4</sup> In experimental drowning of minks, muskrats and beavers, it was shown that not all animals that are trapped fully submerged and "drown" actually inhale significant volumes of water. When dry

drowning occurs, it appears that when a small amount of water enters the larynx or trachea, immediate laryngeal spasm (as a vagal reflex) occurs. An immediate out-pouring of thick mucus occurs, foam and froth develop and physically obstruct the airway.

## Diving Reflex

The facility with which diving birds and cetaceans can dive for long periods has intrigued physiologists for over 100 years.<sup>5</sup> Dr Brett Gooden (of Adelaide) an acknowledged authority on the diving reflex, notes that the Weddell seal is capable of swimming under ice for periods of one hour on a single breath of air. The protective diving reflex is triggered centrally, and by immersing the face in water (even wetting the face in some cases), the subject in effect becomes a transient "heart-brain preparation".<sup>5</sup> In childhood near-drownings, the diving reflex still operates, and its effects can be demonstrated in human adult volunteers. The submerged body manifests bradycardia and shunting of blood from the cutaneous and splanchnic vascular beds to the cerebral and coronary circulation. Blood pressure starts to rise immediately. These reflexes are independent of baroreceptor and chemoreceptor input, but depend on both sensory afferents in the trigeminal nerve and reflexive voluntary inhibition of the medullary respiratory centres. Water temperatures above 20°C (the most common temperature in the case of real-life drownings) do not influence the brain-protecting diving reflex, but progressively lower temperatures augment it. The hypothermic brain-sparing effects of near-drowning in cold water may be mediated through an augmented diving reflex as much as through physical chilling of the body core.

## Lung Changes

Two Australians, Colebatch and Halmagyi, have done much to clarify the lung changes in near-drowning.<sup>8</sup> After water enters the lungs, rapid changes occur. A series of reflexes are triggered which cause practical consequences for the resuscitation and the intensive care expert (and of course no less for the victim). Vagal efferents cause obstruction to peripheral airways. Aspiration of even very small amounts of fresh water (1 ml/kg in experimental animals) is followed by pulmonary vasoconstriction with the immediate development of pulmonary hypertension due to a parasympathetic reflex. This latter phenomenon is seen particularly following aspiration of fresh water into the mammalian lung and, to a lesser extent, in the case of sea-water aspiration but does not occur with aspiration of amniotic fluid.

Sea water, and water containing sodium chloride concentrations that are approximately iso-osmolar with plasma do not denature pulmonary surfactant but may dilute it or wash it out. Fresh water or significantly hypo-osmolar fluid aspiration causes marked change in surfactant activity.

With the aspiration of larger volumes of water (eg. 2.5 ml/kg), intrapulmonary reflexes cause shunting of blood through non-ventilated areas. This shunt (vagus-mediated), combined with surfactant loss or inactivation and with alveolar collapse, causes a significant reduction in mechanical compliance, a phenomenon that further compounds and adds to the effective right-to-left shunt. A mechanical blockage of the bronchial tree is caused by a combination of bronchial spasm, changes in the elastic properties of

lung tissue, and increases in the non-elastic resistance due to physical blockage of the airways by fluid and foam.

The intrapulmonary shunt (ie. percentage of cardiac output that passes through perfused but unventilated lungs and is therefore unoxygenated) increases dramatically after the aspiration of water. It increases from the normal range of 5 to 18 per cent to levels approaching 75 per cent within minutes of fresh-water inhalation. Clinical studies in humans have shown that the shunt takes at least several days to revert fully to pre-accident levels, even although the victim appears normal.

