# Diving and Hyperbaric Medicine

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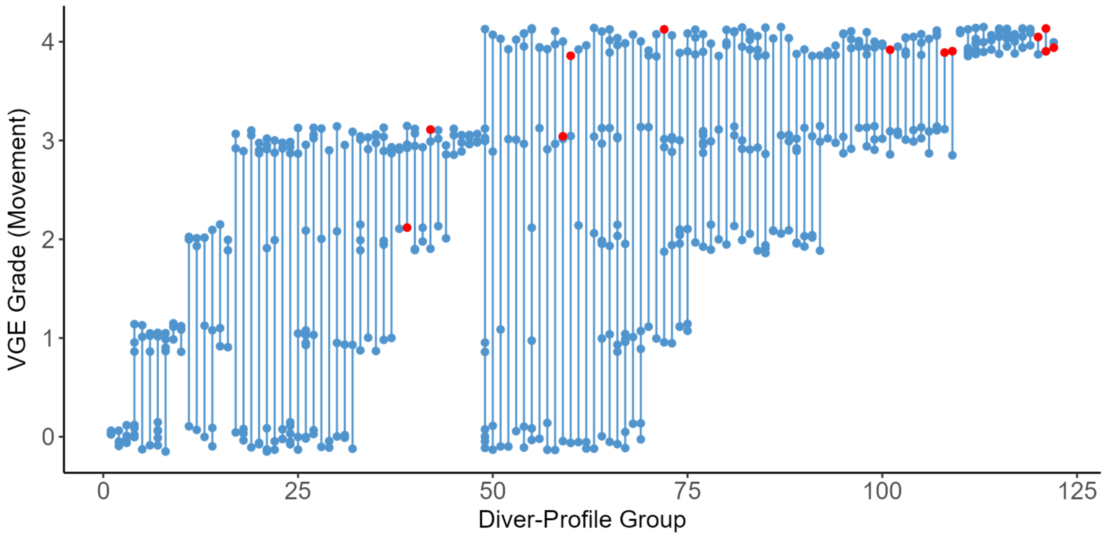

Marked within-diver variability in post-dive VGE

Evaluating a Swedish Navy dive table

Impaired decision-making by nitrogen narcosis

Hypoxia and hypercapnia using full face snorkel masks

Measuring whole body inert gas washout

Capnography in hyperbaric chambers

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**To promote and facilitate the study of all aspects of underwater and hyperbaric medicine**
**To provide information on underwater and hyperbaric medicine**
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## DIVING AND HYPERBARIC MEDICINE

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# The Editor's offering

Welcome to the final issue of DHM for 2023.

It is rare to claim we are publishing a 'landmark paper', but I believe that to be the case in this issue with the publication of the manuscript by David Doolette and Greg Murphy in which they report on within-diver variability in post-dive venous gas emboli (VGE) production. The authors extracted relevant data from their large databases of experimental dives that included recording of post-dive bubble grades and recording of clinical outcomes. These datasets included many instances of individual divers repeating identical dives multiple times. Importantly, these dives were typically closely controlled for important variables that might influence bubbling like depth, time, decompression, temperature, thermal protection, and exercise levels. The defining finding was that the same diver commonly produced markedly variable bubble grades after essentially identical dives. Some may claim not to be surprised by these findings, but until now there was a paucity of reliable data demonstrating the phenomenon. The study also showed that when decompression sickness occurred it was almost always on those occasions when the victims produced high bubble grades. Considered together, these results suggest that seeking strategies to reduce post-dive bubbling remains an important goal in prevention of decompression sickness, but more importantly, they also beckon us to identify and potentially manipulate the as yet unknown pre-dive factors that are responsible for the reported VGE variability. This paper has multiple important implications for our knowledge of decompression pathophysiology, and for defining future research directions.

In other articles Carl Hjelte and colleagues evaluated the safety of a new Swedish armed forces dive table and found its use may be associated with a higher risk of decompression sickness than assumed in its design. In a second paper the same group (this time fronted by Oscar Plogmark) describe a relatively simple method / equipment configuration for measuring whole body inert gas washout. This tool may prove valuable in researching decompression pathophysiology.

In another publication double, our own group has two papers in this issue. In the first, Janneke Grundeman and colleagues investigated rebreathing in full face snorkel masks (FFSMs) as a possible cause of hypercapnia or hypoxia during their use. Both conditions were more likely when breathing via a FFSM than a conventional snorkel. In the second, Xavier Vrijdag and colleagues compared end-tidal $CO_2$ measurements in a hyperbaric chamber at 284 kPa made simultaneously with an in-line EMMA capnograph and via side stream sampling to an analyser outside the chamber. In a subset of subjects an arterial blood gas sample was also taken simultaneously. These three measurements showed clinically acceptable confluence between methods. Measurement of end-tidal $CO_2$ in ventilated patient in hyperbaric chambers is important yet long portrayed as difficult, but this study and others recently published are reassuring in relation to use of the simple in-line EMMA device.

In the remaining original article Pauliina Ahti and Jan Wikgren demonstrated measurable impairment of decision-making during air breathing at 30 m depth. The study included a control group of divers at 5 m, making the difference in performance between 5 and 30 m a compelling finding. It has long been speculated that relevant degrees of narcosis occur during air diving from about 30 m, and this study appears to confirm that.

In a short communication, Feiko de Jong and colleagues extend their work on measurement of exhaled volatile organic compounds after diving into the field and report outcomes for naval heliox dives to 81 m. Hopefully this represents a beginning of refinement of this methodology for use in operational environments.

There are three interesting case reports in this issue. In the first Neil Banham and colleagues report a case of pulmonary barotrauma leading to cerebral arterial gas embolism (CAGE) and small apical pneumothoraces arising during controlled emergency swimming ascent training on an open water diver course. The diver had neurologically recovered when seen at the hyperbaric unit, generating an interesting decision-making conundrum (given the presence of the pneumothoraces) about whether to recompress an asymptomatic CAGE victim in this particular circumstance. In the second, Kubra Canarslan Demir and colleagues report a case of apparent delayed hypoxic encephalopathy in whom recovery appeared associated with hyperbaric oxygen treatment. The authors are appropriately cautious with their conclusions, and advocate further investigation of this indication. Finally, Arnaud Druelle and colleagues report three cases (from two incidents) who suffered hypoxic loss of consciousness after breathing from a scuba tank in which corrosion had consumed almost all oxygen from the air within. Although a known phenomenon, careful documentation and reporting of such cases is very rare.

The editorial team wish SPUMS and EUBS members, and all readers of the journal a happy and safe festive season.

*Professor Simon Mitchell*
*Editor*

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**Cover photo:** Figure 2 from the seminal Doolette and Murphy paper published in this issue demonstrating remarkable variability in post-dive bubble grades in divers completing identical profiles; each vertical line represents a single diver completing several identical dives and each dot represents the bubble grade after a dive; blue dots are dives with no DCS and red dots are dives in which DCS occurred.

# Original articles

## Risk assessment of SWEN21 a suggested new dive table for the Swedish armed forces: bubble grades by ultrasonography

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**Keywords**
Decompression; Decompression illness; Decompression tables; Diving; Echocardiography; Risk; Venous gas emboli

**Abstract**
(Hjelte C, Plogmark O, Silvanius M, Ekström M, Frånberg O. Risk assessment of SWEN21 a suggested new dive table for the Swedish armed forces: bubble grades by ultrasonography. Diving and Hyperbaric Medicine. 2023 December 20;53(4):299–305. doi: 10.28920/dhm53.4.299-305. PMID: 38091588.)
**Introduction:** To develop the diving capacity in the Swedish armed forces the current air decompression tables are under revision. A new decompression table named SWEN21 has been created to have a projected risk level of 1% for decompression sickness (DCS) at the no stop limits. The aim of this study was to evaluate the safety of SWEN21 through the measurement of venous gas emboli (VGE) in a dive series.
**Methods**: A total 154 dives were conducted by 47 divers in a hyperbaric wet chamber. As a proxy for DCS risk, serial VGE measurements by echocardiography were conducted and graded according to the Eftedal-Brubakk scale. Measurements were made every 15 minutes for approximately 2 hours after each dive. Peak VGE grades for the different dive profiles were used in a Bayesian approach correlating VGE grade and risk of DCS. Symptoms of DCS were continually monitored.
**Results**: The median (interquartile range) peak VGE grade after limb flexion for a majority of the time-depth combinations, and of SWEN21 as a whole, was 3 (3–4) with the exception of two decompression profiles which resulted in a grade of 3.5 (3–4) and 4 (4–4) respectively. The estimated risk of DCS in the Bayesian model varied between 4.7–11.1%. Three dives (2%) resulted in DCS. All symptoms resolved with hyperbaric oxygen treatment.
**Conclusions:** This evaluation of the SWEN21 decompression table, using bubble formation measured with echocardiography, suggests that the risk of DCS may be higher than the projected 1%.

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### Introduction

Decompression tables are of fundamental importance for diving safety and to avoid decompression sickness (DCS). To mitigate the risk for DCS they suggest maximum time and depths combinations. In the Swedish armed forces (SwAF) there are several branches and services with units conducting diving operations. Traditionally the SwAF has adopted the United States Navy (USN) decompression tables and the current tables used in the SwAF, called RMS-dyk 13 tables 1 and 2, are a metric conversion of the USN decompression table revision 6 (USN 6). In 2017 the USN introduced a new decompression table called USN revision 7 (USN 7) and now the SwAF needs to decide if they will adopt this novel table, stay with the current table or choose a third option.

Decompression tables are mainly developed in two ways; either by a probabilistic approach where a database of previous dives is used as a guide to decide which time and depth combinations have an acceptable safety profile or through a deterministic model where the knowledge of gas physiology is used to estimate risk for DCS. In 2021 mathematicians and engineers employed by the SwAF constructed a probabilistic model based on a new database consisting of 2,953 dives which in turn was used to create a deterministic algorithm comprising nine tissue compartments, the details of this work are described in a recent paper.[1] This was then used as a framework to develop a new decompression table called SWEN21 which has a projected risk level of 1% for DCS. To verify a decompression table with an incidence of 1% for DCS by examining the dichotomous outcome of DCS or no DCS with an acceptable confidence interval would require several hundreds of dives to be performed.[2,3] This would be highly time consuming and it is therefore widely accepted to use a proxy, namely intravascular bubbles, usually referred to as venous gas emboli (VGE). Previous studies have shown that an increasing amount of VGE detected in the right side of the heart correlates with the incidence of DCS.[2,4–6] The largest published dataset to date correlating DCS and VGE consists of 3,234 dives and indicates a stepwise increase of DCS risk with 0.1% risk when there are no detectable bubbles up 11.5% risk when there is a large amount of VGE.[4]

Venous gas emboli detection is most commonly performed using Doppler flow signals or by two dimensional (2D) ultrasonic imaging of the heart. The amount of VGE is then classified according to a scale and given a grade. When using the Doppler method the grading systems most frequently used are the Spencer or the Kisman-Masurel scale.[2] When using 2D echocardiography the most common grading system is the Eftedal-Brubakk scale.[7]

Though the risk for DCS when using SWEN21 was estimated to be 1% the true operational risk was not known. To ensure the safety of this novel decompression table validation was needed before implementation.

The aim of this study was to evaluate the safety of SWEN21 through the measurement of VGE in a dive series in a hyperbaric wet chamber. The data in this study are from the validation dive series called ValTKLHN2021.

## Methods

### ETHICAL CONSIDERATIONS

The Swedish Ethical Review Authority approved the application "*New decompression tables for the Swedish Armed Forces*" (Dnr: 2020-06865). All subjects provided their informed written consent to participate before the start of the study. The study was conducted in accordance with the Declaration of Helsinki.

### DIVE PROCEDURES

Eight profiles were identified to examine the safety of SWEN21. These combinations were chosen to test the underlying algorithm of the deterministic model which consists of the nine tissue compartments characterised by a unique halftime and supersaturation quota. The respective time-depth combination resulted in a single compartment being the rate limiting step, or 'leading tissue', from becoming supersaturated in that particular dive. Compartments with halftimes exceeding 40 minutes were not tested at this stage of table development since this was considered too time-consuming.

All dives were performed at the SwAF Diving and Naval Medicine Center (DNC) located on the naval base in Karlskrona in a hyperbaric wet chamber (HAUX 2300). The chamber is a horizontal cylinder 2.6 m in diameter and the desired pressure was set at 0.3 m from the chamber floor. Divers were directed stay at that depth but could deviate to 0.3 m below or 2.3 m above since they swam freely in the water. If divers were deviating from the intended depth the chamber operator directed them to go to the intended depth. Water temperature was 10 degrees Celsius ± 1 degree to mimic typical Swedish operational conditions. Each dive involved two subjects fitted with dry suits, undergarments, wet gloves and the Divator MkIII open circuit breathing apparatus (Interspiro, Taby, Sweden). In the dive profiles with oxygen decompression the gas was supplied via a built in breathing system (BIBS) mask with the diver standing with the head out of water but the rest of the body submerged. Compression and decompression were performed in accordance with USN 7 at 23 metres of seawater (msw)$\cdot$min$^{-1}$ and 9 msw$\cdot$min$^{-1}$ respectively. To replicate operational conditions all divers performed low intensity fin swimming during bottom time and decompression. This was accomplished with the diver suspended with an elastic cord to the back off the diving pack or the diver swimming up against the front of the chamber.

### ASSESSMENTS

Venous gas emboli in the right heart were recorded by 2D echocardiography and cases of DCS were clinically assessed. Within 5–15 minutes of surfacing VGE measurements were obtained from each subject and thereafter every 15 minutes for at least a total of seven sessions (spanning approximately two hours). The images were obtained with the subject lying in the left lateral decubitus position with the probe positioned for an apical four chamber view. If this position produced an inadequate view the subject was shifted to supine position and the probe positioned in the subcostal position. The 2D cardiac images were obtained using a portable echocrdiography device with a cardiac probe (EDGE II, Fujifilm SonoSite). Harmonic imaging was used since previous studies have shown that this may increase the sensitivity of bubble detection.[7] The VGE grading was done according to the Eftedal-Brubakk scale by two physicians in real time and a grade between 0–5 was given (Table 1). Resting grade was measured first then the flex grade after three vigorous knee extensions with the subject laying in the same position. For the flex grade the highest amount of VGE sustained for two consecutive heart cycles was used for grades of 4 and higher, for grades of 3 or lower the highest amount of VGE sustained for four consecutive cardiac cycles was used. Only the highest VGE grade at any time point from the two conditions (rest or limb flexion) from each man-dive was noted and the median of these values from the respective dive profile was used in this report and will be referred to as 'VGE grade' henceforth. Symptoms consistent with DCS were recorded every 15 minutes in a standardised form

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**Footnote:** * Appendix 1 and 2 are available on DHM Journal's website: https://www.dhmjournal.com/index.php/journals?id=323

**Table 1**
Eftedal-Brubakk Scale for grading venous gas emboli (VGE)

| Grade | Description |
|---|---|
| 0 | No bubbles visible |
| 1 | Occasional bubbles |
| 2 | At least one bubble every four cardiac cycles |
| 3 | At least one bubble every cardiac cycle |
| 4 | At least one bubble per cm$^2$ in every image |
| 5 | Single bubbles cannot be discriminated |

(Appendix 1). If divers showed symptoms a dive physician examined the subject and in a dialogue with the research team decided if the symptoms could be classified as DCS. The reason the research team was involved in the diagnosis of DCS was due to the fact that the diagnosis is based primarily on subjective reporting and many of the clinical observations are ambiguous. Therefore, DCS diagnosis is prone to interobserver variance and there are few accepted objective criteria; this is especially true with cutaneous manifestations according to the clinical experience of this research team. To minimise the risk of misclassification due to subjectivity we decided that classification of DCS in this study would be done in accordance with the definition in Sawatzky's thesis (Appendix 2).[4] The exception was the classification of cutaneous manifestations which was done in accordance with the description by Hartig et al.[8] since we deemed this detailed description was less susceptible to subjective interpretations. The decision to initiate hyperbaric oxygen treatment (HBO) or normobaric oxygen treatment (NBO) was made by the dive supervisor or dive physician.

## STUDY SUBJECTS

The subjects were aged 20–59 years and all except two were men. The fitness level varied between subjects; some were exercising an hour per week whereas others did over 10 hours per week. The majority were exercising 3–4 hours per week. Most subjects, with the exception of seven relatively recent graduates from SwAF diving school, had extensive experience with diving with several hundreds of logged dives in the military context. Many were also recreational divers. Two of the divers reported that they previously had DCS, during this series none of them experienced new events of DCS. All divers met the SwAF fitness to dive standard. Exclusion criteria were diving within the preceding 48 hours to avoid residual nitrogen load which could result in more bubble formation, ongoing infection and physical training the last 24 hours as previous work has shown this could lower bubble grade.[9,10] Subjects were recruited by mail inquiry to the respective diving units in the SwAF by the staff of DNC. Participation was voluntary and that participants could discontinue the study at any time without any explanation, and neither participation nor discontinuation would affect their military career.

