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INTERPRETATION OF GAS IN DIVING AUTOPSIES

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Key Words

Accidents, bubbles, death, investigations.

Introduction

Recent autopsy protocols for diving fatalities have emphasised the importance of the detection of gas in the body to diagnose cerebral air gas embolism (CAGE), either by erect chest X-ray, CT Scan or by dissection underwater.[1-3]

Boyle's Law states that at a constant temperature the volume of a gas is inversely proportional to the pressure. Cerebral air gas embolism occurs during an uncontrolled ascent without exhalation because the volume of the gas in the lungs expands as the ambient pressure falls, forcing gas into the pulmonary circulation and thence into the cerebral circulation.

Unfortunately, very little critical analysis has been made of the significance of intravascular gas at autopsy. Intravascular gas was detected in 12 out of 13 diving fatalities autopsied at the NSW Institute of Forensic Medicine. In 5 of the 12, the history and autopsy findings did not suggest cerebral air gas embolism. What then is the significance of gas?

Could the gas be artefactual?

Forensic pathologists have long recognised that the process of decomposition causes gas formation. Bacteria proliferate in the dead body, particularly in the blood vessels, breaking down blood and tissues and generating gas in a process of putrefaction. If decomposition was responsible for the intravascular gas then this gas should also be seen in non-diving fatalities.

Resuscitation, using endotracheal intubation, positive pressure ventilation and intravenous cannulation, can cause subcutaneous emphysema and even air emboli. Eight out of 13 of the divers were subject to vigorous resuscitation. If resuscitation was responsible for the intravascular gas then it should also be present in non-diving fatalities.

Finally, at increased pressure the body absorbs nitrogen. Normally during ascent nitrogen diffuses out of the tissues and is breathed out, part of the process of decompression. However, if death occurs at depth and the body is brought rapidly to the surface, nitrogen bubbles will evolve in blood vessels and in soft tissues and are not removed because the circulation has stopped. Decompression would appear to be capable of generating intravascular gas in diving fatalities, either during or after death.

Methods

All diving fatalities in NSW are autopsied at the NSW Institute of Forensic Medicine. In the cases presented here erect chest and abdominal x-rays were taken before autopsy. Autopsies were commenced as soon as possible after death, however there were often delays in transporting the body. The body was positioned with a block under the upper back so that the chest was the highest point. The chest was opened first taking care not to cut the superficial veins of the neck. Gas was aspirated from the heart, using a Hamilton "gastight" syringe (Hamilton Company, Reno, Nevada 89502, USA).

The inferior vena cava and portal vein were opened once the block was removed from under the body. Air aspirated was analysed by the Department of Mineral Resources, Lidcombe. The diving equipment was examined and tested by NSW Police Divers, Sydney Water Police. Where dive computers were used they were down loaded and the dive profiles recorded. Air from the tanks was also tested by the Department of Mineral Resources.

Results

Twelve of 13 diving fatalities had intravascular gas.

The time between death and post mortem varied from 8 hours to 5 days. The average time was 41 hours. Two bodies were recovered from a wreck at 51 m after being missing for 3 days. If these two cases are excluded, the average delay to post mortem was 26 hours, still a significant delay.

In all 12 of the diving fatalities gas was present in the heart, neck veins, inferior vena cava and portal/hepatic veins, often with as much as 100 ml of gas in the right

ventricle. In 4 cases where a cardiorespiratory arrest had occurred on the bottom at a depth greater than 40 m, gas was also present in peripheral vessels, in skeletal muscles and in joints.

The results in 13 unselected non-diving fatalities were quite surprising. Ten of the 13 cases showed gas on X-ray and autopsy. Of these, 5 cases had small amounts in the portal and hepatic veins and 5 cases had larger amounts in the heart, neck veins and hepatic and portal veins. The interval between death and post mortem was 5 hours to 8 days (average 35 hours).

Eight of the non-divers were not resuscitated. In this group, extensive gas was only seen in the presence of obvious decomposition, however small amounts of gas were usually present in the portal hepatic veins after 24 hours.

Five of the non-diving fatality cases had endotracheal intubation and intravenous cannulae; Three of these showed extensive gas. In 2 cases autopsied at 4 and 8 hours after death, there was gas in the heart and neck veins. The short post mortem interval suggested that the gas was not due to decomposition and suggested that resuscitation could introduce gas.

In the last 5 non-divers analysis of the intravascular gas was performed. In 3 cases the analysis was close to normal body percentages. The two cases with decompositional gas yielded quite different results (Table 1).

Discussion

Intravascular gas formation does occur in non-diving fatalities. In unresuscitated cases it appears at around 12-24 hours in the hepatic/portal venous system. This is consistent with what we understand about putrefaction as the source of the bacteria appears to be the gastro-intestinal system. The amount of gas seen is small compared to the diving fatalities unless decomposition was obvious macroscopically. Analysis of the intravascular gas shows high levels of CO_2 and hydrogen which may be the best markers of decomposition.

It appears that resuscitation can cause the early appearance of large amounts of intravascular gas. This was a surprising result. Intravascular gas may represent vigorous positive pressure ventilation in a dying patient or perhaps gas introduced by the intravenous cannulation, although in all cases the cannulae had been capped or were still attached to giving sets. Since eight of the diving fatalities underwent resuscitation I cannot exclude resuscitation as a cause for gas in some of the diving fatalities.

