The effect of breathing hyperoxic gas during simulated submarine escape on venous gas emboli and decompression illness.

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Blogg SL, Gennser M, Loveman GAM, Seddon FM, Thacker JC, White MG. The effect of breathing hyperoxic gas during simulated submarine escape on venous gas emboli and decompression illness. Undersea Hyperb Med 2003; 30(3): 163-174 - Raised internal pressure in a distressed submarine rapidly increases the risk of decompression sickness (DCS) following submarine escape. The hypothesis that breathing a hyperoxic gas during escape may reduce the risk of DCS was tested using goats. Shallow air saturation and simulated submarine escape dives were carried out either singularly or in combination (saturation, escape, or saturation followed by escape) using air or 60% / 40% oxygen (O₂) / nitrogen (N₂) mixture as breathing gas during the escapes. Post-surfacing, animals were observed for signs of DCI and O₂ toxicity. Precordial Doppler ultrasound was used to score venous gas emboli (VGE) using the Kisman Masurel (KM) scale. Following escape from 2.5 MPa, the rate at which VGE disappeared in the hyperoxic group (n = 8) was significantly faster (p < 0.05) than the air group (n = 7). One case of pulmonary barotrauma with arterial gas embolism occurred in the air group, but no cases of DCS were observed. After saturation at 0.18 MPa followed by escape from 2.5 MPa, DCS occurred in four of 15 animals in the air group and in two of 16 animals in the hyperoxic group. The rate of disappearance of VGE was significantly faster (p < 0.01) in the hyperoxic group. O₂ toxicity was not discernible in any of the animals.

decompression illness, submarine escape, hyperoxia, oxygen toxicity, saturation, venous gas emboli.

INTRODUCTION

The current submarine tower (alternatively known as lock or trunk) escape method has been shown to be successful at depths down to 180 meters of seawater (msw) (1.9 MPa) from normobaric pressure in the submarine (1). However, it is most likely that escape will have to be made from a disabled submarine (DISSUB) with raised internal pressure (the Pacocha and the Kursk are recent examples). Increasing pressure in a DISSUB rapidly increases the risk of decompression sickness (DCS) (2).
The possibility of using oxygen-enriched gas mixtures to reduce the risk of DCS was suggested by Donald, Davidson, and Shelford (see 1). A few successful escapes (without prior saturation) were carried out with a 34% O₂ / 66% N₂ mixture from 300 feet of seawater (fsw) (1 MPa), using a slow profile that had caused a high incidence of bends in air breathing animals. However, no full scale series comparing air and hyperoxic escape gas was carried out.

During decompression from saturation, an increased decompression rate can be balanced by an increased PO₂ in the inspired gas (3). However, the beneficial effect of increased O₂ is probably less in deeper and shorter dives (4). Vann (5) noted that breathing 100% O₂ from 18 msw (0.28 MPa) to surface reduced decompression time by 40%. Working on this basis, Leitch (6) suggested that 40% O₂ breathed during escape might reduce decompression time by 10%, though the theory remained untested. In addition to the possible benefits of breathing a high PO₂ gas during decompression, there is also a possibility that survivors may be vulnerable to O₂ toxicity. Modelling indicates that most of the N₂ loading in the tissue takes place during the deepest part of the ascent (1, 7), so to be beneficial, the highest concentration of O₂ needs to be present at the start of the escape, consequently increasing the risk of toxicity.

Another potential problem is that high concentrations of O₂ may increase the risk of DCS. It is tempting to think of O₂ purely as a metabolic gas, rather than having the potential to act as an inert gas, like N₂, when supersaturation occurs. In fact, O₂-induced DCS has been reported (8). Recent modelling has factorised the contribution of O₂ as an inert gas and also its capability to alter inert gas kinetics (9) in order to estimate its effect on DCS development. The results showed that elevated levels of O₂ do contribute to the risk of DCS, although to a lesser degree than the equivalent amount of N₂ (9).

The present study compared the effect of breathing hyperoxic gas during submarine escape upon the evolution and time course of venous gas emboli (VGE) in a range of scenarios, including escape without prior hyperbaric saturation and escape following hyperbaric saturation. The subjects were also closely monitored for signs or symptoms of AGE and O₂ toxicity.