## STATISTICAL ANALYSIS

Venous gas emboli grades are ordinal data. Median and interquartile range (IQR) of the VGE grades were calculated for each separate diving profile, all direct ascent dives, all decompression dives and all the dives combined.

To estimate the risk of DCS we implemented the method described by Eftedal.[3] In this approach the risk of DCS after diving a specific profile $T$ was modeled as

$$P(DCS \mid T) = \sum_{i=0}^{5} P\,(DCS \mid BG_i)P(BG_i \mid T) = \sum_{i=0}^{5} p_i\, q_i(T)$$

where $p_i = P(DCS \mid BG_i)$ was the risk of DCS when the maximum observed bubble grade was $i$, and $q_i(T) = P(BG_i \mid T)$ was the probability of developing bubble grade $i$ after dive profile $T$. The probabilities $p_i$ and $q_i(T)$ were treated as random variables, whose probability distributions were estimated from a prior and empirical data using Bayesian statistics. A Monte Carlo simulation ($n = 500{,}000$ samples) was used to approximate the resulting probability distribution of $P(DCS \mid T)$ and determine a 95% credible interval.

The inputs to this algorithm were:

1. A prior distribution for the probabilities $p_i$. The prior suggested by Eftedal et al.[3] in their Equation 4 was used, namely a point mass of weight 0.1 at each of the endpoints $p_i = 0$ and $p_i = 1$ and the remaning mass distributed uniformly.
2. A prior distribution for the probabilities $q_i$. The prior suggested by Eftedal et al.[3] in their Equation 5 was used, namely a Dirichlet distribution with parameters: $\gamma = [0.5051, 0.3503, 0.3132, 0.6264, 0.2785]$.
3. A dataset $D_{BG \rightarrow DCS}$ of dives with recorded bubble grades and DCS outcomes. These empirical data, together with the prior distributions of $p_i$ were used to compute a posterior distribution for each $p_i$. For this input we used two different datasets:
    (a) The data from Sawatzky, cited by Eftedal et al.[3] in their Table 1.
    (b)The data cited by Doolette in his Table 2.[11]
4. A dataset $D_{T \rightarrow BG}$ of dives performed according to a specific dive profile $T$ with recorded bubble grades. These empirical data, together with the prior distribution of $q_i$ were used to compute a posterior distribution for $q_i$. For this input we used the dives from the present trial.
5. We assumed the correspondence between VGE grades from the Doppler data obtained by Sawatzky, which was graded with the Kisman Masurel scale, and our own VGE data, graded with the Eftedal-Brubakk scale, to be zero to zero, one to one and so forth. The same correspondence was assumed with the VGE grades from Doolette's data, graded according to Naval Experimental Diving Unit

**Table 2**

Venous gas emboli (VGE) grades (Eftedal-Brubakk scale) for SWEN-21 from the validation dive series ValTKLHN2021; [#] decompression with air (see Appendix 3 for respective decompression schedules); [¤] decompression with oxygen (See Appendix 3 for respective decompression schedules); [a] numbers of divers with cutaneous manifestation of diving not to be classified as decompression sickness (DCS), e.g., redness and pruritus; [b] diver who received prophylactic NBO because of arterial bubbles; HBO – hyperbaric oxygen treatment; IQR – interquartile range; min – bottom time minutes; msw – metres of seawater; NBO – normobaric oxygen treatment

| Dive profile msw / min | *n* dives | Median (IQR) VGE grade rest / flexing | *n* DCS | DCS-type (peak VGE grade of individual DCS cases) | *n* cutaneous stress[a] | Treatment |
|---|---|---|---|---|---|---|
| **Direct ascent profiles** | | | | | | |
| All direct ascent profiles | 100 | 3 (1–3) / 3 (2–4) | 2 | 2 musculoskeletal | 1 | 2 HBO<br>1 NBO |
| 18 / 59 | 20 | 3 (2–3) / 3 (3–3.5) | 2 | Shoulder pain (3)<br>Hip pain (4) | | 2 HBO |
| 24 / 33 | 20 | 3 (1–3) / 3 (2–4) | | | | – |
| 33 / 17 | 24 | 3 (2–3) / 3 (3–4) | | | 1 | 1 NBO |
| 39 / 12 | 20 | 3 (1.5–3) / 3 (1.5–4) | | | | – |
| 45 / 8 | 16 | 2 (1–3) / 3 (2–3) | | | | – |
| **Decompression profiles** | | | | | | |
| All decompression profiles | 54 | 3 (2–4) / 3 (3–4) | 1 | 1 neurological | 8 | 1 HBO<br>2 NBO |
| 39 / 20[#] | 22 | 3 (2.5–4) / 3.5 (3–4) | | | 1 | 1 NBO |
| 51 / 10[#] | 16 | 2.5 (1.5–3) / 3 (2–4) | 1 | Sensory loss leg and torso (4) | | 1 HBO |
| 57 / 15[#] | 8 | 4 (3.5–4) / 4 (4–4) | | | 5 | 1 NBO[b] |
| 57 / 15[¤] | 8 | 3 (1.5–3.5) / 3 (2–4) | | | 2 | – |
| **All profiles combined** | | | | | | |
| All profiles | 154 | 3 (2–3) / 3 (3–4) | 3 | 1 neurological,<br>2 musculoskeletal | 9 | 3 HBO<br>3 NBO |

(NEDU) 2-D echocardiography VGE scale, namely zero to zero, one to one and so forth. Both the Kisman-Masurel and the NEDU 2-D scales consist of grades 0–4 whereas the Eftedal-Brubakk consists of grades from 0–5. In the present study we saw no VGE of grade 5 and to the authors' knowledge this has only been seen in animal studies. Therefore, we did not consider this grade or its corresponding value in the other grading scales.

## Results

A total of 154 dives were performed by 47 divers. There were three cases of DCS. Two of the events were classified as musculoskeletal (joint pain) and the third was classified as neurological with symptoms in the form of sensory loss in a leg and parts of the torso. All DCS cases were treated with HBO according to the US Navy Treatment Table 6 with complete resolution of findings and symptoms. One of the divers who experienced DCS after a 18 msw / 59 minute dive showed arterial bubbles later in the series after a 57 msw / 15 minute dive with air decompression. Follow up medical examination showed that this individual has a patent foramen ovale (PFO). The diver who developed neurological DCS was upon examination also shown to have a PFO. Nine divers showed cutaneous manifestations in the form of red skin and or pruritus, none were classified as DCS but instead as decompression stress.

Three divers received prophylactic treatment with NBO by the attending dive physician, two because of non-DCS cutaneous manifestations and one because of visible arterial bubbles on ultrasonography. The two divers who developed musculoskeletal DCS after the 18 msw / 59 minute profile received NBO at 105 minutes after surfacing, prior to HBO-treatment. Receiving NBO may lower VGE grades but since all the divers in the above-mentioned cases, except one, had a grade of 4 in at least in one of the conditions these were kept in the Bayesian DCS risk calculations. The exception was one diver in the 18 msw / 59 minutes profile who had a VGE grade 3 prior to NBO, he was excluded from the

**Table 3**

Distribution of individual peak venous gas emboli (VGE) grades (Eftedal-Brubakk scale) by dive profile; # decompression with air (see Appendix 3 for respective decompression schedules); ¤ decompression with oxygen (See Appendix 3 for respective decompression schedules); min – bottom time minutes; msw – metres of seawater

| Profile msw / min | *n* dives | VGE grade | | | | |
|---|---|---|---|---|---|---|
| | | 0 | 1 | 2 | 3 | 4 |
| 18/59 | 20 | 1 | 2 | 1 | 12 | 4 |
| 24/33 | 20 | 0 | 4 | 2 | 8 | 6 |
| 33/17 | 24 | 0 | 2 | 2 | 11 | 9 |
| 39/12 | 20 | 2 | 3 | 1 | 6 | 8 |
| 45/8 | 16 | 0 | 3 | 4 | 6 | 3 |
| 39/20# | 22 | 0 | 0 | 0 | 11 | 11 |
| 51/10# | 16 | 1 | 1 | 4 | 5 | 5 |
| 57/15# | 8 | 0 | 0 | 0 | 1 | 7 |
| 57/15¤ | 8 | 0 | 0 | 3 | 2 | 3 |
| Total | 154 | 4 | 15 | 17 | 62 | 56 |

**Table 4**

Estimated risk of DCS for the different dive profiles using Bayesian statistics; * a database of dives correlating VGE grade with risk of DCS; # decompression with air (see Appendix 3 for respective decompression schedules); ¤ decompression with oxygen (See Appendix 3 for respective decompression schedules); CI – credible interval; min – bottom time minutes; msw – metres of seawater

| Dive profile msw / min | $D_{BG\rightarrow DCS}^{*}$ with Sawatzky's dataset[4] | | $D_{BG\rightarrow DCS}^{*}$ with Doolette's dataset[11] | |
|---|---|---|---|---|
| | Estimated DCS risk % | 95% CI | Estimated DCS risk % | 95% CI |
| **Direct ascent profiles** | | | | |
| All direct ascent profiles | 7.6 | 5.0–10.5 | 5.3 | 3.5–7.2 |
| 18 / 59 | 7.2 | 4.5–10.2 | 5.6 | 3.4–8.0 |
| 24 / 33 | 7.3 | 4.3–10.6 | 5.1 | 3.2–7.2 |
| 33 / 17 | 8.5 | 5.2–12.1 | 5.7 | 3.7–7.9 |
| 39 / 12 | 7.7 | 4.2–11.6 | 4.7 | 2.9–6.8 |
| 45 / 8 | 5.9 | 3.3–8.8 | 4.7 | 2.9–6.8 |
| **Decompression profiles** | | | | |
| All decompression profiles | 9.3 | 5.6–13.3 | 5.5 | 3.6–7.6 |
| 39 / 20# | 10.1 | 6.2–14.4 | 6.2 | 4.0–8.6 |
| 51 / 10# | 6.8 | 3.6–10.4 | 4.7 | 2.8–6.7 |
| 57 / 15# | 11.1 | 5.4–17.5 | 5.3 | 2.8–7.9 |
| 57 / 15¤ | 7.0 | 3.1–11.3 | 4.7 | 2.6–6.9 |
| **All profiles combined** | | | | |
| All profiles | 8.3 | 5.3–11.5 | 5.4 | 3.6–7.3 |

Bayesian DCS risk calculation because the NBO might have lowered his VGE grade.

The median VGE grades for each dive profile are shown in Table 2. The majority of the depth-time combinations resulted in a grade of 3 both at rest and after flexing which was also the median of the SWEN 21 table as a whole. The exceptions were the 57 msw / 15 minutes profile with decompression on air which resulted in a median bubble grade of 4 at rest and after flexing (IQR 3.5–4 and 4–4 respectively), the 51 msw / 10 minutes profile with air decompression which at rest resulted in median grade 2.5 (IQR 1.5–3) and the 39 msw / 20 minutes profile which after flexing resulted in median grade 3.5 (IQR 3–4). The distribution of individual VGE grades per profile can be viewed in Table 3.

The estimated DCS risk for the different dive profiles is shown in Table 4. Using the data from Doolette[11] correlating VGE grade and DCS outcome as inputs to the Bayesian model suggests the overall risk of DCS using SWEN 21 is 5.4% (95% CI 3.6–7.3); varying between 4.7–6.2%

risk between the different profiles with the decompression profiles having slightly higher risk. Using the Sawatzky data[4] as inputs suggests a DCS risk that is substantially higher with an overall risk of 8.3% (95% CI 5.3–11.5) and varying between 5.9–11.1% risk between the different profiles. Once again the decompression profiles have a higher estimated DCS risk.

## Discussion

The main finding in this study is that according to VGE grades the estimated risk of DCS when using SWEN21 may be higher than the projected 1%. Using Doolette's data as input in the Bayesian model the risk of DCS when using SWEN 21 is around 5% and all of the profiles have a credible interval over 1% which indicates that the DCS risk is likely higher than this. Using the data from Sawatzky as input the overall risk is around 8% with all credible intervals well above 1% which also indicates that the risk is higher than that. The advantage of using the Sawatsky dataset is that it is large and could therefore be assumed to more accurately correlate VGE grade to DCS risk. The downside is that dive conditions and VGE grading might not be consistent with the present trial. The experiments by Sawatzky and colleagues at the Defense and Civil Institute of Environmental Medicine (DCIEM) were done during the 80s and 90s with Doppler flow signals.[4] In our study we used 2D echocardiography. Previous studies have confirmed that there is a good correlation between Doppler flow signals and 2D cardiac imaging, but new advances in echocardiography technology may have made 2D imaging more sensitive in detecting VGE.[7,12] In Sawatzky's material 47% of 1,726 dives produced no detectable bubbles[4] whereas in our study absence of bubbles was found in only 2.5% (4/154) of the dives. This difference may be attributed to confounders such as different depth-time combinations, age and fitness levels of the subjects, all which influence bubble grades, but it may also be due to contemporary technology being more sensitive in detecting bubbles.[2] It might therefore be more suitable to use a more recent dataset like the one from Doolette which also uses 2D ultrasound imagine to grade VGE.[11] The DCS risk is substantially lower using this dataset but still well over 1%. A limitation in using Doolette's dataset is that the VGE grading scale used has not been validated against the Eftedal-Brubakk grading system. In the Bayesian calculation we assumed that these two ordinal scales directly correlate but this may not be the case and therefore the corresponding DCS risk may not translate between the two datasets.

Both the Sawatzky and Doolette datasets used in the Bayesian calculation measured VGE less frequently compared to our dataset. The Sawatsky study made the first measurement 30 min after surfacing and thereafter every 40 min. In the Doolette study the first measurement was done 30 min after surfacing and then at the two-hour mark. In our study four monitoring sessions were completed within the first hour and at least three during the second hour which generated greater temporal resolution. The mean time to reach peak VGE grade in our study was approximately 20 minutes and the 10 minutes delay in first measurement when compared to the Sawatzky and Doolette data could in theory lead to transient early peak VGE grades having been missed which may alter the indicated DCS risk. Other experts in the field have suggested the first monitoring session should be done within 20 min from decompression and that 40-min intervals between measurements may be too sparse and risk underestimating VGE grades.[2] If we examine our VGE grades at 30, 70 and 110 minutes, the same measurement frequency as in Sawatzky's data, and exclude all other measurements, a total of 24 VGE grades were lowered by one or two points. When excluding all measurements except the ones closest to 30 and 120 minutes, as in the Doolette dataset, a total of 32 VGE grades were lowered by one or two points. Using these lower VGE grades from the Sawatzky data in the Bayesian model all of the profiles, except two that were unchanged (51 msw / 10 min and 57 msw / 15 min with oxygen decompression), lowered their relative DCS risk in the range of 4–15% (see Appendix 4). The same analysis with the lowered VGE grades from the Doolette data showed a relative risk reduction in six of the nine profiles, in the range of 2–11%. However, two profiles, 39 msw / 12 min and 57 msw / 15 min with air decompression, showed an increased risk in DCS with 6% and 4% respectively (see Appendix 4). This is explained by the fact that in the original Doolette dataset the risk for DCS is lower with a VGE grade of 4 (5%) than with a grade of 3 (7%).[11] Therefore, the lowering of VGE grades from 4 to 3 in any profile leads to higher estimated DCS risk. This explains why the tendency for DCS risk reduction was more pronounced in the reanalysis of the Sawatzky dataset than in the Doolette dataset. The overall tendency to lower the DCS risk assessments with a reduced measurement frequency would have likely reduced our own DCS risk estimates with the Bayesian approach even further if our own dataset was larger.

Assuming the true incidence of DCS when using SWEN21 is 1% we can, with the help of binominal statistics, calculate that there is a 95% probability that the number of DCS cases would be in the range of 0–3 when performing 151 dives (the three divers who received prophylactic NBO were excluded from this calculation because NBO may alter the risk of developing DCS). Using the estimated risk suggested from Sawatzky's data (8.3%) with the same assumptions there is a 0.12% chance that there would be three or fewer cases of DCS. Using the suggested DCS risk from the Doolette data (5.4%) the probability of seeing three or less cases of DCS is 3.4%. This discrepancy in risk suggested by binominal statistics compared to the Bayesian model, especially in the case of the Sawatzky's dataset, could in part be explained by the fact that the reference material, which links VGE grades and DCS, is overestimating the risk of DCS at the respective VGE grade. Data supporting this line of reasoning comes from NEDU where a larger dive series ($n$ = 96) evaluated a similar dive profile (40 msw / 20 min, 9 min deco at 6 msw) to one in the present trial (39 msw / 20 min, 13 min decompression at 6 msw) resulting in similar VGE grades but with a low incidence of DCS.[13] A prerequisite for estimating DCS risk for a dive profile by means of VGE measurement is that a relation between VGE grade and DCS risk can be derived from a larger dataset of dives. This approach is based on the assumption that the risk of DCS for a diver with a given VGE grade is independent of the dive profile. Furthermore, it is assumed that measurements of VGE grades are so consistent that they can be compared between different datasets which may or may not be true. The two different datasets used in this study correlating DCS risk to VGE grade indicate widely different risk from one another possibly indicating that the VGE measurements are not comparable between the two datasets. Experts in the field of decompression theory question the validity of inferring DCS risk from VGE grades and have shown in big data sets that VGE grade is an imperfect surrogate for DCS risk.[11] Together there are several factors that make the estimate of DCS risk from VGE grade uncertain and therefore these data should be interpreted cautiously.

