ORIGINAL PAPERS

DOPPLER DETECTION OF CIRCULATING BUBBLES IN ATTENDANTS, DECOMPRESSED ON OXYGEN, FOLLOWING ROUTINE HYPERBARIC TREATMENTS.

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Abstract

A pilot study was carried out to examine the incidence of doppler-detectable circulating bubbles in hyperbaric chamber attendants following routine chamber treatment profiles. Bubbles were detected in 44% of exposures at 2 ATA, and 68% of exposures at 2.8 ATA, however most were of low grade only. The consequences of chronic exposure to low-grade circulating bubbles require further investigation.

Introduction

In Hyperbaric Medicine Units around the world, recompression chamber attendants are regularly exposed to increased ambient pressure, sometimes for prolonged periods, and sometimes more than once daily. The treatment tables which are in widespread use have been empirically developed over a number of years, based on both their therapeutic efficacy, and their safety for the attendants. For most conditions treated with hyperbaric oxygen, a pressure of between 2.0 and 2.8 atmospheres absolute (ATA) is used in order to provide optimal therapeutic oxygen partial pressures, and the time of exposure of air breathing attendants to these pressures is limited to comply with the "no-decompression" bottom times quoted in the more conservative dive tables. In some units, including the Royal Adelaide Hospital, the attendant also breathes 100% oxygen during the ascent to facilitate off-gassing of the nitrogen load accumulated during the period of breathing air under pressure.

The reported incidence of clinical decompression illness (DCI) in chamber attendants exposed to these treatment tables is very low. At the Royal Adelaide Hospital Hyperbaric Medicine Unit, 3900 treatments were performed between 1985 and 1991, and two cases of DCI occurred in the chamber attendants. However, a review of the literature shows that using doppler ultrasound, bubbles have been detected in the circulation of subjects exposed to pressures of the order of those routinely used as treatment tables. Eckenhoff[1] found that all subjects exposed to 1.7 ATA pressure for 48 hours developed doppler-detectable circulating bubbles after decompression over 2 minutes (3.5 m (11.5 ft) per minute , although none developed clinical symptoms of DCI. At the Defence and Civil Institute of Environmental Medicine (DCIEM) in Canada, where doppler studies have been performed on all their routinely used diving tables, circulating venous bubbles have been detected in nearly all the recommended "safe" dive tables. They consider that dive tables which produce bubbles of grade 2 or less in 50% of the subjects constitute an acceptable risk, as the reported incidence of decompression illness associated with bubbles of this grade is very low.[2]

Many human studies have been carried out attempting to define a relationship between circulating bubbles and clinical manifestations of DCI. It was initially thought that doppler monitoring could be used to control the decompression profiles and prevent DCI, but the studies performed by Eatock and Nishi[2,3] showed that bubbles tend to be detected after the decompression has been completed, usually up to an hour later, with a peak incidence at 2 hours after decompression.

The implications of subclinical bubble formation are not well understood. It has been suggested that repeated exposure to circulating bubbles may produce cumulative deleterious effects over time, especially in the central nervous system.[3-5] This may occur in the absence of any acute symptoms of DCI as an insidiously progressive loss of higher mental function, such as has been reported in some occupational (professional) saturation divers.[5]

The pathophysiology of DCI is now thought to be complex and involve bubbles both in the blood and tissues, as well as complement activation, vascular endothelial effects, histological and haematological changes. The detection of intravascular bubbles is only an indicator that a decompression stress has occurred. Intravascular bubbles do not mean that clinical DCI will occur.[3,6,7] Susceptibility to DCI appears to be an individual matter, as some people will develop DCI with low-grade or no detectable bubbles, whereas others have higher-grade bubbles with no evidence of clinical DCI.[3]

Although most hyperbaric treatment profiles are of much shorter duration then those studied by Eckenhoff, the pressure to which our attendants are exposed is greater, 2.0 - 2.8 ATA. We were interested to discover if any circulating bubbles were detectable following decompression after our routine chamber treatments, as at the time of planning this investigation (1992) we had been unable to find data about the incidence of bubble formation following short-term exposures at these pressures. A study was performed to examine recompression chamber attendants after their routine hyperbaric exposures, to determine if circulating bubbles are induced following existing treatment tables.

Method

The study, carried out in 1992, was approved by the Royal Adelaide Hospital Human Ethics Committee. Subjects were the nurse attendants and medical staff who were then carrying out the routine chamber treatments. All subjects were passed fit for hyperbaric exposure by the hospital's Occupational Health and Safety Unit, and entered the study with informed consent. None of the subjects had had a hyperbaric exposure for at least 24 hours before the monitored exposure.

The subjects were assessed with a non-invasive doppler probe before each "dive", to obtain a baseline recording of background sounds. The sites used for the doppler recordings were:

1 Over the left sternal edge in the fourth intercostal space, where pulmonary artery flow is best heard.
2 Over the right and left subclavian veins.

