ORIGINAL ARTICLE

Relevance of postmortem radiology to the diagnosis of fatal cerebral gas embolism from compressed air diving

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Aims : To test the hypothesis that artefact caused by postmortem off-gassing is at least partly responsible for the presence of gas within the vascular system and tissues of the cadaver following death associated with compressed air diving.

Methods : Controlled experiment sacrificing sheep after a period of simulated diving in a hyperbaric chamber and carrying out sequential postmortem computed tomography (CT) on the cadavers.

Results : All the subject sheep developed significant quantities of gas in the vascular system within 24 hours, as demonstrated by CT and necropsy, while the control animals did not.

Conclusions : The presence of gas in the vascular system of human cadavers following diving associated fatalities is to be expected, and is not necessarily connected with gas embolism following pulmonary barotrauma, as has previously been claimed.

In the absence of trauma or medical illness, diving associated fatalities are usually caused either by asphyxia from drowning, or by cerebral arterial gas embolism (CAGE) secondary to pulmonary barotrauma. The latter occurs as a result of air trapping and expansion during a rapid ascent from depth. While the circumstances of the accident may give clues as to which is the most likely cause, more conclusive evidence is often needed to indicate the precise cause of death.

Several groups of investigators have published accounts of the results of postmortem medical imaging, (plain films, computed tomography (CT), or both), on the subjects of fatal diving accidents.^{1–6} All have claimed that the presence of intravascular gas in their subjects indicated that antemortem gas embolism must have occurred, and that this was the cause of death. All 10 individual cases reported by these investigators had gas in the vascular spaces. It is our contention that such findings are likely to be commonplace and are merely a reflection of the off-gassing process. None of us are aware of any published data showing a lack of vascular gas on CT following a death associated with diving. An animal experiment was devised to test the hypothesis that the intravascular gas identified by postmortem imaging was essentially artefactual.

METHODS

Seven adult Merino sheep (five female, two castrated male) were used in the study. Sheep were chosen for the study for convenience rather than for physiological or anatomical similarity to humans. Earlier studies have shown their suitability as a model for studying respiratory decompression illness.⁷ Ethics approval was obtained from the ethics review committee of James Cook University.

In the initial experiment a single sheep was placed in a hyperbaric chamber and anaesthetised, using intravenous pentobarbital (pentobarbitone). The animal was accompanied by an anaesthetist who ensured that it was able to breathe chamber air freely at all times, to obviate any possibility of pulmonary barotrauma. The chamber was pressurised to 18 metres of sea water (MSW). After 45 minutes the animal was killed by the lethal intravenous injection of a concentrated pentobarbitone solution. The chamber was then depressurised to sea level over 15 minutes to allow a safe ascent for the attendant. The profile was chosen to simulate a typical recreational reef dive with significant nitrogen tissue loading.

The sheep cadaver was then subjected to CT of the head, neck, thorax, abdomen, and pelvis, at approximate intervals

of one, eight, and 24 hours postmortem. Between scans the sheep was held in a mortuary refrigerator.

The experiment was subsequently repeated using three pairs of sheep, employing one of each pair as a subject (treated in the same way as the initial subject, as described above), and the other as a control. The first control animal was anaesthetised, monitored, and killed at the same time as the subject animal, but was not placed in the hyperbaric chamber, being kept at sea level pressure. Each of the second and third control animals was anaesthetised and killed, and then placed in the hyperbaric air chamber with the live anaesthetised subject animal.

The CT images were then assessed by an experienced radiologist, for the presence of gas within vascular spaces and elsewhere in the soft tissues.

A veterinary necropsy was subsequently carried out on each animal, within three hours of the 24 hour CT.

RESULTS

The CT studies are illustrated in figs 1–3. None of the initial scans, carried out at about one hour postmortem, showed any significant evidence of gas collections in the tissues or vascular spaces, either in the subjects or in the controls, with the exception of a trace of gas in a single neck vein in some of the animals, corresponding to the site of the intravenous injections. However, at eight hours significant amounts of gas were seen in the vessels of the subject sheep. Numerous vascular spaces were identified as containing gas, including cardiac chambers and venae cavae, aorta and major branches, intracranial dural sinuses, portal veins, and numerous smaller vessels in the face and extremities. No new collections of gas were seen in the controls on the eight hour scans.

The 24 hour CT studies of the subject animals demonstrated a similar distribution of gas, but there was a generalised increase in gas volume compared with the scans carried out at eight hours. Gas was identified in the bone marrow of the femur in subject animals on the 24 hour scan, but not in the eight hour or one hour scans.

By 24 hours relatively small amounts of gas were beginning to appear in some areas within the cadavers of the control animals. Figure 3C shows pockets of gas in blood vessels and fascial planes in the pelvis and proximal lower extremities, though the amounts are considerably less than in

Abbreviation: CAGE, cerebral arterial gas embolism

Figure 1 Postmortem computed tomography of the head. (A) Subject at one hour postmortem shows no vascular gas collection. (B) Control at 24 hours shows no vascular gas collection. (C) Subject at 24 hours shows extensive gas collections in the vessels of the soft tissues (white arrow) and cranium, notably the cavernous sinuses (black arrow).

the subject animals. Figure 2C shows two tiny pockets of gas in the heart of one control animal. However, no gas was seen in the cranial cavities of the control animals at 24 hours.

