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*Address for correspondence*
*Dr Douglas Walker, Project Proteus, P.O. Box 120, Narrabeen, New South Wales 2101, Australia.*

## AN INTERESTING CASE OF DECOMPRESSION ILLNESS

Neil Banham

During the final dive of her initial training course a 35 year old female novice diver who developed constitutional and neurological symptoms. As the history of the incident, obtained on admission to the Emergency Department was unclear, the narrative here was compiled from the patient when she presented to hospital, on completion of treatment and at follow up a month later, from the diver's buddy and also from thedive master.

The dive which resulted in the presenting problems was to a maximum depth of 4.4 m for a total dive time was 37 minutes. Of this ten to twelve minutes at least was spent on the surface for instruction and most of the dive was spent around 2 m practising underwater skills such as mask clearing and controlled octopus ascent. Such a profile is well with in all recognised decompression tables and there was no suggestion of a rapid ascent.

Before entering the water she had been quite well. There were some problems with her new mask leaking and slow clearing of her left ear. Some nausea developed during the dive.

Her buddy reported later that the diver complained of feeling absolutely exhausted while she was snorkelling to shore but her was speech normal at this time. It was noted that, when she left the water, she was somewhat unco-ordinated but she was able to carry her gear up to the bus, wash it and to stow it. She complained of feeling unwell with nausea and headache.

At the dive shop, about an hour after the dive, she complained of feeling "weird" and had to sit down. A sensation of numbness developed over the dorsum of her left hand which progressed to paraesthesia extending up her left arm and there was some tingling on the back of her right arm.

The dive master reported that although she was able to answer questions appropriately he was concerned about the progression of symptoms so he took her by car to Fremantle Hospital, some five minutes away.

On examination in the emergency department she was noted to be alert but confused. Her Mini Mental State score of 19/30 indicated a significant psychometric deficit. Peripheral nervous system examination while she was recumbent was normal and while on oxygen in the emergency department her paraesthesia resolved.

On being stood up to assess her co-ordination it was noted that her heel-toe gait was poor, she was unable to perform a sharpened Romberg test and her overall condition deteriorated. She became drowsy, confused and agitated. Her speech was unintelligible. She had been erect for no more than thirty seconds

The differential diagnoses considered were cerebral arterial gas embolism (CAGE) and decompression sickness (DCS). DCS was thought to be unlikely because of the absence of a significant nitrogen load. The possibility of paradoxical embolism of venous bubbles through a patent foramen ovale was later excluded by a normal bubble contrast echocardiography.

Against CAGE were the absence of a history of rapid ascent, no clinical or radiological evidence of barotrauma and the long delay before obvious symptoms. CAGE is usually almost immediately apparent, although delayed cases have been reported many times. Rapid

deterioration on standing has also been reported many times and is presumed to be due to redistribution of bubbles under the influence of gravity.

I would like suggestions about the diagnosis from the audience and then I will tell you what actually happened.

**Audience participation**

Veale

You did not comment at all about her ventilation nor whether she had a normal $PAC0_2$. I see a lot of people referred with funny neurological symptoms and syndromes who are hyperventilating and I just wonder whether this woman was a nutter or not. I think you can clearly exclude DCS on the basis of her nitrogen load. It is not at all impossible that she could have a cerebral arterial gas embolism from barotrauma as a silent event and in the absence of detectable abnormality in the lung.

Banham

When she presented she was not hyperventilating. She had no evidence of carpopedal spasm. However we did not do a $PAC0_2$.

Bove

It is a strange exercise to propose that this woman had decompression sickness. First of all if it was decompression sickness it was purely cerebral decompression sickness and we are not even sure that exists as an entity, let alone in a situation where somebody comes up from four metres.

To me this is a clear cut classic case of arterial gas embolism following pulmonary barotrauma. The woman was a novice diver using scuba doing things like octopus breathing, ascents and descents. It is very easy to slip for a few seconds and ascend one and a half metres and get an air embolism, even though she would have been totally unaware that it occurred. I think the obvious first diagnosis ought to be CAGE and the patient ought to be taken as quickly as possible, even at the beginning of the behavioural changes, to a chamber for treatment. Again, I think it is a futile exercise to try to propose decompression sickness would be even a possibility in this case.

