WHY DIVERS DIE
Two more cases

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Key Words
Cerebral arterial gas embolism, case reports, deaths, decompression illness, incidents.

Introduction

The Poor Knights Islands lie 25 km offshore from the Northland coast of New Zealand. The surrounding waters are bathed by the East Auckland current which brings warm clear water from subtropical areas of the eastern Australian region, and with it eggs and larvae of species of invertebrates and fish seldom seen on the mainland coast. The islands are nature reserves and the surrounding waters are marine reserves. As a consequence this island group is a Mecca for divers, both from New Zealand and overseas tourists.

During the summer tourist season, commercial operators together take up to a total of 100 divers per day to these islands with maybe another 50 sport divers going out in private boats. Popular dive sites range in depth from one or two metres (Lost World) to 40 m (Northern Arch). In past years fatal dive accidents were not unknown at the Poor Knights, but since the mid 1980s there have been only two recent deaths that I am aware of. These are presented here as case histories and show features which may indicate that the victims should perhaps not have been scuba diving.

Case 1

On 26th December 1997 diver A, a Caucasian male aged 55, was on a trip on a commercial dive boat with four companions, including two sons who had given the trip as a birthday gift to their father, a certificated diver with some years' experience. The dive site was at Northern Arch and the dive commenced at 1100. The group descended slowly as diver A had been feeling unwell before the dive. The maximum depth reached was stated to be 36 m. The total dive time was stated to be 25 minutes. During the ascent, at a depth of about 20 m it was noted that he had separated from the group. He was subsequently found floating on the surface apparently unconscious. He was rescued onto the dive boat and resuscitation was commenced. The Emergency Services helicopter was called and a paramedic was lowered who pronounced him dead.

EXTERNAL EXAMINATION

There was an old upper mid-line laparotomy scar. Otherwise there was no significant abnormality. A computerised axial tomography (CAT) scan was performed before the formal post-mortem examination 24 hours after death. This showed gas, (presumably air, but no analysis was undertaken) within intracranial arteries (Figure 1) and within the ventricles of the heart and aorta (Figure 2) confirming arterial gas embolism (AGE). The lungs showed shadowing consistent with haemorrhage or aspiration of gastric contents.

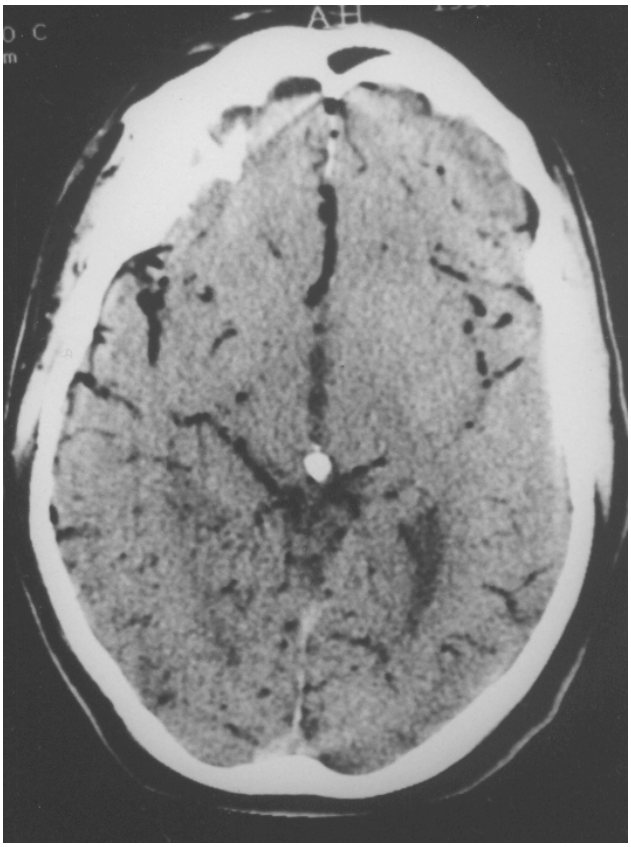

Figure 1. CAT scan of diver A's head before autopsy. The front of the head is to the top. Gas, black areas, can be seen in the intra-carnial arteries.

INTERNAL EXAMINATION

Cerebral artery branches on the surface of the brain showed small bubbles consistent with the CAT scan findings. There was no other abnormality of brain either externally or when sectioned.

The heart was slightly enlarged, weighing 450 g. All three of the major coronary arteries showed atherosclerosis with considerable narrowing of the right and of the anterior descending branch of the left coronary arteries. The left ventricular myocardium showed a healed infarct posterior-inferiorly, adjacent to the septum. Microscopy showed this to be at least three months old. The valves and chambers were otherwise unremarkable; in particular a foramen ovale was not demonstrable.

