HOW DIVER'S DIE

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Key Words

Accidents, case reports, cerebral arterial gas embolism, deaths, drowning, injuries

Introduction

Several years ago the Hillary Commission for Sport and Recreation surveyed the New Zealand population about their sporting and recreation pursuits. They produced meaningful statistics of population involved in activities from archery to shooting and included scuba diving.

The Accident Compensation Insurance Corporation (ACC) provides in New Zealand a no-fault accident insurance for fatal and non-fatal accidents in every sphere of activity. Statistics for 1989-1990 show that claims for compensation scuba diving accidents under New Zealand's Accident Compensation Scheme are relatively low both in total numbers and in comparison to other outdoor sports and leisure activities. It is unlikely that any dive accidents in this period requiring medical treatment or involving death would not have been recorded. Payments for other water sport fatalities including boating, swimming (presumably non-competitive athletic events) and fishing are very high.

Figures from Project Stickybeak and the NZ Underwater Accident recorder show a spread of causes of Australasian diving accidents not dissimilar to other figures such as DAN (Divers Alert Network) USA, except for higher representation of asthma in Australasia.

The Northland environment favours year round diving by local residents, overseas tourists and most recent graduates of Auckland dive schools. The underwater attractions also bring many divers from elsewhere in New Zealand. With the large amount of diving in Northland it also means that this is where many dive accidents occur.

Non-fatal accidents are usually transferred rapidly to the Slark Hyperbaric Unit facilities at the Royal New Zealand Navy (RNZN) Hospital, Devonport by Northland Emergency Helicopter. Fatal accident victims are usually brought to Whangarei for autopsy if the body is recovered.

It is important to establish the cause(s) of death in any fatal accident, and especially in diving accidents, as the death has occurred in a hostile environment. Associated factors include level of training and experience, with medical, psychological and equipment factors. Equipment, if available, is usually examined by the Navy or police divers. Assessments of the other factors, for the coroner, are obtained by police interviews of witnesses (the results are of varying adequacy). The pathologist's role is to establish the mechanism and mode of death. The more information, especially history and equipment examination, one has the more likely is a firm diagnosis.

Autopsy

Empirical and eyewitness evidence indicates that pulmonary barotrauma leading to arterial gas (air) embolism is a likely cause of loss of consciousness in divers. Recent dive profile examination, if available, may suggest possible causes of death but in recent years protocols for autopsies on divers have suggested that efforts be directed to detecting air in the vascular system.

These range from performing the post mortem under water e.g. in an hydraulic bath, or opening the chest cavity through a water seal maintained by the reflected skin flap and using X-rays, both conventional and computerised axial tomography (CAT). In the absence of X-rays pneumothorax has been demonstrated by direct aspiration of the chest before any skin incision.

Demonstration of gas within the cranial cavity before any skin incision or removal of the cranium, prior to the introduction of CAT scans has been especially difficult. These were first used in Australia by Ansford et al in 1990 for investigation of a naval diving accident and later investigating scallop diving fatalities in Tasmania. Three recent scuba diving fatalities are presented to show the usefulness of computerised axial tomography (CAT) scanning to increase gas embolus detection within the vascular system before the body is opened. Northland Base Hospital (now Whangarei Area Hospital) obtained a CAT scanner in 1992.

Before the availability of CAT scans locally, lateral and AP standard X-ray views of skull and thorax were done. These were occasionally of considerable interest, two examples being one with gas outlining Circle of Willis, another with gas in a coronary artery. Both also showing gas throughout the thoracic great vessels.

The factors leading up to the deaths are considered with the significance of the gas detection in relation to the mode of death. No gas analyses were undertaken. This would be essential in the future to distinguish between nitrogen, air and decompositional gases. In these cases the latter is cause is thought unlikely because of the short time interval between death and refrigeration and post mortem examination.

Case reports

Three recent fatalities, each illustrating different problems, which have occurred in widely separated areas of Northland are presented as examples of the usefulness

(or otherwise) of CT (computerised tomography) in the diagnosis of cerebral arterial gas embolism (AGE).

