ORIGINAL ARTICLES

STUDENT DIVERS, AN AT RISK POPULATION TWO CASE REPORTS

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Introduction

Cerebral arterial gas embolism (CAGE) resulting from pulmonary barotrauma is well documented.[1-3] Predisposing factors cited include inadequate exhalation, uncontrolled buoyant and underlying lung pathology (cysts, bullae).4 A number of accidents however, result from apparently normal ascents with adequate exhalation or normal breathing patterns.[4] The most common presentation of CAGE is an acute cerebral insult occurring within minutes of the diver reaching the surface.[5] Typical symptoms and signs include an altered level of consciousness, sensory and motor changes and chest pain.[4] Spontaneous improvement of these focal neurological deficits frequency occur, however these improvements are not always sustained. Early recompression is the definitive treatment.[4]

A common misconception is that divers only experience difficulties performing deep dives. Air embolism however tends to occur most often in people who dive less frequently and especially in inexperienced divers.[4]

The following two case reports reinforce the dangers of shallow water and demonstrate that student divers are at risk.

Case 1

A 19 year old female, undertook an open water diving course. On days one to three she underwent pool training sessions to a maximum depth of 3 m. Days 4 and 5 involved an overnight trip to the Great Barrier Reef. The first two ocean dives to a maximum of 12 m were completed without incident. On the first dive of the next day she performed a giant stride entry into the water, with her regulator in her mouth. Immediately on surfacing from this she complained of left sided chest pain which was increased in severity by deep inspiration and coughing. She swam to the stern of the boat, unwilling to continue the dive because of the pain. After leaving the water, she was given an analgesic tablet and lay down on her bunk.

Several hours later she sat up and immediately noticed a heaviness in her body. She felt weak down the left side and had no sensation in her left arm and leg. She was unable to speak and felt dizzy. These symptoms persisted unchanged for fifteen minutes and then gradually improved over a number of hours.

On examination at Townsville General Hospital ten hours after the initial incident she was found to be alert and orientated. Her left arm was weaker than the right arm, grade 4/5, and power in both lower limbs was also decreased to grade 4/5. She had anaesthesia to pin-prick and light touch on the left in a C3-T7 distribution.

A diagnosis of CAGE was made and she was recompressed in a Dräger Duocom Portable Recompression Chamber and transferred to Prince Henry Hospital, Sydney.

TABLE 1

RECOMPRESSION TREATMENT REGIME FOR CASE 1
(information supplied by Prince Henry Hospital)

6-10-89	Extended Table 62
7-10-89	Extended Table 62
	Table 61
8-10-89	9 m soak 120 minutes, 30 minute ascent
	9 m soak 105 minutes, 20 minute ascent
9-10-10	9 m soak 120 minutes, 5 minute ascent
10-10-89	9 m soak 120 minutes, 5 minute ascent
11-10-89	9 m soak 125 minutes, 5 minute ascent
12-10-89	9 m soak 125 minutes, 5 minute ascent
16-10-89	9 m soak 125 minutes, 5 minute ascent

The treatment profile is shown in Table 1. Significant improvement occurred during these treatments with complete resolution of her motor weakness, however, there was minimal recovery of her sensory deficit.

Investigations performed during her hospital stay included CT head scan, MRI head scan and chest X-ray, none of which showed any abnormality.

On review one month following the incident, she was found to have residual anaesthesia of the left arm with total proprioceptive loss and she had an abnormal gait. There was evidence of sympathetic disturbance in the left hand. She was referred for continuing physiotherapy.

Case 2

A 25 year old female, also undertook an open water certification course. The class had practised basic skills in their first pool session and all appeared comfortable in the

water. The instructor swam the class into deeper water (maximum 3 m) and then indicated to them that they should surface. Close to the surface the instructor noted her to be struggling and assisted her to the side of the pool. She was unable to pull herself out of the pool and had to be assisted out by the training staff. Witnesses noted that her face exhibited marked left sided drooping. She was unable to move her left arm, nor could she stand unsupported because of weakness in her left leg. Oxygen therapy was commenced. She was placed in a head down position.

