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ORIGINAL ARTICLE

Pre-dive normobaric oxygen reduces bubble formation in scuba divers

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Abstract Oxygen pre-breathing is routinely employed as a protective measure to reduce the incidence of altitude decompression sickness in aviators and astronauts, but the effectiveness of normobaric oxygen before hyperbaric exposure has not been well explored. The objective of this study was to evaluate the effect of 30-min normobaric oxygen (O_2) breathing before diving upon bubble formation in recreational divers. Twenty-one subjects (13 men and 8 women, mean age (SD) 33 ± 8 years) performed random repetitive open-sea dives (surface interval of 100 min) to 30 msw for 30 min with a 6-min stop at 3 msw under four experimental protocols: "air–air" (control), "O_2–O_2", "O_2–air" and "air–O_2" where "O_2" corresponds to a dive with oxygen pre-breathing and "air" a dive without oxygen administration. Post-dive venous gas emboli were examined by means of a precordial Doppler ultrasound. The results showed decreased bubble scores in all dives where preoxygenation had taken place ($p < 0.01$). Oxygen pre-breathing before each dive ("O_2–O_2" condition) resulted in the highest reduction in bubble scores measured after the second dive compared to the control condition (–66%, $p < 0.05$). The "O_2–air" and "air–O_2 "conditions produced fewer circulating bubbles after the second dive than "air–air" condition (–47.3% and –52.2%, respectively, $p < 0.05$) but less bubbles were detected in "air–O_2 "condition compared to "O_2–air" ($p < 0.05$). Our findings provide evidence that normobaric oxygen pre-breathing decreases venous gas emboli formation with a prolonged protective effect over time. This procedure could therefore be beneficial for multi-day repetitive diving.

Keywords Oxygen · Diving · Bubbles · Decompression sickness

Introduction

Neurological decompression sickness (DCS) and its potential sequelae represents the predominant diving disorder encountered by scuba divers. This pathological event is caused by bubble formation that may occur in the tissues or blood vessels of the brain and spinal cord during decompression. It is generally accepted that gas bubbles grow from pre-existing nuclei attached to the vessel walls (Blatteau et al. 2006) and that venous gas emboli (VGE) magnitude is linked to a high risk of DCS (Neuman et al. 1976; Gardette 1976; Eatock 1984; Nishi and Tikuisis 1996). Nevertheless, there is also medical evidence that the presence of intravascular bubbles alone is not sufficient to induce the symptoms of DCS (Bayne et al. 1985; Dunford et al. 2002). It has been suggested that these asymptomatic bubbles formed in nearly all decompressions and called "silent bubbles" may be indicative of gas phase formation elsewhere in the body (Nishi et al. 2003) and could also have long-term negative effects on endothelial function (Brubakk et al. 2005). Hence, circulating bubble detection with Doppler systems can be considered as a valuable indicator of overall decompression stress and used as a tool for improving existing decompression procedures, thereby reducing the potential risk of developing DCS (Eftedal et al. 2007).

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Recent studies in divers have reported several preventive measures that may decrease the number of vascular gas bubbles generated by decompression such as exercise before diving (Blatteau et al. 2005; Dujic et al. 2008), pre-dive hydration (Gempp et al. 2008) or heat exposure (Blatteau et al. 2008). The possibility that oxygen (O_2) breathing affects DCS risk has been extensively investigated before altitude decompression (Webb and Pilmanis 1999; Bateman 1951) or before extravehicular activity in space (Webb and Pilmanis 1998). This method is also routinely employed during decompression for deep air operational diving to accelerate the washout of nitrogen loaded in tissues, thus shortening the decompression time and lowering the incidence of DCS (Hamilton and Thalmann 2003). In the same way, using oxygen in saturated condition for developing safe escape procedures may have relevance to submarine rescue (White et al. 1999; Gennser and Blogg 2008; Soutiere et al. 2005). Recently, several studies have shown that a single hyperbaric oxygen exposure before diving also appeared beneficial for preventing the occurrence of DCS in animals (Katsenelson et al. 2007; Butler et al. 2006; Arieli et al. 2002) and reducing bubble generation in humans (Landolfi et al. 2006). However, this approach does not seem as effective when normobaric oxygen is used as pretreatment before a simulated dive in rats (Butler et al. 2006) or in pigs (Broome and Buttolph 1996). To our knowledge, no human data are currently available on the influence of pre-dive normobaric oxygen on DCS risk as estimated by Doppler measurement.

