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ORIGINAL ARTICLE

Effect of in-water recompression with oxygen to 6 msw versus normobaric oxygen breathing on bubble formation in divers

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Abstract It is generally accepted that the incidence of decompression sickness (DCS) from hyperbaric exposures is low when few or no bubbles are present in the circulation. To date, no data are available on the influence of in-water oxygen breathing on bubble formation following a provocative dive in man. The purpose of this study was to compare the effect of post-dive hyperbaric versus normobaric oxygen breathing (NOB) on venous circulating bubbles. Nineteen divers carried out open-sea field air dives at 30 msw depth for 30 min followed by a 9 min stop at 3 msw. Each diver performed three dives: one control dive, and two dives followed by 30 min of hyperbaric oxygen breathing (HOB) or NOB; both HOB and NOB started 10 min after surfacing. For HOB, divers were recompressed in-water to 6 msw at rest, whereas NOB was performed in a dry room in supine position. Decompression bubbles were examined by a precordial pulsed Doppler. Bubble count was significantly lower for post-dive NOB than for control dives. HOB dramatically suppressed circulating bubble formation with a bubble count significantly lower than for NOB or controls. In-water recompression with oxygen to 6 msw is more effective in removing gas bubbles than NOB. This treatment could be used in situations of "interrupted" or "omitted" decompression, where a diver returns to the water in order to complete decompression prior to the onset of symptoms. Further investigations are needed before to recommend this protocol as an emergency treatment for DCS.

Keywords Diving · Decompression sickness · Bubble · In-water recompression

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Introduction

Divers are at risk of decompression sickness (DCS) caused by bubbles of inert gas that may evolve in the tissues or blood due to supersaturation during decompression. It is generally hypothesized that gas bubbles grow from pre-existing nuclei attached to the vessel walls or by hydrodynamic cavitation resulting mainly from musculoskeletal activity (Blatteau et al. 2006a, b). The detection of venous circulating bubbles is considered as a valuable indicator of decompression stress and used as a tool for validation of the safety of decompression procedures. It is generally accepted that the incidence of DCS is low when few or no bubbles are present in the circulation (Nishi et al. 2003).

Paul Bert is the first to state that recompression using oxygen is the optimal treatment for decompression injuries and pointed out that this treatment was very effective for getting rid of the gas from vascular system (Bert 1878). Vascular bubbles formed as a result of DCS continue to grow for hours after their initial formation and mainly damage the endothelium with numerous secondary effects related to biochemical or immunological responses developed with time (Francis and Mitchell 2003). There are two theoretical considerations supporting recompression using oxygen. First, the rapid removal of the bubbles from recompression can prevent some of these secondary effects and avoid permanent tissue damage; and second there is the advantage of increased inspired oxygen partial pressure

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when pure oxygen is breathed, which can help counteract the effects of tissue hypoxia that may result from DCS-induced endothelium damage.

Delay in starting treatment may influence results. It appears that more severely injured divers are dependent on the early treatment to maximize improvement (Ball 1993; Moon and Gorman 2003) and that after 6 h or more, a further hold-up of treatment does not influence outcome significantly (Stipp 2004; Ross et al. 2004). To achieve the optimal outcome, the diver should be treated promptly and longer delay than a few hours should be avoided.

The treatment of DCS remains a serious problem in remote locations, especially in situations where the initiation of a therapeutic recompression in a hyperbaric facility may take several hours or days. In-water recompression (IWR) is defined as any attempt to treat or relieve suspected symptoms of DCS by returning an afflicted diver to the water. The published methods of IWR used pure oxygen breathing for prolonged periods of time at a depth of 9 msw (Edmonds 1999; Pyle 1999). IWR should be used in remote localities as an immediate measure to stop the evolution of DCS before evacuating the victim for subsequent treatment to the nearest hyperbaric facility. However, there are many problems associated with IWR that are well recognized by both divers and medical advisers. Resulting from environmental conditions, the risks of drowning and hypothermia are the most often quoted, and pure oxygen breathing at 9 msw can also expose to acute oxygen toxicity. Moreover, the IWR effectiveness in comparison with standard recompression techniques has not been assessed.

Actually, it is commonly accepted that normobaric oxygen should be administered immediately after a DCS and continued until the patient reaches the hyperbaric chamber. This may significantly reduce the symptoms for mild DCS, but this initial treatment is not sufficient for patients with severe neurological symptoms. The value of substituting IWR for normobaric oxygen, in emergency treatment of DCS has never been studied.

