serious misadventure occurring in such good circumstances (there was only a slight slop) so they never really "registered" the significance of the calls for help. Second, discussion with the victim revealed that his scuba diving experience had been 20 years previously. He had also omitted to reveal that in the past three years he had become a chronic asthmatic, a fact he endeavoured to conceal from everyone whenever possible. His surface problem apparently resulted from this cause. Incidentally, his wife had observed everything which had occurred and even so remained reluctant to reveal this important fact.

Now I have learned to not only ask about the dates of the diving experience of those who seek to borrow my equipment, I ask about their health most carefully!

RETINAL ISCHAEMIA DUE TO HYPERBARIC OXYGEN

Karin Herbstein and John Murchland

There has been a recent resurgence of interest in the use of oxygen at increased partial pressures, both by divers and for medical conditions, including the treatment of Multiple Sclerosis by medical and non-medical persons.

It has been assumed that the conventional pressures and times at which divers or patients breathe 100% oxygen are safe.

For 100% oxygen the maximum recommended safe pressure (to avoid CNS oxygen toxicity) according to Edmonds, Lowry and Pennefather, and other authorities, is 10 metres or 203 kPa (2 atm) absolute pressure.[1,2] The time element is of course also relevant.

Complications have been reported but mainly when breathing hyperbaric oxygen beyond recommended safety limits.

However as demonstrated in this case report, and more extensively described in another recent article,[3] a healthy diver developed retinal ischaemia at a “safe” level of hyperbaric oxygen. Review of the literature suggests that this complication could proceed to a more permanent loss of vision.

As there is the notorious variability of susceptibility to nitrogen narcosis and decompression sickness, not only between different divers but for the same person on different occasions, similarly there is great variability to the vaso-constrictive influence of hyperbaric oxygen.2 Also the young[4] and the healthy with no vascular disease[5] and migraine sufferers[6] are more susceptible to vaso-constriction.

Case Report

The patient was a fit healthy man aged 44, a Scuba-diver, who had accompanied multiple sclerosis patients during their one hour treatment at 2ATA in a chamber. He did this on ten consecutive days, on two occasions using a spare oxygen mask and so became exposed to 2 ATA oxygen for two one hour periods. Several days later he became aware of a visual defect in his left eye. This was found to be due to a retinal “cotton-wool spot”, whose position in the eye was consistent with the field defect that was plotted.

Discussion

A "cotton-wool spot” is an ischaemic area in the retina. The greater the partial pressure of oxygen inhaled the greater the vasoconstriction. The retinal vasculature is the most sensitive to the vasoconstrictive influence of hyperbaric oxygen.[5] Paradoxically therefore, hyperbaric oxygen, instead of causing increased tissue oxygenation, may cause ischaemia due to vasoconstriction. Ischaemia, a temporary state due to hypoxia due to decreased blood flow, may progress to infarction, or death of the tissue, if sufficiently severe or prolonged vasoconstriction occurs. Blindness has been reported from vaso-constriction due to migraine.[6] Bilateral blindness has also been reported after prolonged inhalation of 80% oxygen at normal atmosphere pressure due to a central retinal artery occlusion.[8]

Oxygen toxicity, both from pulmonary and CNS effects, is well documented. It is for this reason that rebreather units using 100% oxygen are not advised for use below 25-30 feet of seawater, the dangers being increased by adverse factors such as cold, fatigue, raised CO_2 tension, or anxiety. Pulmonary considerations limit the therapeutic use of oxygen, though the inclusion of "air breaks” is believed to reduce the risks. For chamber therapeutic use 3 ATA is the usual maximum, while in-water use at 1.8 ATA (9m) is allowed. The growing availability of apparatus allowing 100% oxygen at 1 ATA (on land or in a boat) as an initial management option may not be totally devoid of risk, apart from the possibility of fire or explosion. There has been an increase in use of 100% oxygen at 2 ATM, for medical and non-medical treatment, with no reported complications at these concentrations and pressures.