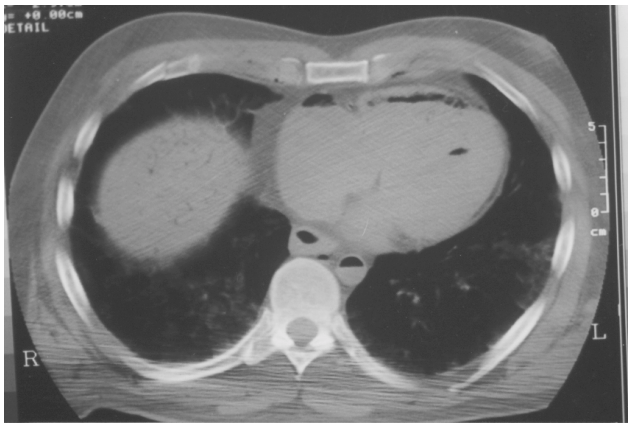

Figure 2. CAT scan through diver A's chest. The left side of the body is on the right of the illustration. The grey area in the right lung is liver. The spotty grey areas in the lung fields are haemorrhages or aspirated fluid.

The trachea and bronchi contained slightly bloodstained fluid and the lung substance showed widespread areas of haemorrhage confirmed by microscopy. The appearances were consistent with pulmonary barotrauma.

With the exception of adhesions around the stomach from previous surgery, there was no other significant abnormality.

SUMMARY OF FINDINGS

Evidence of pulmonary barotrauma leading to cerebral arterial gas embolism. Coronary artery atherosclerosis. Old myocardial infarction.

COMMENT

It was subsequently established that diver A had experienced severe angina about ten years before his death, resulting in his having an angioplasty which completely abolished his symptoms. As he was a keen diver he asked his cardiologists at the time if it would be safe for him to continue diving and being assured that it would be so, he dived regularly, usually during the summer vacations until his death. His wife and sons said that he had not attended any cardiologist for follow up from the time of his angioplasty until his death. I assume that because of the state of his coronary arteries and the demonstrated old myocardial infarct, that he may have experienced some cardiac related symptoms leading to his unnoticed ascent.

I would welcome comments from cardiologists as to management of post-angioplasty divers especially as to advice relating to increased risks from scuba diving, and what follow up is desirable for such patients, e.g. should a stress ECG be performed before allowing their return to diving, and at what intervals, taking into account that he had had trouble free diving for ten years.

Case 2

On 26th March 2000 diver B, a Caucasian female aged 58, was diving from a charter boat at the Poor Knights Islands at a site known as Anne's Reef. She and her buddy were approximately 16 m deep when she was seen to stop swimming and began waving her outstretched arms through a clump of seaweed; she then clutched the seaweed in both hands and her dive buddy noticed that there were no bubbles coming from her second stage regulator although there was said to be a small stream of bubbles exiting her mouth past it.

Her buddy surfaced with her after dropping her weight belt. She was retrieved to a nearby boat where she was given oxygen and CPR for approximately 75 minutes until an Emergency Services helicopter arrived and lowered a paramedic who administered adrenaline and atropine. There was no response and after confirming asystole by ECG the paramedic pronounced her to be dead. At the time of rescue and during CPR it was noted that fine creamy-white foam was coming from her mouth indicating probable inhalation of water, with drowning, and/or pulmonary oedema.

EXTERNAL EXAMINATION:

There was no external abnormality.

A CAT scan of head and thorax was performed before the formal post-mortem examination eight hours after her death. This showed small amounts of air in the basilar and anterior cerebral arteries (Figure 3), in the ventricles of the brain (figure 4) and in the left and right ventricles of the heart; as well as portal venous system and the azygous and hemiazygous veins (Figure 5), all indicating that some air embolisation had occurred.

INTERNAL EXAMINATION

The convolutions of the surface of the brain were flattened and the sub-arachnoid vessels were congested, both appearances consistent with anoxia with oedema. There was no free blood in the sub-dural or sub-arachnoid spaces, nor in the lateral ventricles. The sectioned brain showed no significant macroscopic abnormality.

The ribs from the second to the eighth were fractured in the mid clavicular line on each side; most likely this had occurred during CPR as there was little associated bleeding or damage to underlying lung. Both lungs were markedly oedematous, the left weighing 790 g and the right 890 g. The right lung substance showed an area of intra-parenchymal bleeding into the lower lobe. There was no evidence of previous lung disease. The appearances are consistent with aspiration of water with drowning and probable pulmonary barotrauma. In the absence of any macroscopic heart disease, left ventricular failure causing pulmonary oedema can be excluded.