The water flux occurs across the alveolar epithelium, through a basement membrane and finally through the endothelial capillary lining into the capillary lumen where hemodilution occurs. This flux causes rapid and severe distortion of pulmonary ultrastructure. Histologic studies show that cells which produce surfactant are damaged. If the victim is successfully resuscitated, this loss (or denaturation) of surfactant can lead to "secondary drowning".<sup>7</sup>

There is no question that the phenomenon of "secondary drowning" is one of the most feared complications which can occur. It always seems terribly unfair when this complication develops in a rescued victim who appears to be doing so well for the first few hours after rescue. The condition is potentially fatal unless anticipated and treated. Symptoms vary considerably. In the conscious patient the development of tachypnoea, the inability to take a deep breath, a burning retrosternal pain, pleuritic pain with a hoarse rasping cough, and expectoration of increasing amounts of frothy, pink sputum, herald the presence of this condition. Cyanosis may develop if it is not already present, and respiratory failure may approach rapidly. The single most important sign, and one of the very serious prognostic concern indeed, is the return of coma in a near-drowned patient who has had a lucid period. Before the full implications of secondary drowning were realised, this complication had a uniformly fatal outcome.

### Brain Hypoxia

Obviously, the most important link in the drowning chain,<sup>8</sup> is that of brain death. Brain death may occur either before or after cardiac asystole. There are four links in the final sequence of brain death in immersion accidents:

1. State of Decreased Neuronal Metabolism  
After consciousness is lost a short period of reversibility of the altered neuronal metabolism occurs, before intracellular hypoxia and acidosis cause permanent damage. Not all neurones are equally vulnerable, and those associated with more subtle forms of intellectual activity are affected first. The electroencephalogram (EEG) becomes flat during this stage but this condition is reversible. Oxygen depletion arrests neuronal activity, and it is probable that the depletion of oxygen stores progresses more rapidly and is more responsible for these early changes than the effect of the accumulation of potentially lethal quantities of hydrogen ion. It is also probable that the order of deleterious influence is hypoxia, acidosis, and hypercarbia.
2. Vasogenic Stage  
This stage is a short period during which

extravasation of large molecules from cerebral vessels occurs. The blood-brain barrier breaks down, with both fluid and macromolecules passing into the parenchyma.

3. Stage of Oedema  
Dissolution of vascular and cell wall integrity (functional if not anatomical) leads to both intracellular and intercellular increases in osmotic pressure. If the patient survives, oedema develops. It is thought that cerebral oedema caused by ischaemia is harmful only in that it may cause sufficient hemispheric swelling to result in mass effect and herniation. There is no evidence to date to indicate that oedema, per se, increases the extent of neuronal death. In survivors, the speed with which significant cerebral oedema develops remains uncertain, and in several recent studies this phenomenon has been reported as not being a real problem in the first 24 hours after rescue.
4. Stage of Permanent Cerebral Death  
This condition occurs either within minutes of submersion, and the victim is taken to the mortuary rather than the emergency room of the hospital, or is an iatrogenic clinical state created by therapeutic technology. It is important to appreciate that early (up to several hours) post-rescue signs of apparent brain death (flat EEG, fixed and dilated pupils, no vital signs except the ECG trace) are not fully interpretable with our current knowledge. Some 30 to 50 per cent will still survive, and of these a significant number will not suffer major brain damage.

### Post-Rescue Care

A near-drowned victim will have some or all of the above changes in lungs and brain. Triage after rescue is popularly grouped into the Modell-Conn Classification of "A" (awake); "B" (obtunded or "blunted" consciousness); and "C" (comatose). Respiratory support with positive end-expiratory pressure (PEEP) in those not breathing spontaneously (a constant positive airways pressure, "CPAP", is used for the conscious patient), and intensive care monitoring are universally used. At this stage of knowledge, induced hypothermia, barbiturate rescue and invasive monitoring of intra-cranial pressure are controversial,<sup>10</sup> and results from different series can be scientifically interpreted. Management should be directed at watchful expectancy for arterial hypoxia, secondary drowning, hyperthermia, seizures, raised intra-cranial pressure, septicaemia (often with exotic marine organisms) and gastrointestinal bleeding, all of which can be anticipated on the pathophysiological grounds enumerated above, and treated aggressively.