When I examined her forty-five minutes later at Townsville General Hospital, a left hemiparesis with left facial paresis was evident, but no sensory abnormalities were detected. Recompression was undertaken, as shown in Table 2.

After this course of treatment the facial paresis had resolved completely. There was however residual spastic changes in the left leg but she was able to walk with the aid of a stick. Minimal recovery of power was seen in the left arm.

TABLE 2

RECOMPRESSION TREATMENT REGIME FOR CASE 2

9-04-90	Extended table 62
10-04-90	18 m soak 60 minutes, 30 minute ascent
	18 m soak 60 minutes, 30 minute ascent
11-04-90	18 m soak 60 minutes, 30 minute ascent
	18 m soak 60 minutes, 30 minute ascent
12-04-90	18 m soak 60 minutes, 30 minute ascent
	18 m soak 60 minutes, 30 minute ascent
13-04-90	18 m soak 60 minutes, 30 minute ascent
14-04-90	18 m soak 60 minutes, 30 minute ascent
15-04-90	18 m soak 60 minutes, 30 minute ascent
16-04-90	18 m soak 60 minutes, 30 minute ascent

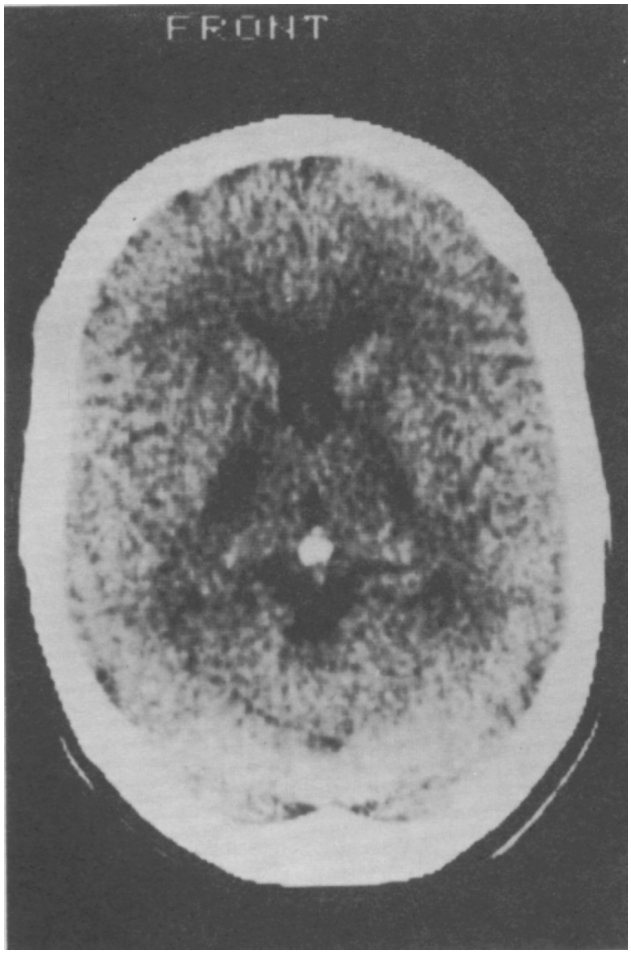

Figure 1. CT head scan of Case 2 showing an area of low attenuation adjacent to the right internal capsule.