Strengths of this study includes that it was a large series done in a controlled and standardised way with frequent measurements graded by two diving physicians, one of whom has several decades of experience in this field of research. A potential limitation was that the test population may not fully be representative of operational divers in the SwAF. The divers in this validation series tended to be older than operational divers and higher age is a factor that may cause a higher bubble grade and an increased risk of DCS.[10] In summary, this warrants further validation of SWEN21 to ensure its safety.

## Conclusion

This evaluation of the novel SWEN21 dive table, using VGE formation measured with echocardiography suggests that the DCS risk may be higher than the projected 1%.

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# Rapture of the deep: gas narcosis may impair decision-making in scuba divers

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## Abstract

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**Introduction:** While gas narcosis is familiar to most divers conducting deep (> 30 metres) dives, its effects are often considered minuscule or subtle at 30 metres. However, previous studies have shown that narcosis may affect divers at depths usually considered safe from its influence, but little knowledge exists on the effects of gas narcosis on higher cognitive functions such as decision-making in relatively shallow water at 30 metres. Impaired decision-making could be a significant safety issue for a multitasking diver.

**Methods:** We conducted a study exploring the effects of gas narcosis on decision-making in divers breathing compressed air underwater. The divers ($n = 22$) were evenly divided into 5-metre and 30-metre groups. In the water, we used underwater tablets equipped with the Iowa Gambling Task (IGT), a well-known psychological task used to evaluate impairment in decision-making.

**Results:** The divers at 30 metres achieved a lower score (mean 1,584.5, standard deviation 436.7) in the IGT than the divers at 5 metres (mean 2,062.5, standard deviation 584.1). Age, body mass index, gender, or the number of previous dives did not affect performance in the IGT.

**Conclusions:** Our results suggest that gas narcosis may affect decision-making in scuba divers at 30 metres depth. This supports previous studies showing that gas narcosis is present at relatively shallow depths and shows that it may affect higher cognitive functions.

## Introduction

Diving requires constant evaluation of the changing underwater environment. The level of alertness and consciousness changes as the diver moves through the vertical water column. A scuba diver is dependent on the breathing gas they carry, and the breathing gas affects the nervous system and cognitive functions of the diver. While gas narcosis is familiar to many divers conducting ‘deep’ dives (> 30 metres [m]), its mechanisms and effects at shallower recreational depths (often considered as maximum 30 m) are poorly understood.[1]

Both in the hyperbaric literature[2–4] and in the dive community, dives to 30 m or shallower are often considered relatively safe from the effects of narcosis. However, research has shown that immersion at depths as shallow as 5 m can cause cognitive impairment in divers (not necessarily due to gas narcosis),[5] and many other chamber,[1] animal,[6] and open water studies[7–9] show various changes in cognition under hyperbaric pressures. Many of these studies have focused on the effect of narcosis on memory, and research suggests that it is not the short-term memory but the long-term memory that is affected by narcosis.[10,11] More recent research has shown that narcosis particularly affects the free-recall memory, but only when the information was learned at depth under narcosis.[9] The retrieval of information, whether under narcosis or at shallow water, was not impaired.[9] These results and further studies suggest that narcosis affects the encoding of information into long-term memory, rather than the information retrieval.[7]

While memory is an integral part of most cognitive functions, successfully conducting a dive requires several cognitive and executive functions such as coping with new situations, self-regulation, and decision-making. Both Divers Alert Network (DAN) annual reports on diving fatalities, injuries, and incidents,[12] and British Sub-Aqua Club (BSAC) diving incident reports from previous years[13] reveal that nitrogen narcosis has been a contributing factor in many accidents and incidents that have occurred at depth (20–60 m). The incidents varied from slight confusion that was resolved by ascending, to lethal entanglement. The most remarkable issue with narcosis is that it impairs the diver's capacity to respond to a potential issue underwater.[4,12,14]

While initial studies show that decision-making can be impaired as shallow as 5 m,[5] it is known that other higher executive functions, namely inhibitory control ability, are impaired at 20 m.[15] Making decisions requires multiple cognitive functions: retrieving information from and encoding information into long- and short-term memory, assessing the information and the options available in the current context while potentially experiencing various emotions, and deciding the best course of action. Because of decompression and gas limitations, decisions underwater must be made quickly. Understanding the effect of narcosis on such higher cognitive functions could greatly improve diver safety and training.

The Iowa Gambling Task (IGT)[16] is a widely used neuropsychological task designed to assess impairments in decision-making.[17] The IGT was originally used to assess decision-making in patients with injury to the ventromedial prefrontal cortex, an area known to be linked to poor decision-making in complex and uncertain situations. The IGT has been specifically designed to predict real-life decision deficits. It has since been used as a clinical diagnostic measure to assess decision-making in individuals with neurological disorders, psychiatric disorders, nonclinical populations, and animal models.[17] It is the most commonly used tool to assess decision-making in people with alcohol use or gambling disorders.[18]

Given the similarities in the symptoms of gas narcosis and alcohol intoxication, and that both alcohol and nitrogen are thought to affect areas in the prefrontal cortex, an area important for decision-making,[16] the IGT could be a suitable tool to study the effect of gas narcosis on decision-making in divers. To address the knowledge gap on the effects of gas narcosis on decision-making, we trialled the use of IGT to study decision-making in scuba divers potentially experiencing narcosis. Specifically, we addressed the following question: does gas narcosis affect the performance in IGT in scuba divers breathing compressed air at 30 m compared to a control group at 5 m? To our knowledge, this is the first time the IGT is used to study decision-making in scuba divers.

## Methods

The study protocol was approved by the University of Jyväskylä Ethics Committee and all participants were volunteers and provided written informed consent.

### PARTICIPANTS

A total of 25 participants (male $n$ = 18, female $n$ = 7) were recruited through local Finnish dive clubs. The participant had to be a minimum of Professional Association of Diving Instructors (PADI) advanced open water diver or equivalent (i.e., certified to dive to 30 m), capable of diving in a dry suit, answer 'no' to all questions in the PADI medical statement or get a doctor's certificate to prove fitness to dive, be over the age of 18, and their last dive must have been within a year. The least experienced diver had 40 dives, and the most experienced diver had > 2,000 dives (median 180, mean 360, standard deviation [SD] 454). The certification level of participants varied from advanced open water diver to technical diving instructor. The mean body mass index (BMI) was 25.6 (SD = 3.9). The BMI was included because the correlation between body fat and other pressure related issues such as decompression sickness is well-known, so we wanted to see any potential effect, however unlikely, it could have on narcosis.

### LOCATION, CONDITIONS, AND MATERIALS

The experiments were carried out over three days in October 2019. The study took place at the Kaatiala quarry in Ostrobothnia, Finland. Kaatiala is a popular dive site among Finnish divers, it has relatively stable and clear conditions, and a gravel bottom which limits silting up. The maximum depth of the open water quarry site is 30 m. On our test dates, the water temperature was approximately 6°C at 5 m and 4°C at 30 m. The horizontal visibility was measured using a secchi disc and a measuring tape, and was between 6 and 7 m both at the control depth of 5 m and at the bottom at 30 m. We used four identical Samsung Galaxy A 8" tablets in underwater housings (Alltab, Valtamer Ltd, Helsinki), that had the Iowa Gambling Task[16] application (https://www.apkmonk.com/app/com.zsimolabs.iowa) installed. One housing got damaged and flooded on the first dive, so for the rest of the experiment, only three tablets were used. All participants used compressed air as a breathing gas and were on open-circuit scuba with their own, standard dive equipment, including their own dry gloves.

### STUDY DESIGN

Each participant filled out a Finnish translation of the Behavioural Inhibition System (BIS) and Behavioural Activation System (BAS) questionnaire[19] before the dive. The BIS/BAS questionnaire is a 20-point self-report questionnaire, designed to assess the motivational systems underlying individual behaviour. Questions measuring the inhibition system assess the motivation to avoid aversive outcomes, while questions measuring the activation system assess the motivation to approach goal-oriented outcomes. The answer scale is a 4-point Likert scale. The participants' BAS scores were then ranked in order from the lowest to the highest, and every other participant was allocated to the 5-metre or 30-metre group respectively. Those in the 5-metre group would take the IGT test at 5 metres, and those in the 30-metre group would take the IGT at 30 metres. The results of the BIS/BAS questionnaire are known to be linked to the results of the IGT,[20] so the group allocation was done to reduce the effect of individual variation in risk-taking tendency. In total 12 participants were allocated to 5 m and 13 to 30 m. Regardless of whether the participant had been allocated to the 5 m or 30 m group, the test dives were done in groups of two to five people, including the researcher. Each participant was given a tablet that had been turned on at the surface and the test had been set ready so that it could be started with two presses of a (touch-screen) button underwater. The task had been explained to the participants in advance as part of the standard pre-dive briefing, but they had not seen the actual task. The groups descended to their target depths following regular dive guidelines and a maximum descent rate of 20 m·min$^{-1}$. The groups descending to 5 m did a free descent with a wall as a visual reference and the 30 m group used a fixed line as a visual reference.

The IGT test has four buttons representing card decks, labelled A, B, C, and D. The participant is given a USD$2,000 ‘loan’ to start with, and their aim is to make as much money as possible. The participant has a total of 100 cards in four decks and they choose (i.e., press the deck label) one card at a time. With each card, they either lose or win money. They have no prior knowledge of which decks will yield the most money and which decks will lose the most money. The decks A and B are ‘high risk’ decks, as they always win USD$100 but also incur occasional large losses of up to USD$1,250. Decks C and D are ‘low risk’ decks resulting in small wins of USD$50 in each trial and occasional small losses of up to USD$250. The decks A and B will result in a net loss of USD$250 over 10 trials on average, while decks C and D will gain USD$250 over 10 trials. The decision-maker receives win and loss information and total current gain or loss after each trial. Completing the 100 trials took approximately 10 minutes, and after that, the participant locked the tablet screen and ascended to the surface together with the group following regular dive guidelines. Divers ascending from 30 m did a 3-minute safety stop at 5 m. Consequently, the average total dive time was as follows: descent (20 m·min$^{-1}$) + IGT (10 min) + ascent following computers (10 m·min$^{-1}$) + safety stop for the 30-metre group (3 min). Due to gas constraints, no acclimatization time was included.

The IGT software automatically recorded the participant’s random ID, number of trials, the chosen deck (A, B, C, or D), reward, penalty, gain, total amount of money, response time, touch speed, and time (with one-second accuracy). All these data were saved on the tablet memory and retrieved after all the experiments were completed.

### DATA ANALYSIS

To test the effect of depth (categorical variable with two levels: 5 m and 30 m), and any potential confounding influence of dive experience (number of dives), BMI, age, and gender (categorical variable with two levels: male and female) on the response variable of IGT score (i.e., the total amount of money the diver ‘won’ in the IGT), a generalised linear model (GLM) from the R package ‘MASS’[21] was used. A negative binomial distribution was used to account for overdispersion. The model assumptions were checked by visually inspecting model diagnostics plots. A backwards stepwise model selection and Akaike Information Criterion (AICc and QAIC) from the R package ‘MuMIn’[22] were used to select the model of best fit. A $\chi^2$-test for the difference in null deviance and residual deviance was done to check for the goodness of fit of the model. All analyses were conducted using R version R-4.0.3.[23]

## Results

As a result of complete tablet and housing malfunction underwater, the results of three participants had to be excluded from the analyses. A total of 22 participants were therefore included in the final analyses. Of these 22 participants, three had their tablet malfunction before they reached the end of the task. In order to keep the sample size as high as possible, we did not exclude those three. Out of one hundred IGT card trials, these three had completed 99, 81, and 79 trials respectively. Their test results were treated as 99, 81, and 79% of the full score and their final scores were corrected respectively. The IGT score was therefore achieved from a total of 22 participants, 11 of them at 5 m and 11 at 30 m. The score used was the total amount of money achieved at the end of the task. The IGT software also recorded the response time, touch speed, and total time. While these could be interesting variables to investigate, we omitted them from the analyses due to the practical challenges faced by participants when handling the underwater housing with dry gloves. The participants wore their own personal gloves

**Table 1**

Summary of the number (n) of participants, gender distribution, number of dives, age, body mass index (BMI) and Iowa Gambling Task (IGT) scores, by test depth (5 m or 30 m) and all divers combined; F – female; M – male; SD – standard deviation

| Depth | *n* | M/F | Previous dives Mean (SD) | Age (years) Mean (SD) | BMI Mean (SD) | IGT score Mean (SD) |
|---|---|---|---|---|---|---|
| Total | 22 | 15/7 | 360 (454) | 35.2 (10.3) | 25.6 (3.9) | 1,823.5 (559.6) |
| 5 m | 11 | 8/3 | 326 (570) | 33.6 (11.1) | 25.0 (3.5) | 2,062.5 (584.1) |
| 30 m | 11 | 7/4 | 393 (324) | 36.8 (9.6) | 26.3 (4.4) | 1,584.5 (436.7) |

**Figure 1**

The lack of relationship between the IGT score and the number of dives; due to the few extreme values in the number of dives, this parameter is shown on a logarithmic scale

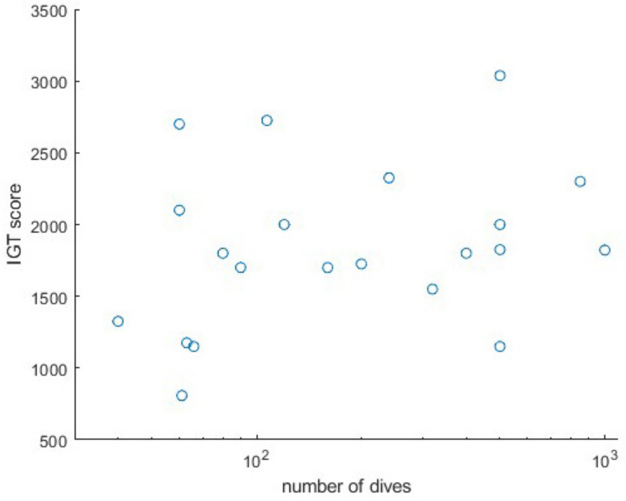
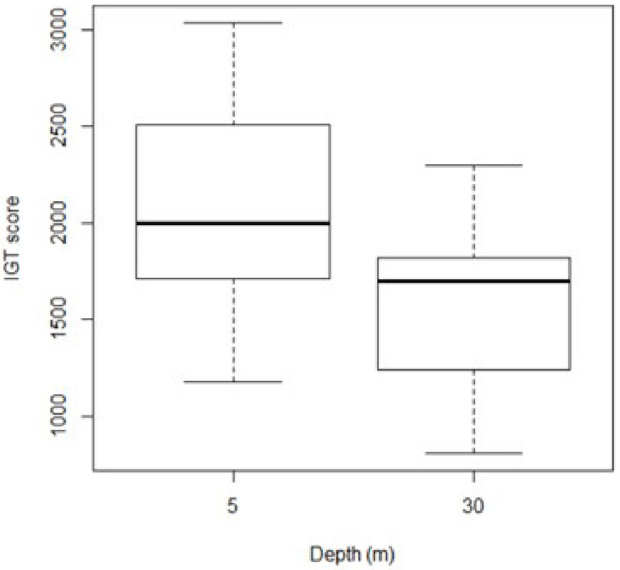


**Figure 2**

Boxplot of the effects of depth on the IGT score; the horizontal line inside the box shows the median, the ends of the box show the upper and lower quartiles, and the whiskers show the highest and lowest scores



and it was obvious that the underwater housing responded better to some dry gloves than others, and therefore any time-related variable would not be reliable. The break down summary of participant gender, number of dives, age, BMI and the IGT score by depth is detailed in Table 1.

The best fitting model was $Y_i \sim \beta_0 + \beta_1 D_i$ where $Y_i$ = the IGT score, $\beta_0$ = the intercept, and $D_i$ = depth as a categorical variable with two levels (either 5 or 30 m). The effect of the number of dives (Figure 1), BMI, age, and gender were not statistically significant, but the model showed a significant relationship between depth and the IGT result so that participants at 30 m has lower scores than participants at 5 m (Figure 2, Table 1) (GLM, $z = -2.243$, $P = .025$, $DF = 20$).

The $\chi^2$-test for the difference between the null deviance and residual deviance was $P = 0.025$, indicating that the model is an adequate fit for the data.

## Discussion

Our results show that divers at 30 m achieved lower scores in the IGT than divers at 5 m. This means that at 30 m the divers chose ‘riskier’ decks, i.e., those that yield high rewards but also high losses and always result in a loss in the long term. In contrast, those at 5 m forewent the immediate rewards and chose decks that yield low rewards but also low losses, and in the long term earn the decision-maker more money. The difference in the scores at 30 m and 5 m suggests that gas narcosis could affect the decision-making process in divers breathing compressed air at 30 m of fresh water, compared to divers at 5 m. While our study did not compare the difference in the performance between the surface and underwater, our results are in line with other studies showing impaired cognitive functions at modest depths: such as a study that showed impaired control of the inhibitory system in divers when measured with the Stroop test at 20 metres,[15] and a study that showed that performance-related cognitive skills, sustained attention in particular, are affected at as shallow as 5 m.[5] We also assessed the effect of the number of previous dives, BMI, age, and gender, and none of these had a significant effect on the IGT result.

