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ORIGINAL ARTICLE

Oxygen breathing or recompression during decompression from nitrox dives with a rebreather: effects on intravascular bubble burden and ramifications for decompression profiles

Jean-Eric Blatteau · Julien Hugon · Emmanuel Gempp · Olivier Castagna · Christophe Pény · Nicolas Vallée

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Abstract Preventive measures to reduce the risk of decompression sickness can involve several procedures such as oxygen breathing during in-water decompression. Theoretical predictions also suggest that brief periods of recompression during the course of decompression could be a method for controlling bubble formation. The aim of this study was to get clearer information about the effects of different experimental ascent profiles (EAPs) on bubble reduction, using pure oxygen or recompression during decompression for nitrox diving. Four EAPs were evaluated using bubble monitoring in a group of six military divers using Nitrox 40% O_2 breathing with a rebreather. For EAP 1 and 2, 100% O_2 was used for the end stage of decompression, with a 30% reduction of decompression time in EAP 1 and 50% in EAP 2, compared to the French navy standard schedule. For EAP 3 and 4, nitrox 40% O_2 was maintained throughout the decompression stage. EAP 3 is based on an air standard decompression schedule, whereas EAP 4 involved a brief period of recompression at the end of the stop. We found that EAP 1 significantly reduced bubble formation, whereas high bubble grades occurred with other EAPs. No statistical differences were observed in bubbles scores between EAP 3 and 4. One diver developed mild neurological symptoms after EAP 3. These results tend to demonstrate that the "oxygen window" plays a key role in the reduction of bubble production and that breathing pure oxygen during decompression stops is an optimal strategy to prevent decompression sickness for nitrox diving.

Keywords Diving · Decompression sickness · Bubble · Oxygen · Recompression

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Introduction

Decompression sickness (DCS) after scuba diving is due to the formation of inert gas bubbles that may evolve in tissue or blood due to supersaturation when returning to the surface pressure. Although currently available decompression tables and algorithms are capable of reducing the risk of DCS, they cannot eliminate it completely. Inert gas bubbles cause a variety of symptoms ranging from mild skin rash to serious neurological impairment (Blatteau et al. 2010). It is generally accepted that the incidence of DCS is low when few or no bubbles are present in the circulation. Accordingly, circulating bubble detection with Doppler systems is considered as a valuable indicator of decompression stress (Nishi et al. 2003).

In France, mine clearance divers use a new semi-closed circuit rebreather, the complete range autonomous breathing equipment (CRABE; Aqualung, Carros, France), made

with non-magnetic materials. This device operates with predefined gas mixtures (trimix or nitrox) that determine the maximum depth at which they can be used. We have recently observed that the dives using nitrox 40% O_2 mixture are associated with an increased risk of neurological DCS in depths between 30 and 40 msw (Gempp et al. 2011). Accordingly, the French navy decided to evaluate a new nitrox procedure for the CRABE apparatus, based on the French navy MN90 schedule for air diving using the method of equivalent air depth. This method allows one to use existing air tables for decompression involving different oxygen–nitrogen mixtures, and new tables do not have to be calculated. MN90 was developed using a Haldanian model and has been validated with an overall risk estimated at one case of DCS for 30,000 dives (Blatteau et al. 2005).

With the aim of optimising the safety of the nitrox 40% dives, we evaluated preventive measures to reduce the risk of DCS such as oxygen breathing during in-water decompression (Blatteau and Pontier 2009). Pure oxygen breathing increases the arterial oxygen partial pressure (O_2 pp) and causes the inert gas partial pressure to drop, reducing the degree of supersaturation and bubble formation from gas nuclei (Hamilton and Thalmann 2003; Arieli et al. 2011). On the other hand, the predictions of the tissue bubble dynamics model suggest that an intermittent recompression (pressure effect) during the course of decompression could be another effective method for controlling the gas phase growth and decompression stress (Gernhardt 1991).

In this article, we report the possibility of reducing post-dive bubble formation using oxygen breathing or a brief recompression during in-water decompression, by testing a series of experimental ascent profiles (EAPs) designed to reduce bubble formation. For EAPs 1 and 2, 100% oxygen breathing was used for the end stage of decompression, with a reduction of decompression time by 50% with EAP 2 and a 30% reduction in EAP 1. The choice of these reductions was based on the French Ministry of Labour Tables (Direction des Journaux Officiels 1992) that offers oxygen breathing during in-water decompression with a reduction of the decompression time by 50% for the shallow dives and by 30% for the deep dives, compared with the corresponding air table. For EAP 3 and 4, nitrox 40% O_2 was maintained throughout the decompression stage. Decompression stops were based on the MN90 air schedule for EAP 3, whereas EAP 4 was based on the bubble dynamics model with a brief period of recompression at the end of the decompression stop, as proposed by Mollerlokken et al. in an animal model (Mollerlokken et al. 2007).