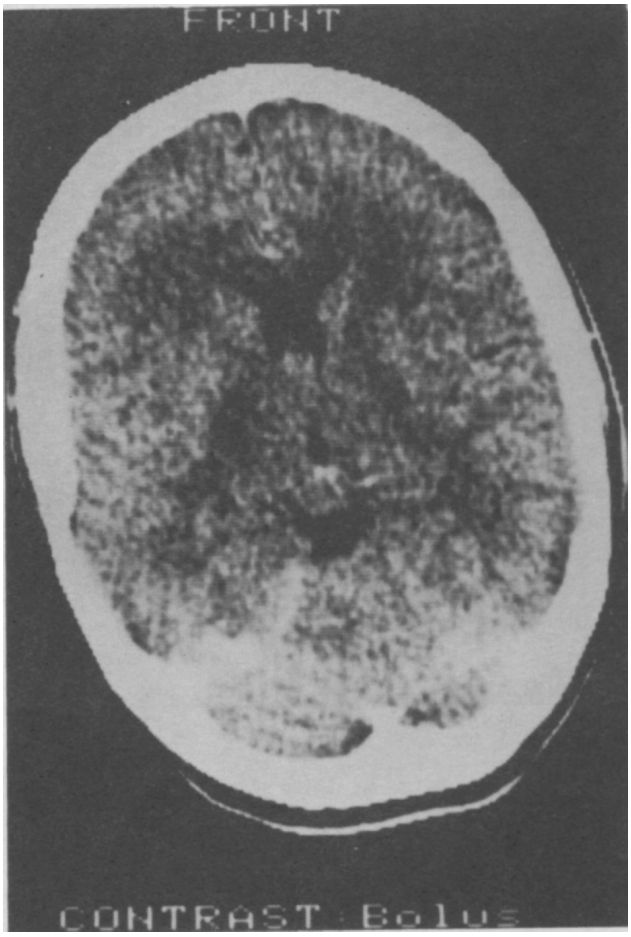

Figure 2. CT head scan, with contrast medium, of Case 2. The area of low attenuation in figure 1 is not enhanced, suggesting a small right limbic infarct consistent with the clinical findings.

Intensive physiotherapy and occupational therapy continues. A CT scan of her head performed eleven days after the incident revealed a moderate sized area of low attenuation adjacent to the right internal capsule (Figure 1). This was not enhanced with any contrast media and no mass effect was demonstrated (Figure 2). These appearances are consistent with a small limbic infarct which can explain her clinical presentation.

Discussion

Underwater panic and exhaustion of air supply followed by an uncontrolled ascent are typical scenarios used by recreational diving instructors to illustrate the origins of CAGE in divers. It is not well appreciated that an apparently uncomplicated swimming pool dive or a boat entry into deep water, while using compressed air breathing apparatus may result in embolism.

During ascent lung volumes will expand according to Boyle's Law and can lead to pulmonary barotrauma. A rise in alveolar pressure of 80-100 mm Hg is sufficient to force air into pulmonary capillaries.[4] This corresponds to an ascent from a depth of one metre to the surface with the lungs fully inflated and gas trapping present.

The sequence of events in Case 1 suggest that the diver was carried a metre or more below the surface (probably because she was overweighted) following her giant stride entry, took an involuntary breath in response to the stimulus of sudden immersion and was then swept rapidly to the surface by her partially inflated buoyancy compensator before she could exhale. Such a sequence easily explains her CAGE. I have not been able to find any previous report of this complication of giant stride entry.

Case 2 was observed by her instructor to exhale during the ascent. The ascent rate was controlled and a follow up chest X-ray revealed no gross lung pathology. In the absence of such predisposing factors for CAGE, the role of localized air trapping as opposed to a generalized overpressure injury must be considered. She was noted to be in difficulty in mid water, and, knowing the ascent was otherwise uncomplicated, this suggests embolization occurred before she surfaced. Walker[7] reported two fatal cases of mid-water embolization occurring in open water.

The use of a head down position in the first-aid management of CAGE is debatable. Many authors support the use of this posture.[2,8-10] Anecdotal reports have documented sudden deterioration in the condition of victims with a change in posture from head down to head up.[10] This clinical observation is supported by animal studies in which gas bubbles were observed to travel "backwards" up the aorta in head up animals against the direction of blood flow.[10] This would indicate bubbles distribute according to their buoyancy at least in the great vessels. However, increasing opposition to the head down posture is emerging. Results from animal studies by Dutka et al.[11,12] have led them to conclude a prolonged head down position following CAGE results in increased intracranial pressure and blood brain barrier damage despite hyperbaric therapy.