This has been available for use in these diving related deaths. In one the body was retrieved from 30 m after immersion for 3-4 hours. The other two patients experienced difficulties at 7 and 10 m, ascended to the surface and subsequently died after unsuccessful resuscitation attempts, in one case by an inexperienced lay person, and the other after attempted resuscitation by a general practitioner and then the Northland Emergency Rescue Team.

Case 1

A dive charter boat at Three Kings received a radio message from the 16 year old deckhand of a cray fishing boat close by saying that his skipper had gone diving an hour earlier, to retrieve a trapped cray pot, and had not returned. The cray fishing skipper (male), aged 28, was an inexperienced diver. The charter boat skipper came alongside and commenced a search, finding and retrieving the body quickly from a reef surface at 30 m. It was estimated it had been submerged for approximately 2 hours.

The weight belt was on but not the mask. When examined about 24 hours after retrieval the body was unremarkable except that the appearance of his eyelids was unusual. He had gross haemorrhage into the eyelids with splitting of the skin, and conjunctival haemorrhage and oedema (Figure 1).

CT scans of the head (Figure 2) and trunk were obtained, which showed the vascular system full of gas. The overall significance of this was uncertain, because of the time underwater and the time since retrieval. It is highly likely that most of this gas was nitrogen which had off gassed from body tissues since the body was brought to the surface. The superficial cerebral blood vessels were full of bubbles (Figure 3 page 115). The lungs show gross oedema consistent with drowning and areas of haemorrhage which suggest probable pulmonary barotrauma leading to AGE.

WHY DID HE DIE?

This diver was totally untrained and this was his first dive! He carried scuba gear on his boat as safety equipment! I was told by the Police that his tank still contained air and his regulator was satisfactory.

My hypothesis is that he descended without equalising the pressure in his mask, producing a gross mask squeeze. This resulted in haemorrhage into his lids and tearing of skin with bleeding into his mask. In pain from the swelling and having suddenly gone blind, he panicked, removed his mask and drowned.

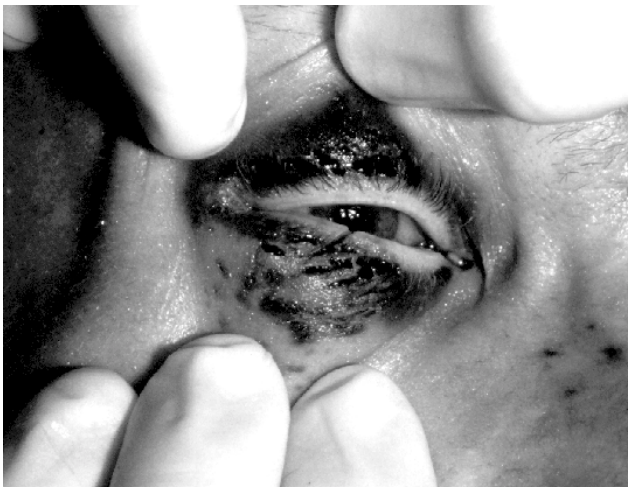

Figure 1. Case 1. Haemorrhage into lids with split skin of upper lid.