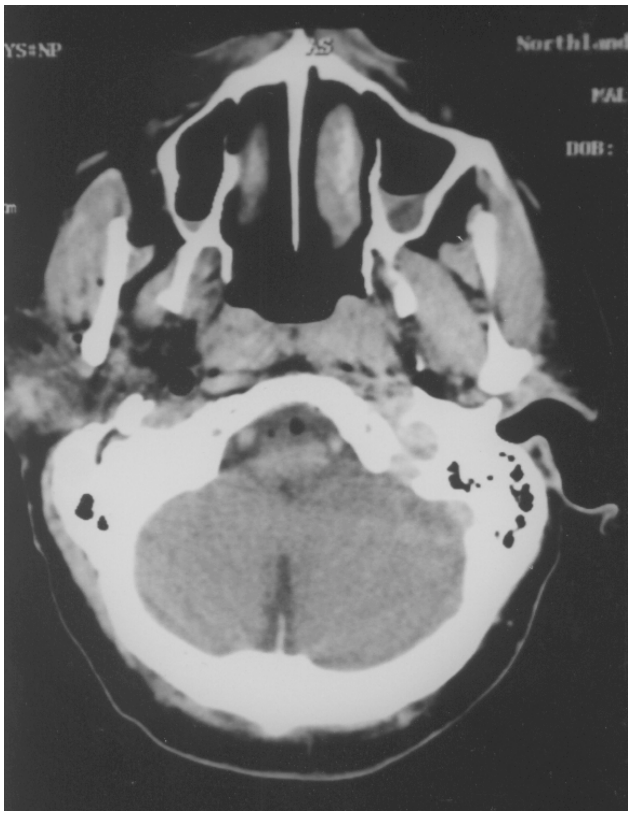

Figure 3. CAT scan through diver B's maxillary sinuses showing air, black dots, in the basilar artery and the anterior cerebral arteries .

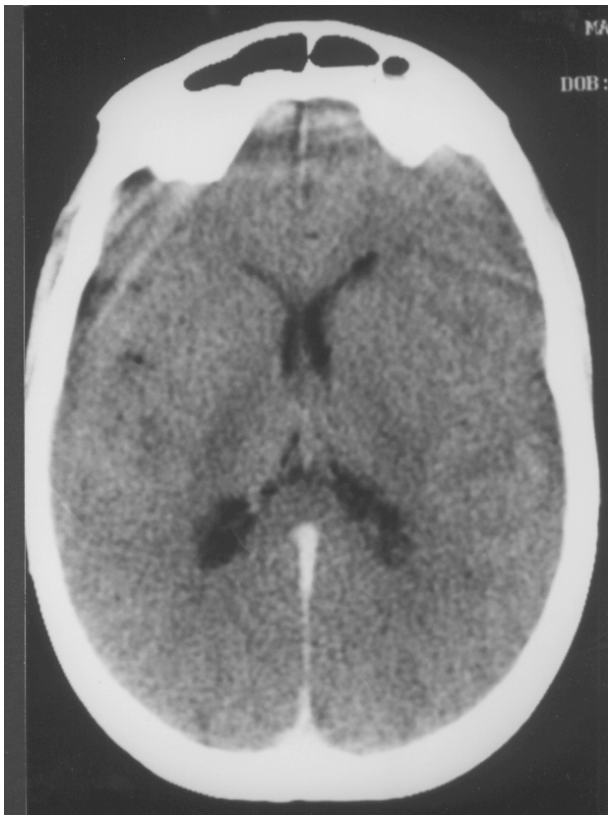

Figure 4. CAT scan through diver B's the upper part of the head showing gas, black areas, in the ventricles of the brain.

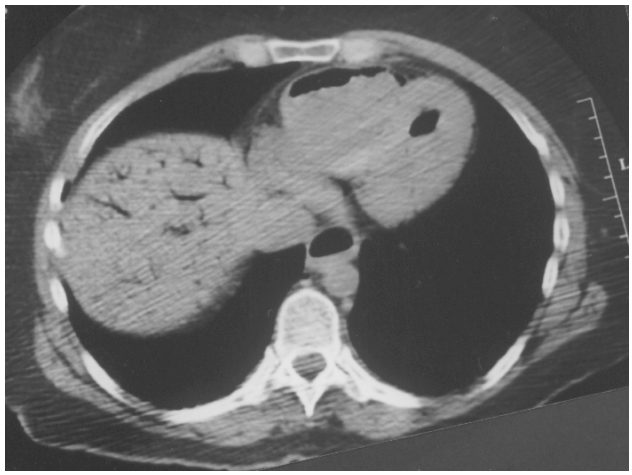

Figure 5. CAT scan through the lower chest of diver B. The left side of the body is on the right of the illustration. The grey area in the right lung is liver with the black streaks gas in the portal venous system. There is gas in both cardiac ventricles and in the azygos and hemi-azygos veins.

The heart was of normal size and appearance, weighing 300 g. All three major coronary arteries were free of atherosclerosis. The valves and chambers were unremarkable. A foramen ovale could not be demonstrated.

There was no other significant abnormality.