We emphasise, as illustrated by this case, that due to the exquisite sensitivity of retinal vessels to the vaso-constrictive influence of hyperbaric oxygen and due to the possibility of a paradoxical situation of ischaemia secondary to this vasoconstriction, there is a need for caution and awareness of the possible ocular complications of high oxygen concentrations especially at increased atmospheric pressures, even within the recommended “safety” limits, and especially in the young and healthy and in migraine sufferers.

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EDITOR'S NOTE

The authors made a MEDLARS search of the literature before writing their paper and were unable to discover any medically reported cases (as contrasted with medico-legal cases, where a judge decides) of blindness following hyperbaric oxygen therapy. Any readers who know of cases of blindness following short term hyperbaric oxygen therapy in adults are asked to communicate with the authors.

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AN UNRECOGNISED BEND

A case report and comments based on the records of the New Zealand Underwater Association

Douglas Walker

The victim was a 38 year old male who had spent several days diving to 20-30 feet, undertaking 2-3 dives a day, on the East coast of the North Island. On the morning in question he had dived at 30 feet for about 90 minutes. He then surfaced and went by boat to a spot where he had been told the depth was 70 feet. He dived to a depth he felt was in excess of 100 feet for about 10 minutes but he had neither watch nor depth gauge. He then made a rapid ascent to 15 feet where he spent a few minutes before surfacing.

Later that day he drove to an inland town which was at an altitude of 1200 feet. On arrival he noticed some numbness and tingling in his feet and felt cold. The next day he returned to the coast and had a shallow dive, to 20 feet. However his symptoms persisted, so he consulted a local doctor. The doctor told him he had "A touch of the benz" and should come back if he felt dizzy.

He then returned home, where he sought the advice of another doctor. This practitioner told him he had "a bit of a bend" and he was reassured that he would get better. He then advised the victim to go for a deep dive in the nearby Lake Taupo, to go to 100 feet and come up in stages. Six days after the initial onset of symptoms the victim carried out a decompression dive, using a marked line, in fresh water to 100 feet. There was no improvement in his condition.

Several days later his employer, himself a diver, contacted the medical team on duty at the Devonport Naval Base. Their opinion was that it was unlikely to be decompression sickness (DCS) and that recompression would be of no value to him at this late stage even had the problem been DCS. He again consulted his own doctor and fourteen days after the onset of his symptoms he was referred to a Base Hospital for investigation. Subsequent neurological investigations were stated to reveal that he was suffering from a condition which is not related to diving and that his problems were not therefore caused by decompression sickness.

COMMENT

This report is submitted for several reasons, the most important being to remind divers of the critical importance of safe diving procedures. Such includes an intelligent awareness of the diving related problems which can occur, married to a (cynical?) awareness that THEY (the divers) may have to both make and defend any diving medicine diagnosis. The following Critical Points in the story are identified as a basis for consideration:-

1. The diver was careless in having neither watch nor depth gauge and nevertheless diving in an unknown-depth area. There is nowhere any mention of a buddy and the probability arises of this being an "experienced" but "can do" type of diver. A rapid ascent increases the risk of DCS.

2. The onset of symptoms after arriving at altitude was significant.

3. The doctor told him he had "benz". It was HIS (the diver's) responsibility to get informed advice as the symptoms indicated the possibility of spinal DCS. As every diver knows, recompression is the specific therapy. Like most divers he did not apply this information to himself.

4. The second doctor told him his symptoms would get better and that he should treat himself by a "therapeutic" dive to 100 feet in the lake. The diver should have been aware, even if the doctor was not, that such attempts at treatment almost invariably worsen the problem. He should at this stage have been completely aware of the need to contact Devonport Naval Base and done so on his own initiative. He does not seem to have discussed his troubles with any other divers.

5. His employer was the first to take the correct course of action. It is not known what tale the victim presented to the Naval Base over the phone but many diving doctors would advise a trial of recompression even at such a late stage for a possible spinal bend.

6. Neurological investigations would reveal the results of spinal cord damage, not the actual cause. With respect to the neurologist, unless he was aware of diving-related CNS problems his opinion on such matters would be secondary to the history-supported probability of DCS.