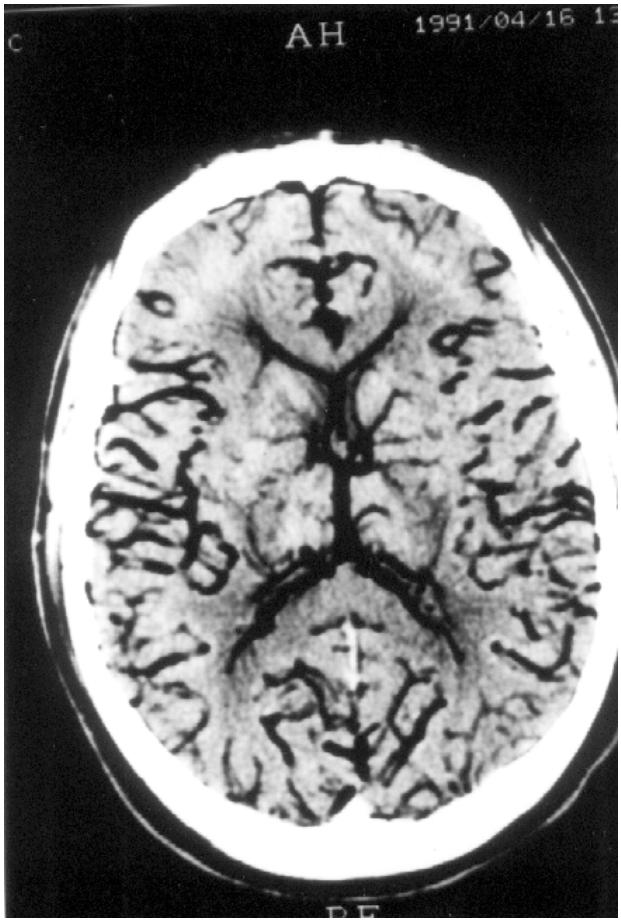

Figure 2. Case 1. CAT scan head. Large amounts of gas (black) in all intracranial vessels.

Case 2

A couple were diving off Rauol Island in the Kermadec Group 400 miles North East of New Zealand.

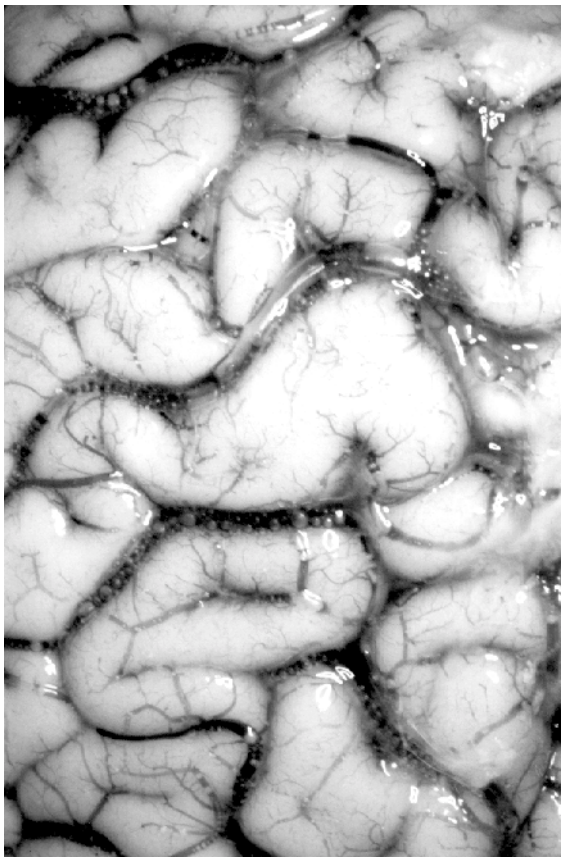

Figure 3. Case 1. Cylindrical and spherical bubbles in cerebral vessels.

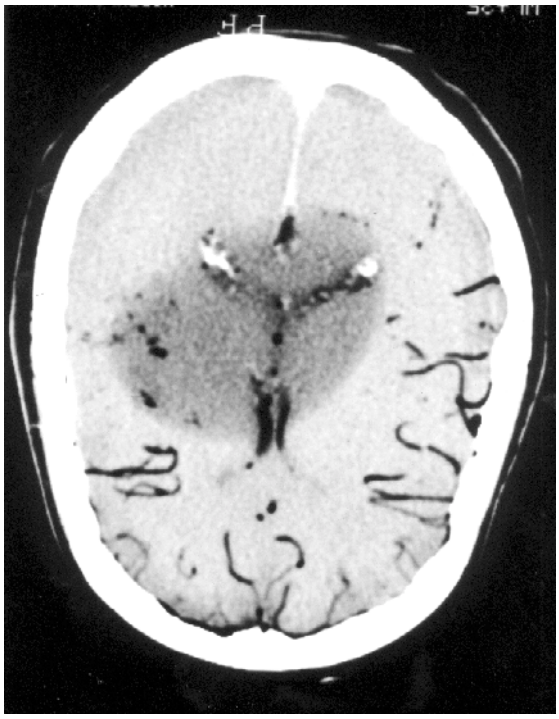

Figure 4. Case 2. CAT scan of frozen head. Gas (black) within intracranial vessels. Compare with Case 1.

