10. Oxygen therapy is only emergency treatment. If symptoms are suggestive of decompression sickness and they respond either partially or temporarily to 100% oxygen, then that should be sufficient to convince the attendant that recompression is essential.

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ANOTHER WAY TO GET BENT

John D.McKee

This 32 year old patient had been abalone diving for sixteen years, mostly just south of Nowra and south of Eden. He had had seven abalone diving days during 1988, of which four were in March, and his most recent dive had occurred south of Eden on 21.3.88.

The history

His diving profile for the day had involved an initial dive to 18 m for 45 minutes, after which he brought his abalone bag back to the boat. He then immediately descended again. The second dive was to 21 m for 70 minutes, although in fact he did admit later that he spent approximately 10 minutes of the 70 minutes at 27 m. He then surfaced, gave his abalone to his sheller, and immediately descended to between 6 and 9 m where he spent the next ten minutes.

I saw this patient and his sheller on 23.3.88, and the sheller said that while he was aboard their boat at Cape Howe off the New South Wales-Victorian border, a boat, a shark cat, had approached at great speed, rammed their boat. The shark cat driver jumped aboard, pushed the sheller out of the way, and then drove their boat off at high speed, dragging the diver along below for a distance of about 100 metres. During this alleged episode, the sheller kept yelling out that there was a diver below.

Eventually, the "pirate" driver stopped the boat, allegedly indicated that he was a "Fisheries Inspector", and dragged the diver out of the water. He then drove the boat and its occupants to Gabo Island. Subsequently the patient and his sheller were interviewed for two hours, by Inspectors, and the boat was confiscated.

That night, 21.3.88, the patient complained of fairly severe pains in his hips, shoulders and right elbow, and he had a troublesome headache. He was seen by nursing staff at a local hospital, and apparently he was given oxygen to breathe for 20 minutes, but at no stage was he seen by a doctor.

The following day he was no better, and when referred to me the next day, 23.3.88, he seemed to be rather vague with a poor memory, and most of the information and history I obtained from his sheller, who was an amateur diver.

His mate had known him for some years, and he had observed a pronounced loss of alertness, lethargy, definite memory loss, a slowness in doing all things, especially manual activities, and he had observed the diver's hands shaking from time to time. The diver complained of a whistling sound in both ears, and he still had pain in both hips, both soulders, the right elbow and the upper abdomen.

On examination the patient was fully conscious, there was no gross neurological abnormality, he walked with a "wide base", but he was tender in both groins, and I suspected his abnormal gait was due to tearing of ligaments when he was towed through the water. His response to questions and his response to commands was slow, but all movements were normal, there was no obvious muscle weakness, but there was a suggestion of diminution of light touch perception in the lower limbs.

Treatment

I formed the opinion that this patient almost certainly had cerebral decompression sickness, and I arranged for his transfer by air ambulance to Sydney, by a fixed wing aircraft, pressurised to sea level, during which time he received 100% oxygen.

Following his arrival at the Prince Henry Hospital in Sydney, he was treated in the hyperbaric unit recompression chamber for 265 minutes at 2.8 atmospheres. At the commencement of treatment, he still seemed to have poor short term memory, he was complaining of a buzzing"in the left ear, fairly severe pain in his right shoulder, hip, and abdomen. He complained of some paraesthesia and numbness in both legs.

After 10 minutes at depth he indicated that he was feeling much better, his shoulder pain had almost completely disappeared, his hip pain was 50% better, and his abdominal pain had ceased.

Six hours after the commencement of treatment he had voided urine, he appeared to be much more alert, he had lost his headache, and he only had minimal residual pain in the right hip. As well, there was only minimal residual auditory hallucination.

Finale

This diver, who had had normal long bone x-rays in

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.

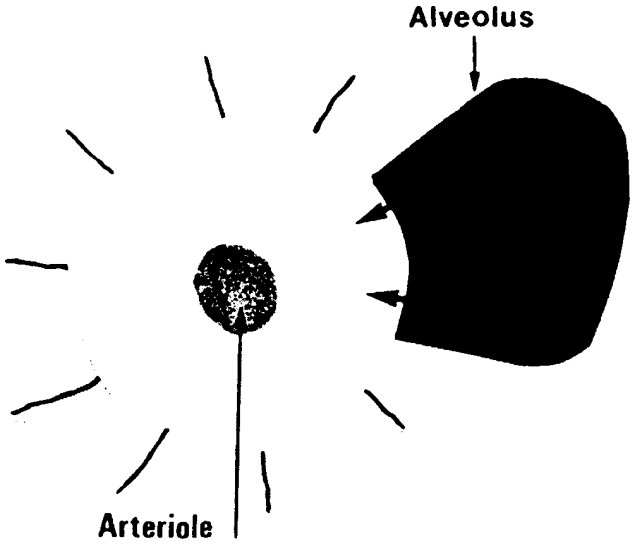

FIGURE 1 Where an overdistended marginal alveolus abuts on to a perivascular sheath in the lung, a pressure gradient may develop in favour of gas movement through the (disrupted?) base membrane (arrowheads) into the perivascular space (shown distended with gas). This will explain pulmonary interstitial 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.