Previous non-diving related studies have shown that participants with impediment to the prefrontal cortex, an area important in decision-making, gain lower scores in the IGT compared to those with no damage to the prefrontal cortex.[17] Gas narcosis is the result of exposing neurological tissue to high partial pressures of gases, but the exact mechanisms behind gas narcosis are still unknown. Those mechanisms are likely similar to those of other anaesthetics and take place at the brain synapses.[4] In the current study, it is likely that the impaired decision-making at 30 m could be a result of abnormal activity in the prefrontal cortex, caused by the elevated partial pressure of nitrogen.

Memory is an integral part of decision-making, and previous studies have shown that impairments in working memory can influence the performance in IGT and therefore affect decision-making.[24] However, narcosis affects mainly long-term memory, and not short-term memory.[7,10,11] Given that working memory is part of short-term memory,[25] this could indicate that the impaired decision-making at depth is not simply a relic of impaired memory at depth, but that narcosis also affects higher cognitive functions.

The experience of the diver, measured in the number of dives, did not influence the performance in IGT. While experience usually improves diving skills, and therefore may make it easier to respond to an incident, research shows little correlation between dive experience and adaptation to narcosis,[26] in line with our results.

It is also important to recognise that narcosis has a subjective and objective component and that the relationship between these is not clear. Anecdotal tales of gaining tolerance to narcosis can probably be attributed to the subjective rather than the objective component of narcosis. Similarly, narcosis may affect the metacognition of the diver, and the diver's confidence to perform well may depend on the difficulty of the task at hand. In contrast, it has been shown that although narcosis causes impairment in cognitive functions at 33–42 m, the divers' awareness of it is good, and therefore they might be able to compensate for it.[27] Given that our results show impairment in decision-making, the question of whether they would decide to compensate for the impaired cognitive functions or have the skills to do so while under narcosis, requires further research. However, experience may affect how well the diver is aware of their cognitive degradation underwater, and experienced divers might be more aware of their cognitive functions in the water than inexperienced divers.[5]

The diver's awareness of self and the surroundings is an important part of diver training at all levels. In the dive community, it is often thought and taught that factors such as dehydration, tiredness, temperature, task loading, current, and visibility may affect how strongly the diver experiences narcosis. It could be argued, for instance, that the 2°C difference at 5 and 30 metres in our study could affect the results. However, there is no scientific consensus on this. Recent research suggests that it is likely that only the breathing gas and the absolute pressure contribute to narcosis.[28] In practice, however, it could be reasonable to assume that the level of (at least) subjective narcosis is affected by various factors, as narcosis, coldness, fatigue, and other factors can accumulate. In the current study, the divers were not faced with any additional tasks such as navigation, highlighting the role of absolute pressure in decision making.

Our results showed no relationship between IGT performance and gender or age. However, outside of the diving context, both are known to affect the performance in IGT. It is thought that the differences in decision-making strategies between men and women have neurobiological and hormonal basis, and the differences are mainly strategic: women decide based on detailed information, while men decide on global information.[29] It could be that narcosis affects decision-making more than just gender does, however, our sample size is too small and unbalanced in terms of genders, and therefore no conclusions on the effect of gender on the IGT results under narcosis can be drawn. Similarly, age is known to affect the performance in IGT, especially during early childhood, adolescent years and after 60 years of age,[30] but the age variation within the participants in the current study was too small to draw any conclusions.

One of the strengths of the present study is that its participants were from the general recreational diving community and were heterogenous in terms of dive experience. A lot of dive-related research is conducted on navy divers. While the advantage of this is that they often have detailed medical evaluations and a detailed record of their dive history, which helps in the standardisation of the experiment, the study population itself tends to be rather homogeneous and mainly consists of healthy and physically fit men of around the same age. The general diving population, however, consists of people of different ages, experience levels and fitness levels. The extrapolation of research results from a homogenous group to a very heterogenous group obviously posits various issues.

## LIMITATIONS

The present study has limitations that should be considered. Due to the small sample size, care should be taken when interpreting the results. A potential issue in the present study is that it was not double-blinded. Both the researcher and the participants knew that the effects of nitrogen narcosis were being studied. There are, however, ethical and practical issues in conducting this kind of study without telling the participants what is being studied. Also, the observed effect could have been explained by an unknown difference in the groups.

Due to the learning effect in the IGT, a within-subject design was not possible. Future studies focusing on the effects of decision-making in divers and using the IGT could, however, probably benefit from two additional groups of test subjects: a group of divers and a group of non-divers who take the IGT on land or just below the surface. Given that cognitive functions are known to impair at as shallow as 5 m,[5] repeating the IGT on land (or just below the surface) could exclude any potential effects on cognitive functions from immersion in shallow water. Including a non-diving group could help in evaluating whether the potential long-term effects of exposure to high partial pressures of gases may extend their influence on performance in the IGT. Additionally, a questionnaire about the subjective feelings of narcosis could also add value to any further studies. Currently, no studies exist on the effect of different gas mixes on the higher cognitive functions. Repeating the current study with different gas mixes at different depths could reveal important information on the effects of pressure and breathing gases on the cognitive functions of a diver.

## Conclusions

In summary, our results demonstrated that divers at 30 m performed worse in the IGT than divers at 5 m. It is likely that this was caused by gas narcosis, but given the relatively small sample size, the results should be interpreted with caution. The number of dives, age, BMI, and gender did not appear to influence the performance in the IGT.

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# Full-face snorkel masks increase the incidence of hypoxaemia and hypercapnia during simulated snorkelling compared to conventional snorkels

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**Keywords**

Diving research; Equipment; Hypercapnia; Hypoxia; Safety

## Abstract

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**Introduction**: Air flow in full-face snorkel masks (FFSMs) should be unidirectional to prevent rebreathing of exhaled air. This study evaluated rebreathing and its consequences when using FFSMs compared to a conventional snorkel.

**Methods**: In a dry environment 20 participants wore three types of snorkel equipment in random order: Subea Easybreath FFSM; QingSong 180-degree panoramic FFSM; and a Beuchat Spy conventional snorkel (with nose clip), in three conditions: rest in a chair; light; and moderate intensity exercise on a cycle ergometer. Peripheral oxygen saturation, partial pressure of carbon dioxide ($PCO_2$) and oxygen ($PO_2$) in the end tidal gas and FFSM eye-pockets, respiratory rate, minute ventilation, were measured continuously. Experiments were discontinued if oxygen saturation dropped below 85%, or if end-tidal $CO_2$ exceeded 7.0 kPa.

**Results**: Experimental runs with the FFSMs had to be discontinued more often after exceeding 7.0 kPa end-tidal $CO_2$ compared to a conventional snorkel e.g., 18/40 (45%) versus 4/20 (20%) during light intensity exercise, and 9/22 (41%) versus 3/16 (19%) during moderate intensity exercise. Thirteen participants exhibited peripheral oxygen saturations below 95% (nine using FFSMs and four using the conventional snorkel) and five fell below 90% (four using FFSMs and one using the conventional snorkel). The $PCO_2$ and $PO_2$ in the eye-pockets of the FFSMs fluctuated and were significantly higher and lower respectively than in inspired gas, which indicated rebreathing in all FFSM wearers.

**Conclusions**: Use of FFSMs may result in rebreathing due to non-unidirectional flow, leading to hypercapnia and hypoxaemia.

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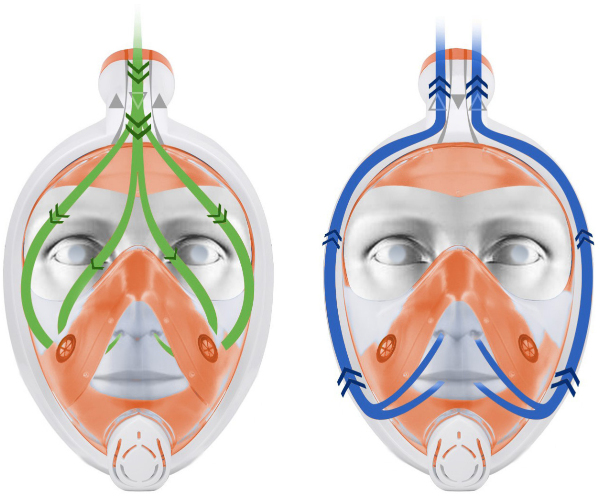
## Introduction

Full-face snorkel masks are widely used, particularly by ‘tourist divers’ and were adopted as protective personal equipment during the COVID-19 pandemic. While these masks provide an ‘easier’ alternative to the traditional mask and snorkel combination, there are concerns about the potential for rebreathing exhaled gas high in carbon dioxide ($CO_2$). There have been several snorkelling accidents including fatalities associated with the use of full-face snorkel masks.[1–3] Accumulation of $CO_2$ in the mask resulting in hypercapnia is a possible contributor to these fatalities. Hypoxia may also be a contributing factor.

When breathing through a full-face snorkel mask, inhaled air is drawn down the snorkel, through the eye-pocket, and then into a sealed oronasal compartment through one-way valves designed to isolate the oronasal compartment from the eye-pocket during exhalation. When exhaling, the expired gas passes through another one-way valve and is expelled up the snorkel via a separate expiration channel (Figure 1). When the mask is functioning as intended, unidirectional air flow should occur, with no mixing of inhaled and exhaled gas. A ‘normal’ tube snorkel has a dead space of about 160 mL. The dead space in the oronasal compartment of a full-face snorkel mask is about 250 mL and can increase to up to 1,470 mL depending on the brand, if the seals or valves are not working properly.[4] A larger ‘equipment dead space’, effectively an extension of the anatomic dead space, results in more rebreathing of exhaled gas before breathing fresh air,[5] which may lead to hypercapnia or hypoxia. Hypercapnia can cause dizziness, shortness of breath, headaches, and loss

**Figure 1**

Schematic design of a full-face snorkel mask. Left: inhalation through the one-way valve in the snorkel through the eye-pocket and one-way valves into the oronasal pocket. Right: exhalation through separate tubes on the side from the oronasal pocket back to the snorkel, exiting through one-way valves in the snorkel (not mixing with inhaled air)



of consciousness, while hypoxia may cause confusion and ultimately loss of consciousness. These effects can be both distressing and dangerous while snorkelling.

One study has evaluated the partial pressure of $CO_2$ ($PCO_2$) in the oronasal pocket of a full-face snorkel mask.[6] Inspired $PCO_2$ levels of 1–2 kPa and increased breathing resistance with any water intrusion were recorded, however participants were able to maintain expired $CO_2$ levels of 4–6 kPa. Although no increase in respiratory rate was observed, tidal volume and minute ventilation were not measured. Minute ventilation usually increases with hypercapnia, but this may be achieved by a larger tidal volume rather than an increased respiratory rate.[7] Other papers typically involving small numbers of participants, examined adapted full-face snorkel mask for use as personal protection equipment against COVID-19,[4,8–11] or to ventilate patients with COVID-19.[12,13] The studies that measured $CO_2$ all noted an increase of inspired or expired $CO_2$ while wearing the full-face snorkel mask.[4,8,10,12,13] It is important to note that adaptations to those full-face snorkel masks by adding filters, may have influenced the gas flow dynamics. None of the existing studies have evaluated the gas composition in the eye-pocket during use.

The primary aim of this study was to evaluate oxygenation and $CO_2$ levels while wearing full-face snorkel masks or a conventional snorkel at rest, and during light and moderate exercise. We also evaluated the potential for rebreathing during use of these devices.

## Methods

The study protocol was approved by the University of Auckland Human Participants Ethics Committee (Reference UAHPEC3497).

### TRIAL DESIGN AND PARTICIPANTS

This was a randomised, intervention study conducted in the Exercise Physiology Laboratory at the University of Auckland during December 2020 and February 2021. Twenty healthy participants aged 18 to 60 years old were recruited. Participants were eligible when no health concerns were ticked on the Recreational Scuba Training Council screening questionnaire for diver training candidates. All participants provided written informed consent.

### SNORKEL AND MEASUREMENT EQUIPMENT

Three items of snorkel equipment were tested: SUBEA Easybreath full-face snorkel mask (Decathlon, Lille, France); QingSong 180-degree panoramic full-face snorkel mask (QingSong, China); Beuchat Spy conventional snorkel. The SUBEA mask represents a recognised market leader in the full-face snorkel mask space, and the Qing-Song mask is more typical of cheaper products marketed on-line. Both full-face snorkel masks had two sampling lines ported, one to the eye-pocket and one to the oronasal compartment. The conventional snorkel had a sampling line ported approximately 5 cm from the mouthpiece. The sampling lines were connected to two respiratory gas analysers (ML206, AD Instruments, Dunedin, New Zealand), to measure the partial pressures of $CO_2$ and oxygen ($PCO_2$ and $PO_2$). Participants were fitted with an earlobe peripheral oxygen saturation sensor connected to an oximeter pod (AD Instruments, Dunedin, New Zealand), and an Equivital $eqO_2$ + LifeMonitor belt (Equivital, Cambridge, UK) to measure ECG and chest expansion.

Chest expansion was calibrated to measure minute ventilation using a pneumotachometer (MLT1000L, AD Instruments, Dunedin, New Zealand) during one minute of normal breathing while resting in a chair. The addition of a pneumotachometer to the full-face snorkel masks would have altered mask volumes and functionality. We therefore calculated minute ventilation based on chest expansion during the experiments.

All data were sampled continuously at 1 kHz using a Powerlab 16/35 and acquired via LabChart Pro data acquisition software version 8.1.24 (AD Instruments, Dunedin, New Zealand).

### EXPERIMENTAL PROCEDURES

Participants performed spirometry (forced vital capacity [FVC] and forced expiratory volume in one second [$FEV_1$], from which the $FEV_1$/FVC ratio was calculated). All participants used all three types of snorkel equipment with the order of full-face snorkel mask type randomised. The Beuchat conventional snorkel was always last. Participants were fitted with the correct full-face snorkel mask size, based on distance between the top of their nose to the bottom of their chin. Mask fitting was tested by drawing negative pressure from the full-face snorkel mask while blocking the snorkel. Participants wore a nose clip whilst wearing the Beuchat conventional snorkel.

Participants underwent baseline measurements while resting in a chair and not wearing any snorkel equipment. Baseline values for gas composition in the eye-pocket were assumed equivalent to room air values since the eye-pocket should only contain atmospheric air if gas is flowing correctly.

Each item of snorkel equipment was tested for five minutes in three conditions (unless a safety threshold was breached): 1) rest in a chair; 2) light intensity exercise on a cycle ergometer at four metabolic equivalents (MET) to simulate slow finning; and 3) moderate intensity exercise at 6 MET to simulate swimming against a current. We determined a 5-minute duration to be long enough to achieve a steady state ventilation at the chosen workloads and therefore ensured participants experienced peak ventilatory rates and respiratory gas concentrations for each workload. Any experimental run was discontinued if peripheral oxygen saturation dropped below 85% or end-tidal $CO_2$ exceeded 7.0 kPa. Before the next type of snorkel equipment was tested participants rested until all parameters returned to baseline.

### OUTCOME MEASURES

The primary endpoint measures were the proportion of participants with hypoxaemia below 95% and 90%, and the measured end-tidal $CO_2$ during the last 10 seconds of each condition.

Secondary outcome measures included end-tidal $PO_2$ in the oronasal compartment, $PCO_2$ and $PO_2$ in the eye-pocket of the mask late in exhalation, respiratory rate, calculated minute ventilation and heart rate averaged during the last 10 seconds of each condition. If the condition was discontinued early because of hypoxaemia below 85% or end-tidal $CO_2$ exceeded 7.0 kPa, time till discontinuation was noted.

While participants were wearing the full-face snorkel masks the seal of the internal skirt between the eye and oronasal pocket was classified as adequate (no visible gaps and no exhalation condensation) or inadequate (visible gaps or exhalation condensation). Participants were asked to describe their experiences with the snorkel equipment, immediately after removal.

### STATISTICAL ANALYSIS

Descriptive statistics were generated to characterise study participants and reported as mean and standard deviation (SD). Normality of outcome measures was established with the Kolmogorov-Smirnov test. Differences between the different snorkel equipment were tested with a one-way ANOVA. Significant results were analysed with a post-hoc test with LSD correction. All tests were two-sided, with $\alpha$ set at 5%. Mean differences with their 95% confidence intervals (CI) are reported. All data were analysed using SPSS Statistics version 25.0 (IBM, Armonk, NY, USA).

## Results

Participants were on average 33 years old (11.7), 14 of the 20 participants were female, average body mass index (BMI) was 24.3 $kg \cdot m^{-2}$ (4.1), and average $FEV_1$/FVC was 0.79 (0.16). Nineteen identified as experienced snorkelers, one participant had used a full-face snorkel mask before. A range of ethnicities were reported being 13 European/New Zealand, three Chinese, two South-East Asians, one Brazilian, one North American. No participant had facial hair.