The purpose of this study performed in field conditions was to evaluate whether 30 min of normobaric oxygen breathing before a single and a repetitive scuba dive could decrease decompression-induced VGE formation.

Methods

Subjects

The study population consisted of 13 males and 8 females (mean age (SD) 33 ± 8 years), all healthy, with no previous DCS and with diving experience (300–3,000 dives). Their body mass index varied between 19.9–21.6 kg/m² for women and 23–24.5 kg/m² for men. The study was approved by the University's Institutional Review Board, and written informed consent was obtained before the experiment.

Experimental design

Each subject served as their own control and performed repetitive open-sea dives under four experimental conditions in counterbalanced order: (1) a control condition without oxygen pre-breathing before the two dives (air–air), (2) an experimental condition in which subjects either breathed normobaric oxygen before both dives (O_2–O_2), (3) or oxygen before the first dive but not before the second (O_2–air), (4) or oxygen only before the second dive (air–O_2). All subjects were tested at the same time of the day to minimise the effects of circadian rhythms and experimental conditions were separated by a minimum of 24 h for each diver, and the dives were conducted over a 2-week period. The location of experiment was the village of Kani in the Maldives.

Test protocol

Subjects were transported to the scuba centre at 9 a.m. after having eaten a standard breakfast (orange juice, bread, cereals with milk and fruits) and drinking 500 ml of mineral water over a 30 min period before each dive and immediately after the second one (total = 1,500 ml during the entire half-day test).

The depth of each dive was set to 30 msw with a 30 min bottom time. The divers used air as breathing gas and were supplied with a diving computer (Suunto D9, Finland). They were instructed to swim a distance of 100 m along a horizontal wreck once they reached the maximum depth. The ascent rate was set at 10 m/min with a decompression stop at 3 m for 6 min. After the first dive, the subjects stayed at rest on the boat during 2 h, and then performed a second dive similar to the previous dive. Data on the dive profiles were downloaded from the diving computer to a personal PC with data acquisition software. During the dive, subjects were told not to perform any strenuous exercise. The sea temperature at the surface and at bottom varied between 28 and 30°C and all subjects were equipped with a 3 mm wet suit.

In the situation where divers received oxygen before diving, they were asked to stay in a supine position at rest and breathe through a high concentration mask during 30 min (flow rate of 10 l/min, 100 kPa inspired PO_2). A 15-min period between the end of preoxygenation and the beginning of the dive was respected.

Post-dive bubble analysis

After the completion of each dive, divers stayed on the boat without doing any exercise. Circulating bubble detection was performed by an experienced operator using a continuous wave Doppler ultrasound system equipped with a 5 MHz probe (Aqualab system GE, Milwaukee, WI) on the precordial area. Monitoring was performed by the same operator every 20 min for 90 min after surfacing and the Doppler signals were stored on a laptop computer for subsequent evaluation. During bubble measurement, divers were supine and at rest for 3 min.

The amount of bubbles was graded according to the Spencer scale (Spencer 1976) before being converted into a Kissman Integrated Severity Score (KISS) according to the following formula: $KISS = (100/4^{\alpha}\ (t_4 - t_1))\ ((t_2 - t_1)\ (d_2^{\alpha} + d_1^{\alpha}) + (t_3 - t_2)\ \ (d_3^{\alpha} + d_2^{\alpha}) + (t_4 - t_3)\ \ (d_4^{\alpha} + d_3^{\alpha}))/2$, where t_i is the time of observation in minutes after reaching the surface, d_i the doppler score (grades 0–IV) observed at time t_i and $\alpha = 3$ (the parameter α takes into account the fact that the bubble grade is not a linear measure of bubble quantity). The KISS was assumed to be a meaningful linearised measure of post-decompression intravascular bubble activity status that may be treated statistically (Nishi et al. 2003).

Statistical analysis

All data were expressed as mean ± standard deviation (SD). After passing normality test, a two-way analysis of variance with repeated measures was performed to test the effect of experimental conditions (O_2 vs. air) and the allocation of diving session (first or second dive) for association with bubble score. A Tukey post-hoc test was used to determine any differences between experimental conditions and each dive. 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.

