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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.¹ 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,² 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.³ 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.⁴

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,^{5,6,7} together with various types of cardiomyopathy, including alcoholic cardiomyopathy, myocarditis including sarcoidosis, and various conduction defects such as the Wolff-Parkinson-White syndrome,^{8,9,10} 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,¹¹ 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.

TABLE 1

SUDDEN DEATH IN THE WATER

Lethal Occurrence; water environment incidental	Non-lethal Occurrence; lethal because of water environment	Lethal Occurrence; caused by the water environment
Cardiac Disease Cerebrovascular Disease Trauma	Epilepsy Cerebrovascular Disease Trauma Head Injury Cardiac Arrhythmia Diabetes Mellitus (hypoglycaemia) Asthma Alcoholism Effects N, CO, CO ₂ , O ₂ Narcotic Drugs Impaired consciousness from other causes Post hyperventilation blackout	Drowning Acute Hypothermia Pulmonary Barotrauma Cerebral Arterial Gas Embolism (CAGE)
Death due to primary event	Death usually due to drowning, secondary to primary event	Death due to primary event

B. Deaths where the presence of water was a major contributing factor

A person may drown following an accident or other event which in itself may not have been fatal. Some medical conditions result in temporary or transient unconsciousness, and if this event occurs even in shallow water, death from drowning may result. In every case of drowning it is important to seek out any such underlying or contributing condition. Epilepsy is commonly considered to be one such disorder¹² although estimates as to its contribution to drowning deaths are variable. One such case is reported in the New Zealand Diving Related Deaths 1983.¹³ Hypoglycaemia following insulin, exertion, and lack of dietary carbohydrate is another possible cause. Transient ischaemic attacks associated with cerebral atherosclerosis, and even vaso-vagal episodes due to exertion and changes in external temperature¹⁴ may also result in unconsciousness followed by drowning. Acute vertigo, produced by cold, may result in complete disorientation and drowning.¹⁵ Head injury, spinal injury, cerebral thrombosis, cerebral and sub-arachnoid haemorrhage may also result in unconsciousness followed by death in the water.

Alcohol is commonly associated with drowning in adults, and must be regarded as a contributing cause in all such cases. Low levels may act simply by causing the victim to have lost a certain degree of caution and to swim further from land. Moderate levels may impair co-ordination and help to produce fatal hypothermia. High levels may be responsible for the victim falling into water and being unable to extricate himself. In these cases, alcohol may be considered the prime cause of death, and the water is only a major contributing factor.

Drowning is a frequent means of suicide, and may be chosen by the deceased when there is a desire to conceal the act of self-destruction. Suicide should always be suspected in any unexplained drowning, particularly if a non-swimmer or poor swimmer has been found inexplicably in deep water.

The following cases illustrate the group of deaths where water is a major contributing factor, and the difficulty that may occur in determining the contribution of each factor where there are several factors involved.

Case 4

A male, aged 18 was found floating face downwards in 1 m of water, 50 m from the shore. He was a known asthmatic, and at post-mortem there was histological evidence of active asthma with mucus plugging of small bronchioles and eosinophils in bronchial walls, together with signs of water inhalation. Death was attributed to drowning associated with acute bronchial asthma.

Case 5

The body of a 60 year old man, a known depressive, was recovered from a lake 2 days after his boat was found run aground. When the boat was examined by police, it was discovered that the wheel was fixed so that it would travel directly forward. It seemed that the deceased deliberately left the boat in the centre of the lake, having first arranged motor and steering so that the boat would travel away from him, perhaps intending that his suicide should appear as an accident.

Case 6

A man in his late sixties fell overboard from a boat travelling across a lake late at night. He and his companions on board had been drinking for some hours. His cries of distress were heard in the darkness for about three minutes, but by the time the boat had changed course and circled back, he was lost. The water temperature was 15°C. At post mortem there was severe coronary-artery disease, evidence of drowning, and a post-mortem blood alcohol of 150 mg%. The alcohol, potential hypothermia, and the coronary artery disease may all have contributed to the death from drowning, and it is difficult to assess the relative contribution of each of these factors.