Participants who completed the whole experiment wore each item of snorkel equipment for a total of 15 minutes. Some experimental runs were discontinued prematurely because the safety threshold of 7.0 kPa end-tidal $PCO_2$ was exceeded (Table 1). No terminations occurred in the resting condition. During light intensity exercise 22 of 60 (36.7%) experimental runs were stopped prematurely, and these participants did not continue to moderate intensity exercise. During moderate intensity exercise another 12 of 38 (31.6%) experimental runs were stopped prematurely. Experimental runs where participants were wearing full-face snorkel masks had to be discontinued more often compared to wearing a conventional snorkel e.g., 18/40 (45%) versus 4/20 (20%) during light intensity exercise, and 9/22 (41%) versus 3/16 (19%) during moderate intensity exercise (Table 1 and Figure 2). This resulted in less total time in experimental runs using the full-face snorkel masks compared to the conventional snorkel (respectively 1.8 and 2.4 minutes shorter). Early termination in the full-face snorkel masks was usually associated with an inadequate seal of the internal skirt of the mask. Even with good external fit, the internal skirt did not always seal adequately for all facial types.

Although no condition was discontinued early because of hypoxaemia, 13 participants experienced hypoxaemia below 95% (five Subea full-face snorkel mask, four QingSong full-face snorkel mask, and four conventional snorkel). Of those, five experienced hypoxaemia below 90% (three Subea full-face snorkel mask, one QingSong full-face snorkel mask, and one conventional snorkel), with two participants

**Table 1**

Average values during baseline (no snorkel equipment), and during rest, light and moderate exercise during use of snorkel equipment; baseline (no snorkel equipment) values for the eye-pocket are environmental air values. FFSM – Full-face snorkel mask; $PCO_2$ – carbon dioxide partial pressure; $PO_2$ – oxygen partial pressure; $P_ECO_2$ – carbon dioxide pressure late in exhalation; $P_EO_2$ – oxygen pressure late in exhalation

| **Parameter** | **Equipment** | **Baseline** | **Rest** | **Light** | **Moderate** |
|---|---|---|---|---|---|
| Started in condition (*n*) | FFSM Subea | 20 | 20 | 20 | 12 |
| | FFSM QingSong | 20 | 20 | 20 | 10 |
| | Snorkel | 20 | 20 | 20 | 16 |
| Discontinued (*n*) | FFSM Subea | 0 | 0 | 8 | 5 |
| | FFSM QingSong | 0 | 0 | 10 | 4 |
| | Snorkel | 0 | 0 | 4 | 3 |
| | | **Total** | **Rest** | **Light** | **Moderate** |
| Time in each condition (min) | FFSM Subea | 10.9 (0.8) | 5 (0) | 3.8 (0.4) | 2.1 (0.5) |
| | FFSM QingSong | 10.3 (0.8) | 5 (0) | 3.5 (0.4) | 1.8 (0.5) |
| | Snorkel | 12.7 (0.8) | 5 (0) | 4.3 (0.3) | 3.4 (0.5) |
| | | **Baseline** | **Rest** | **Light** | **Moderate** |
| Oxygen saturation (%) | FFSM Subea | 99.8 (0.4) | 99.9 (0.8) | 99.3 (1) | 98.9 (1.8) |
| | FFSM QingSong | | 100 (0.4) | 98.5 (2.2) | 97.9 (1.9) |
| | Snorkel | | 99.8 (0.7) | 98 (3.2) | 97.1 (3.3) |
| End-tidal $PCO_2$ oronasal pocket (kPa) | FFSM Subea | 5.76 (0.37) | 5.55 (0.51) | 6.13 (0.25) | 6.49 (0.5) |
| | FFSM QingSong | | 5.83 (0.32) | 6.29 (0.36) | 6.61 (0.61) |
| | Snorkel | | 5.69 (0.24) | 5.84 (0.34) | 5.81 (0.57) |
| End-tidal $PO_2$ oronasal pocket (kPa) | FFSM Subea | 14.55 (1.29) | 14.88 (0.92) | 14.33 (0.5) | 14.02 (1.1) |
| | FFSM QingSong | | 14.4 (0.73) | 14.04 (0.45) | 13.85 (1.1) |
| | Snorkel | | 14.79 (0.71) | 14.42 (0.63) | 14.83 (0.71) |
| $P_ECO_2$ eye-pocket (kPa) | FFSM Subea | 0.03 (0.0) | 3.13 (1.2) | 2.61 (0.87) | 2.62 (1.27) |
| | FFSM QingSong | | 3.14 (0.88) | 3.48 (1.28) | 3.75 (1.46) |
| $P_EO_2$ eye-pocket (kPa) | FFSM Subea | 21.0 (0.0) | 17.72 (1.33) | 18.35 (1.1) | 18.32 (1.55) |
| | FFSM QingSong | | 17.5 (0.96) | 17 (1.19) | 16.67 (1.51) |
| Respiratory rate (breaths·min$^{-1}$) | FFSM Subea | 12 (4.1) | 15.4 (4.3) | 18.4 (3.6) | 19.4 (5.3) |
| | FFSM QingSong | | 15.3 (4.1) | 21.6 (5.9) | 22.9 (7) |
| | Snorkel | | 13.5 (3.9) | 19.4 (3.4) | 22.6 (4.9) |
| Minute ventilation (L.min$^{-1}$) | FFSM Subea | 18.4 (4.8) | 23.9 (13.3) | 35.4 (10.2) | 42.8 (13.6) |
| | FFSM QingSong | | 23.7 (10.6) | 36.8 (8.2) | 38.4 (8.6) |
| | Snorkel | | 22.3 (12.5) | 30.6 (11.6) | 34.4 (9) |
| Heart rate (beats·min$^{-1}$) | FFSM Subea | 66.3 (14.1) | 71.7 (13.5) | 106.8 (14.5) | 128.3 (27.4) |
| | FFSM QingSong | | 70.4 (19.2) | 105.4 (23.9) | 128.2 (28.4) |
| | Snorkel | | 76.9 (16.7) | 112.8 (15.4) | 141.3 (16.7) |

**Figure 2**

Survival curve showing exercise termination while wearing each type of snorkel equipment; FFSM – Full-face snorkel mask

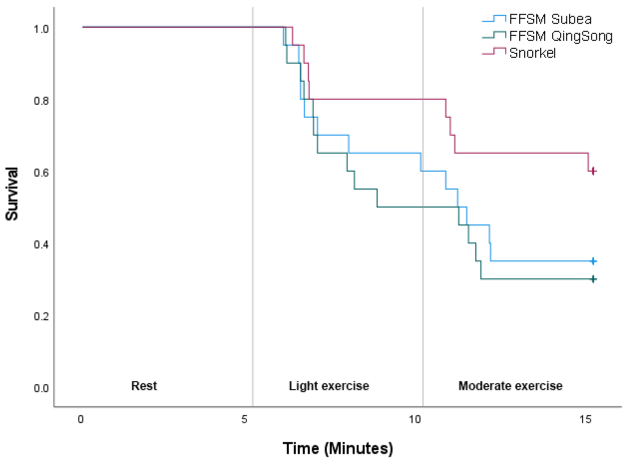


**Figure 3**

Typical inhale and exhale patterns in the eye-pocket and oronasal compartment of the full-face snorkel masks; the eye-pocket values should only be static lines representing atmospheric air ($PCO_2$ of 0.03 kPa and $PO_2$ of 21.0 kPa) if gas was flowing correctly (unidirectional flow). $E_T$ – end tidal; $PCO_2$ – carbon dioxide partial pressure (kPa); $PO_2$ – oxygen partial pressure (kPa); s – seconds

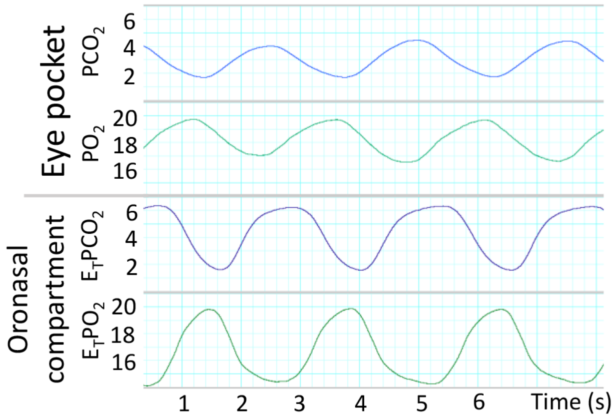


**Table 2**

Differences between full-face snorkel masks and the conventional snorkel. Overall differences were tested with a one-way ANOVA (overall P-value). Significant results were analysed with a post-hoc test with LSD correction and reported as mean difference (MD) with 95% confidence intervals (CI). Differences in the eye-pocket are comparisons between pressures of carbon dioxide and oxygen in the eye-pockets of the full-face snorkel mask and environmental air values. FFSM – Full-face snorkel mask; $PCO_2$ – carbon dioxide partial pressure; $PO_2$ – oxygen partial pressure; $P_ECO_2$ – $CO_2$ pressure late in exhalation; $P_EO_2$ – oxygen pressure late in exhalation

| Parameter | Overall *P*-value | FFSM Subea | | FFSM QingSong | |
|---|---|---|---|---|---|
| | | MD (95% CI) | *P*-value | MD (95% CI) | *P*-value |
| Time in each condition (min) | < 0.001 | -1.8 (-3.0 to -0.5) | 0.007 | -2.4 (-3.7 to -1.1) | < 0.001 |
| End-tidal $PCO_2$ (kPa) | 0.004 | 0.3 (0.0 to 0.5) | 0.019 | 0.4 (0.2 to 0.6) | 0.001 |
| End-tidal $PO_2$ (kPa) | 0.024 | -0.4 (-0.9 to 0.1) | 0.092 | -0.7 (-1.2 to -0.2) | 0.007 |
| $P_ECO_2$ eye-pocket (kPa) | < 0.001 | 3.2 (2.8 to 3.6) | < 0.001 | 3.8 (3.4 to 4.2) | < 0.001 |
| $P_EO_2$ eye-pocket (kPa) | < 0.001 | -3.4 (-3.9 to -2.9) | < 0.001 | -4.5 (-4.9 to -4.0) | < 0.001 |

desaturating to 86% for half a minute after respectively 0.5 and 3.0 minutes of wearing the Subea full-face snorkel mask during light intensity exercise.

The end-tidal $PCO_2$ in the oronasal pocket of the Subea and QingSong full-face snorkel masks was significantly higher compared to the conventional snorkel (mean difference respectively, 0.3 kPa (95% CI 0.0 to 0.5) and 0.4 kPa (95% CI 0.2 to 0.6). The end-tidal $PO_2$ measured in the oronasal compartment of the QingSong full-face snorkel mask was significantly lower compared to the conventional snorkel (mean difference, 0.7 kPa [95% CI 0.2 to 1.2] [Table 2]).

In the full-face snorkel mask eye-pockets, the $PCO_2$ was higher, while the $PO_2$ was lower (both about 3 kPa) compared to atmospheric air in both full-face snorkel masks (Table 2). The $PCO_2$ and $PO_2$ in the full-face snorkel mask eye-pocket fluctuated with each inhalation and exhalation in all subjects, even where there was a seemingly adequate internal skirt fit (Figure 3).

There was no overall difference in hypoxaemia ($P = 0.528$), respiratory rate ($P = 0.201$), minute ventilation ($P = 0.451$) or heart rate ($P = 0.379$) between the different types of snorkel equipment.

Despite an excellent external fit of the mask, the internal skirt between eye-pocket and oronasal compartment did not always seal adequately as noted by either a gap between the skirt and the face or fogging in the eye-pocket. Seven participants had an inadequate internal seal between the pockets while wearing the Subea mask, and 11 had an inadequate seal while wearing the QingSong mask. The majority of those experiencing an inadequate seal were female (seven Subea, nine QingSong). All five participants of Chinese or South-East Asian ethnicity experienced an inadequate internal seal.

All participants reported a subjectively higher work of breathing during use of both full-face snorkel masks compared to a conventional snorkel (e.g., "*harder-*" or "*difficult to breathe*", "*more resistance*", "*takes more effort*"). Participants were divided in their rating of highest work of breathing when comparing both full-face snorkel masks (seven Subea, seven QingSong, six equal). Some participants expressed discomfort while wearing a full-face snorkel mask: e.g., hot on their face (two Subea, five QingSong, "*this is a sauna*"); claustrophobic (three Subea, one QingSong); headache (two Subea); lightheaded (two Subea); nauseous (one QingSong).

## Discussion

We investigated the development of hypercapnia and hypoxaemia during rest and exercise whilst wearing full-face snorkel masks and a conventional snorkel. Thirteen participants experienced mild hypoxaemia, and our safety endpoint of 7.0 kPa end-tidal $CO_2$ was reached more frequently and earlier when wearing a full-face snorkel mask compared to a conventional snorkel. All participants experienced a significant increase in end-tidal $CO_2$ accompanied by a decrease in end-tidal oxygen in the oronasal compartment, compared to a conventional snorkel. We also measured a significant increase in $PCO_2$ and decrease in $PO_2$ in the eye-pocket of both full-face snorkel masks compared to atmospheric air. This latter finding suggests that the internal oronasal mask seal is not completely competent, and that rebreathing occurred when wearing these full-face snorkel masks.

Previous studies that measured $CO_2$ noted an increase in inspired and/or end-tidal fractions in participants while wearing a full-face snorkel mask.[4,6,8,10] Some authors argue that short term exposures to higher inspired $CO_2$ fractions are physiologically inconsequential. The British Standard for respiratory protective devices including full-face snorkel masks considers an inspired $PCO_2$ of 1 kPa the safe limit.[14] Most humans respond to an inspired $PCO_2$ of 1 kPa by subconsciously increasing their minute ventilation to maintain normocapnia, but from about 2 kPa onwards increased ventilation is perceptible.[7,15] This in turn may be uncomfortable for the wearer as indicated by the experiences of our participants that it was "*difficult to breathe*", "*more resistance*", or "*taking more effort*". Previous studies that have examined the subjective effects of wearing full-face snorkel masks have included similar reports of shortness of breath[9] and perceived discomfort according alongside higher inspiratory pressures.[4]

Rebreathing seems a common and undesirable consequence of using full-face snorkel masks.[6,8] Even when the full-face snorkel mask was used as a ventilation device on healthy volunteers, inspired $PCO_2$ increased up to about 3 kPa, while inspiratory $PO_2$ decreased.[12,13]

Two of the three existing studies assessing oxygen saturation did so in a non-continuous manner with long intervals.[10,11] If we had taken a similar approach instead of continuous monitoring, we might have failed to detect hypoxaemia in our participants, as the average values were similar in this study. We acknowledge that there is a form of survival bias in our study since we stopped multiple exposures due to high end-tidal $CO_2$. Perhaps if we had allowed hypercapnia to progress, the effect of lower inspired oxygen concentrations on hypoxaemia would have become more pronounced. Our results are in line with the other snorkelling simulation study,[6] although oxygen saturations were not measured in all their participants. They reported one participant with mild hypoxaemia (peripheral saturation 93%), who was a smaller female.

In our study we noted that females and participants of Chinese or Asian ethnicity more frequently experienced an inadequate oronasal seal, and subsequently rebreathed expired gas within the mask. This is consistent with a previous study in which participants with higher inspired $PCO_2$ and lower inspired $PO_2$ were all of Asian ethnicity.[8] Since facial size and proportion vary greatly by ethnicity and gender,[16] a three-size-fits-all approach to designing full-face snorkel masks may require reconsideration.

In theory, full-face snorkel masks are designed to produce unidirectional airflow. However, our measurements spanned the respiratory cycle in the eye-pocket for all participants, demonstrating failure of unidirectional flow. If the oronasal mask seal and one-way valves were truly competent, the gas in the eye-pocket should conform to the composition of inspired atmospheric air. Our findings suggest that this is not the case, and that exhaled gas mixes with incoming air resulting in rebreathing to various degrees. Even when there were no obvious signs of leakage through either the internal skirts or valves, such as fogging, lowered $PO_2$ and elevated $PCO_2$ in the eye-pocket were observed. The consequently larger dead space probably explains why full-face snorkel masks performed worse than conventional snorkels in this and the other snorkel study.[6] If this tendency to rebreathing is combined with a smaller tidal volume, as would be the case in children, a greater tendency to hypercapnia and hypoxia might be apparent. Tidal volume in small adults or children might not be sufficient to offset the effects of rebreathing and dead space (the latter which may be as large as 0.7 to 1.5 L in full-face snorkel masks).[4]

There are several limitations to this study which need to be acknowledged. Firstly, participants were predominately healthy, experienced divers. Considering most divers are familiar with breathing through regulators and snorkel equipment, it is possible that participants may have been more aware of, and able to control their breathing more confidently, maintaining normal tidal volumes and ventilation and experiencing less $CO_2$ accumulation, therefore performing better than novice snorkelers. This is largely speculation, but at the very least we need to acknowledge that our study cohort were not novice snorkelers who are the most frequent users of full-face snorkel masks. Similarly, our subjects were adults, and as discussed above, there are plausible reasons to believe that children might be more adversely affected by rebreathing in full-face snorkel masks. Second, snorkelers with underlying co-morbidities which may affect breathing, like asthma or chronic obstructive pulmonary disease, may influence the performance of full-face snorkel masks. Third, exercise was completed on a cycle ergometer rather than in water. While levels of exertion should be comparable, there was no hydrostatic pressure. It is possible that the negative static lung load associated with head-out immersion may reduce lung compliance, increase work of breathing, and negatively impact $CO_2$ sensitivity thus blunting the ventilatory response to rebreathing.[17] On the other hand, it is also possible that when submerged in water, the pressure on the mask might result in a better oronasal seal, possibly reducing airflow between the pockets. Fourth, we only tested two full-face snorkel mask brands, other brands may perform differently. However, no mask on the market takes into account all different facial types for a perfect internal skirt fit and all seem to use the same technology to create one-way valves. We therefore hypothesise that this is an issue applicable to more full-face snorkel mask brands than the two tested in our study. Lastly, we could not add a pneumotachometer to directly measure minute ventilation without altering the functionality of the full-face snorkel masks. We calculated minute ventilation indirectly from the measured chest expansion.