**MATERIALS AND METHODS**

The initial trials reported on here, involving saturation dives (24 hours at depth) or saturation immediately followed by simulated escapes, were executed to determine a submarine ‘safe-to-escape’ curve for the Royal Navy (RN). Subsequent studies involving hyperoxic breathing gas involved collaboration between the United Kingdom (UK) Defence Evaluation Research Agency (DERA) and the Swedish Defence Research Agency (FOI).

The goat was chosen for this series of studies because it historically has been used as a model for decompression research, and there is an extensive comparative database of goat exposures. The animals were females or castrated males having a mean weight of 45.8 kilograms (kg) ± 7.7 (range 36 - 64 kg, n = 67). There was no significant difference in weight between experimental groups (Table 1). All trials were conducted according to the regulations of the UK Animals Act (1986). Prior to pressure exposures, all animals were familiarized with the Submarine Escape Simulator (SES), trained to stand in restraints, and wear oro-nasal masks.
Table 1. Summary of subject details, profile protocols and results

<table>
<thead>
<tr>
<th>Breathing gas (#)</th>
<th>Mass (Kg)</th>
<th>Sat depth (MPa)</th>
<th>Escape depth (MPa)</th>
<th>Compression time (s)</th>
<th>Maximum KM-score (median)</th>
<th>DCS/n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air (n=20)</td>
<td>48±8.6</td>
<td>0.18</td>
<td>-</td>
<td>-</td>
<td>3</td>
<td>0/20</td>
</tr>
<tr>
<td>Air (n=7)</td>
<td>43±8.1</td>
<td>0.10</td>
<td>2.5</td>
<td>24</td>
<td>3</td>
<td>0/7*</td>
</tr>
<tr>
<td>Hyperoxic (n=8)</td>
<td>42±7.0</td>
<td>0.10</td>
<td>2.5</td>
<td>24</td>
<td>3</td>
<td>0/8</td>
</tr>
<tr>
<td>Hyperoxic (n=16)</td>
<td>46±6.2</td>
<td>0.18</td>
<td>2.5</td>
<td>30</td>
<td>4</td>
<td>2/16</td>
</tr>
<tr>
<td>Air (n=15)</td>
<td>47±8.3</td>
<td>0.18</td>
<td>2.5</td>
<td>30</td>
<td>4</td>
<td>4/15</td>
</tr>
</tbody>
</table>

* An eighth animal developed AGE after pulmonary barotrauma and is not included in the statistics.

Saturation period - 24 h
Time at depth during all escapes - 4 s
Ascent time for all escapes - 87 s

Pressure exposures took place within the SES, which consists of two spherical chambers (one of 3 m diameter and one of 2 m) joined via an interconnecting door, with doors to the outside at both ends. The SES allows the rapid pressure changes commensurate with submarine escape to be simulated precisely by transfer of gas from larger to smaller sphere via a computer-controlled valve system. Saturation phases took place in the 3 m sphere, while simulated escapes were conducted in the 2 m sphere. During all saturation phases, the goats were free in the 3 m sphere with free access to water. All food, apart from hay, was withheld for 24 h before the dive, but concentrated pelleted food was available for the first 12 h of the saturation in the 3 m sphere. The animal’s behavior could be monitored at all times via an array of video cameras. A 24 h saturation period was used, which is sufficient for all tissues to be saturated in the goat (3, 10). In prior studies using goats (10), it was shown that 0.18 MPa was the final incremental saturation pressure at which no cases of DCS or AGE occurred. Therefore, all saturation phases took place over 24 h at 0.18 MPa. The saturation pressure was maintained to ±0.5 kilopascals (kPa) by addition of air, while carbon dioxide (CO₂) was measured by infrared spectroscopy (ADC) and kept below 0.5 kPa. O₂ levels were measured with an analyser (Servomex) and maintained at 21 kPa ± 0.3 kPa by an automatic injection system (Analox). The chamber atmosphere was scrubbed by an external life support system containing activated charcoal, silica gel and soda lime to remove organic gases and excess water vapor. Simulated submarine escapes were made from 2.5 MPa, based upon previous experience (1, 10). At 2.5 MPa, it was expected that VGE would evolve, but that life-threatening DCS would not occur.