The two cases presented here do not illustrate any advantage of a head down posture over a horizontal posture. Case 1 certainly shows that CAGE victims should not be sat up, while Case 2 did not demonstrate any resolution nor deterioration of symptoms or signs with a head down position. The poor response of this diver to recompression highlights the inadequacy of conventional treatment to ameliorate the known secondary effects of gas bubbles on vascular endothelium and blood constituents.[13]

Both divers have been left with significant neurological abnormalities which limit their employment and interfere with their day to day activities. Their medical histories had no identifiable risk factors which would have identified them as being at increased risk for CAGE.

Conclusion

These two cases demonstrate that CAGE can occur in shallow water. Prevention requires that instructors emphasise to their students the importance of exhaling immediately inhalation has been completed. Correct weighting of students so that they are neutrally buoyant on the surface, will prevent deep descent with giant stride entry.

Acknowledgements

I wish to thank Dr G. Gordon, Dr V. Callanan and Dr D. Gorman for their editorial assistance and M. Shapter for the medical photography.

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This paper was written when Dr R M Walker MB.BS., Dip DHM., was holding the position of Locum Director of Hyperbaric Medicine at the Townsville General Hospital, Townsville, Queensland, Australia.

CLINICAL MANIFESTATIONS OF THE DECOMPRESSION ILLNESSES

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Introduction

"Many cases are on record to show the danger of lost time".

This sombre warning first issued in 1982[1] still holds good today. Despite training of both amateur and commercial divers in the recognition of decompression illness, delays at all stages are all too frequent. The implications in the light of the work of Palmer, Calder and Hughes are obvious in attempting to avoid permanent residual spinal or cerebral damage.[2]

Dysbarism embraces all the illnesses caused by changes of pressure and volume: barotrauma and decompression sickness.

Barotrauma

Changes in volume in air or gas containing spaces cause the largest proportion of morbidity and mortality, by tearing lung tissue and leading to cerebral arterial gas embolism (CAGE). This is seen in its purest form in submarine escape training where there has been no prior inert gas load taken up by the tissues. In the diving context, the distinction between arterial embolism and decompression sickness may be less clear cut when there is an inert gas tissue load as well. Collapse and unconsciousness, with or without convulsions may occur (the commonest presentation in the Royal Naval submarine escape tank training series), due to gas in the cerebral or coronary arteries. This usually occurs on ascent or immediately on surfacing and the patient may die before being recompressed.[3]

A second form may have variable symptoms, cortical in origin and may respond to recompression initially but relapse later and a third form may make a full recovery on recompression.

Where there is a fast or uncontrolled ascent followed by a rapid onset of serious symptoms, arterial gas embolism should be suspected. This, in most cases, will have occurred within 5 or at the outside 10 minutes after reaching the surface.

The following diagnostic criteria are suggested.[4]
1 Collapse or unconsciousness occurring without warning immediately or within the first few minutes after decompression.
2 Inco-ordination.
3 Confusion.
4 Weakness or paralysis of limbs.
5 Visual disturbance.
6 Unilateral paraesthesiae.

An unresponsive stupor, possibly with eyes open, has been described.

Decompression Sickness

AETIOLOGY

Gas as bubbles in the circulation or separated "autochthonous" gas in tissue can occur when local supersaturation is reached during decompression.[5-7] Extra- vascular bubbles can cause damage by occlusion of the circulation with tissue hypoxia, disruption of cells, compression of adjacent tissues as in the spinal cord, tearing of tissue or tissue planes. Intravascular bubbles can trigger a cascade of secondary events. These include activation of complement, haemorrhage or clotting, and respiratory changes causing reduction of venous drainage of the spinal cord.[8] The differing aetiology can explain both the quite bewildering variety and severity of presenting symptoms and signs described in decompression sickness (DCS).