Since circulating bubble detection with Doppler systems is considered a tool for the validation of the safety of decompression procedures (Nishi et al. 2003), the EAPs were evaluated using bubble monitoring in a group of military divers. The results were verified using a global decompression approach modelling bubble formation in the body (Hugon 2010). The aim of this study was to get more definite and clear information about the effects of different ascent profiles, using pure oxygen or a brief episode of recompression during decompression. The results may allow operational divers to develop optimal procedures for limiting levels of bubble formation and consequently DCS risk.

Methods

Study population

Subjects were eight medically fit military divers who gave their consent; procedures conformed to the Declaration of Helsinki. All the subjects were trained mine clearance divers or combat swimmers and none of them had experienced DCS in the past. The group's average age was 35.5 ± 3 years with a body mass index of 24.5 ± 1.4 kg m^{-2} (mean ± SD), each diver had approximately 10 years of experience. A total of six divers (two for one dive) participated in a given protocol while the other two stood by to provide rescue if necessary. Each diver performed a calibrated effort during the dive using heart rate monitoring-Galileo (with fin-swimming at a determined leg frequency). Physical exertion and diving were precluded for 48 h before participation; experimental dives were separated by a week.

Pressure profiles

The dives were simulated in the wet section of a hyperbaric chamber at a water temperature of 17°C. All subjects were compressed in 1.5 min with nitrox 40% O_2 breathing to 520 kPa (5.2 ATA, 42 msw) for 30 min (EAPs 1 and 2, Fig 1a) or 25 min (EAPs 3 and 4, Fig 2a), respectively. Decompression was then performed at a rate of 150 kPa min^{-1} (15 msw min^{-1}).

For both EAPs 1 and 2, 100% oxygen breathing was used during the ascent profile from 10 msw with an initial rinsing procedure during 1 min, consisting in 3 ventilatory cycles with exhalations out of the rebreather. For EAP 1, decompression stops were performed at 6 msw for 1 min and at 3 msw for 14 min (equivalent to 30% reduction in decompression time calculated with MN90), whereas a stop at 3 msw for 9 min was performed for EAP 2 (equivalent to 50% reduction in decompression time calculated with MN90). The order of the experimental dives EAPs 1 and 2 was randomly allocated.

For EAP 3, Nitrox 40% O_2 breathing was maintained during the ascent profile including the decompression stop

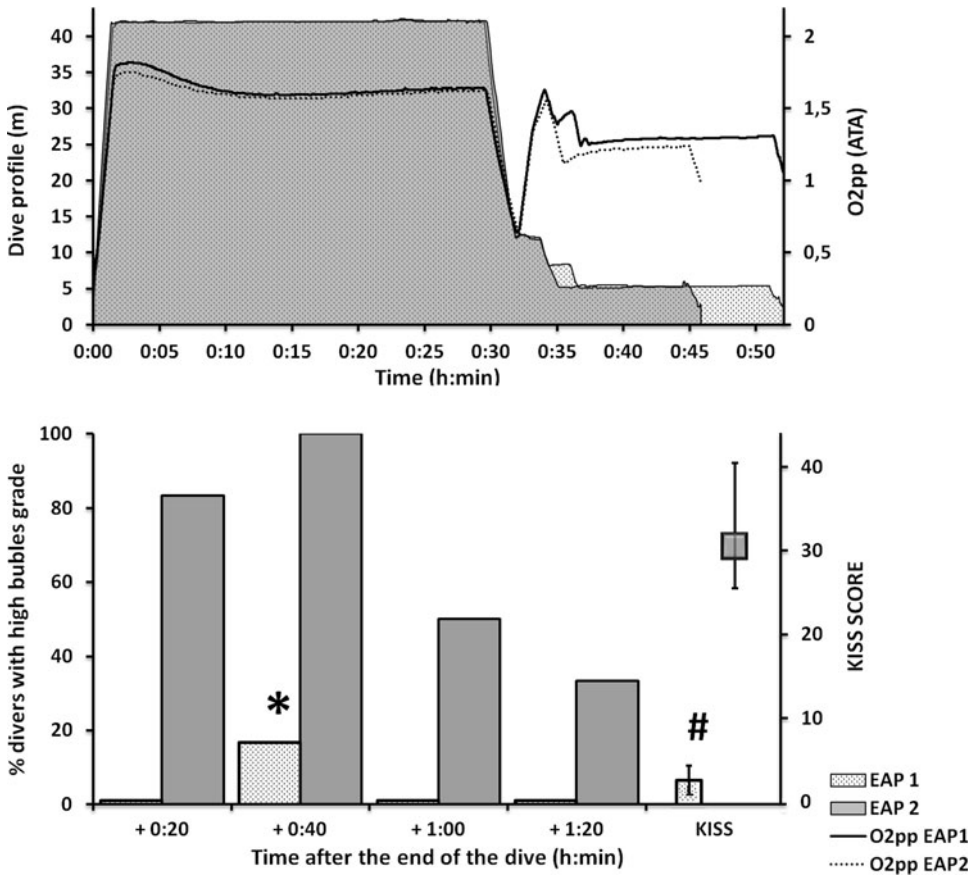

Fig. 1 Profiles of depth (msw) versus elapsed time (min) for EAP 1 and EAP 2. *White* represents the EAP 1 and *grey* the EAP 2. *Dotted line* represents O_2pp values for EAP 2 and solid line for EAP 1. The percentage of subjects with high bubble grades (Spencer ≥ 3) is given at specified times after surfacing from each profile, with the resulting bubble KISS values. * and # denote $p < 0.05$ for comparisons between Spencer grades at bubble peak and KISS values, respectively

of 11 min at 3 msw, according to the MN90 air schedule. Nitrox 40% O_2 breathing was also used during decompression for EAP 4 with stops at 3 msw for 6 min followed by a recompression to 9 msw for 5 min. The order of the experimental dives EAPs 3 and 4 was randomly also allocated.