To date, no human clinical data are available on the influence of in-water oxygen breathing on bubble formation following a provocative dive. The purpose of this study was to investigate whether oxygen breathing at 6 msw is more effective than normobaric oxygen in reducing post-dive venous circulating bubbles.

Methods

Study population

Nineteen healthy military divers aged 23–48 years (34 ± 7 years, mean ± SD), gave their written informed consent to participate. All the subjects were trained divers and none of them had experienced DCS in the past. Their body mass index varied between 21.3 and 26.8 kg m^{-2} (24.2 ± 1.4 kg m^{-2}, mean ± SD). All experimental procedures were conducted in accordance with the declaration of Helsinki.

Diving protocol

Scuba divers used open-circuit breathing air and were all provided with the same diving material and thermal protection equipment (5 mm neoprene wetsuit). The dive protocol consisted of an open-sea field dive to 30 msw (400 kPa) breathing air for 30 min (sea temperature 15°C) with a decompression rate of 15 msw min^{-1} and a 9-min stop at 3 msw (French Navy MN90 procedure). During bottom time divers performed a constant fin-swimming at a frequency that was reproduced across all the dives. Each diver performed three dives 3 days apart: one control dive, and two dives followed by 30 min of hyperbaric (HOB) or normobaric oxygen breathing (NOB); both HOB and NOB started 10 min after surfacing. The divers did not do any diving during the 3-day intermissions.

For HOB, divers carried out in-water recompression to 6 msw at rest (160 kPa PO_2). NOB was performed in a dry room with a stable environmental temperature (20°C) in supine position (flow rate of 15 l/min, 100 kPa PO_2). Supine NOB was chosen to be as close to the underwater weightlessness HOB for the similarity of increased cardiac output and blood volume distribution within the body.

The order of the three dives was randomly allocated. Divers were instructed to avoid physical exertion and diving during the 2 days that preceded each trial.

Bubbles analysis

Decompression bubbles were examined by a pulsed Doppler device equipped with a 2 MHz probe on the precordial area (MicroMaxx, Sonosite Inc, Bothell, WA). Monitoring was performed by the same blinded operator 40, 60, and 80 min after surfacing in supine position for 3 min at rest and after two lower limbs flexions. The signal of bubbles was graded according to the Spencer scale (Spencer 1976) before to be converted into Kissman Integrated Severity Score (KISS). This score takes into account the kinetics of the bubbles at the different recording times and was assumed to be a meaningful linearized measure of post-decompression intravascular bubble activity status that may be treated statistically (Nishi et al. 2003).

Statistical analysis

All data are presented as mean ± SD. For statistic processing, we used the Sigmastat 3.0 software program (SPSS

inc., Chicago, IL, USA). Data were analyzed using non-parametric statistics because of the small sample-size. Comparisons for difference in bubble grade were evaluated by Friedman test (repeated measures ANOVA on ranks) and Tukey's test for all pairwise multiple comparisons. The level of significance was set at $P < 0.05$.

Results

None of the divers suffered from DCS after the dives or presented signs of CNS oxygen toxicity. The kinetics of the bubble scores at 40, 60, and 80 min revealed a bubble peak at 60 min for the control dives, whereas the bubble peak for the two post-dive experimental conditions (HOB and NOB) was observed at 40 min.

Kissman Integrated Severity Score bubble count was significantly different in these three different conditions ($P < 0.001$) and significantly lower for post-dive NOB than for control dives. In-water recompression with oxygen to 6 msw (HOB) dramatically suppressed circulating bubble formation. Bubble count was significantly lower for post-dive HOB than for the control dive and even the post-dive NOB.

Discussion

The main finding in this study is that in-water recompression with oxygen to 6 msw is more effective in removing gas bubbles than air or even NOB.