In five of the diving fatalities, the individual was observed unconscious on the bottom at over 40 m and the body was then brought to the surface rapidly. In two of the cases, the divers became trapped in a wreck at 51 m, ran out of air and were not recovered for 3 days. These two cases showed advanced decomposition. In two cases, drowning due to oxygen toxicity at 47 m and drowning due to using air and poorly maintained equipment at 76 m, the body was brought rapidly to the surface. X-Rays at autopsy showed extensive gas in soft tissues, muscles, joints and peripheral vessels, as well as in the heart. The gas here is most probably due to peri- or post-mortem decompression. In the 5th case, the cause of unconsciousness was not established, however, given the rapid ascent of the body from 46 m, I am unsure whether the gas seen represents a cerebral air gas embolus or post mortem decompression.

Of the remaining 7 diving fatalities, four had strong evidence of cerebral air gas embolism. Two lost consciousness suddenly on reaching the surface. In one of these the dive computer demonstrated a 20 second ascent from 30 m. In two other cases, the presence of a perforated ear drum and an empty tank suggested a rapid ascent.

One case, who had a 70% stenosis of the left anterior descending coronary artery, became unwell on the bottom and unconscious during the ascent. The presence of the gas suggests a cerebral air gas embolism during ascent even though the primary cause of death appears to be ischaemic heart disease. In the remaining 2 cases; the presence of the intravascular gas implies a cerebral air gas embolism, although there is no history of a rapid ascent.

TABLE 1

GAS ANALYSIS OF NON-DIVING AUTOPSIES SHOWING GAS ON X-RAY

Gases	Three normal bodies		Two decomposing bodies	
			Case 1	Case 2
Nitrogen	77.3%	(range 75.9 - 78.8%)	23.0%	46.0%
Oxygen	18.7%	(range 16.8 - 20.6%)	5.0%	8.0%
Carbon dioxide	4.3%	(range 3.8 - 4.5%)	45.0%	3.02%
Hydrogen			26.8%	14.0%
Methane				0.1%

Large amounts of gas were seen in the venous circulation in all of these cases. If we postulate that, in a rapid ascent,barotrauma causes expanding gas in the alveoli to leak into the pulmonary veins, then one would expect most of the gas to be in the arterial system, but this is not the case. This pattern of distribution of the gas may indicate that the lungs trap gas bubbles more effectively than other tissues, such as the brain,[4] once the gas has passed thought the systemic circulation. Alternatively the gas may enter the venous circulation through the pulmonary lymphatic system as has been suggested in neonatal systemic air embolism.[5]

Conclusions

Decomposition and resuscitation can result in intravascular gas at autopsy. In view of the possible confusion arising from the production of decompositional gas, in performing diving autopsies it is important to autopsy the body as soon as possible after death. Analysis of the gas for hydrogen and carbon dioxide may indicate whether the gas is due to decomposition or not. In deep diving fatalities some gas formation due to decompression does occur.[2,3] It is not clear whether decompression makes any contribution to intra-vascular gas formation in diving fatalities during more superficial dives.

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OPEN CHAMBER CARDIAC SURGERY: A CLINICAL INJURY MODEL FOR ARTERIAL GAS EMBOLISM

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Summary

Cerebral arterial gas embolism (CAGE) can occur in divers breathing compressed gas and as a consequence of some medical procedures. Open chamber left heart surgery, in particular, is invariably complicated by CAGE, although manifestations may be "subclinical". This paper discusses the utility of open chamber left heart surgery as a clinical injury model for CAGE in divers and other groups.

Key Words

Bubbles, CAGE, cardiovascular, investigations, research.

Review

Cerebral arterial gas embolism (CAGE) may occur in compressed gas diving following either pulmonary barotrauma or right to left shunting of venous inert gas bubbles.[1] Clinically, in divers, there is rapid onset of neurological phenomena such as altered consciousness, visual changes, cognitive changes, and sensorimotor deficits. The natural history of CAGE in divers is complex and may include death (often by drowning), persistent neurological symptoms, spontaneous recovery and relapse despite initial spontaneous recovery. CAGE is one of several mechanisms of bubble-induced injury collectively referred to as decompression illness (DCI).[2] The contribution of CAGE to morbidity in many cases of DCI is difficult to assess since several injury mechanisms may be involved simultaneously. This difficulty complicates the study of therapeutic intervention targeted at CAGE and is compounded by the usual absence of any pre-morbid data, a variable extent of injury and an even greater variation in the time from onset of illness in divers to presentation for treatment.

Cerebral arterial gas embolism also occurs in cardiac surgery. Any operation supported by a cardiopulmonary bypass (CPB) circuit carries the risk of CAGE as there are numerous ways in which air can be introduced into the body from the circuit. These include: incomplete pre-bypass de-airing of the circuit;[3] the use of a bubble oxygenator;[4] low blood volume in the venous reservoir;[5] high blood flow rates and entrainment of air into the venous inflow to the CPB machine.[6] In operations where the heart is not opened (closed chamber procedures), such as coronary artery bypass grafting (CABG), the CPB circuit is the main source of emboli. However, there is much