This study also had a number of strengths, including a head-to-head comparison of two full-face snorkel masks with a conventional snorkel, a relatively large sample size, a randomised order of the full-face snorkel masks, and measurements at rest, light intensity exercise (comparable to normal snorkelling) and moderate intensity exercise (comparable to fighting a current).

## Conclusions

These results suggest that at least some full-face snorkel masks enhance the risk of hypercapnia and possibly hypoxia due to rebreathing arising primarily from communication between the eye and oronasal pockets. Manufacturers and future snorkelers should be made aware of this new information to prevent unsafe situations.

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### Conflicts of interest and funding

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# Measuring whole body inert gas wash-out

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Decompression sickness; Diving research; Gas kinetics; Nitrogen; Physiology; Pressure

**Abstract**
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**Introduction:** Quantifying inert gas wash-out is crucial to understanding the pathophysiology of decompression sickness. In this study, we developed a portable closed-circuit device for measuring inert gas wash-out and validated its precision and accuracy both with and without human subjects.
**Methods:** We developed an exhalate monitor with sensors for volume, temperature, water vapor and oxygen. Inert gas volume was extrapolated from these inputs using the ideal gas law. The device's ability to detect volume differences while connected to a breathing machine was analysed by injecting a given gas volume eight times. One hundred and seventy-two coupled before-and-after measurements were then compared with a paired *t*-test. Drift in measured inert gas volume during unlabored breathing was evaluated in three subjects at rest using multilevel linear regression. A quasi-experimental cross-over study with the same subjects was conducted to evaluate the device's ability to detect inert gas changes in relation to diving interventions and simulate power.
**Results:** The difference between the injected volume (1,996 ml) and the device's measured volume (1,986 ml) was -10 ml. The 95% confidence interval (CI) for the measured volume was 1,969 to 2,003 ml. Mean drift during a 43 min period of unlaboured breathing was -19 ml, (95% CI, -37 to -1). Our power simulation, based on a cross-over study design, determined a sample size of two subjects to detect a true mean difference of total inert gas wash-out volume of 100 ml.
**Conclusions:** We present a portable device with acceptable precision and accuracy to measure inert gas wash-out differences that may be physiologically relevant in the pathophysiology of decompression sickness.

## Introduction

The ability to measure inert gas turnover is crucial to studying and understanding the pathophysiology of decompression sickness (DCS).[1] Physiologic interventions that alter cardiac output and/or tissue perfusion have been shown to affect inert gas turnover[2–7] and the risk of developing DCS.[7,8] However, the correlation between quantitative differences in inert gas turnover and the risk of DCS is not known. Measuring inert gas turnover can help us understand how to integrate physiological factors into mathematical decompression models and improve their predictive accuracy. Due to the technical complexity of the required equipment, the number of trials measuring inert gas turnover in the context of diving is limited.

To our knowledge, a very limited number of research groups have studied and published on techniques of measuring whole body inert gas wash-out and/or uptake.[2,9–12] More studies with continuous measurements of inert gas wash-out and/or uptake are needed to better understand gas dynamics and how they relate to the pathophysiology of DCS. These studies would benefit from devices able to continuously measure and statistically analyse changes in inert gas volumes over time.

The primary aim of this study was to present a portable closed-circuit device for quantifying inert gas wash-out over time and evaluate its performance both with and without human subjects. A secondary aim was to determine the required sample size to achieve statistical power for future cross-over studies.

## Methods

The study was approved by the Swedish Ethical Review Authority (Dnr: 2020–06865) and all subjects provided informed, written consent to participate before the start of the study.

## NITROGEN WASH-OUT

All measurements were performed at an ambient pressure of 101.3 kPa (1.0 atmosphere absolute [atm abs]). Nitrogen wash-out was detected as alterations in volume within a closed rebreathing system. To determine the proportion of volume changes attributable to nitrogen wash-out, adjustments based on the principles of the ideal gas law was used. The adjustments were based on the following sensors and system specifications; relative humidity and temperature in the total system volume, temperature in the carbon dioxide scrubber, total volume and changes in the counterlung volume. The volume conversion to standard temperature ($V_{ST}$) was performed for each section (hoses, scrubber and counter lung) in time increments as the temperature in the system and scrubber changed over the period of measurement according to equation 1 where T is the measured temperature in degrees Celsius and $V_{AT}$ is the measured volume at ambient temperature.

$$V_{ST} = V_{AT}\left(\frac{273}{273+T}\right) \qquad \text{Equation 1}$$

To further standardise the volume changes due to pressure difference and humidity a conversion to standard temperature and pressure, dry (STPD) was performed according to eq. 2 where $P_B$ is the measured ambient pressure in kPa, $P_{H2O}$ is the water vapour pressure for saturated gas at the ambient temperature in kPa, and RH is the measured relative humidity.

$$V_{STPD} = V_{ST}\left(\frac{P_B - P_{H2O} \cdot RH}{101.3\ kPa}\right) \qquad \text{Equation 2}$$

The measured volume change was then multiplied with the fraction of nitrogen (1 – measured oxygen fraction) to determine nitrogen wash-out.

The closed circuit (see Figure 1) consisted of a mouthpiece, a modified soda-lime scrubber to remove carbon dioxide (Inspiration Tempstik scrubber, AP Valves, Cornwall UK), an ASVPOD for oxygen injection (device manufactured by Poseidon Diving Systems AB, Gothenborg Sweden with oxygen sensing and dosage, and temperature sensing), a counterlung (ISMIX counter bellows, Interspiro AB, Täby, Sweden) with a volume sensor and a CPOD (Poseidon Diving Systems AB, Gothenburg, Sweden) for oxygen, temperature and water vapour sensing. The oxygen fraction was regulated at a setpoint of 21% by the two Poseidon PODs via a computerised algorithm for sensing and dosage.

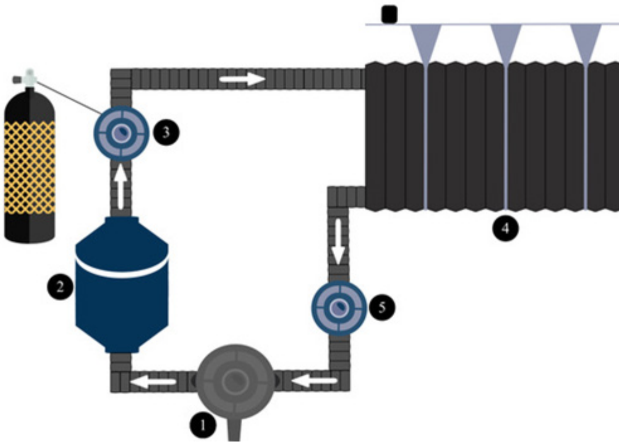
## DESIGN AND SUBJECTS

### *Experimental measurements for evaluation of volume detection*

To evaluate the device's sensors, software, and mechanical setup, we utilised a breathing machine and known gas flows.

**Figure 1**

Closed-circuit device for measuring inert gas wash-out volume; 1 – mouthpiece; 2 – soda-lime $CO_2$ scrubber; 3 – ASVPOD , with $PO_2$ sensing and dosage, and temperature sensing; 4 – counterlung with a position sensor for volume sensing; 5 – CPOD with $PH_2O$, temperature and $PO_2$ sensing



The following experiment presents a case in which the closed circuit was connected to a breathing machine (Life Support Equipment Test Facility, Ansti Test Systems Ltd, Fareham UK) with a fixed respiratory rate of 15 breaths per minute and a tidal volume of 700 ml. Paired volume registrations were analysed before and after injections of 1,996 ml, STPD air.

### *Human measurement for evaluation of potential drift and a quasi-experimental crossover study*

Three subjects meeting the Swedish Armed Forces physical standards for diving were recruited. All were male, non-smoking and between 35–43 years old (Table 1).

To evaluate the drift the three subjects underwent one control measurement each. During these measurements they remained at rest in a supine position.

A quasi-experimental crossover study was conducted with four distinct diving interventions, with nitrogen wash-out measurements immediately after each dive. The four interventions were: 18 metres of sea water (msw) with bottom time of 50 minutes performed either dry or immersed (18 msw / 50 min, dry/immersed) and 39 msw with bottom time of 10 min performed either dry or immersed (39 msw / 10 min, dry/immersed).

All dives were performed in a hyperbaric chamber (HAUX 2300) at the naval base in Karlskrona, Sweden. During the dry dives, the subjects remained either lying down or seated at rest. The immersed dives occurred in water at 10 (± 1)°C with the subjects wearing undergarments, wet gloves, and dry suits; and snorkeling while engaged in low intensity finning. Compression and decompression were completed in accordance with US Navy Diving Table revision 7 at 23 $msw{\cdot}min^{-1}$ and 9 $msw{\cdot}min^{-1}$ respectively.

**Table 1**
Characteristics of participants

| Subject | Age (years) | Weight (kg) | Height (cm) | Training (min·week$^{-1}$) | Resting heart rate (beats·min$^{-1}$) | Body fat (%) | Body mass index (kg·m$^{-2}$) |
|---|---|---|---|---|---|---|---|
| 1 | 37 | 90.3 | 185 | 200 | 45 | 16.4 | 26.1 |
| 2 | 43 | 75.1 | 180 | 100 | 60 | 15.3 | 23.2 |
| 3 | 35 | 85.3 | 185 | 500 | 65 | 12.5 | 24.9 |

To decrease inter- and intraindividual variance of respiration volumes, all nitrogen volume measurements were performed with the subject resting in a supine position. The volume in the system was measured at the end of each expiration as end-expiratory volume tends to be more reliable (less variance) relative to end-inspiratory volume.[13] The zero setpoint was defined as the median of the first ten extracted data points.

### STATISTICAL ANALYSES

The statistical methods were devised through a collaborative effort involving two biostatisticians.

#### *Experimental measurements for evaluation of volume detection*

The device's ability to detect known injected gas volumes was analysed with a paired *t*-test. Measurements before were paired with measurements after gas injections of known volumes. The difference between the injected and the measured volumes indicates the accuracy of the device. The 95% confidence interval (CI) of the measured volume is an indication of the precision.

#### *Human measurement for evaluation of potential drift and a quasi-experimental crossover study*

End-expiratory plots with grouped linear regressions were used to visualise the control measurements in relation to the measurements of nitrogen wash-out after each intervention. We used time periods 0–15 minutes and 33–43 minutes, allowing us to observe both the difference in nitrogen wash-out flow rates (greater in the beginning) and total volume difference which was more distinct in the end of the wash-out period.

Inert gas volume drift during control measurements with human subjects was analysed with a multilevel linear regression model. The grouped linear regression line (with random individual intercepts) from the end expiratory plots were compared with the expected zero-line. The drift was expressed as the model's estimate which reflects the mean nitrogen volume difference from zero (ml), and the model's time factor which reflects the mean nitrogen flow rate difference from zero (ml·min$^{-1}$).

The device's ability to detect inert gas wash-out volume differences in a crossover study with three subjects was analysed with a multilevel linear regression model (random individual intercepts). The model's estimate for the period 33–43 min (when the wash-out curves flatten) was used to detect mean differences in total inert gas wash-out volumes. The model's time factor was used to analyse the flow rate differences (ml·min$^{-1}$), within a given period.

The secondary aim was analysed using Monte Carlo based simulations using the multilevel linear regression models and the data obtained from the dive profile of 18 msw / 50 min, performed immersed and dry, during the interval 33–43 minutes. The required sample size was estimated to detect a given inert gas volume difference with 80% power and a false positive rate (alpha) of 0.05, to inform the design of future experimental cross-over studies using the device.

## Results

### EXPERIMENTAL MEASUREMENTS FOR EVALUATION OF VOLUME DETECTION

The difference between the injected volume (1,996 ml) and the measured volume (1,986 ml) was -10 ml. The 95% CI for the measured volume was 1,969–2,003 ml. In total eight injections of 1,996 ml and 11–34 paired before-and-after measurements (a total of 172 paired volume registrations) were analysed.

#### *Human measurement for evaluating potential drift and a quasi-experimental crossover study*

During one of three control measurements a gas leak was discovered. This was assumed to be constant and was corrected for in the data processing. The three individual control measurements (0–43 min) for drift analyses showed a mean nitrogen volume difference of -19.24 ml (95% CI -37.15 to -1.49) and a flow rate difference of -0.23 ml·min$^{-1}$ (95% CI -0.93 to 0.56) (Table 2).

The quasi-experimental crossover study could significantly detect mean nitrogen wash-out volume differences between the different interventions. The mean differences in inert gas wash-out (total volumes and flow rates) are shown in Table 3 and the end-expiratory plots with grouped linear regression lines during the wash-out periods 0–15 min

**Table 2**
Control measurements with three subjects for drift analyses with mean nitrogen ($N_2$) volume and flow rate differences in relation to the expected zero; CI – confidence interval

| Time period | Mean $N_2$ volume difference ml (95% CI) | Mean $N_2$ volume flow rate difference ml·min$^{-1}$ (95% CI) |
|---|---|---|
| Drift (33–43 min) | -73.91 (-105.46 to -39.11) | 2.46 (-3.29 to 7.98) |
| Drift (0–43 min) | -19.24 (-37.15 to -1.49) | -0.23 (-0.93 to 0.56) |
| Drift (0–15 min) | -40.25 (-68.39 to -11.02) | 0.68 (-2.11 to 3.92) |

**Table 3**
Mean differences of inert gas wash-out total volumes and flow rates; CI – confidence interval

| Intervention | Mean nitrogen total volume difference (33–43 min) ml (95% CI) | Mean nitrogen volume flow rate differences (0–15 min) ml·min$^{-1}$ (95% CI) |
|---|---|---|
| Dry, 18 msw / 50 min vs controls | 409 (356 to 458) | 20 (15 to 26) |
| Dry, 39 msw / 10 min vs controls | 542 (487 to 596) | 32 (27 to 37) |
| Immersed, 18 msw / 50 min vs controls | 660 (600 to 715) | 32 (27 to 37) |
| Immersed, 39 msw / 10 min vs controls | 719 (664 to 774) | 28 (22 to 35) |
| Immersed 18 msw / 50 min vs dry 18 msw / 50 min | 264 (215 to 309) | 12 (7 to 18) |
| Immersed 39 msw / 10 min vs dry 39 msw / 10 min | 104 (36 to 172) | -5 (-13 to 2) |

**Table 4**
Power simulations regarding detection of mean inert-gas wash-out volume differences; *n* – participants required for 80% power (alpha = 0.05)

| Effect size (ml) | *n* |
|---|---|
| 400 | 2 |
| 300 | 2 |
| 200 | 2 |
| 100 | 2 |
| 50 | 4 |

and 33–43 min are shown for the different dive profiles in Figures 2 and 3. All measurements were recorded between 5–48 minutes (with 5 minutes after surfacing set as zero for all recordings) after surfacing for all interventions. One out of three recordings for the profile 39 msw / 10 min immersed, was only possible between 0–13 minutes because of battery problems.

The dives at 18 msw / 50 min immersed had an increased mean nitrogen wash-out volume of 264 ml (95% CI 215 to 309 ml) compared to the dry dives, and the mean flow rate during the first 15 min was 12 ml·min$^{-1}$ (95% CI 7 to 18 ml) higher. The dives at 39 msw / 10 min immersed had an increased mean nitrogen wash-out volume of 104 ml (95% CI 36 to 172 ml) compared to the dry dives and the flow rate during the first 15 min was 5 ml·min$^{-1}$ (95% CI -13 to 2) lower.

Our power simulation, based on this cross-over study design, found a sample size of two subjects would be able to detect a mean difference of total inert gas volume of 100 ml with 80% power and a false positive rate (alpha) of 0.05 (see Table 4).

## Discussion

### MAIN FINDINGS

We present a portable closed-circuit device for quantifying inert gas wash-out volumes following decompression. Performance without and with human subjects demonstrated acceptable precision and accuracy to measure relevant differences. With this device, a sample size of two is sufficient to detect a mean difference of 100 ml. The device is easy to transport and suitable for use in the field.