O_2 pp and equivalent air depth

The rebreather CRABE incorporates a counterlung with two concentric bags allowing the breathing gas to be periodically vented into the water in proportion to the volumetric ratio of the two bags. Due to dilution of the breathing gas in the counterlung, the oxygen partial pressure is lower than in the cylinders. The CRABE allows the diver to go down to 55–80 msw with trimix mixture, however, the majority of dives are performed using a nitrox mixture of 60% O_2 up to 24 msw, 40% O_2 from 25 to 45 msw, and 32% O_2 between 45 and 55 msw. Analysis of O_2 pp measured with a "black box" in this device has previously made it possible to highlight a theoretical relationship of O_2pp/Nitrogen (N_2)pp between the counterlung and the cylinders:

$$b = a \times [P + (\mathrm{K} \times \mathrm{Co})]/P - \mathrm{Co}$$

where b is N_2pp in the counterlung, a is N_2pp in the cylinders, P is absolute pressure at depth, K = 11.42 (volumetric ratio between the two bags), Co = 0.055 (coefficient based on oxygen consumption).

Decompression profiles were calculated using the method of equivalent air depth. It involves determining the inert gas N_2pp at a specific depth, then selecting a standard air table that has the same N_2pp and using that table to perform the decompression. For nitrox mixtures with more oxygen than air the resulting table will be for a shallower depth and will thus require a shorter decompression. For each profile, we calculated the theoretical O_2pp/N_2pp within the rebreather and performed a conversion in equivalent air depth. For a dive to 42 msw with a rebreather- O_2pp calculated at 1.65 ATA, N_2pp = 3.55 ATA, thus the equivalent air depth is 3.55/0.79 = 4.5 ATA or 35 msw. Decompression stops are based on this depth according to the MN90 air schedule, i.e., 11 min at 3 msw for 25 min of bottom time.

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 20, 40, 60 and 80 min after surfacing in supine position for 3 min at rest and after two lower limbs flexions. The signal