Preoxygenation was linked with a significant decrease in post-dive circulating bubble production whatever the dive considered (21.1 ± 9.8 vs. 11.1 ± 6.7 for air diving and dives preceded by oxygen breathing respectively, $p < 0.01$) (Fig. 1).

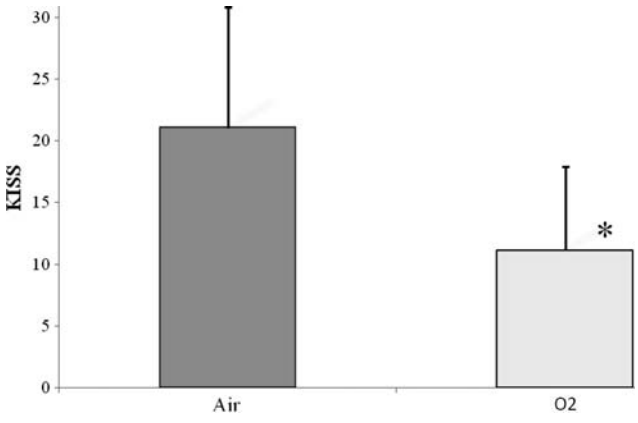

Fig 1 Post-dive circulating bubble detection (KISS) for all dives with O_2 pre-breathing (*white bar*) or without (*grey bar*). Values are expressed as mean ± SD. *Significant difference between conditions, $p < 0.01$

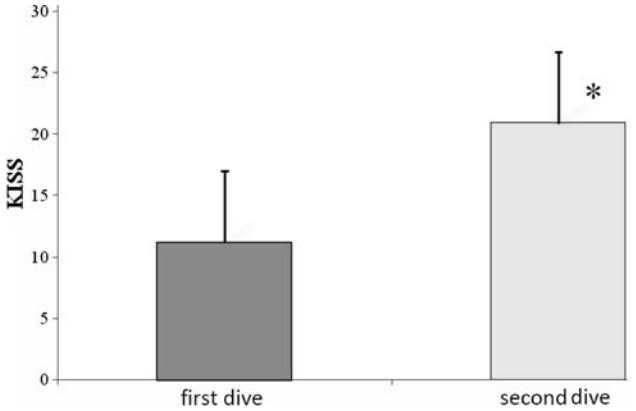

Fig 2 Post-dive circulating bubble detection (KISS) for all divers after the first and the second dive, respectively, whatever the experimental condition (with or without O_2 pre-breathing before dives). Values are expressed as mean ± SD. *Significant difference between conditions, $p < 0.01$

Figure 2 shows that bubble formation was always significantly higher after the second dive than after the first whatever the experimental condition (20.9 ± 10.6 vs. 11.2 ± 5.8, respectively, $p < 0.01$).

The post-hoc test analysis indicates that in "O_2–air" and in "air–O_2" conditions, the bubble score for the second dive was significantly lower when compared with the "air–air" condition (18.8 ± 5.2 and 17.02 ± 5.2, respectively, vs. 35.6 ± 6.6, $p < 0.05$) (Fig. 3). When the two dives were performed with prior oxygen breathing ("O_2–O_2 "condition), the bubble score after the second dive was significantly lower than that measured in the two situations where only one of the two dives was performed with prior oxygen breathing ("O_2–air" and "air–O_2" conditions, respectively)

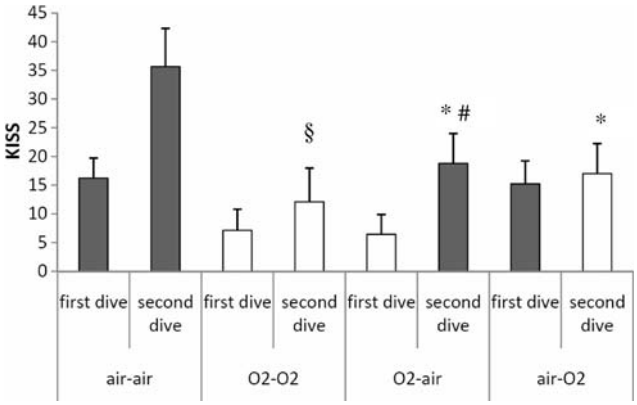

Fig 3 Post-dive circulating bubble detection (KISS) for all divers after the first and the second dive, respectively, in all experimental conditions. Values are expressed as mean ± SD. *Significant difference between second dive in air–air condition with "air–O_2" and "O_2–air" conditions, respectively, $p < 0.05$. §Significant difference between second dive in "O_2–O_2" condition with "air–O_2" and "O_2–air" conditions, respectively, $p < 0.05$. #Significant difference between second dive in "air–O_2" and "O_2–air" conditions, $p < 0.05$