**Figure 2**

End-expiratory plots with grouped linear regression lines for the three subjects during nitrogen wash-out period 0–15 min and 33–43 min, for the dive profile 18 msw / 50 min and control measurements; a – control measurements versus measurements 0–15 min after a dry dive; b – controls measurements versus measurements 33–43 min after a dry dive; c – control measurements versus measurements 0–15 min after an immersed dive; d – control measurements versus measurements 33–43 min after an immersed dive; e – measurements 0–15 min after a dry dive versus measurements 0–15 min after an immersed dive; f – measurements 33–43 min after a dry dive versus measurements 33–43 min after an immersed dive

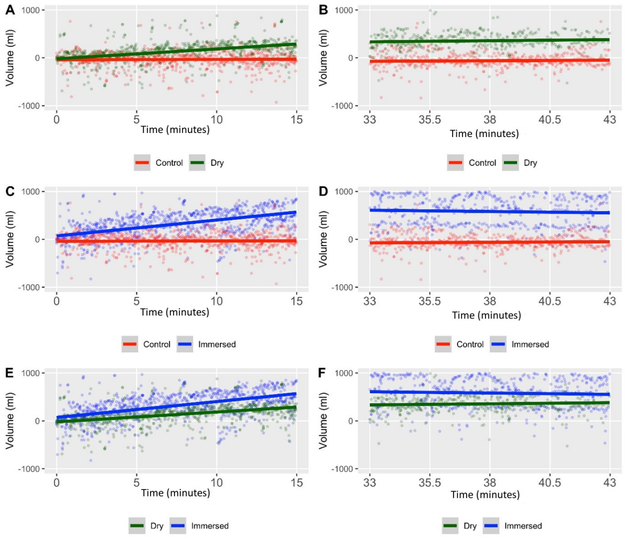
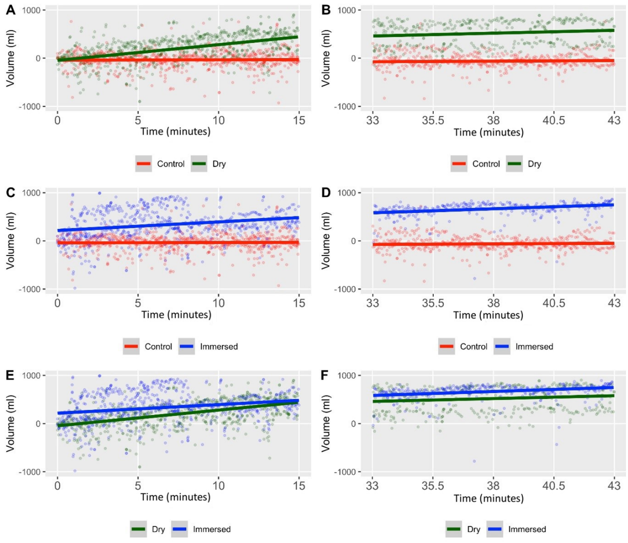


**Figure 3**

End-expiratory plots with grouped linear regression lines for the three subjects during nitrogen wash-out period 0–15 min and 33–43 min, for the dive profile 39 msw / 10 min and control measurements; a – control measurements versus measurements 0–15 min after a dry dive; b – control measurements versus measurements 33–43 min after a dry dive; c – control measurements versus measurements 0–15 min after an immersed dive; d – control measurements versus measurements 33–43 min after an immersed dive; e – measurements 0–15 min after a dry dive versus measurements 0–15 min after an immersed dive; f – measurements 33–43 min after a dry dive versus measurements 33–43 min after an immersed dive



The evaluation without human subjects demonstrated that the device detected 1,996 ml as 1,986 ml (95% CI 1,969–2,003). This accuracy (a discrepancy of only 10 ml) exceeds the requirements needed to detect significant differences in gas wash-out.

The quasi cross-over study revealed significant differences in nitrogen wash-out volumes between both control versus interventions and interventions versus interventions. The difference between dry dives and immersed dives after the profile 18 m / 50 min, was significant for both total wash-out volume and the wash-out flow rate for the first 15 minutes.

## IMPLICATIONS

Our small quasi cross-over study, analysed with multilevel linear regression, suggests a sample size of two subjects could be sufficient to detect mean inert gas wash-out differences after immersed versus dry dives and between different dive profiles. However, these findings are related to the device's observational error and may not account for differences between our study subjects and the general population.

In future studies with larger sample sizes our device together with a multilevel linear regression model could probably detect effects related to divers' anthropometry.

Since earlier studies have shown significant changes in DCS risk after cold versus warm immersed decompression[8] and dry versus warm immersed pre-denitrogenation[7] we argue that inert gas volume differences in our quasi cross-over study may play a role in the pathogenesis of DCS. Equal volume differences have also been observed in cold versus warm exposures and negative pressure breathing during denitrogenation at 1 atm abs.[4,5]

## STRENGTHS AND LIMITATIONS

A strength of the device is its ability to extract data points that correspond to end exhalation and to continuously calculate STPD volume changes of inert gas. This methodology generates a data point for every breath, a more frequent data collection interval than achieved in prior studies.[3,9,12] The intra- and inter-individual variance at the end of a normal passive exhalation (functional residual capacity) has been shown to be more stable compared to other spirometrically defined points.[13] All extracted data points were analysed with a multilevel linear regression model which minimises bias from breathing instructions or manual selection of data points.

One limitation of our evaluation on human subjects is a small sample size that is not representative of the general population. It is possible that other subjects have greater variance of end-expiratory plots, smaller nitrogen volume differences or problems breathing through the mouthpiece. Another limitation is the manual adjustment that needed to be done to correct for a gas leak during one of the control measurements. This adjustment may not have affected precision, but likely had an effect on accuracy. A third limitation is the counterlung of the device, which had a tendency to catch when gliding in the suspension device. This may have had an effect on the device's ability to provide exact data points. A fourth limitation is that our statistical model needs to define appropriate time periods to analyse the differences in total nitrogen wash-out volumes and flow rates, respectively.

## Conclusions

We present a portable device with acceptable precision and accuracy to measure inert gas wash-out differences that may be physiologically relevant in the pathophysiology of decompression sickness.

When using a cross-over study design, our power simulations estimated that a sample size of two subjects may be sufficient to detect physiologically relevant differences of inert gas wash-out volumes.

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# Comparing the EMMA capnograph with sidestream capnography and arterial carbon dioxide pressure at 284 kPa

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**Keywords**
Capnography; Hyperbaric chamber; Intensive care; Patient monitoring

## Abstract

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**Introduction:** Capnography aids assessment of the adequacy of mechanical patient ventilation. Physical and physiological changes in hyperbaric environments create ventilation challenges which make end-tidal carbon dioxide ($ETCO_2$) measurement particularly important. However, obtaining accurate capnography in hyperbaric environments is widely considered difficult. This study investigated the EMMA capnograph for hyperbaric use.

**Methods:** We compared the EMMA capnograph to sidestream capnography and the gold standard arterial carbon dioxide blood gas analysis in a hyperbaric chamber. In 12 resting subjects breathing air at 284 kPa, we recorded $ETCO_2$ readings simultaneously derived from the EMMA and sidestream capnographs during two series of five breaths (total 24 measurements). An arterial blood gas sample was also taken simultaneously in five participants.

**Results:** Across all measurements there was a difference of about 0.1 kPa between the EMMA and sidestream capnographs indicating a very slight over-estimation of $ETCO_2$ by the EMMA capnograph, but fundamentally good agreement between the two end-tidal measurement methods. Compared to arterial blood gas pressure the non-significant difference was about 0.3 and 0.4 kPa for the EMMA and sidestream capnographs respectively.

**Conclusions:** In this study, the EMMA capnograph performed equally to the sidestream capnograph when compared directly, and both capnography measures gave clinically acceptable estimates of arterial $PCO_2$.

## Introduction

End-tidal carbon dioxide monitoring is vital when monitoring mechanically ventilated patients to ensure adequate ventilation.[1] This applies to patients in intensive care, but potentially more so during treatment inside a hyperbaric chamber due to physiological and pressure changes[2] which frequently necessitate adjustments to ventilator parameters.[3] Unfortunately, not all technology can be brought into the hyperbaric chamber environment due to physical incompatibilities (like pressure and temperature), electrical power restrictions and increased fire risks.[4]

Capnography aims to measure the pressure of carbon dioxide ($PCO_2$) in the expired gas at the end of each exhalation (the end-tidal $CO_2$ [$ETCO_2$]). This is accepted as an adequate surrogate for the $PCO_2$ in arterial blood. The sensors used for measuring $CO_2$ in this context are typically spectroscopic and discern $CO_2$ molecules by their characteristic absorption of infrared light. These sensors can be deployed in mainstream or sidestream configurations. Mainstream positioning puts the sensor in the main flowpath for exhaled and inhaled gas at the end of the endotracheal tube. Sidestream positioning puts the sensor outside the main flowpath, with a continuous gas sample drawn from the end of the endotracheal tube to the sensor via a narrow bore tube. In the hyperbaric setting the sidestream capnography sensor is commonly placed outside the chamber, with the sampling line ported through the chamber wall. The pressure difference during the hyperbaric treatment will force gas through the tubing. The $PCO_2$ measurements made at normobaric pressure need to be multiplied by the absolute pressure inside the chamber to obtain the actual value, even if the device displays a fraction of $CO_2$.

Measuring carbon dioxide under pressure using a mainstream device has the downside of interference by both collision and pressure broadening.[5] Collision broadening is known to affect the accuracy of capnography negatively (underestimating the result) due to the increased presence of oxygen molecules that collide with carbon dioxide molecules, causing a transfer of energy that results in broadening of the carbon dioxide absorption peak.[6,7] Pressure broadening, on the other hand, causes an overestimation of the result due to a pressure-induced shift in the absorption spectrum for carbon dioxide.[8] One study determined a measured increase in $PCO_2$ of 0.4 kPa per 101 kPa total pressure.[6] The results can be automatically compensated if the device has an integrated pressure and oxygen sensor. However, it requires the compensation algorithm to accept the large pressure changes commonly used inside the hyperbaric chamber versus the small atmospheric changes commonly programmed into these devices. Alternatively, mainstream capnography can be manually adjusted based on the gas mixture's oxygen content and pressure with a compensation formula/graph/table. However, it has been reported that each device works differently and would require its own compensation values.[5,8,9] One obvious advantage of sidestream capnography in this setting is that the $PCO_2$ is measured at normobaric pressure and the result is not influenced by collision and pressure broadening.

Mainstream and sidestream capnography have been compared extensively in the normobaric environment.[10] In the hyperbaric environment, only a few studies have been conducted. One study compared mainstream capnography with arterial blood samples and found a good correlation ($r^2 = 0.83$) but an expected large overestimation of arterial $PCO_2$ by the capnography of 2.22 kPa at 284 kPa treatment pressure in patients ventilated with 100% oxygen.[11] Another assessed a mainstream capnograph with various calibration gases at hyperbaric pressure and found a persistent overestimation.[5] Similarly, a recent study investigated the EMMA capnograph for use inside the hyperbaric chamber using multiple calibration gases and found consistent over-estimation at 284 kPa.[12]

The EMMA capnograph is a lightweight, mobile, battery-powered (two AAA alkaline batteries) mainstream capnograph developed for pre-hospital and mobile care. The small device contains a sensor and display, providing the end-tidal carbon dioxide pressure and respiratory rate. Previous use in our hyperbaric studies has confirmed that the device functions at the highest pressures achieved in that work (557 kPa).[13] Also, oxygen breathing at 284 kPa did not cause obvious problems.[14] Those studies did not assess the accuracy of the device.

This study aimed to validate the mainstream EMMA capnograph under pressure by comparing it with sidestream capnography and, in a small convenience sample, to the gold standard arterial blood gas sampling.

## Methods

### TRIAL DESIGN AND PARTICIPANTS

This prospective methods comparison sub-study was part of a randomised cross-over study investigating the interaction of nitrogen and $CO_2$ in producing narcosis during 608 kPa exposures at the Slark Hyperbaric Unit, Te Whatu Ora Waitemata, from August to October 2022. The study protocol was approved by the Health and Disability Ethics Committee, Auckland, New Zealand (reference 16/NTA/93), and was registered with the Australian New Zealand Clinical Trial Registry (ANZCTR: U1111-1181-9722, http://www.anzctr.org.au/, RRID:SCR_002967).

Participants ($n = 12$) were healthy, certified technical divers aged between 18 and 60 years. Candidate participants currently using recreational drugs, tobacco, psychoactive medication, excessive alcohol, or over five caffeine-containing beverages a day were excluded. Prior to each hyperbaric exposure, participants had at least six hours of sleep, abstained from any caffeinated drink on the day and refrained from diving and alcohol for 24 hours prior. All twelve participants provided written informed consent, and five participants provided additional informed consent for the arterial blood gas sampling.

### EXPERIMENTAL PROCEDURES

All 24 measurements (two per participant) were conducted inside a cylindrical five-person hyperbaric chamber (W.E. Smith Engineering PTY LTD, Australia). The measurements were taken at the 284 kPa stop during decompression from 608 kPa while breathing environmental air. Two hundred and eighty-four kPa was chosen as it is the most common maximum hyperbaric oxygen treatment pressure. The measurements consisted of a simultaneous analysis of $ETCO_2$ with the EMMA capnograph and sidestream capnography for five breaths through a breathing tube (Figure 1). The subjects were at rest throughout the experiment. They were instructed to seal their lips around the breathing tube and simply breathe normally for five breaths. In five participants, an additional arterial puncture during the breaths provided arterial carbon dioxide levels. In these five subjects, the breath measurements were timed to coincide with the drawing of the arterial specimen.

*Mainstream EMMA capnography*

Mainstream $ETCO_2$ was measured with the EMMA capnograph (Masimo, Irvine, CA, USA). Calibration was not needed according to the manufacturer's recommendations. Data points were manually transcribed.

**Figure 1**

Breathing tube with EMMA capnograph and sidestream sampling tube

*Sidestream capnography*

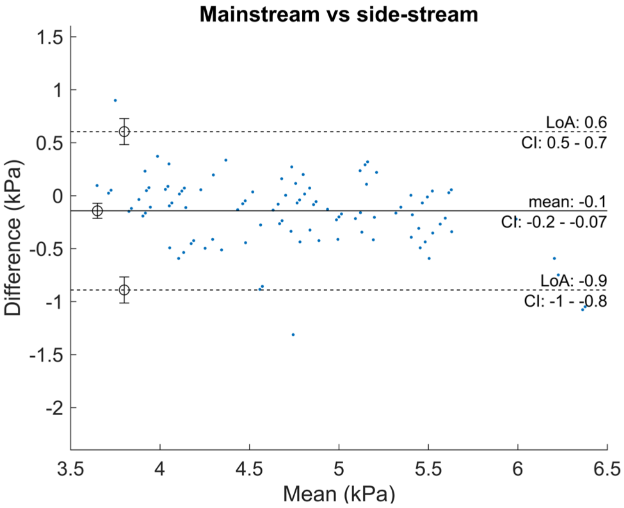
A sampling tube was attached to the breathing tube of the EMMA capnograph. This sampling line was ported through the chamber wall, and connected to a gas analyser (ML206, ADInstruments, Dunedin, New Zealand, RRID:SCR_001620) via a t-piece to limit the gas flow to the analyser. The sampling pump was set to the maximum (approximately 200 ml·min$^{-1}$) to minimise the response time. Data were recorded with a PowerLab 4/25T (ADInstruments) and LabChart Pro version 8.1.24 (ADInstruments, RRID:SCR_017551) software. At the start of each measurement, calibration was performed according to the manufacturer's recommendations with a known reference gas. End-tidal carbon dioxide values were derived from the continuous carbon dioxide measurement by automated breath-by-breath detection. The $ETCO_2$ values of the five breaths were manually exported and multiplied by the environmental pressure inside the hyperbaric chamber (284 kPa).

*Arterial carbon dioxide*

In a convenience sample of five consenting participants, an arterial blood sample was taken. Flow in the radial and ulnar arteries was checked prior to compression using colour-flow ultrasound (Butterfly iQ, Guildford, CT, USA). The radial arterial puncture (23g needle) was performed under local anaesthesia (2% lignocaine) by an experienced anaesthetist (SJM) using palpation to locate the non-dominant radial artery. It was recorded during which of the five breaths exactly the 2 ml blood was drawn. After ensuring there was no gas in the syringe, the blood sample was depressurised and analysed directly outside the hyperbaric chamber with an iStat Alinity point-of-care blood gas analyser (Abbott, Abbott Park, IL, USA, RRID:SCR_008392).