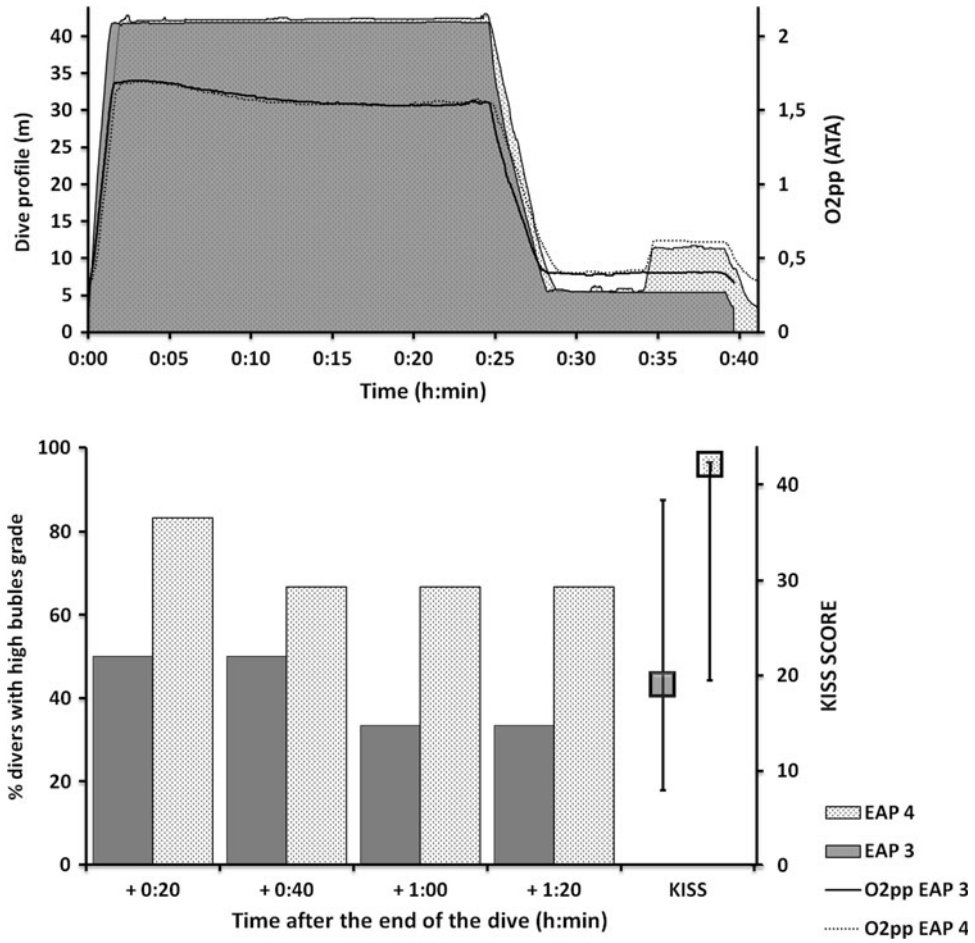

Fig. 2 Profiles of depth (msw) versus elapsed time (min) for EAP 3 and EAP 4. *Grey* represents the EAP 3 and *white* the EAP 4. *Dotted line* represents O_2pp values for EAP 4 and *solid line* for EAP 3. The percentage of subjects with high bubble grades (Spencer ≥ 3) is given at specified times after surfacing from each profile, with the resulting bubble KISS values

of bubbles was graded according to the Spencer scale before being 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 linearised measure of post-decompression intravascular bubble activity status that may be treated statistically (Nishi et al. 2003).

Statistical analysis

All data are presented as median ± interquartile range. For statistical processing, we used the Sigmastat 3.0 software program (SPSS inc., Chicago, Illinois). Data were analysed using non-parametric statistics because of the small sample size. Comparisons for differences in bubble grade were evaluated by a Wilcoxon Signed Rank test. The level of significance was set at $p < 0.05$.

Results

The maximum bubble score at rest (bubble peak) was observed at 40 min after surfacing for all protocols. Only one diver produced Spencer bubbles grade IV following the EAP 3.

Under the EAP 1 conditions, maximum Spencer bubbles grades at rest were significantly lower than under the EAP 2 conditions [$\text{median}_{\text{Spencer}}$ 1 ± 1 vs. 2 ± 0.1, $p = 0.03$]. Furthermore, KISS values (at rest and after flexions) under the EAP 1 conditions were significantly lower than under the EAP 2 [$\text{median}_{\text{Kiss at rest}}$ 0.6 ± 1.3 vs. 10.5 ± 3.6, respectively, $p = 0.03$] (Fig. 1).

Due to high bubble grades occurring with EAP 2, it was decided to reduce the bottom time to 25 min (Fig. 2a) for subsequent dives using nitrox 40% O_2 breathing during decompression for EAPs 3 and 4.

No statistical differences in bubbles scores (Spencer and KISS) were observed when comparing EAP 4 and EAP 3 conditions [$\text{median}_{\text{Kiss at rest}}$ 17.4 ± 0.21 vs. 10.5 ± 3.6, respectively, $p = 0.3$] (Fig. 2).

Additionally, we tested the percentage of divers exhibiting Spencer grade III and IV bubbles at bubble peak (after flexions) as a simple criteria for estimating the acceptability of dive profiles. We found only 16.6% of divers with grade III for EAP 1 but 100% for EAP 2. The results were 50% for EAP 3 and 66.6% for EAP 4 (Fig. 1, 2).

One diver experienced neurological symptoms while exhibiting grade IV bubbles after surfacing from the EAP 3. He presented a burn sensation in the right side of his abdomen and paresthesia in the right lower limb, however, motor and sensory functions were intact and reflexes were present and symmetrical. He was recompressed (GERS B French table at 4 ATA) and symptoms resolved completely at the end of the treatment. Since this diver had previously performed EAP 4 and had finished EAP 3 (DCS came after his last dive), his bubble detection was included in the results.

O_2pp values are shown in Figs. 1a, 2a; the data are in accordance with the theoretical relationship described in methods section. The average O_2pp in the breathing gas during the decompression period is 1.3 ATA for EAP 1 and EAP 2 but only 0.4 ATA for EAP 3 and 0.5 ATA for EAP 4.

Discussion

The main finding of this study is that oxygen can be used during in-water decompression to reduce decompression time and bubble formation. The benefits of oxygen in decompression have been known for over a century (Bert 1878) and it is almost axiomatic in decompression table calculation that for a given table, the higher the O_2pp breathed, the lower the risk of DCS. However, many decompression models do not take into account oxygen effects on bubble dynamics, and track only inert gas partial pressure (Hamilton and Thalmann 2003). Naturally, the increased arterial O_2pp causes the inert gas partial pressure to drop, increasing the elimination gradient. This reduces the degree of supersaturation and thus, the possibility of gas phase separation and bubble formation (Hamilton and Thalmann 2003).

Moreover, when living animals are in steady state, the sum of the partial pressures of dissolved gas in the tissues is usually less than atmospheric pressure, a phenomenon known as "the oxygen window" (Behnke 1951) or "inherent unsaturation" (Hills 1977). This is because metabolism lowers partial pressure of O_2 in tissue below the value in arterial blood and the binding of O_2 by haemoglobin causes a relatively large O_2pp difference between tissue and arterial blood. When a tissue is clamped by bubbles (the tissue inert gas has been rapidly stored in numerous bubbles, the tissue inert gas tension has dropped and is in equilibrium with the bubble pressure i.e., close to the ambient pressure), the "oxygen window" is the only driving force that permits the elimination of the inert gases (Hills 1966; Van Liew and Burkard 1993). The "oxygen window", the difference between the sum of oxygen and carbon dioxide tensions on the arterial side and the sum on the venous side, increases proportionally to the O_2pp in the breathing gas. As a consequence, breathing oxygen is an efficient way to reduce the dimensions of the bubbles formed in case of profuse bubbling and to increase locally the tissue desaturation rate in any case.