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## SUDDEN DEATH IN THE WATER

John Hayman

### INTRODUCTION

This review outlines conditions which may result in sudden death in the water. Frequently several factors may be involved in such a fatality, and in assessing a death, the contribution of each of these factors should be evaluated. "Sudden death in the water" includes deaths in divers, swimmers, bathers rather than swimmers, and deaths in those who inadvertently entered water.

The term "sudden death" has been variously defined. The most commonly accepted definition is death occurring in a previously apparently healthy person within 24 hours.<sup>1</sup> However sudden death is more frequently thought of as death occurring within minutes. The term "instantaneous or immediate death" refers to death occurring within seconds; such deaths are nearly always cardiac or traumatic in origin.

In considering the causes of sudden death in the water, it is helpful to group the deaths in one of three categories:

- A. Deaths occurring in the water, often associated with exertion or stress, but where the water itself was not a major contributing factor.
- B. Deaths occurring in water where the presence of water was a major contributing factor.
- C. Deaths occurring in water where the water was the essential factor.

These groupings are not absolute, but are intended to emphasise the variable role of the aqueous environment in sudden death.

## CAUSES OF DEATH IN THE WATER

### A. Deaths associated with exertion or stress

These deaths are mostly cardiac in origin, but in some cases this may be difficult to confirm at autopsy. Swimming either at the surface or at depth requires a deceptive amount of exertion,<sup>2</sup> and the professional diver working at depth expends even greater energy. Such exertion may induce a fatal arrhythmia in a previously asymptomatic individual with ischaemic

heart disease, particularly in a person with a low level of habitual activity.<sup>3</sup> The fatal event may well have occurred during exertion in another area, and in these cases death in the water may be simply fortuitous. However, additional factors such as anxiety or panic associated with the initial pain or breathlessness, hypoxia resulting from water inhalation, or malfunction of diving apparatus, and hypothermia may also contribute to the development of a fatal arrhythmia.<sup>4</sup>

Cardiac lesions other than ischaemic heart disease may cause sudden death in the water. Valvular heart disease, including Barlow's syndrome and Marfan's syndrome,<sup>5,6,7</sup> together with various types of cardiomyopathy, including alcoholic cardiomyopathy, myocarditis including sarcoidosis, and various conduction defects such as the Wolff-Parkinson-White syndrome,<sup>8,9,10</sup> may all be responsible for a sudden cardiac death in the water.

Cerebral lesions, such as a cyst of the third ventricle may infrequently produce instantaneous death,<sup>11</sup> but most cerebral or vascular catastrophes result in loss of consciousness, with death occurring from drowning as a secondary event.

The following case reports illustrate this group of deaths.

#### Case 1

A schoolboy aged 12 was found lying in 1.5 m of water in a public swimming pool during his school's swimming sports. He was immediately brought to the surface where expert attempts to revive him were unsuccessful. He was not seen to be in difficulty and had been beneath the water for no more than one or two minutes.

#### Case 2

One year later, his younger brother, then the same age, swimming in the same sports, was seen to collapse during a race. Immediate attempts were made to revive him, but these were unsuccessful. Although never proven, it would seem probable that both deaths were due to arrhythmia associated with a conduction defect. Both hearts were examined in detail, and conditions such as myocarditis, cardiomyopathy, valvular heart disease, and anomalous origin of coronary arteries were excluded.

#### Case 3

A professional diver, aged 42, was working in 30 m of water when his companion found him to be unresponsive. He was brought to the surface and placed in a decompression chamber, where attempts to resuscitate him proved unsuccessful. At post-mortem, there was an area of fibrosis in the inferior wall of the left ventricle associated with atheromatous occlusion of the circumflex branch of the left coronary artery and right main coronary artery. The body showed widespread intravascular and soft tissue bubble formation with explosive disruption of both cerebral hemispheres. Death was due to ischaemic heart disease, and the bubbles would not have been present when the diver died at 30 m, illustrating that bubble formation is a physical rather than a physiological event. With occlusion of two major coronary vessels, this diver could have died at any time, but the exertion and stress of working at depth were held to be precipitating causes.