Doppler recordings were taken at the precordial site for one minute at rest, and then for 30 seconds after each of three deep knee bends, and at the subclavian sites for 30 seconds at rest, and then for 15 seconds after each of three hand clenches. All doppler records were taped on audio-cassettes.

No subject had any doppler-detectable bubbles before their hyperbaric exposure and all subjects were used as their own controls.

The subject then carried out a routine treatment dive in the recompression chamber. This consisted of either

10:90:30. (2.0 ATA (10 m) for 90 minutes on air followed by a 30 minute ascent to surface (1 ATA) on 100% oxygen.)

or

18:60:30. (2.8 ATA (18 m) for 60 minutes on air, followed by a 30 minute ascent to surface (1ATA) on 100% oxygen).

After the treatment had been completed, the subject was monitored again using the same procedure. Recordings were made immediately on leaving the chamber, and at 30 minutes, one hour and hourly intervals thereafter until no bubbles could be detected. No participating attendant was exposed to pressure more than once in 24 hours.

Two blinded observers individually assessed the tapes and any bubbles detected were quantified using the method of Kisman and Masurel (K-M code).[2,8]

Results

Tape recorded doppler sounds were obtained from 37 chamber treatments, with a total of 21 subjects. There were eighteen 10:90:30 runs, and nineteen 18:16:30 runs. The doppler findings are shown in Table 1. During the study one subject, who had no doppler-detectable bubbles at any time, developed clinical static neurological DCI, manifested by short term memory loss and higher mental function impairment (especially calculation skills), headache and impaired co-ordination, which was successfully treated.

TABLE 1

SUBJECTS WITH DETECTABLE BUBBLES AFTER EXPOSURE TO PRESSURE

Treatment	Number of subjects	Maximum Bubble Grade 0	I	II	III	IV
10:90:30	18	10	4	3	1	0
18:60:30	19	6	7	4	2	0

Discussion

These results show that circulating bubbles are formed in RCC attendants following routine treatments. Bubbles were formed in 8 of 18 exposures (44%) at 2 ATA, and 13 of 19 exposures (68%) at 2.8 ATA. These bubbles are mostly of low grade only (grade 1 or 2) and in the one case of DCI recorded, no bubbles were detected at all. No bubbles or bubbles of grade 2 or less were found in 17 of 18 exposures (94%) at 2 ATA, and 17 of 19 exposures (89%) at 2.8 ATA, which fulfils the DCIEM criteria for a decompression profile with an acceptable risk of DCI.

A Chi-squared analysis comparing the incidences of bubbles detected in the 10 and 18 m tables showed no significant difference. The sample size was too small to detect a difference. A difference, if any, would be not be detected until the sample size approached 150.

It is interesting that bubble formation has occurred following these very conservative tables, indicating that a significant decompression stress has occurred. Those subjects with detectable bubbles, even of a higher grade, reported no symptoms of DCI. The single subject with clinical DCI had no doppler-detectable bubbles.

This pilot study shows that with conservative treatment tables, in more than half the attendants exposed, doppler-detectable bubbles are occurring, although they are of low grade. This may have important consequences regarding the safety of repetitive chamber "diving" by hyperbaric attendants. It would seem prudent to adopt work practice standards which minimise the exposure of chamber attendants to even low-grade circulating bubbles,

as the consequences of long term repeated exposure to asymptomatic low-grade bubbles are still unknown. It is the practice at the Royal Adelaide Hospital to limit the number of dives in the chamber to one a day for each attendant, on a maximum of 4 days a week. Certainly, more than one hyperbaric exposure per day would not be recommended, although we have not examined the incidence of Doppler-detectable bubbles under this circumstance.

Further investigation is warranted examining other treatment tables currently in common use. More detailed higher mental function testing may be useful in excluding any "subclinical" deleterious effect of exposure to these low-grade bubbles, and confirming the safety of the treatment tables currently in use.

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FUNCTIONAL ENDOSCOPIC SINUS SURGERY IN DIVERS WITH RECURRENT SINUS BAROTRAUMA.

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Abstract

Advances in endoscopic technology combined with computerised tomography have dramatically changed our understanding of sinus disease. Functional endoscopic sinus surgery (FESS) significantly enlarges the maxillary sinus ostia, the drainage pathways from the frontal sinuses and removes obstructed or diseased ethmoid cells. If necessary, the ostia of the sphenoid sinuses can also be enlarged. FESS techniques have treated successfully aviators with recurrent sinus barotrauma. Divers who suffer from recurrent sinus barotrauma should also benefit from these techniques. This paper documents early FESS experience with two divers suffering from recurrent sinus barotrauma.

Introduction

Divers are at risk of developing sinus barotrauma while diving due to repeated large fluctuations in pressure. Acute sinus barotrauma which commonly occurs following a recent viral upper respiratory tract infection responds to conservative medical management and is usually self limiting. Infrequently, recurring attacks of sinus barotrauma may occur. Recurrent sinus barotrauma may be refractory to conservative medical measures. Newer endoscopic sinus surgical techniques have treated successfully aviators with recurrent sinus barotrauma.[1] FESS techniques should also be useful in divers suffering