However, these positive effects may be counterbalanced by O_2 vasoconstriction that may decrease inert gas elimination with the elevation of O_2pp (Anderson et al. 1991). In reality, human diving data are limited and the real impact of pure oxygen breathing during decompression is not completely established.

Oxygen breathing during decompression has been used to reduce the in-water exposure time for working dives in cold water. An in-water oxygen decompression procedure was developed at DCIEM (Nishi et al. 1984) with oxygen breathing at 9 msw. The results of this study, using both wet and dry divers and involving over 300 man-dives, showed that the decompression stress is reduced compared to the corresponding profiles for compressed air. Moreover, it has been demonstrated that the incidence of DCS with oxygen decompression was two to three times lower than with air decompression for dives of the same depth and bottom time (Imbert and Bontoux 1987). Using a validated probabilistic model to estimate the risk of DCS for planned decompression dives with pure oxygen or 50% nitrox as a single decompression gas, Walker et al. (Walker et al. 2010) recently found that decompression with pure oxygen decreased the probability of DCS compared to decompressing with 50% nitrox, irrespective of overall decompression time. Our results with the presence of high bubble grades and the occurrence of one DCS case during EAP 4 (nitrox breathing) compared to EAP 1 (pure oxygen breathing) are in accordance with these data.

The 1986 COMEX tables, which are also the 1992 French Ministry of Labour Tables (Direction des Journaux Officiels 1992), have an option that offers oxygen breathing during in-water decompression at 6 msw (20 fsw). Compared to the corresponding air table, the oxygen table reduces the decompression time by 50% for 15 msw dives and by 30% for 60 msw dives. Our findings indicate that the most efficient reduction in the decompression time should not exceed 30% for 40 msw dives.

Breathing oxygen underwater does incur the risk of oxygen convulsions, but experience has shown that oxygen decompression can be both safe and efficient if diver selection, training and equipment are appropriate. For the last 50 years, the French navy has used semi-closed circuit rebreathers with a high level of O_2pp, i.e., mine clearance divers usually tolerate O_2pp of 1.8 ATA using a nitrox mixture of 60% O_2 up to 25 msw for 3 h, with no case of hyperoxic seizure reported (Gempp et al. 2011). It seems that the addition of nitrogen with oxygen may improve cerebral oxygen tolerance (Arieli et al. 2005).

Our study demonstrates that adding a brief period of recompression at the end of the decompression stop did not reduce bubble formation in our protocol. The predictions of a bubble dynamics model suggest that intermittent recompression during the course of a saturation decompression would be a more effective method for controlling the gas phase growth and decompression stress (Gernhardt 1991). This theoretical concept was supported by an experimental study using bubble detection in 12 pigs compressed to 500 kPa (5 ATA) for 90 min (Mollerlokken et al. 2007). The experimental procedure followed the same profile except that a 5 min recompression of 50 kPa (0.5 ATA) was added at the end of each of the last three decompression stops before ascending to the next stop depth. This brief recompression during the late decompression stops reduced the amount of bubbles (Mollerlokken et al. 2007).

From a Haldanian point of view, such a recompression would not give any advantage. On the contrary, increasing pressure during decompression will only increase tissue gas tensions and hence the risk of exceeding the critical tension values during the subsequent ascent. These last authors suggest that such results support rather two-phase models i.e., a bubble size decrease induced by the recompression, with an associated increase of tissue inert gas pressure and desaturation rate in case of clamping configuration. Our results are not in accordance with this experimental study in pigs, and suggest that there is a need for a better understanding of bubble formation during decompression, supported by an exhaustive gas phase model.

If we consider the basic bubble dynamics, the standard equation describing the condition of spherical bubbles (Epstein and Plesset 1950) applied to the case of a tissue totally or partially saturated with a single inert gas is the following:

$$\frac{dR}{dt} = \frac{\Re TDS}{R} \frac{P_t - P_{amb} + \beta - \frac{2\gamma}{R}}{P_{amb} + \frac{4\gamma}{3R}}$$

where, $\Re$ is the radius of a spherical bubble (m), $\Re$ is the perfect gas constant, T is the temperature (K), D is the diffusion coefficient of the inert gas in the tissue (m^2/s), S is the solubility of the inert gas in the tissue (ml/ml/Pa), P_t is the inert gas tension in the tissue (Pa), P_{amb} is the ambient pressure (Pa), β is the sum of vapour pressure, oxygen and carbon dioxide tensions and in the tissue (Pa), γ is the surface tension in the tissue (N/m)

This equation has been widely used in decompression theory, and adapted to take into account blood perfusion effects (Van Liew and Hlastala 1969). Its reliability has previously been discussed (Srinivisan et al. 1999). Gernhardt (1991) introduced a new approach by modelling a gas diffusion barrier representing a low-permeable shell surrounding the bubbles, slowing the growth process. This model requires the introduction of very low diffusion coefficients ($D = 10^{-12}$ m^2/s), which is debatable from a physical point of view (layer of proteins and/or phospholipids). The form of the bubble dynamics equation remains nevertheless similar to the relation proposed above. A simple analysis of the recompression process can be conducted with a similar reasoning.

