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INVESTIGATING DIVING FATALITIES A CASE REPORT

Peter Lewis

Summary

A case report is presented with discussion of the autopsy technique and the evidence needed at the coronial inquest.

Introduction

Investigation of a skin-diving accident requires assessment of:

1 medical history;
2 diving experience;
3 dive profile;
4 environmental conditions;
5 diving equipment;
6 the autopsy.

Finally a correlation of all facts needs to be made.

Case report

The victim was a 28 year old male who died while using hookah, or, more properly, surface supplied air from a compressor. The autopsy showed signs of drowning but otherwise did not indicate the underlying cause of death.

MEDICAL HISTORY

He was an overweight 28 year old electrician, who had been an Australian Rules football (Victorian Football League) player. He had had a knee reconstruction. His father died of heart attack when in his 50s.

DIVING EXPERIENCE

Unknown but at least of 4 years duration and he could free-dive to 12 m.

DIVE PROFILE

His buddies were two experienced snorkellers and one novice. He used hookah to dive to 9 m for 25 minutes. After a surface interval of 30 minutes a second dive was made to 9 m for 10 minutes and then, after a further surface interval of 2 minutes, a third dive was made to 9 m for 5 minutes. At this time it was discovered that he was not breathing and he was pulled to the surface by the air-hose and brought, with enormous difficulty, into the boat. The face mask was 1/4 full of froth and blood. Resuscitation was attempted but to no avail.

ENVIRONMENTAL CONDITIONS

He was diving off an island, 2 km offshore, in a marine reserve with no current, 20 m visibility and a 5-10 knot breeze.

DIVING EQUIPMENT

His face mask was perished, the booties had broken zips and the compressor was in poor condition. The regulator hose was, inappropriately, tied directly to his weight belt. The hookah unit was sent to the police diving unit in Sydney for assessment and this revealed four of the five engine mounts were broken. This allowed the compressor to pivot, which in turn caused the air intake hose to disconnect. This hose had no clamps, was a poor fit and had multiple cracks. Numerous other compressor defects were noted. Compressed air was taken for analysis and this revealed a carbon monoxide concentration of 144 p.p.m. while the hose was connected and 633 p.p.m. when the hose was disconnected. Australian Diving standards require a maximum of 10 p.p.m.

AUTOPSY

Blood taken revealed a carbon monoxide saturation of 50%. It is of interest was that the body, tissues and blood did not show the classic cherry-red colour.

CORRELATION

The history, police reports, autopsy, toxicology and equipment testing allowed the conclusion of “death due to drowning due to carbon monoxide poisoning due to diving using defective equipment”.

Comments on Carbon Monoxide Levels.

Non-smokers have carbon monoxide levels of <1% while smokers average 5% (range 1 to 9%, although super-inhalers can have up to 16%:). Blood levels less than 10% usually produce no symptoms while higher levels produce headache, lethargy and nausea. Levels over 50% produce increasing confusion, then syncope and coma. Exhaust fumes from older cars contain 5 to 6% carbon monoxide which if led to the sealed interior of a car (a method of suicide) have caused death in approximately 10 minutes.[1] The toxic limits of carbon monoxide by varying ambient and oxygen partial pressure have not been established.[2] A comprehensive review of carbon monoxide poisoning has been published in the SPUMS Journal.[3]

Post mortem examination

A technique has been published by the Royal College of Pathologists of Australasia[4] and is described in the book “Diving and Subaquatic Medicine”.[5] In summary, this involves a full assessment of the history and circumstances of the accident, a detailed autopsy, including histological examination, and special tests.

Before the autopsy the body should have X-rays of neck, chest and abdomen to detect intravascular air. CT scans are excellent if one can get them. The external examination should include assessment of colour and any subcutaneous emphysema around the root of the neck. Opening the cranium underwater is recommended,

however, this is impractical for the majority of prosectors. It is important to dissect carefully so that assessment of the distribution and significance of any air within the cerebral vessels can be made. The chest cavity can be opened underwater by creating a shallow pool by pouring water within the reflected flaps of thoracic skin. Aspirating the ventricles of the heart by using a syringe partly filled with water will reveal any intracardiac air. The presence and size of any patent foramen ovale should be recorded. Blood should be taken for estimation of alcohol, carbon monoxide and other drugs. Vitreous humour biochemistry may sometimes be of value.

Correlation of findings for the Coroner

The post-mortem findings are interpreted with the knowledge of all the circumstances of the accident so that the event or events leading to the fatality may be completely defined. This correlation, however, needs to be made by a knowledgeable person.

I urge SPUMS members to volunteer their services to the pathologist or government medical officer at the time of an autopsy of a diver and if necessary to write a definitive correlation for the coroner. In this way relevant findings should be handed down and appropriate lessons learned.

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OXYGEN AS A DRUG: A DOSE RESPONSE CURVE FOR RADIATION NECROSIS.

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Background

Hyperbaric oxygen is a well known adjunct in the treatment of human clinical radiation necrosis. Its mechanism of action has been determined to be a stimulation of macrophage derived angiogenesis factor and macrophage derived growth factor by establishing physical oxygen gradients in radiated tissue. Angiogenesis is directly proportional to the oxygen pressure in the hyperbaric environment.

Methods

42 New Zealand white rabbits (*Oryctologus cuniculus*) received 320 cGy fractions of cobalt (^{60}Co) radiation twice weekly for a total dose of 5,440 cGy. Resulting tissue damage which develops over six months does not produce overt necrosis. The 42 animals were divided into 6 groups of 7 animals each. Each group of animals was then exposed to oxygen at 1, 1.5, 1.75, 2.0, 2.5, and 3.0 ATA. The animals were then killed painlessly with infusion of 30% Barium Sulfate and 5 ml of Hypaque. The radiated tissue was harvested and prepared for tissue microradiographic angiography. The radiographs were coded and analysed by a blinded investigator (MP) using a random point analysis of vessels per tissue area (V/T). A mean score V/T versus dose of oxygen in atmospheres was derived (Table 1 and Figure on page 87.

TABLE 1

ATA	1.00	1.50	1.75	2.00	2.50	3.00
Vessels (V/T)	0.09	0.12	0.25	0.43	0.74	0.91

Conclusions

The results indicate that oxygen does indeed behave as a drug. The optimum dose in this model is 3.0 ATA