C. Deaths due to water

Here, it is the deceased's presence in the water which is the essential cause of his death. Most of these deaths are due to drowning, with other factors contributing to a variable extent. Thus alcohol ingestion, hypothermia, and exhaustion may be significant contributing factors either in isolation or in combination. In divers, nitrogen narcosis, carbon monoxide poisoning and carbon dioxide retention may all contribute to death by drowning. In breath-hold divers, hyperventilation prior to the dive may lead to anoxia, unconsciousness and subsequent drowning.¹⁷

The mechanisms of drowning have been investigated clinically^{18,19} and experimentally.²⁰ A small amount of aspirated water may lead to pulmonary vascular spasm, pulmonary hypertension, pulmonary venous bypass and anoxia. These physiological reactions may have a more profound effect on consciousness than the actual presence of fluid in the lung. A small amount of fluid, particularly fresh, may be absorbed so that at post-mortem it would appear that no water has entered the lung. After apparently successful resuscitation attempts there may be progressive dyspnoea, reduced lung compliance, anoxia, and death from a progressive form of adult respiratory distress syndrome.²¹

In the aqueous environment, heat may be rapidly lost from the body,²² the rate of loss depending on the temperature of the water, the amount of insulation due to clothing and body fat, and the amount of movement in the water. In very cold water, hypothermia with fatal cardiac arrhythmia may occur within minutes. Sudden, particularly sudden unexpected entry into very cold water may result in immediate death due to cardiac arrest, cardiac arrhythmia, or to uncontrolled respiration and inhalation of water. In these cases, death has occurred before there has been significant cooling of the body; death is due to cold exposure rather than true hypothermia.

In divers, pulmonary barotrauma with air embolism may be a cause of sudden death and may occur following an uncontrolled ascent from relatively shallow depths.²³ Air embolism may in itself be fatal, or may lead to convulsions, loss of consciousness, and drowning. Air embolism may occur where there is partial obstruction of one bronchus,²⁴ leading to over inflation of one or more lung segments despite apparent adequate exhalation on ascent. Decompression sickness may occur acutely,²⁵ but this is unusual and symptoms do not usually appear before surfacing from depth.

In both swimmers and divers, death may occur from trauma, stings and bites from marine animals, shark and crocodile attack. The passing speedboat and the companion's speargun are obvious and not infrequent causes of death or serious injury.

INVESTIGATION OF SUDDEN DEATH IN THE WATER

The investigation of a sudden death in the water requires more than a final post-mortem examination. All the circumstances of the deceased's entering the water should be determined, including the time since the last meal, alcohol ingestion, and the taking of medication. The water conditions, including temperature, currents, wave heights and time spent in the water must be known. The interval of time

during which the deceased was missing, and the nature of rescue and resuscitation attempts should be recorded. If a diver, all details of training, experience and equipment should be known, together with the state of function of this equipment.

The autopsy procedure for a diver has been described in detail.²¹ If a cardiac death is suspected, the coronary arteries and heart valves should be examined in detail, and tissue blocks for histological study should be taken from each of the four quadrants of the left ventricular wall. If necessary multiple blocks are taken through the sino-atrial nodes and atrio-ventricular nodes and conducting bundles. Other arteries, including the common carotids and carotid bifurcations should be inspected. Material for histological examination should also be obtained from lungs, and all abdominal and endocrine organs. Samples should be taken of lung, liver, kidney and bone marrow for diatom analysis.²⁷

The complete investigation of a fatal diving accident involves more than a pathologist carrying out a post-mortem examination and producing a report in isolation. In reaching a conclusion, the findings of the autopsy should be considered together with all the known circumstances of the accident. The complete and correct evaluation of these circumstances is important in understanding such fatalities and preventing future occurrences.