During a decompression, microbubbles are formed in tissues from a phenomenon induced by a supersaturation state. It is accepted that these microbubbles are generated from pre-existing gas nuclei (Blatteau et al. 2006). Two main extreme situations can arise for the tissue: (1) the gas nuclei population recruited is not very large so that the bubble dynamics do not modify to the tissue desaturation rate, (2): the gas nuclei population recruited is very large so that the bubble dynamics modify the tissue desaturation rate significantly (clamping phenomenon).

For the first configuration, the tissue is not clamped; the bubbles grow continuously during the decompression, more or less rapidly depending on the diffusion characteristics at bubble-tissue interface introduced in the model. If the diffusion process is not slowed ($D = 10^{-9}$ m^2/s) and the tissue structure does not restrain the growth, the radius of the microbubbles can reach non negligible dimensions even for moderate $P_{ss} = P_t - P_{amb} + \beta$ supersaturation levels (>100 μm). A recompression of a few tenths of ATA should have a very low impact on microbubble dimension (radius decreased by only 13% at best for a 5 m recompression) and the surface tension effects probably remain negligible. Moreover the supersaturation level P_{ss} is reduced only slightly (potentially negative in some cases) and its impact is noticeable only for slow tissues having a rather low inert gas content at the end of the exposure. As a consequence, the dimensions of the bubbles cannot change to a great extent during a recompression of short duration (a few minutes) and, additionally, the tissue desaturation rate is not favoured by the procedure as foreseen by the Haldanian model. If the diffusion process is slowed ($D = 10^{-12}$ m^2/s), rapid bubble shrinkage and a positive effect on the tissue desaturation rate are no longer conceivable.

For the second configuration, the tissue is clamped. The numerous microbubbles formed grow rapidly during the decompression and stabilise rapidly to a rather small dimension (<100 μm). A stabilised population with radii lower than 5 μm is not conceivable without the introduction of huge micronuclei densities $>10^{13}$ nuclei/m^3, which is unrealistic. As a consequence, the surface tension effects on bubble pressures can be assumed minor. For the clamped configuration, the dissolved and separated phases are in equilibrium, which implies a tissue gas tension close to the ambient pressure. As explained above, a

recompression of a few tenths of ATA has a very low impact on the dimensions of the microbubbles so that the amount of inert gas returned to the surrounding tissue by re-dissolution increases the gradient only slightly allowing the elimination of the gas by perfusion via the blood stream. As remarked before, once a tissue is clamped, the only driving force to transfer the inert gas from the tissue to the blood is the "oxygen window". This inherent unsaturation can be expressed as follows in first approximation (Hills 1966):

$$\Delta_w = (Pa_{O_2} + Pa_{CO_2}) - (Pv_{O_2} + Pv_{CO_2}) \approx f_{O_2}(P_{amb} - P_{H_2O}) - \alpha$$

with f_{O_2} the oxygen fraction in the breathing gas and α a constant ($\approx$0.12 bar). As the oxygen window increases with the ambient pressure P_{amb} the desaturation rate can indeed be increased by the recompression. Nevertheless, a short duration cannot provide a significant effect on the desaturation for tissues that desaturate with a moderate to slow speed during the decompression stages, like muscles and fat. This conclusion is not applicable if the pressure increase level is non negligible (>1 ATA) and/or the duration of the recompression period is sizeable (Gernhardt 1991), a real positive effect being then conceivable.

What is noticeable is the important role of the "oxygen window". It is clear that a rise in the pressure increases this "oxygen window" but only to a few tenths of ATA if air is breathed (around 0.1 ATA for a 5 m recompression). Conversely, an increase of the oxygen fraction to 100% opens this "window" significantly: breathing pure oxygen at 6 m increases the "oxygen window" to 1.6ATA, which is an appreciable driving force for tissue desaturation compared to air breathing at the same depth.

To summarise, this analysis shows that in the case of profuse bubbling, (1) the effect of a short recompression on tissue desaturation or on microbubble dynamics is probably very small. (2) it is highly preferable to open the "oxygen window" (i.e., to enhance tissue desaturation rate by breathing pure oxygen than choosing a recompression strategy. The first point raises the question of the representativeness of the experimental results on pigs mentioned (Mollerlokken et al. 2007). The second statement tends to justify and clarify the results found with our experiments: EAP 1 protocol with oxygen is more efficient than EAP 4 recompression procedure. The average O_2pp in the breathing gas during the decompression period is 1.3 ATA for EAP 1 and EAP 2 but only 0.4 ATA for EAP 3 and 0.5 ATA for EAP 4. This implies large differences in the "oxygen window" and so in the elimination rate.