(12.1 ± 5.9 vs. 18.8 ± 5.2 and 17.02 ± 5.2, $p < 0.05$). Finally, if we compare the conditions when only one of the two dives was carried out following preoxygenation, the bubble score after the second dive was significantly lower when oxygen pre-breathing preceded the second dive compared to the case when it only preceded the first dive ($-9.5 \pm 2.2\%$ in percentage changes in KISS, $p < 0.05$).

Discussion

The main finding of this descriptive study is that breathing normobaric oxygen for 30 min before an open-sea air dive provided a significant reduction in decompression-induced bubble formation. This beneficial effect was observed after the first dive and was maintained after the second dive even when the latter was not preceded by preoxygenation. Additionally, it has been well-established that VGE production is higher for repetitive dives than for a single dive (Dunford et al. 2002) with a greater risk of DCS following multi-day repetitive dives (Hamilton and Thalmann 2003). This is consistent with our results in the "air–air" condition but also in all other experimental conditions when oxygen was breathed. Our results, however, emphasise that the magnitude of bubble generation after repetitive dives was limited, compared to the control condition, when dives were performed with preoxygenation (Fig. 3). From the findings above, several hypotheses can be considered to explain how the reduction of VGE could be attributable to oxygen pre-breathing.

Denitrogenation

Denitrogenation is a procedure designed to facilitate the washout of inert gas which has accumulated in saturated tissues by increasing the gradient for nitrogen elimination between tissues and blood. This mechanism is based on the principle of opening the "oxygen window", initially described by Behnke (1967), which depends on the partial pressure of inspired oxygen and the tissue metabolic rate. A rise in arterial oxygen pressure when breathing pure oxygen encourages the diffusion of dissolved inert gas from tissues into the blood. As a consequence, the resolution of resident inert gas is accelerated and tissue nitrogen supersaturation during decompression is reduced, thus limiting bubble generation. It has also been reported that exercising to improve perfusion, ventilation and diffusion during preoxygenation before altitude decompression may provide improved protection against DCS (Webb and Pilmanis 1998).

On the basis of these theoretical considerations, denitrogenation is supposed to be effective immediately after oxygen exposure, therefore in the present study, this process could be involved when oxygen was given prior to the first dive. However, a recent work showed that this procedure appeared ineffective when normally saturated body tissues in atmospheric conditions were exposed to a subsequent compression following oxygenation (Landolfi et al. 2006). In that study, the authors mathematically demonstrated that the partial pressure of dissolved nitrogen in fast tissues (5 and 10 min half-times) at 400 kPa after 25 min of compression was similar between the groups of tissues that received previous hyperbaric oxygen and those that did not receive preoxygenation, suggesting that the nitrogen washout effect by oxygen at ground level is quickly neutralised at depth.

Furthermore, denitrogenation does not explain the reduction in VGE observed in Fig. 3 after the "O_2–air" condition compared with controls that breathed air only and, in the situation where oxygen was only breathed prior to the second dive, preoxygenation may also have contributed more to reducing the population of residual bubbles produced by the first dive than to denitrogenation per se. Thus, our data may indicate that the role of denitrogenation alone does not appear preponderant in the effectiveness of oxygen pre-breathing in the removal of decompression-induced VGE.

Oxygen pre-breathing as a method to remove gas nuclei population

Oxygen prior to diving could have a protective effect through the elimination of pre-existing gas micronuclei before they are able to grow into bubbles. The proposed mechanism is based on the ability of oxygen to replace nitrogen in the nucleus by diffusion. Reduction of tissue oxygen pressure after switching from oxygen to air could enhance the consumption of oxygen from the nucleus, thus eliminating it completely (Arieli et al. 2002). This method for DCS prevention called "oxygen pretreatment" has been primarily verified in hyperbaric conditions in rats (Katsenelson et al. 2007) and in goats (Butler et al. 2006). Similar results were also found in humans with a reduction of post-decompression bubble production from 400 kPa after pretreatment with oxygen at 160 kPa (Landolfi et al. 2006). However, the effectiveness of pre-exposure to normobaric oxygenation is uncertain. In a previous experimental work, using transparent prawns, Arieli et al. (2002) demonstrated that saturation with normobaric oxygen followed by air just before hyperbaric exposure significantly reduced the bubble size in decompressed prawns but not bubble density. In a goat model, Gennser and Blogg (2008) have shown that animals breathing normobaric oxygen for 15 min before hyperbaric exposure simulating submarine escape were prone to producing less Doppler-detected VGE after surfacing than the controls which breathed air. Nevertheless, no dysbaric disorders were observed in either of