A computer decompression model predicted that 60% O₂ was the lowest concentration to allow safe ascent from 250 msw (2.6 MPa) after saturation at 10 msw (0.2 MPa) for 24 h (11). Therefore a mixture of 60% O₂ and 40% N₂ was used as breathing gas during the escape phases.
Prior to compression control Doppler ultrasound measurements were made using the Kisman Masurel (KM) scoring system (12) and the audio output recorded onto digital audiotapes (DAT) using DAT recorders (Sony). Continuity of measurement was maintained throughout the studies by using the same Doppler operators. Any peculiarities in an individual goat’s gait were noted so that limb ‘bends’ (joint DCS) could be differentiated from normal posture or movement.

**SATURATION PROTOCOL**

**0.18 MPa saturation**

Goats (n = 20) were placed in the chamber, the doors secured, the chamber was pressurised to 0.18 MPa at a rate of 0.05 MPa per min. After 24 h, the chamber was brought to the surface at a rate of 0.05 MPa/s and the animals were moved to a holding pen where they were observed closely for 2 h. Doppler recordings were taken immediately on surfacing, and at 15 min, 30 min, 1 h, 1.5 h and 2 h post surfacing, then again at every hour until two successive measurements detected no VGE or eight hours had passed.

**ESCAPE PROTOCOL**

**2.5 MPa escape on air or hyperoxic gas**

Pairs of goats were placed in restraints in the 2 m sphere and oro-nasal masks were placed over their snouts and secured behind their heads. One mask had a sample line inserted near the nostrils that lead to a mass spectrometer outside the chamber so the breathing gases could be monitored. Both the air control (n = 8) and hyperoxic (n = 8) groups wore masks to eliminate differences caused solely by the mask. Breathing gas was supplied to the masks via the hood inflation system (HIS). The HIS (which provides breathing air and buoyancy in the escape suit) in SES replicates that of UK submarines. Once the animals were settled in the restraints, the chamber doors were sealed and the 3 m sphere was compressed to the backing pressure necessary to complete the 2.5 MPa escape profile. During this period of around 30 min, the 2 m sphere remained at atmospheric pressure with the animals breathing air. Once the backing pressure was reached, in the case of the hyperoxic group only, the HIS gas supply was switched to a mixture of 60% O₂ / 40% N₂ one to two minutes before the compression began. Gas from the 3 m sphere was then rapidly transferred to the 2m sphere, effecting a compression to 2.5 MPa in 24 s. Following a short hold at depth (4 s bottom-time), the 2 m sphere was decompressed to surface at a rate of 2.75 meters of seawater/second (msw/s) (0.0275 MPa) and breathing gas switched back to air (Figure 1). The remaining backing gas was emptied from the 2 m sphere and the goats taken from the 2 m sphere into holding restraints. They were held in restraints for 30 min., during which time Doppler recordings were made every five minutes. Expired CO₂ and O₂ were monitored intermittently and recorded using a sample line connected to an oro-nasal mask and a capnograph (Datex-Ohmeda). At the end of this period, the goats were placed in an observation pen and Doppler monitoring continued as described above.

**SATURATION + ESCAPE PROTOCOLS**

**AIR - 0.18 MPa saturation followed by 2.5 MPa escape on air**

Goats (n = 15) were introduced to the chamber. Following 24h saturation, two attendants ‘locked down’ to 0.18 MPa in the 2 m sphere and transferred the goats from the 3 m sphere into holding restraints within the 2 m sphere via the interconnecting door. The attendants then moved into the 3 m sphere, locked the middle door and were brought back to surface. The subjects remained in the 2 m sphere for around 30 min. before the escape, while the 3 m sphere was...
charged with gas to perform the 2.5 MPa escape profile. The escape involved a computer controlled rapid compression to 2.5 MPa over 30 s, followed by a short hold at depth (4 s) then decompression to surface at a rate of 2.75 msw/s (0.0275 MPa) (Figure 1). Once at surface, the goats were removed from the restraints and transferred into an observation pen for 2 h and monitored for any signs of DCS or O₂ toxicity. Precordial Doppler measurements were made using the same schedule as in the saturation protocol.

**Figure 1.** Plot A shows the computer generated profile used for escape only protocol (compression time 24 s), while plot B describes 24 h saturation at 0.18 MPa followed by escape (compression time 30 s). Note that in B, the x-axis (time scale) is interrupted to accommodate the entire dive period.
**HYPEROXIC GAS- 0.18 MPa saturation followed by 2.5 MPa escape on hyperoxic gas**

This trial followed the protocol for escape on air, as described in the preceding paragraph. However, once the animals (n = 16) were settled into the restraints in the 2 m sphere following post-saturation transfer, oro-nasal masks were fitted to deliver hyperoxic breathing gas during the escape phase. Again, a sample line was attached in order to verify that the animals breathed gas during escape of the correct composition (60% O$_2$/ 40% N$_2$). The escape profile and subsequent protocol was performed as described for saturation and escape with air.