While the proposed reasoning focused on bubbles formed in tissues, the conclusion would be the same with a model considering microbubble formation at the capillary endothelium level (Chappell and Payne 2006). To illustrate this discussion, a new global decompression model modelling bubble formation in the body has been used (Hugon 2010). For a given exposure, this biophysical mixed gas model estimates both during and after the decompression the global bubble flow rate on the mixed venous side. This flow rate reflects the contribution of the different tissues involved in microbubble formation, and in particular muscles and fat. The physiological tissues of the body can be characterised by a micronuclei population that can be partially recruited into microbubbles during the decompression (Yount and Hoffman 1986). The micronuclei densities have been selected in order to make a clamping phenomenon possible in case of high supersaturation levels. Finally, microbubble dynamics are modelled. A set of model parameters has been proposed and justified in the mentioned work. It has been re-used here. EAP 1 to EAP 4 has then been simulated to determine the global bubble flow rate at right heart level. The results are presented in

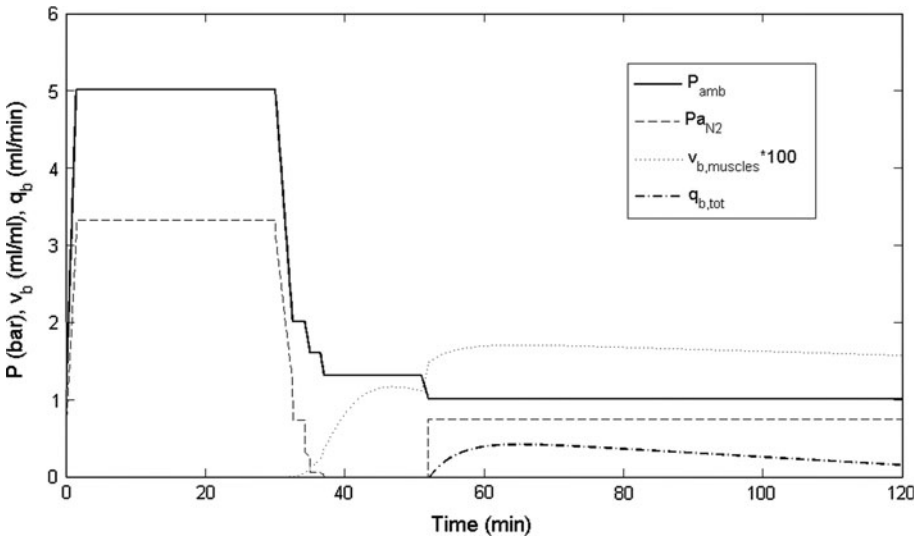

Fig. 3 Simulation of EAP 1 procedure using a biophysical decompression model according to Hugon (2010): evolution with time of **a** the ambient pressure, **b** the arterial inert gas tension, **c** the microbubble volume formed in muscles $v_{b,muscles}$ (ml/ml), **d** the estimated bubble flow rate at right heart level $q_{b,tot}$ (ml/min) merging the contribution of muscles and fat tissues

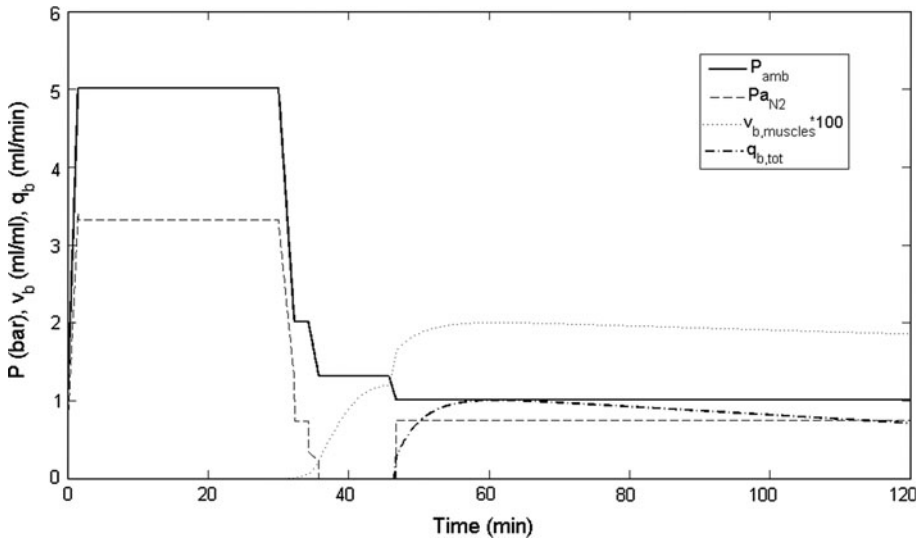

Fig. 4 Simulation of EAP 2 procedure using a biophysical decompression model according to Hugon (2010): evolution with time of **a** the ambient pressure, **b** the arterial inert gas tension, **c** the microbubble volume formed in muscles $v_{b,muscles}$ (ml/ml), **d** the estimated bubble flow rate at right heart level $q_{b,tot}$ (ml/min) merging the contribution of muscles and fat tissues

