September, 1987, was considered to have cerebral decompression sickness, and he was treated with Table No. 6, resulting in a complete resolution of his symptoms, although subsequently he was still found to be slow in speech and thought. These findings were thought to be normal for him.

I contacted him by telephone at the end of May, and he is certainly still very slow in speech and thought. He has not dived again perhaps he is following the recommendation of Dr Ian Unsworth, Director of the Hyperbaric Unit, however the main reason is that his boat remains impounded somewhere in Victoria! Currently he is unemployed. He plans to take the Inspectors to the High Court, with charges of attempted murder, claiming near drowning because of the constant kinking of the hookah hose while he was being towed, and in addition the cerebral decompression sickness complication, and the fact that his boat had been illegally impounded.

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## ARTERIAL GAS EMBOLISM FROM PULMONARY BAROTRAUMA: WHAT HAPPENS IN THE LUNG?

John Williamson

**ABSTRACT**

The precise sequence of events in the lung parenchyma which precede and accompany arterial gas embolism remain unknown. Alveolar-vascular membrane disruption is still the favoured mechanism. The base of marginal alveoli which sit on pulmonary vascular sheaths appear to be one area of weakness in the face of alveolar overdistension; this does not necessarily explain the access of alveolar gas into the lumen of pulmonary blood vessels. Clinically arterial gas embolism in divers has no clear association with pre-existing pulmonary scarring, pneumothorax, or the depth of the dive. Some association exists, or is suspected, with reduced pulmonary compliance, rapid ascents, and air trapping. Autopsies, are not good at detecting the pulmonary consequences of such events, and diving medical examinations may be missing still unrecognised predisposing factors in some susceptible would be scuba divers.

While there is at present general agreement with the assumption that in arterial gas embolism (AGE) diving gases gain entry into the pulmonary venous system via the lungs, during pulmonary barotrauma of ascent, this event has never been directly demonstrated to date. The pathogenesis of these events remain unconfirmed and yet has direct relevance to both the prevention and treatment of the disorder.

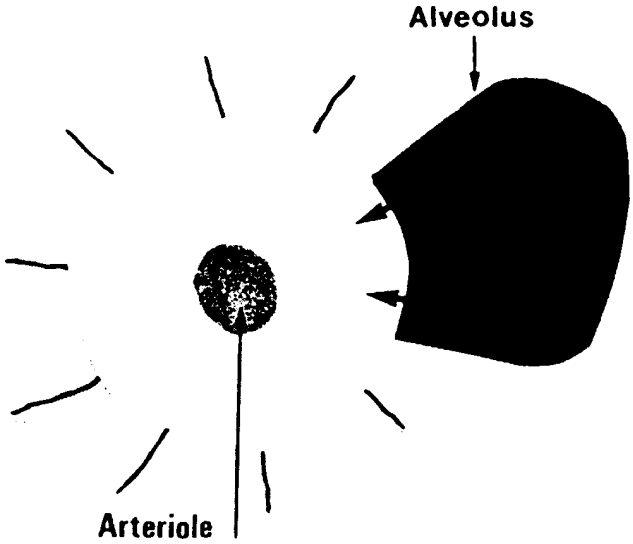


**FIGURE 1** Where an overdistended marginal alveolus abuts on to a perivascular sheath in the lung, a pressure gradient may develop in favour of gas movement through the (disrupted?) base membrane (arrowheads) into the perivascular space (shown distended with gas). This will explain pulmonary interstitial emphysema, but not necessarily pulmonary gas embolism.

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A search of the diving and medical literature will show that some well recognised papers[1,2] make only vague reference to how the gas actually gets into the vascular spaces. The author was able to find only one readily available text that made any attempt to grapple with this aspect of the problem[3]. Here it is suggested that once the first breath is taken (intrathoracic pressure is lowered) following pulmonary barotrauma, any extralveolar gas "can intravasate into torn vessels". (Further light has recently been thrown upon where and how the gas goes, once in the blood stream, by Gorman and his colleagues[4].)

With the steady appearance of published research (albeit mostly on experimental animals) combined with the rapidly accumulating clinical experience of diving medical physicians, who are now coping with the explosive increase world wide of recreational scuba diving, a reconsideration of certain pointers and clinical associations which are beginning to emerge may prove helpful.

**Micro-anatomy of lung parenchyma**

While the vulnerable site to overdistension in human lung tissue remains uncertain, animal work[1,2] suggests that at least one such site is located in the "marginal" alveoli that abut upon pulmonary vascular sheaths (Figure 1).