The wife, aged 49, was an inexperienced, untrained diver. At 10 m she signalled to her husband that she wished to surface. They both ascended. On the surface she told him that she felt uneasy and wished to stop the dive. She also felt too weak to swim against the slight current to the boat. Her husband inflated her buoyancy compensator, then swam to their yacht's dinghy and returned with it to her, by which time she was unconscious. A dive charter boat (with the same skipper as in Case 1) came around the corner of the island at this time. The victim was taken aboard and resuscitation attempts started. These included intracardiac adrenalin (advised by medical personnel using radio). This was unsuccessful. Her body was taken ashore and on Police instructions placed in the walk-in deep freeze at the Department of Conservation Lodge.

As death occurred in "suspicious" circumstances the police arranged for retrieval by Emergency Rescue Helicopter, which refuelled during its return journey from a RNZN fuel dump on Esperance Rock. This involved hovering with only 1 skid on the rock!

No external injuries were evident at post mortem. A CT scan showed much gas in the cerebral circulation and great vessels (Figure 4). There was nothing obvious in coronary arteries, but there was air in ventricles. The lungs showed haemorrhagic areas suggestive of pulmonary barotrauma as a source of AGE. The post mortem was technically difficult due to frozen organs.

WHY DID SHE DIE?

The likely sequence of events was an inexperienced untrained diver in a remote environment ascending, in an emotionally upset state, while breath holding. The resulting pulmonary barotrauma led to embolism with loss of consciousness and respiratory and cardiac arrest on the surface.

Case 3

A 34 year old female, with five years diving experience, was on a scallop dive from a launch in Rawhiti Channel, Bay of Islands in 8-9 m. She was diving with her father after lunch on New Year's Eve. He noticed, ten minutes into the dive, that she was actively ascending but continued his dive.

Her non-diving husband was on their boat and saw her surface about 50 m away. She waved, rolled onto her back and swam to the boat. He helped her aboard where she immediately collapsed and lost consciousness. He radioed for help and went ashore at Urapukapuka Island where a tourist helicopter arrived bringing a Paihia general practitioner who continued resuscitation attempts.

The Northland Emergency Rescue helicopter team arrived some time later with an anaesthetist and assisted, but resuscitation attempts were declared unsuccessful after about 2 hours. The body was transferred to Northland Base Hospital for a Coroner's post mortem. CAT was undertaken on skull and thorax. The skull views showed a single segmental bubble obstructing the right internal carotid artery at the siphon (Figure 5). The opposite side was clear. Lung fields showed areas of pulmonary infiltration. There was gas in the aorta, vascular tree and the left ventricle and hepatic artery (Figure 6).

The tracheobronchial tree contained a considerable quantity of poorly digested food similar to that in her stomach down to the peripheral bronchi. The gastric mucosa was reddened, consistent with an acute gastritis.

WHY DID SHE DIE?

The likely sequence of events was that, following a salad lunch, for some reason, probably gastritis, she felt like vomiting soon after submerging. Not being trained in the techniques of vomiting underwater she ascended to the surface while holding her breath and trying not to vomit. She developed pulmonary barotrauma, embolised during the ascent, vomited at the surface and aspirated. This eliminated any chances of survival by preventing adequate ventilation during resuscitation.