SUMMARY OF FINDINGS

Salt water aspiration leading to drowning; complicated by pulmonary barotrauma with intravascular air embolism. Multiple rib fractures from CPR.

COMMENT

The CPR was administered by a Master Dive Instructor and three nurses, one of whom worked in an Emergency Room. The multiple rib fractures due to the CPR are disturbing. Had she recovered she would have been in respiratory distress with a flail chest and in considerable pain.

She was making her ninth dive since completing her Open water Diver certificate. She was a competent swimmer and was said to be a confident diver. She swam laps every day in a large metropolitan public swimming pool with no apparent problems. However her husband recalled that approximately one year before her death she blacked out while doing her laps and was pulled from the pool unconscious. She was taken to the Emergency Department of a nearby hospital by ambulance and after a few hours observation, without formal admission, was allowed to go home. Her husband described her as "very groggy" for a day or two afterwards. Around this time her adult children told a general practitioner friend that she had experienced chest pain with shortness of breath. It appears that she has been well since the blackout incident until her death.

Unfortunately her Emergency Department notes are not available, nor are any details as to her pre-dive course medical examination, if she had one.

I think it likely that she, for whatever reason, experienced a change in consciousness during her final dive and aspirated water even though she apparently did not lose her second stage regulator. I think it likely that air embolisation occurred during ascent while unconscious. Air in the basilar artery would have involved her respiratory centre in the brain stem producing cessation of respiratory muscle activity.

Assuming that the history of blackout one year earlier was provided to her training agency or to her examining doctor, should she have been passed as fit to dive? I think not!

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This is the second paper in the Why Divers Die series by Dr Rees Jones. The first will be found in SPUMS J 1998; 28 (2): 113-117.

PEAK EXPIRATORY FLOW AT INCREASED PRESSURE

Paul S Thomas, Clifford Ng and Michael Bennett

Key Words

Equipment, hyperbaric research, medical conditions and problems.

Abstract

It has been estimated that asthma is as common amongst divers as in the general population. Thus, asthmatic individuals may require hyperbaric therapy and monitoring in this treatment. Asthma is usefully monitored by the Wright's standard and mini-peak flow meters. These devices are versatile and can be used under conditions where electrical supply is difficult or inadvisable. In a hyperbaric chamber electrical sources are restricted due to the risk of fire. We therefore compared the performance of Wright's mini- and standard peak flow meters with a rolling seal spirometer, especially adapted for use in the chamber. The hypothesis tested was whether the peak flow meters, which are density dependent, would over-read compared with the spirometer which is unaffected by changes in density because it is volume dependent.

Seven normal subjects performed volume-dependent spirometry to derive peak expiratory flow (PEF) and PEF manoeuvres using standard and mini-PEF meters at sea level and at 3, 2.5, 2, 1.5 and again at 1 atmosphere (ATA). There was a progressive and significant decline in PEF with increasing pressure as measured by the spirometer (69.5+/-0.8% baseline at 3 ATA), while the PEF meters showed a progressive increase in their readings (107.9+/-1.7% at 3 ATA). Using these data points we were able to derive a correction factor which allows the appropriate PEF values to be calculated from the Wright's PEF meters, if the pressure is known. Thus, the Wright's PEF meters can be used under conditions of hyperbaria, if a suitable correction factor is used.

Introduction

It is well recognised that flow is dependent upon density and the flow of gas from the lungs is no exception. In situations of increased gas density, flow is reduced and becomes increasingly turbulent, which can result in an increased time to exhale and less efficient gas exchange. If there is already a critical narrowing of the airway, then this reduced flow can impede exhalation, so that it is incomplete before there is a need for the next inspiratory cycle to begin. This can lead to gas trapping in the airspaces or alveoli. Asthma is the most common reversible disorder with airway narrowing. Airway obstruction in the older population is usually secondary to smoking and only partially reversible.

In situations where gas density is reduced airway obstruction can be measured under both field and experimental conditions, e.g. mountaineering expeditions and hypobaric chambers.[1] In hyperbaric situations where gas density is increased, e.g. diving, studies must be confined to hyperbaric chambers.

Previous work has indicated that the peak expiratory flow (PEF) and forced expiratory volume in the first second (FEV_1) are increased under conditions of reduced gas density. Therefore, as gas density increases the PEF and FEV_1 will fall. This has been demonstrated previously by breathing mixtures of gases of different density at sea-level and in hyperbaric chambers.[2,3]

The number of indications for hyperbaric oxygen therapy is increasing,[4] and therefore some individuals with airway obstruction may have indications for such treatment. In addition, since an unknown number of asthmatic subjects dive, it is evident that some may well develop problems relating to decompression and may require treatment in a hyperbaric unit. A subject with asthma or airway obstruction may require airway monitoring while in the chamber. To reduce the risk of fire, equipment within a hyperbaric