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THE IDEAS BEHIND THE NEW USN TABLES

John Knight

The USN air decompression tables are used by sports divers around the world. No one really knows how good they are because no one knows how many dives are made to what schedule every year and how many cases of decompression sickness (DCS) result. These figures are not even reliable for the USN in spite of the requirement to record every dive and forward the figures to the Navy Safety Center. These are many cases of people getting bent using the USN no-decompression tables as they are written. That is straight to depth and staying there for the full bottom time and returning to the surface. Some will have ascended faster than 60 feet (18 metres) a minute, but others have kept to that limit. Looking at the testing that was done for these tables¹ there were 27 bends in 568 dives, with an average of 6 dives a schedule, covering 88 schedules. This gives a 4.75% incidence of DCS. So one could perhaps expect about 5% bends using the USN tables as they are written. Two thirds of the schedules were never tested!

Repetitive dives were even less thoroughly tested and later experience is much less than for single dives.

Dembert et al² published the 1981 USN statistics. There were 35 cases of DCS in 92,484 dives, approximately 0.038%, which is 100 times safer than

the acceptance trials and various other series where the USN no-decompression tables have been used. How can one explain the difference. Much of the air diving is shallow, and well within the no-decompression limits. For decompression dives USN divers seldom dive the dive in the book, they often add either a depth increment or a time increment, or both, to the dive and decompress for this fictitious dive. If they get close to the no-decompression limits they often decompress for the first decompression dive in the table.

In an effort to find out the incidence of decompression sickness the USN did a study of decompression dives, as being more likely to be recorded than no-decompression dives, and the incidence of DCS.³ The results, covering the years between 1 October 1971 and 30 November 1978 were published in January 1980. The overall incidence of DCS was 1.25%. The average incidence was 1.1% for the 43 schedules that were dived more than 100 times. Four schedules produced a statistically significant increased incidence, ranging from 3.1 to 4.8%. The conclusions were that there were a very limited number of schedules that were being used frequently, and that the incidence of DCS was very low (at 1.25%). There was a trend to an increase in DCS incidence with longer dives.

Weathersby et al have published a review of a large number of sets of experimental dives, including: A: the original testing of the current USN Table 1, B: the original testing of the exceptional exposure tables, C: Royal Navy submarine escape trials,⁶ D: dives at the Defence and Civil Institute of Environmental Medicine, Ontario, Canada,⁷ E: sea trials of a proposed Royal Navy decompression table,⁸ F: trials to see whether exercise during decompression affected the incidence of DCS,⁹ G: testing of surface decompression using oxygen,¹⁰ and H: a very conservative Royal Navy table which was not accepted for fleet use.¹¹

They predicted, using a relatively simple mathematical model, the probability of DCS in these dives of known profile and known DCS incidence. This allowed them to alter the values in the model to get best fits to the data. Their method of integrating risk over the course of an entire decompression differs fundamentally from the usual practice of trying to avoid a specific point, a given supersaturation ratio in the current USN tables and any supersaturation in Brian Hills' theories, all through the decompression. They assumed that a given decompression stress is more likely to produce symptoms of DCS the longer it lasts. This also means that a large number of different decompressions from the same dive may result, after integration, in the same probability of DCS. They did not invoke specific mechanisms of bubble formation, number, or growth. They assumed that when the computed inert gas partial pressure was less than ambient pressure there was no risk of DCS.

They used 6 models to examine their data. The first had one well mixed compartment for calculating the tissue partial pressure of inert gas. The second had a threshold of supersaturation, that had to be exceeded for symptoms, added. The third had two tissue compartments, which meant that the probability of no DCS was the joint probability of no DCS in both tissues. The Fourth was the third with a threshold added. The fifth was a two compartment model with three kinetic parameters rather than a single exponential for each compartment. The sixth was the same as the fifth with a threshold added.