**STATISTICS**

Mann Whitney U tests were employed for the analysis of KM Doppler scores. Further information on statistical testing is provided in the results section.

**RESULTS**

A summary of results is provided in Table 1.

**Saturation - 0.18 MPa**

Of the 20 goats subjected to this dive profile, none showed any signs or symptoms of DCI post-surfacing. However, VGE were detected on first measurement in every animal. At this point (15 minutes post-surfacing) the lowest KM score was 1-, the median was 2 and the maximum 4-, spanning almost the full range of the KM scale (12). Figure 2 shows the median Doppler scores for each time point up to 8 h post-dive. The median onset time to maximum Doppler score was 60 minutes. The median values were calculated and plotted by converting the KM scores to a numerical scale, where a "+" value added 0.33 and a " -" value subtracted 0.33.

**Escape - 2.5 MPa escape on air or hyperoxic gas**

Eight goats initially entered the 2.5 MPa escape trial on air. However on bringing one pair to surface, one animal was found to have suffered from a serious barotrauma (cerebral air embolism) and was immediately euthanised. Of the remaining seven, none showed any signs or symptoms of DCS or barotrauma. All showed normal ventilatory patterns and respiratory gas exchange. At 5 minutes post-surfacing, the median Doppler score was 3 (see Figure 2 and Table 1), which was also the maximum Doppler score noted for this dive series. At 4 hours post-surfacing, no precordial VGE were detectable by Doppler ultrasound.

Eight goats were also used for the 2.5 MPa escapes using hyperoxic breathing gas. All of these animals completed the escapes and did not show any signs of O$_2$ toxicity during the dives. On surfacing, none of the goats showed any signs or symptoms of DCI, or had respiratory problems. The median Doppler score of 3 was reached at 5 min., as was the maximum Doppler score (see Figure 2 and Table 1). However, unlike the air group, bubble evolution had ceased in all of the animals by 60 min. This rapid cessation, when compared with that of the air group, is shown in Figure 2.
Figure 2. Time course of post-dive detectable precordial venous gas emboli (VGE) for each dive profile. Values are converted (see Table 2) median Kisman Masurol (KM) Doppler scores.

Sat = saturation  
Esc = escape  
HYPEROXIC = group breathing 60% / 40% O₂/N₂ during escape  
AIR = group breathing air during escape profile  
Dotted line denotes entire dive conducted only on air  
Solid line denotes escape profiles conducted using hyperoxic HIS gas

To describe more fully the relationship between bubble cessation and the type of gas breathed, Figure 3 shows a box and whisker percentile plot describing the range of KM scores for each time point for both the air and hyperoxic groups. Although a good deal of overlap occurred in the magnitude of KM scores in the early stages post-escape, it can be seen that precordial VGE evolution and the range of KM scores fell in the hyperoxic breathing subjects much sooner than in the air group.

A modified Mann Whitney U test was utilized to determine any significant difference between the air and hyperoxic groups, in terms of disappearance of VGE. Tests were performed across the time period from which animals in the hyperoxic group had started to score KM zero (no precordial bubbles found), to the point at which all animals in this group had ceased to bubble and had zero scores. Therefore, paired data at four points, 25, 30, 45 and 60 minutes post surfacing, were tested. Consequently, the significance level of the test was reduced from 0.05 to p<0.0125 (0.05/4) to compensate for repeated testing. Data of the 45 and 60 minutes time points showed that p < 0.01, therefore from 45 minutes onwards, the amount of circulating bubbles was significantly lower in the group of animals that had been breathing hyperoxic gas during the
escape, than in the air breathing group. The median time for all VGE to disappear was significantly shorter in the hyperoxic group (p<0.05).

**Figure 3.** Box and whisker percentile plot comparing the range of precordial KM Doppler scores against time for both the hyperoxic and air groups following 2.5 MPa simulated escape.