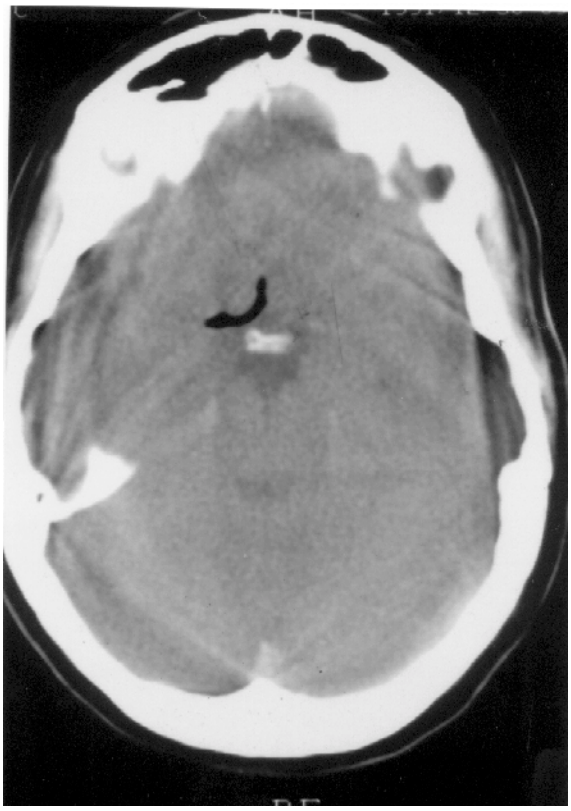

Figure 5. Case 3. CAT scan head. Single segmented bubble of gas (black) within right internal carotid artery. Compare with Cases 1 and 2.

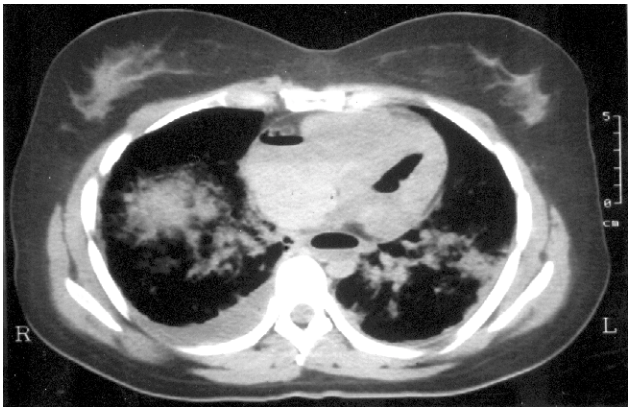

Figure 6. Case 3. CAT scan thorax. Gas (black) within aorta, left and right ventricles. Pulmonary infiltrates secondary to aspiration of stomach contents.

Discussion

I have endeavoured to show that diving deaths can result in a number of injuries which one would not necessarily expect, that CT scans are a good way of examining the body before starting the post mortem but can show air in unexpected places which makes diagnosis of the original injury less certain as off-gassing has to be considered.

In Case 1 drowning was the probable mechanism of death, contributed to by mask squeeze, panic and perhaps gas embolism. The other two cases undoubtedly died on the surface so drowning can be confidently eliminated. Case 3 involved aspiration of stomach contents which undoubtedly interfered with ventilation. Regrettably no gas analysis of samples from their vascular system bubbles were undertaken in any instance.

These three cases show that intravascular gas can be readily demonstrated and localised by Computerised Axial Tomography.

Does delay in retrieval of a body (Case 1) allow continuing uptake of nitrogen into tissues of lung and blood? If this is so, retrieval from depth with release of pressure allows production of gas from blood in the lung vessels, pushing gas into pulmonary arteries and pulmonary veins, and so into the chambers of heart, and perhaps more peripheral spread.

The sources of intravascular gas in these cases can only be guessed at.[1] Is it as a result of vigorous resuscitation? If intravascular gas is introduced during

resuscitation, what is the mechanism? Should resuscitation procedures be changed to avoid or prevent this? Should resuscitation attempts be advised against because of the risk of initiation or worsening of air embolism?

Analysis of gas samples from great vessels and ventricles should be able to confirm the origin of gas. Air would be from pulmonary barotrauma. Gas with a high nitrogen content would have evolved from dissolved gas in tissues. Various complex mixtures, involving CO_2, hydrogen sulphide and short chain fatty acids, develop from putrefactive changes.

References

1 Lawrence C. Interpretation of gas in diving autopsies. *SPUMS J* 1997; 27 (4): 228-230

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