At necropsy, considerably more intravascular gas was present in subject sheep compared with controls. On reflection of the fore and hind legs at the start of the examination, gas bubbled out of the severed brachial and femoral vessels in the subject animals but not in the controls. Subsequent examination revealed more gas in subject than control animals in the vessels of thoracic and abdominal cavities as well as in the subcutaneous tissue of the chest and in the fascial planes of the abdominal wall.

DISCUSSION

During compressed air diving the amount of nitrogen dissolved in the tissues of the individual's body increases as a function of time, depth, and type of tissue. The readiness with which the various tissues saturate with nitrogen varies depending upon their perfusion with blood, the tissue solubility for nitrogen, and the rate nitrogen diffuses into and through the tissue.

For the reader less familiar with diving physiology it should be noted that a considerable amount of nitrogen is taken up into various tissues. The following calculation makes some typical assumptions and is included to emphasise this point: the solubility of nitrogen in water is 12.9 μl per ml at 1 absolute atmosphere (ATA) at 37°C. The solubility of nitrogen in lipid is 5.2 times greater, or 67 μl per ml under the same physical conditions.⁸ The solubility increases with partial pressure in a linear fashion. The practical significance of this can be illustrated using the hypothetical example of a 70 kg human. At sea level, the 70% water content would contain, at equilibrium and breathing air, 499 ml nitrogen dissolved within it. For every 1 ATA above this pressure, the amount dissolved at saturation would increase by the same amount. Assuming a fat

compartment of 10% body weight, this would contain another 477 ml nitrogen per ATA at 100% saturation. Even a relatively low saturation of adipose tissue can therefore be expected to make a significant contribution to the excess nitrogen load within the body of a diver. For example, assuming 80% water compartment saturation and 40% fat compartment saturation, the 70 kg individual diving at 30 metres (approximately 4ATA), for perhaps 15 minutes, would carry an excess nitrogen load of 1384 ml on return to sea level. Table 1 demonstrates the calculation.

If the subject dies after some time at depth, the excess nitrogen will come out of solution once the body is returned to sea level, by the process of tissue degassing. This can be identified by postmortem imaging.

The process of degassing evidently occurs relatively gradually, not being detectable by CT at one hour postmortem, though it becomes very obvious by eight hours, with much of the dissolved inert gas evolved into the vascular spaces.

The appearance of gas in the subject animals at eight hours, but not in the controls, represents the process of off-gassing described above. The appearance of small amounts of gas in the control animals at 24 hours is considered to be secondary to postmortem autolysis.^{9 10}

Conclusions

This study shows that postmortem CT or other body imaging techniques are not useful in determining whether or not diving related CAGE has occurred. Intravascular gas is likely to be present after a relatively short period of time, purely from the passive process of off-gassing occurring postmortem and may be present with non-pressure-related causes of death such as drowning. This is at variance with the published view that such gas collections are indicative of death from pulmonary barotrauma and cerebral arterial gas embolism. The usefulness of these techniques in deceased divers is mainly limited to the

Figure 2 Postmortem computed tomography of the thorax. (A) Subject at one hour shows no vascular gas collection. (B) Subject at 24 hours shows extensive gas in the cardiac chambers and great vessels, and numerous smaller collections in the soft tissues of the chest wall and paraspinal muscles. (C) Control at 24 hours shows air in the tracheobronchial tree, but no significant intravascular gas collection, though two tiny adjacent gas pockets are shown superficially in the heart (see text).

Figure 3 Postmortem computed tomography (CT) of the subject animal at 24 hours (A and B). (A) The upper abdominal region shows extensive gas collections in the portal veins of the liver (arrow) and systemic vessels (arrowheads). (B) The pelvic region shows widespread collections of gas in the vessels of the pelvic musculature. (C) 24 hour postmortem CT of control animal shows small pockets of gas in the soft tissues of the pelvis and proximal lower extremities (arrows).

demonstration of evidence of manifestations of pulmonary barotrauma, such as pneumothorax, pneumomediastinum, or pulmonary interstitial emphysema.

It is hypothetically possible that CT carried out within an hour or two of death may be useful if it shows cerebral intravascular gas, because artefactual gas production was not demonstrable in our one hour CT studies. Published case reports suggest that intravascular gas can be demonstrated antemortem following gas embolism from non-diving causes—for example, medical

Table 1 Calculations involved in determining excess nitrogen load

Item	No	Units
Total body mass 70 kg	70	Kilograms
Water	49	Litres
Fat	7	Kilograms
Solubility of N ₂ in water ⁶	13	μl/ml/ATA @ 310K
Solubility of N ₂ in fat ⁶	67	μl/ml/ATA @ 310K
Body fat volume (SG=0.8)	9	Litres
Water compartment N ₂ /ATA	499	ml @ 100% saturation
Fat compartment N ₂ /ATA	477	ml @ 100% saturation
Total N₂/ATA	976	ml @ 100% saturation
Water N ₂ at 4ATA	1597	ml @ 80% saturation
Fat N ₂ at 4ATA	763	ml @ 40% saturation
Total N₂ @ 4ATA	2360	ml
Excess water N ₂ at sea level	1098	ml
Excess fat N ₂ at sea level	286	ml
Total excess N₂ at sea level	1384	ml

Take home message

Intravascular gas found in the cadavers of victims of fatal underwater diving accidents is likely to be due to postmortem artefact and does not necessarily indicate antemortem arterial gas embolism.

procedures such as percutaneous lung biopsy.¹¹ However, the practicability of obtaining such information within one or two hours after a diving accident is highly questionable given the retrieval times usually involved.

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