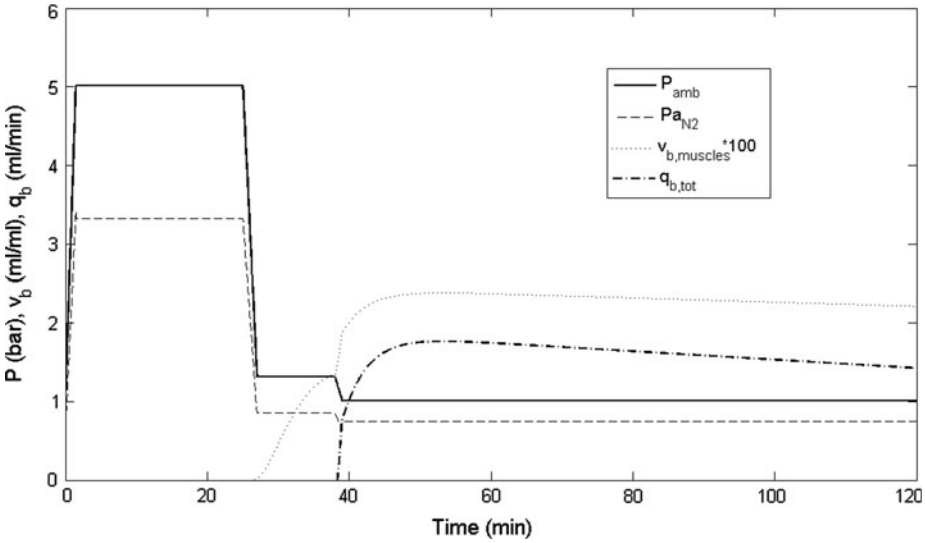

Fig. 5 Simulation of EAP 3 procedure using a biophysical decompression model according to Hugon (2010): evolution with time of **a** the ambient pressure, **b** the arterial inert gas tension, **c** the microbubble volume formed in muscles $v_{b,muscles}$ (ml/ml), **d** the estimated bubble flow rate at right heart level $q_{b,tot}$ (ml/min) merging the contribution of muscles and fat tissues

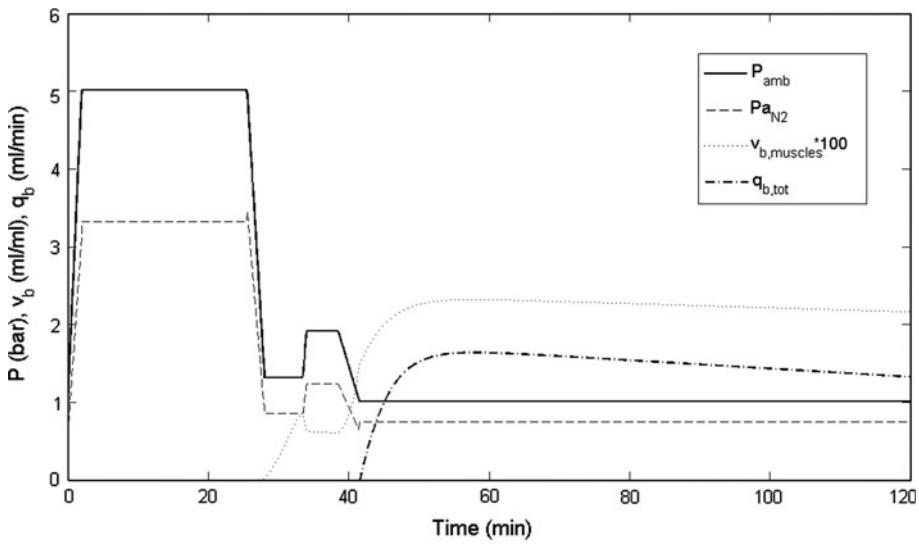

Fig. 6 Simulation of EAP 4 procedure using a biophysical decompression model according to Hugon (2010): evolution with time of **a** the ambient pressure, **b** the arterial inert gas tension, **c** the microbubble volume formed in muscles $v_{b,muscles}$ (ml/ml), **d** the estimated bubble flow rate at right heart level $q_{b,tot}$ (ml/min) merging the contribution of muscles and fat tissues

Figs. 3, 4, 5, and 6. It appears clearly that EAP 2 is more severe than EAP 1 in terms of bubble production and that EAP 3 and EAP 4 are worse, as suggested by the experimental results.

In conclusion, we observed that pure oxygen breathing during the end stage of decompression significantly reduced bubble formation for 40 msw dives using a semi-closed circuit rebreather with nitrox 40% mixture. This effect was optimal with a reduction of decompression time by 30% compared with the corresponding air schedule and was more beneficial than a brief recompression at the end of the decompression stop. Our findings highlight the preponderant role of the "oxygen window" provided by pure oxygen breathing compared to the pressure effect given by a recompression using a poorly enriched nitrox.

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