Black bars denote group breathing air during the escape
Open bars denote group breathing 60% / 40% O_{2}/N_{2} during escape
Boxes show the extent of the 25^{th} to 75^{th} percentile
Whiskers show the extent of the 10^{th} to 90^{th} percentile
Black or white lines within the box denote the median values

**Saturation + Escape**

**0.18 MPa saturation followed by 2.5 MPa escape on air or hyperoxic gas**

15 animals were subjected to 24 h at 0.18 MPa (8msw) followed by escape from 2.5 MPa while breathing air. Four of these suffered from DCS after return to surface. One case of central nervous system (CNS) DCS occurred, with the symptoms (spinal involvement) presenting almost immediately on surfacing (5 min.). The remaining three cases were single limb DCS; one presented at 1 hour post-surfac ing (right fore-leg pain), the second at 1h 20 min. post surfacing (left hind-leg) and the third at 1h 25 min. post surfacing (right hind-leg). All four animals were treated for DCS. The remaining 11 animals showed no signs or symptoms of DCI.

At 15 minutes post-surfacing (the earliest time point measured in this section of the study), the median Doppler score was 3+ (see Figure 2), ranging from 3+ to 4. The circulating VGE then increased to a median KM of 4, which was maintained for the next 90 minutes. At 8 h post surfacing, all of the animals were still producing precordial bubbles and the median KM Doppler score at this time was 3. The full range of KM scores for each time point can be seen in Figure 4.
Sixteen animals entered the study of 24 h air saturation followed by escape using hyperoxic breathing gas. Of these, two suffered from DCS. One animal suffered from severe CNS DCS on reaching the surface and was immediately euthanised. The second case was also CNS DCS (staggering and loss of co-ordination) and presented at 17 minutes post surfacing. None of the other animals showed any signs or symptoms of DCI throughout the 8 h observation period and their respiratory signs were normal. At five min. post-surfacing the median Doppler score was 4 -, and then peaked at 20 min. post-surfacing at KM 4. However, this peak was only maintained for five min.; the Doppler scores then started to decrease gradually. At 4 h post-surfacing, a KM score of zero was recorded in one animal and by 8 h, all but three goats had a score of zero. Figure 4 shows the rate of decline in terms of Doppler scores for the hyperoxic group in comparison to that of the air group, who, as noted previously, retained high KM scores right through to 8 h.

Again, a modified Mann Whitney U test was used at five time points (240, 300, 360, 420 and 480 min.) over which time this hyperoxic group’s KM scores reached zero. The significance level of the test was reduced to 0.01 (0.05/5). At every point tested, p ≤ 0.01, showing that the rate at which the Doppler scores of the hyperoxic group fell was significantly more rapid than that of the air group.

DISCUSSION

VENOUS GAS EMBOLI (VGE) DOPPLER SCORES

All protocols involving simulated submarine escapes promoted large numbers of Doppler detectable VGE. The maximum bubble scores appeared only a short time after surfacing, and in some cases the maximum may have occurred before the first Doppler measurements could be made. It is expected that maximum bubble evolution will occur very shortly after surfacing, since this type of dive profile (rapid ascent) targets the fast tissues (1,7). Therefore, the onset of bubbling may be expected earlier than in saturation dives, as seen in Figure 2, on comparison of the curves for 0.18 MPa saturation and the 2.5 MPa escapes.
There was no discernable difference in the early bubble scores between the groups breathing air or 60% O\textsubscript{2} during submarine escape. This is not surprising, as the total inspired gas load would be similar in both groups, as described below.

As O\textsubscript{2} is metabolized and the more soluble gas CO\textsubscript{2} is produced, the sum of the venous partial pressures of these metabolic gases is less than that on the arterial side, forming the ‘oxygen window’ (12). Thus a higher oxygen partial pressure will theoretically allow more N\textsubscript{2} to be dissolved and transported in the tissues and venous blood without bubble formation. However, when the PO\textsubscript{2} exceeds the amount of O\textsubscript{2} that the tissues can metabolize, venous PO\textsubscript{2} will start to increase. The excess O\textsubscript{2} will then be able to act as an inert gas and contribute to the formation of bubbles (13), though to a lesser degree than the equivalent amount of N\textsubscript{2} (9). The actual PO\textsubscript{2} when this occurs depends on blood flow and metabolic rate of the tissue in question, but very high O\textsubscript{2} partial pressures during the submarine escapes will undoubtedly exceed the metabolic requirements of even the fastest tissues.