The delicate and attenuated alveolar-capillary membrane, particularly of the "partitional" alveoli, is a tempting anatomical site to postulate rupture and entry. However the evolving concepts of "thick" and "thin" components of this layer[5] and the existence of pore systems in this membrane[6] (pores of Kohn), together with certain theoretical pressure considerations[2], make this site as a portal for direct intravascular entry of gas an uncertain one at present.

Retrospective human studies suggest that fatal lung parenchymal barotrauma is not consistently related to pre-existing parenchymal scar tissue, nor to fixed parenchymal structures detectable by imaging and/or autopsy techniques[7].

**Analogous non-diving clinical correlates**

As every intensivist knows, the incidence of spontaneous gas embolism via the lungs (excluding direct penetration) in ventilated patients is a rarity. However the other pulmonary barotraumatic events, particularly, mediastinal air, pulmonary interstitial emphysema and pneumothorax, are not. These latter events are known to be associated with conditions of reduced pulmonary compliance and high peak inspiratory airway pressures[8]. Their association with positive end expiratory pressure is less clear.

Cerebral arterial gas embolism (CAGE) arising from direct lung injury (e.g. blast injury, penetrating chest wounds) does occur, although uncommon and is still frequently not thought of by attending clinicians. It usually occurs at the time of the injury, but may occur or recur during management (e.g. ventilation). The resulting central neurological disturbance may be attributed to co-incident injury and/or resultant hypoxia from other co-existing causes.

**Diving related clinical correlates**

As a glance through recent diving medical literature will show, it is now apparent that among recreational divers sudden impairment of consciousness associated with breathing compressed gases is by no means a rare event. It would seem likely that some of these are a result of CAGE from pulmonary barotrauma. Many of these persons recover consciousness and apparent well-being acutely and spontaneously, a fact now rendered comprehensible by Gorman's work[9]. Their subsequent medical course remains unknown, as they seldom present after that to a diving medical, nor perhaps to any, physician. However one would expect significant central neurological sequelae[4,9].

Table 1 outlines the Townsville Recompression Chamber teams' experience with recognised CAGE in scuba divers, since the chamber commenced operation in 1977. Eight (8) of these cases have been dealt with in the last 4 years. Several other episodes of sudden loss of consciousness in the water which were reported to team members during the 11 years could not be followed up.

**TABLE 1**

**The Townsville Diving Medicine Team's Experience Cerebral Arterial Gas Embolism in Divers* 1977-March 1988**

| | |
|---|---|
| Total Number of Cases (all diagnoses) | 68 |
| Total Number of CAGE Cases | 9 |
| CAGE Fatalities | 3 |
| Identified Ascent Problems | 5 |

*****This pattern is similar to world wide experience[11].

From our experience, and from the documented experience of others, we can say that:

1. In sport scuba-divers, the combination of clinically demonstrable CAGE with other forms of pulmonary barotrauma (e.g. pneumothorax, pneumomediastinum and subcutaneous air) is decidedly uncommon. In our experience it is less than 5%. It seems as if the development of any of the latter intrathoracic phenomena "protects" against the occurrence of CAGE.

2. CAGE in scuba-diving invariably occurs in association with an ascent, planned or accidental. CAGE may occur in the absence of any recognised problem during planned ascent, and in divers of all levels of experience with no known or previously detectable (at present) predisposition.

3. There appears to be no association between CAGE in diving and:

   (a) pre-existing scar tissue in lung or pleura[8]
   (b) measured lung size[10]
   (c) depth of the dive.

4. An association does appear to exist between CAGE in scuba-diving and:

   (a) decreased pulmonary compliance[11]
   (b) rapid ascents, irrespective of the depth of the dive. This may perhaps be related to the rate of intrapulmonary gas pressure rise.
   (c) premature airways closure[11] which may occur during forced exhalation during ascent.

It should be remembered that CAGE occurring during breath-hold diving (snorkeling) is well documented[12], although still poorly appreciated.

5. Present macroscopic and microscopic autopsy techniques and training, although improving[7,13,14], remain inadequate for the investigation of parenchymal lung damage in fatal cases of diving-related CAGE.

6. Because of the prevailing ignorance surrounding the pathology of gas embolism in pulmonary barotrauma, and allowing for those cases of CAGE resulting from errors in safe diving technique, diving medical examinations, no matter how carefully performed (and many still are not!), may not be excluding all would-be scuba divers who are medically predisposed to this potentially lethal complication.

Epidemiological, clinical and experimental research into this problem is warranted.

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