Our results are in accordance with a previous study including 17 pigs breathing air and submitted to a strenuous dive to 600 kPa for 30 min (Mollerlokken et al. 2007). The animals in the experimental group were recompressed in a dry hyperbaric chamber to 160 kPa breathing 100% oxygen while the control group remained at surface breathing air. The recompression treatment was initiated an hour after the provocative dive, when the number of vascular bubbles were at peak values. Following recompression, the bubbles in the pulmonary artery were rapidly reduced and no bubbles reappeared after ending the treatment. However, this study did not compare the experimental group with a NOB group and recompression was not performed in water but in a dry hyperbaric chamber (Mollerlokken et al. 2007). There is some evidence that immersion might enhance the rate at which nitrogen is eliminated, however, exposure to cold can limit this effect. Indeed, cold results in the constriction of peripheral circulatory vessels and decreased perfusion reducing the efficiency of nitrogen elimination (Balldin and

Table 1 The individual bubble score (Spencer scale and KISS) after the experimental post-dive conditions (HOB and NOB) and control dives

Divers	Control dives				Post-dive NOB				Post-dive HOB			
	40 min	60 min	80 min	KISS	40 min	60 min	80 min	KISS	40 min	60 min	80 min	KISS
1	3	3	3	42.12	0	0	0	0	0	0	1	0.39
2	3	3	3	42.12	3	3	3	42.12	1	1	1	1.56
3	3	3	3	42.12	2	2	2	12.48	0	0	0	0
4	2	2	2	12.48	1	1	1	1.56	0	0	0	0
5	1	1	0	1.17	1	1	1	1.56	0	0	0	0
6	2	2	2	12.48	3	3	3	42.12	0	0	0	0
7	3	3	2	34.71	1	0	0	0.39	0	0	0	0
8	1	2	0	6.63	0	0	0	0	0	0	0	0
9	2	1	0	3.9	0	0	0	0	0	0	0	0
10	3	3	3	42.12	3	2	1	17.16	1	0	0	0.39
11	1	3	3	31.98	1	1	0	1.17	0	0	0	0
12	2	2	1	9.75	1	2	1	7.02	0	1	0	0.78
13	1	1	1	1.56	0	0	0	0	0	0	0	0
14	2	2	1	9.75	3	2	1	17.16	0	0	0	0
15	3	4	3	70.98	2	2	2	12.48	0	0	0	0
16	3	3	2	34.71	1	2	0	6.63	1	1	0	1.17
17	3	3	2	34.71	0	0	0	0	0	0	0	0
18	3	3	2	34.71	0	0	0	0	0	0	0	0
19	3	2	1	17.16	0	0	0	0	0	0	0	0

The higher bubble score were seen with the control dives, however, two divers presented high bubble score with NOB (divers 2 and 6)

Bubble scores were closed to zero for HOB

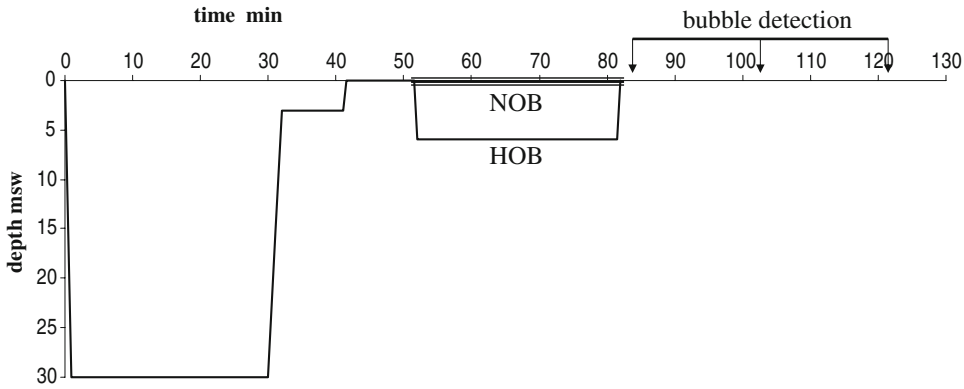

Fig. 1 Experimental protocol including one control dive and two dives followed by 30 min of hyperbaric (HOB) or normobaric oxygen breathing (NOB). Bubble detection was performed at 40, 60, and 80 min after surfacing

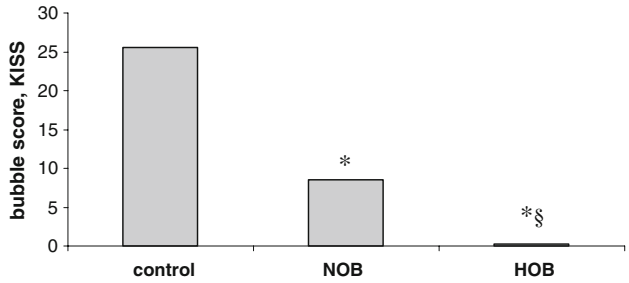

Fig. 2 KISS bubble grades following control dive and post-dive exposures with hyperbaric (HOB) or normobaric oxygen breathing (NOB). *Asterisks* denote $P < 0.05$ from control dives and *dollar* denotes $P < 0.05$ from NOB

Lundgren 1972; Balldin 1973). Diving in 15° with 5 mm wet suit would cause peripheral cooling. During the hyperoxic exposure, some difference may be related to the temperature of the outer shell between NOB and HOB, where at NOB peripheral perfusion may be higher.