As the amount of gas dissolved in the tissues will therefore be similar in both the hyperoxic and air groups, the initial bubbling will also be similar. However the time to resolution of bubbles was much quicker in the group breathing hyperoxic gas (Figure 2). It is assumed that bubbles formed after air dives mostly contain nitrogen, but in the hyperoxic groups, bubbles appearing immediately after ascents will contain a high ratio of O\textsubscript{2} to N\textsubscript{2}. While N\textsubscript{2} in bubbles can only be transported out of tissues via diffusion, O\textsubscript{2} may be consumed in the tissue as time progresses at surface. This will increase the PO\textsubscript{2} gradient between the inside and the outside of the bubbles and accelerate its removal, while also increasing the PN\textsubscript{2} in the bubble. Thus the N\textsubscript{2} diffusion gradient will be increased, and so the combination of these two mechanisms should allow bubbles to resolve more rapidly, hence the swifter time to resolution in the hyperoxic group. A reduction in the overall bubbling period will reduce the risk of DCS (14). Therefore breathing a hyperoxic gas during submarine escape could be of benefit in this respect.

**DECOMPRESSION ILLNESS**

Pulmonary barotrauma with arterial gas embolisation (AGE) is an ever-present risk during submarine escape. Goats usually exhale spontaneously during the ascents (1), however, on occasion, they succumb to AGE. Clinically it is difficult to differentiate between severe neurological DCS or AGE, even after autopsy. In the present study all sudden deaths or cases with CNS symptoms were considered to be DCS if there were no clear signs of pulmonary barotrauma, i.e. rapid onset of symptoms on surfacing (< 20 min.), oral/nasal bloody froth, and/or post mortem findings. Only one case, occurring after submarine escape from 2.5 MPa in the air group, could be clearly classified as barotrauma / AGE (Table 1).

Two cases of CNS DCS occurred after saturation followed by submarine escape with hyperoxic gas, and another occurred after the same dive profile but using air as the escape gas. Thus, no difference in the incidence of CNS DCS between these groups was seen. As CNS DCS is thought to be linked to the initial bubble load, this was in keeping with the fact that there was no difference in the initial bubble scores between the groups.

The saturation and escape on air profile also produced three cases of limb DCS. The symptoms appeared 60 to 120 min. after the animals had reached surface. However, no limb DCS, or any other symptoms were observed in the hyperoxic group past 20 min. post-surfacing. The probability for such a difference to occur between these two groups was calculated using Fischer’s exact test (p = 0.11). Although not statistically significant, it does indicate a trend toward reduced occurrence of, or protection against late DCS when breathing hyperoxic gas.
during the escape. Concurrently, the limb DCS in the air group appeared during a period when their bubble scores were significantly higher than those of the hyperoxic group.

Late DCS usually manifests as limb pain, which although uncomfortable and possibly debilitating, is not as potentially damaging as the early onset CNS DCS. Therefore, although the administration of a hyperoxic gas during submarine escape will reduce the overall bubble load, importantly, it does not appear to reduce the risk of CNS DCS developing. Further experimental work should be carried out to determine how to reduce the initial bubble load.

**OXYGEN TOXICITY**

Pure O₂ at increased pressure is a potent convulsing agent, producing tonic-clonic convulsions after a relatively short exposure (15). O₂ convulsions may be fatal if the upper airways are blocked during a submarine escape ascent, while convulsions at the surface increase the risk of drowning. Therefore, it is important that the use of O₂ to decrease the risk of DCI is balanced against the risk of inducing acute O₂ toxicity. In ordinary diving operations the maximum inspired PO₂ is generally limited to between 140 and 200 kPa to avoid O₂ convulsions. However, there is a minimum latency period before the onset of oxygen convulsions, regardless of the oxygen partial pressure (15, 16, 17, 18). Since the minimum latency to O₂ convulsions is four minutes, even at O₂ pressures as high as 30 ata (18), the rapid course of submarine escape would protect against acute O₂ toxicity.

Appositely, the goats in the present study were exposed to a maximum inspired PO₂ of 1500 kPa (15 ata) for 4 s. The inspired PO₂ was above 140 kPa (the lower limit for O₂ convulsions (16)), for less than 100 s. No signs of acute O₂ toxicity, such as increased skittishness, myoclonic jerks or overt convulsions were observed in any of the goats. Therefore, it is hoped that humans may use this hyperoxic gas protocol without risk of O₂ toxicity, although it must be remembered that goats were tested here.

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