these two groups, limiting the significance of oxygen prebreathing in preventing DCS development. Finally, Broome and Buttolph (1996) failed to demonstrate that 2-h pre-dive oxygen breathing would be of benefit in reducing the incidence of neurological DCS in a swine model. These conflicting results could reflect the differing nature of the oxygen application and hyperbaric exposures used in these animal observations.

In the present study, the significant reduction of VGE after repetitive dives when the first dive was preceded by oxygen breathing compared to VGE production after repetitive dives without preoxygenation tends to indicate that oxygen has a prolonged protective effect over time. This finding is consistent with the mechanism of gas nuclei elimination by oxygen pretreatment. Indeed, previous experimental studies have shown that the delay for regeneration of a depleted gas nuclei population may be of the order of a few hours up to 100 h (Yount and Strauss 1982). In the "O_2–O_2" condition (Fig. 3), the cumulative effect of oxygen upon bubble reduction following repetitive dives could also be related with the additional beneficial role provided by supplemental oxygen upon remaining nuclei population.

Some authors have shown that oxygen could have positive effects on lymphatic vessel activity and edema reduction by enhancing protein removal by the lymphatic system (Balestra et al. 2004). In certain circumstances, DCS may also manifest itself as localised lymphoedema. If we consider that during decompression a large proportion of the bubbles are generated in soft tissues, particularly in the interstitium, we can postulate that these microbubbles formed in the lymphatic fluid from preexisting gas nuclei would be drained into the venous blood. As suggested by Balestra et al. (2004), oxygen may be also beneficial in increasing elimination of even protein-coated bubbles by the lymphatic bed. Thus, it is conceivable in our study that preoxygenation may encourage the reduction of gas nuclei localised in both venous and lymphatic networks.

Haemodynamic effects of oxygen administration

Cardiovascular consequences of normobaric hyperoxia in healthy persons have been studied extensively and it has been shown that 1-h of this condition leads to a significant decrease in heart rate, cardiac output, and increase in systemic vascular resistance. Interestingly, it has been demonstrated that these alterations could persist for up to 1 h after a return to normoxic conditions (Waring et al. 2003; Thomson et al. 2006). If we consider that the uptake or release of gas by a particular tissue depends on both the rate of blood flow to the tissue and the rate of gas diffusion into the tissue from the blood, then the inert gas uptake rate must be slower and consequently bubble formation reduced when blood flow is lower. Hence, we can assume that decreased cardiac output and peripheral vasoconstriction were probably present at the start and during the dive preceded by oxygenation, which thus limited inert gas load and subsequent bubble formation.

Considering the lack of evidence for oxidative stress after short exposures at normobaric hyperoxia in humans (Lemaître et al. 2002) and in rats (Singhal et al. 2002), we assumed that increasing the formation of reactive oxygen species involved in the pathogenesis of oxygen-neuronal toxicity as part of our experimental procedure was safe. Nevertheless, a 15-min surface air interval between the oxygen breathing and diving sessions was respected in order to avoid a theoretical exposure to relative hyperoxia during the dive with oxygen partial pressures ranged from 100 kPa (immediately after normobaric oxygen) to 80 kPa (at 30 msw-depth). It is important to note, however, that rare reports exist of neurological effects in divers breathing pure oxygen at pressures of 280 kPa for 30 min-exposure duration [one episode of convulsion among 6,250 oxygen tolerance tests conducted by the US Navy over 25 years (Walters et al. 2000)].

In summary, this study demonstrates that 30 min-normobaric oxygen prebreathing ending 15 min prior to diving decreases VGE. This procedure could provide protection from DCS, particularly in multi-day repetitive diving. The study of the mechanisms involved for reducing gas nuclei population by oxygen warrants further experiments.

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