The purpose of the initial recompression treatment for DCS is primarily to reduce the bubble formation and consequently to diminish the mechanical effects of the bubbles. Vascular bubbles mainly damage the endothelium with numerous secondary effects, such as activation of leukocytes, aggregation of thrombocytes and initiation of coagulation (Francis and Mitchell 2003). The body regards bubbles as foreign surfaces and responds to them some time after the gas bubbles have been formed. Rapid removal of the bubbles could prevent some of these secondary effects (Nossum et al. 1999, 2002). Despite a recompression treatment, Mollerlokken et al. (2007) found no significant difference in survival time between the experimental and control groups with, however, a trend towards better survival in experimental group. Actually, this study observed that by waiting for 1 h before starting the recompression treatment, the removal of bubbles was not able to prevent arterial endothelial damage. It was hypothesized that due to this latency before the start of treatment, the endothelial function has already been impaired to a point where recompression to 160 kPa with oxygen for 60 min has little effect. Indeed, previous findings showed a relationship between gas bubbles and mechanical endothelial damage related to biochemical or immunological responses developed with time (Nossum et al. 1999, 2002).

We hypothesized that delay in starting treatment could influence results significantly. Most of severe neurologic DCS occur within a period of few minutes after surfacing.(Francis and Mitchell 2003; Aharon-Peretz et al. 1993) and in-water recompression should be performed immediately after the onset of symptoms (Edmonds 1999; Pyle 1999). In the present study, we chose a period of 10 min after the provocative dive to provide a brief delay to prepare the divers to IWR. The aim of our human study was not to evaluate the effect of recompression treatment on DCS or endothelial damage. None of our divers presented symptoms of DCS and we compare only vascular circulating bubbles in three different situations. Actually, we demonstrated a preventive effect on bubble formation when divers breath oxygen after surfacing and that this effect is better when divers return in water to 6 msw for 30 min. Since it is generally accepted that the risk of DCS is low when few or no bubbles are present in the circulation, this treatment could be used to prevent DCS in situations of "interrupted" or "omitted" decompression, where a diver returns to the water in order to complete omitted decompression prior to the onset of symptoms.

However, further experimental investigations are still needed before a similar emergency treatment protocol for DCS can be recommended. Published methods of IWR involve victim returning underwater for a long period of time i.e., 3 h (Edmonds 1999; Pyle 1999). But dehydration and cold due to a long period of immersion can worsen symptoms of DCS and acute oxygen toxicity is also related to the duration of the exposition. In response to these considerations, we have proposed a procedure for IWR at 190 kPa pressure with oxygen for only 1 h (Blatteau et al. 2006a, b). Indeed, previous data indicate that optimal pressure to decrease elimination time for bubbles is 200 kPa, and that additional pressure up to 400 kPa would not influence this elimination time in pigs (Brubakk 2004). Another

important factor is that perfusion-dependent N_2 elimination decreases secondary to vasoconstriction induced by increasing oxygen pressures. Pure oxygen breathing induced a small, insignificant (3.5%) decrease in nitrogen yields, but further increases in oxygen pressure induced significant decreases in nitrogen yields, i.e.,−8.9% for 200 kPa and −16.9% for 250 kPa (Anderson et al. 1991). Protocols including a pressure up to 190 kPa breathing oxygen for 1 h seem, however, a good compromise between bubble reduction and nitrogen elimination. These procedures should be tested in animal model of DCS immediately after the onset of DCS symptoms.

In conclusion, in-water recompression with oxygen to 6 msw is more effective in removing gas bubbles than NOB. This treatment could be useful in situations of "interrupted" or "omitted" decompression in order to prevent DCS (Table 1; Figs. 1, 2).

Conflict of interest statement There is no financial or other relationship that might be perceived as leading to a conflict of interest (i.e., affecting author's objectivity).

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