Banham

Her symptoms are initially quite vague and all of this obviously when it was pieced together became much more apparent. Other things we thought about were did she have a problem with her gas mix but carboxyhaemoglobin was less than one percent. Salt water aspiration crossed our minds but there were no other clinical features to suggest that and a chest x-ray was normal. So, fortunately the diagnosis was made fairly apparent on standing the patient up, despite this being about an hour and a half after the completion of her dive. I was just wondering if anyone else in the audience had actually seen this occur before.

Veale

It is a well documented phenomenon and certainly Des Gorman has a handful of delightful stories he can tell people in the same way. One of the ones I remember vividly is of a female diver at Stony Cove in the UK who lost consciousness at the surface following an abort from a deep rescue dive. She was brought ashore and was fine and regained consciousness and then they stood her up and she promptly died.

Banham

This woman had actually been standing, washing down her gear, sitting in a bus and walked into the dive shop.

Davis

Secondary deterioration is common, and fits very nicely with the modern concept of what is happening to the cerebral vessels as a result of gas embolism. Anybody who has read Des Gorman's papers will appreciate that about this time it is classic for the secondary deterioration in cerebral blood flow. Deterioration does not necessarily have anything whatsoever to do with a residual gas phase in the cerebral circulation, but is a reflection of the pathological injury that occurs as a result of the gas going through in the first place. The shallowest diver I have ever had in the chamber in Christchurch was in two metres from a swimming pool. He was a diving instructor who should never have been diving. He was grossly obese, had a symptomatic hiatus hernia with very clear evidence of recurrent pulmonary aspiration at night who, getting out of the pool during a class, became very dizzy on the pool side and fell over. He walked in to hospital. He began with very vague symptoms, poor on his Mini Mental Score and absolutely hopeless at sharpened Romberg and certainly that assessment is appropriate, in these people it was often the only physical finding. I have seen that in quite a number of divers and I expect Fred Bove has too.

Bove

The Dan reporting system received a case of a woman standing at the shallow end of a pool during a scuba class, water about up to her mid-chest, a mask was put down by her feet in the pool and she was asked, with her scuba tank on, to lie down in the pool, put the mask on, clear it and come up. She embolised and that was about four feet of water. Clear cut classic air embolism. I think that in a pool or shallow water with scuba gear on CAGE is got to be the primary diagnosis any time mental change is seen.

Unidentified speaker

I remember that when I was initially trained as a diver in the Navy in 1958 we had to do an exercise, with a

re-breather set, in a twenty foot tank where we had to take the full face mask off and then swim to the surface. In that short distance I well remember having a gush of cold water on my face, coming half way to the surface, feeling a great distension in my lungs and remembering, at that stage, now is the time to breath out.

Veale

Some of the original studies involved inflating the lungs of cadavers clearly showed lung rupture with a one metre or a 1.3 m ascent so transthoracic pressure changes of 75 to 100 cm of water pressure are enough to rupture lungs.

Banham

The phenomenon of arterial gas embolism from shallow depths is well recognised. What I am really trying to emphasise is the delay in onset of her symptoms which is somewhat atypical of acute arterial gas embolism, and then her sudden dramatic deterioration when she stood up some hour and a half later. Has anyone else actually seen a delayed significant deterioration such as that, rather than acute one?

Veale

Those of you that run, or have run, recompression chambers, hands up if you have seen delayed presentation of CAGE. That is four of us.

**Conclusion**

Despite the delay in onset the diagnosis was made of cerebral arterial gas embolism (CAGE), and urgent arrangements were made for recompression therapy. She was kept supine and given one hundred percent oxygen by non- rebreathing circuit using a mask with a tight seal. Intravenous fluids were started. By the time she was taken into the chamber she had improved but not back to the state she had been in before she was stood up. She was treated with a modified Table RN62 and made a full recovery.

The modifications to Table RN62 are that there is twice the time (one hour) decompressing from 18 m to 9 m at a rate of 0.15 m/minute, with a five minute air break midway. Instead of having periods of an hour on oxygen they are reduced to twenty minutes by five minute air breaks. It is not routinely extended either at 18 m or at 9 m. The total elapsed time is 5 hours and 20 minutes. Since the introduction of these modifications we have not had to extend at either 18 m or at 9 m.

There was no deterioration after the initial treatment. Because of persistent tiredness two follow up treatments (RN61) were given on subsequent days. After discharge the patient felt tired but was otherwise asymptomatic.

The patient was advised of the possible risks of further diving and chose not to continue diving.

*Dr Neil D G Banham, MBBS, FACEM, is attached to the Hyperbaric Medicine Unit, Fremantle Hospital, PO Box 480, Fremantle, Western Australia 6160.*