**Figure 2**

Bland-Altman plot comparing the EMMA and sidestream capnograph; each breath is plotted as the mean and difference between the two measurement methods; CI – confidence interval; LoA – 95%-level of agreement



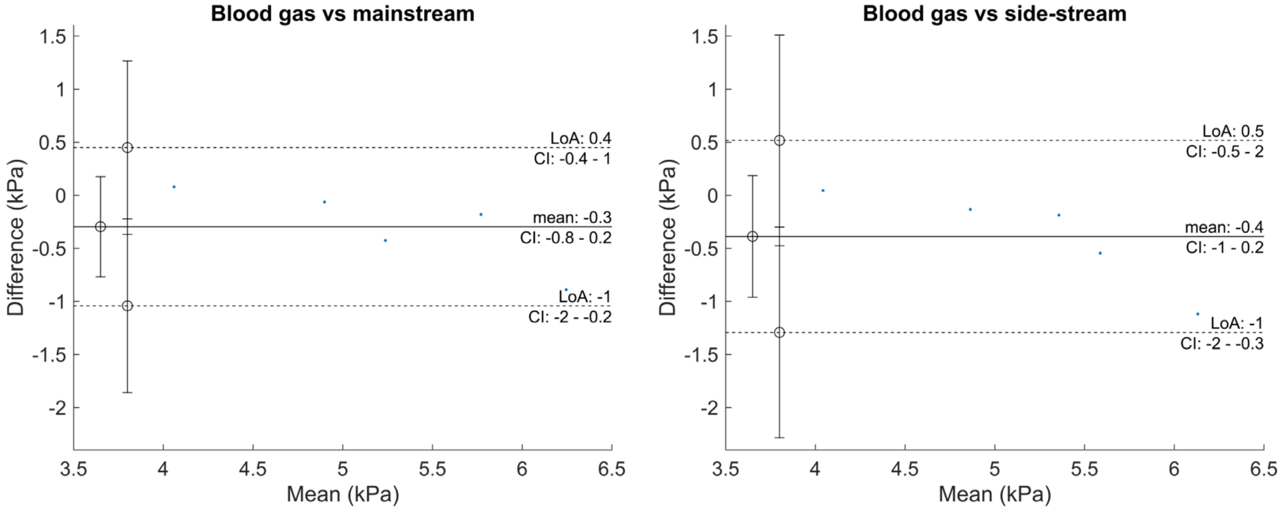
## STATISTICAL ANALYSIS

All data were imported into Matlab version 2022b (Mathworks, Natick, MA, USA, RRID:SCR_001622) for analysis. The gas measurement values from all 120 breaths measured with the EMMA and sidestream capnograph datasets were presented as median and range, because of a non-normal distribution of both datasets (Kolmogorov–Smirnov test). Datasets were compared with the Wilcoxon signed rank test. Breath-by-breath end-tidal carbon dioxide values of the EMMA and sidestream capnograph were compared with a Bland-Altman analysis to determine the agreement between the two methods. We graphically presented the variation of differences between the capnography methods against their average (Bland–Altman plot).

According to the reporting standards for Bland–Altman analysis, to ensure that the 95%-limits of agreement were meaningful summary statistics of the differences, we checked the following assumptions: repeatability, constant variation, and normality.[15] Repeatability represents within-participant variation in repeated capnography measurements in the same participant. We recorded five breath measurements per participant in each of two separate pressure exposures, and assessed the repeatability of end-tidal carbon dioxide by one-way ANOVA. In contrast to the total dataset of

**Figure 3**

Bland-Altman plot comparing the arterial blood gas pressure and EMMA (left) or sidestream (right) capnographs; the End-tidal values in these figures are a subset of the data presented in Figure 2 (the five breaths closest to the point of arterial sampling in the five subjects who had this done); CI – confidence interval; LoA – 95%-level of agreement



120 breaths, each first, second, third, fourth and fifth breath was normally distributed (Kolmogorov-Smirnov test). We graphically checked whether the differences were normally distributed in a histogram and whether variations in the differences were constant across the range of measurements. The differences between the two measures were normally distributed (Kolmogorov-Smirnov test).

Arterial $PCO_2$, as the gold standard, was compared with the averaged $ETCO_2$ values of the breaths during the arterial blood gas sampling of both the EMMA and sidestream capnograph and the difference was calculated as the accuracy. The difference between the two accuracies was calculated ('accuracy difference').

In the non-inferiority comparison of accuracies of the two capnography measurements, we set *a priori* the non-inferiority margin of 0.66 kPa (5 mmHg) in accuracy difference. The size of the margin was determined from a clinical standpoint and previous reports.[16]

## Results

Of the 120 breaths, 114 and 113 were captured by EMMA and sidestream capnography respectively due to recording issues with the LabChart software. The median (range) $ETCO_2$ was 4.8 (3.3–6.9) and 4.6 (3.7–5.9) kPa for the EMMA and sidestream capnographs, respectively. There was a statistically significant (but clinically insignificant) difference of about 0.1 kPa. The Bland-Altman analysis showed a 95%-level of agreement between -0.9 and 0.6 kPa (Figure 2). The visual inspection of the differences did not show skewed data, suggesting no correlation with the outcome size. The square root of the within-participant variance of $ETCO_2$ was 0.2 kPa for the EMMA capnograph and 0.1 kPa for the sidestream capnograph. One-way ANOVA showed no difference between breaths for both capnographs, indicating that repeatability was adequate. The Kolmogorov-Smirnov test of the differences showed normally distributed data.

The median (range) arterial $CO_2$ pressure was 5.45 (4.0–6.7) kPa, and the median $ETCO_2$ at the point of arterial sampling was 5.0 (4.1–5.8) and 5.3 (4.1–5.6) kPa for the EMMA and sidestream capnographs respectively. Compared to the arterial blood gas pressure, the non-significant difference was about 0.3 and 0.4 kPa for the EMMA and sidestream capnographs respectively (Figure 3). The *accuracy difference* between the two methods was 0.1 kPa. The number of data points was too small to analyse the levels of agreement effectively.

## Discussion

In this study, we validated the use of the EMMA capnograph compared to sidestream capnography and the gold standard arterial blood gas sampling. We found a statistically significant but clinically insignificant difference between the EMMA and sidestream capnographs, with the EMMA capnograph overestimating the $ETCO_2$ by about 0.1 kPa compared to sidestream capnography. Both the EMMA and sidestream capnographs underestimated the arterial $PCO_2$ by about 0.3 and 0.4 kPa, respectively. The accuracy difference between these two was only 0.1 kPa, indicating agreement between the two end-tidal measurement methods. Neither was inferior, as the difference was smaller than the preselected threshold of 0.66 kPa.

The difference between the two end-tidal measurement methods and the accuracy difference were small and clinically insignificant.[16] Even in healthy participants it is expected that $ETCO_2$ will be slightly lower than arterial $PCO_2$ due to alveolar dead space, i.e., gas exhaled from lung units with a high ventilation : perfusion ratio dilutes the $CO_2$ measured in the expired mixed alveolar gas.[17] This underestimation has been shown consistently.[18–20] Thus, the slight underestimation of the arterial $PCO_2$ based on $ETCO_2$ measured by either capnography method employed here was expected. In contrast, based on appraisal of previous studies, the accuracy difference was smaller than expected. The most comparable study conducted in ventilated human subjects at 284 kPa showed a much larger difference between $ETCO_2$ and arterial $PCO_2$ (an overestimation of 2.2 kPa).[11] We cannot explain the contrast with our results, except to observe that the subjects in that study were mechanically ventilated with 100% oxygen, and the $ETCO_2$ measurement device was different.

A pressure-broadening effect could explain the small difference found between the two capnography methods in the present study. The increased pressure inside the hyperbaric chamber, to which the EMMA capnograph was exposed, could have caused an overestimation of the $ETCO_2$ value. This effect is consistent so that the difference can be anticipated, as shown by others as a linear relationship between pressure and the results from the EMMA capnograph.[12]

### STRENGTHS AND LIMITATIONS

A limitation of the EMMA capnograph was the inability to be calibrated with a reference gas, which may have contributed to the slight difference between the two capnography measures. Nevertheless, this head-to-head comparison between the EMMA capnograph and a research-grade sidestream capnograph suggested that this small, portable and battery-powered device performs well under the circumstances of our experiment. We undertook two measurements of five breaths with twelve participants allowing us to compare 113 data points. A limitation is that we collected only five arterial blood gas samples due to the complexity of taking blood gas samples inside the hyperbaric chamber. This could have been increased by increasing the number of participants or by taking multiple blood samples via a catheter from the same five participants. The number of data points was too small to analyse the levels of agreement effectively. However, the collected data showed good agreement between both capnographs and the arterial blood sampling.

We measured the $PCO_2$ in end-tidal breaths at the point the arterial blood gas sample was taken. Previous research has shown a short (approximately 15 second – approximately three breath) delay between peripheral arterial $PO_2$ and end-tidal $O_2$ due to the time it takes for blood to flow from the lungs to the puncture site.[21] It seems likely that the potential for error to be introduced due to this delay is minimal in our measurements because our participants were resting in a steady state.

In previous research to 608 kPa,[13] we noted that the EMMA capnograph produced errors over 557 kPa, but sidestream capnography would be a viable option for such higher pressure exposures. Nowadays, pressures beyond 284 kPa are very rarely used in hyperbaric treatments. Based on our data, at the pressures at which most hyperbaric treatments are conducted, the EMMA capnograph provides $ETCO_2$ measurements that are sufficiently accurate for decision-making during ventilation of an intubated patient. One caveat is that our subjects were breathing chamber air and not oxygen. Oxygen breathing can cause an increase in alveolar dead space[22] which would increase the difference between $ETCO_2$ (measured by either mainstream or side stream methods) and the true arterial $PCO_2$, and hyperoxic breathing may also affect collision broadening.[5] In future work, this study can be repeated with oxygen breathing, preferably in ventilated patients.

## Conclusion

This study showed that the EMMA capnograph slightly over-estimated the $ETCO_2$ and slightly underestimated the arterial $PCO_2$ in human subjects spontaneously breathing air in a hyperbaric environment at 284 kPa. The inaccuracies were clinically insignificant and if these findings were replicable in patients ventilated with 100% oxygen, they would establish the EMMA capnograph as suitable for monitoring ventilated patients during hyperbaric oxygen treatment.

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# Within-diver variability in venous gas emboli (VGE) following repeated dives

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## Abstract

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**Introduction:** Venous gas emboli (VGE) are widely used as a surrogate endpoint instead of decompression sickness (DCS) in studies of decompression procedures. Peak post-dive VGE grades vary widely following repeated identical dives but little is known about how much of the variability in VGE grades is proportioned between-diver and within-diver.
**Methods:** A retrospective analysis of 834 man-dives on six dive profiles with post-dive VGE measurements was conducted under controlled laboratory conditions. Among these data, 151 divers did repeated dives on the same profile on two to nine occasions separated by at least one week (total of 693 man-dives). Data were analysed for between- and within-diver variability in peak post-dive VGE grades using mixed-effect models with diver as the random variable and associated intraclass correlation coefficients.
**Results:** Most divers produced a wide range of VGE grades after repeated dives on the same profile. The intraclass correlation coefficient (repeatability) was 0.33 indicating that 33% of the variability in VGE grades is between-diver variability; correspondingly, 67% of variability in VGE grades is within-diver variability. DCS cases were associated with an individual diver's highest VGE grades and not with their lower VGE grades.
**Conclusions:** These data demonstrate large within-diver variability in VGE grades following repeated dives on the same dive profile and suggest there is substantial within-diver variability in susceptibility to DCS. Post-dive VGE grades are not useful for evaluating decompression practice for individual divers.

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## Introduction

Decompression sickness (DCS) is caused by intracorporeal bubble formation from supersaturated dissolved gas. Venous bubbles (venous gas emboli [VGE]) are easily detected by ultrasonic methods and their profusion graded on an ordinal scale. These are widely used as a surrogate endpoint instead of DCS in studies of decompression procedures, both because VGE occur commonly after diving whereas DCS is rare, and because VGE profusion is presumed to be correlated with an increased risk of bubbles forming at or impacting sites where they will cause DCS. Indeed, in large compilations of diving data with both DCS and VGE outcomes, cumulative incidence of DCS increases with increasing peak post-dive VGE grades.[1,2] However, there is no VGE grade that has both good sensitivity and specificity for DCS and peak post-dive VGE are highly variable following dives on the same dive profile (depth/time/breathing gas history).[2]

Despite these limitations, there are emerging trends toward interpreting VGE grades measured in an individual diver. Notably, divers can now purchase equipment used for self-monitoring of post-dive VGE and are using the result to provide feedback on modifying future decompression practice.[3,4] A future application of VGE measurements could be real-time physiological monitoring during diving for real-time control of decompression.[5] Validity of these emerging and potential applications of individual VGE measurements relies on an understanding of the within-diver variability in VGE grades as well as the association of VGE grades to DCS in individual divers. However, little is known about how much of the variability in post-dive VGE grades is proportioned between-diver and within-diver.[6,7]

The current study is a retrospective examination of within-diver variability of VGE grades. The U.S. Navy Experimental Diving Unit (NEDU) has previously published results of several large trials in which several dive profiles were each dived many times under controlled laboratory conditions. In these trials, the same divers often repeated the same dive profile on multiple occasions, separated by at least one week, and VGE were measured after each repeated dive. These data were analysed for within-diver variability in peak post-dive VGE grades.

## Methods

The data analysed in this paper were collected during four dive trials approved by the NEDU Institutional Review Board.[8–12] Informed consent for those trials included consent for de-identified data to be used for future research. Six dive profiles were tested in these four dive trials (see DIVE PROFILES section). Diver-subjects dived these dive profiles one or more times. For the present report, repeated dives are more than one dive by the same diver on the same one of these six dive profiles. Divers refrained from any hyperbaric or hypobaric exposure for two or three days before and after each experimental dive, and in practice, repeated dives were typically at least one week apart. Full details of the dive trials are available in the original reports and only relevant details are summarised here.[8–12]

All diving occurred in the NEDU Ocean Simulation Facility hyperbaric chamber and wet pot complex. Diving depth was simulated by pressurising the chamber complex with air, and pressure was controlled to approximately 0.5 feet of sea water (fsw, 1 fsw = 3.0643 kPa) accuracy. Chamber atmosphere and wet pot water temperature were actively controlled. Temperatures were sampled at ≥ 0.5 Hz and the means for each dive were computed. Descents and ascents, bottom times, and decompression stop times were followed to within a few seconds of the prescribed dive schedule.

### VGE MONITORING

After surfacing from a dive, divers spent 10 minutes adjacent to the chamber before being escorted to a climate-controlled laboratory where they generally remained seated for the remainder of a two-hour observation period. For each VGE examination, the diver reclined in the left decubitus position while the heart chambers were imaged (apical long-axis four-chamber view) with transthoracic two-dimensional (2-D) echocardiography. Generally, the same ultrasound equipment and imaging mode was used by the same trained cardiovascular technician for all 2-D echocardiographic imaging for each dive profile. With repeated diving, the ultrasound operator and the divers themselves became familiar with the best window for obtaining a 4-chamber apical view in each diver.

Venous gas emboli in the right heart chambers were graded according to one of two ordinals scales shown in Table 1.[2,13,14] The same scale was used throughout all testing of a dive profile, but to aid comparison between dive profiles for the present analysis, modified Eftedal-Brubakk grades 3 and 4a and grades 4b and 5 were collapsed to single grades approximately equivalent to NEDU grades 3 and 4 respectively. At each examination, VGE were graded three times: after the diver had been at rest on the examination table for approximately one minute and then after three forceful limb flexions around the right elbow and the right knee to elicit a bubble shower. For the movement conditions, the grade assigned was the highest signal sustained for at least four cardiac cycles for grades 1–3 or for about 0.5 s for higher grades. Grades (NEDU scale) were assigned at the time of measurement by the same author (DJD). Modified Eftedal-Brubakk grades were also assigned by either of the present authors, however inter-rater reliability is high for the Eftedal-Brubakk scale,[13] and the two authors routinely graded ultrasound images together to maintain concordance. For each man-dive, the peak grade of all resting examinations and the peak grade of all resting and limb flexion examinations were analysed; for compactness

**Table 1**
Venous gas emboli grading scales

| Grade | Modified Eftedal-Brubakk | Grade | NEDU |
|---|---|---|---|
| 0 | No bubbles | 0 | No bubbles |
| 1 | Occasional bubbles | 1 | Rare (fewer than 1/s) bubbles |
| 2 | ≥ 1 bubble / 4 heart cycles | 2 | Several discrete bubbles visible |
| 3 | ≥ 1 bubble / heart cycle | | |
| 4a | ≥ 1 bubble / $cm^2$ in all frames | 3 | Multiple bubbles/cycle, not obscuring image |
| 4b | ≥ 3 bubble / $cm^2$ in all frames | 4 | Bubbles dominate image, may blur chamber outlines |
| 5 | Whiteout, individual bubbles cannot be discerned | | |

these are hereafter denoted as 'resting' or 'movement' VGE grades respectively.

DIVE PROFILES

Two dive profiles were air decompression dives to 170 fsw, 52 metres of seawater (msw) (622 kPa) for 30 minutes bottom time.[8] Divers were immersed throughout the dives and the mean water temperatures ranged from 29.5°C to 30.8°C. Divers performed approximately 135 W of continuous work on an electrical-hysteresis-braked cycle ergometer during the time at bottom. Divers rested in a seated position during decompression. Examinations for VGE were at around 30 minutes and two hours post-dive. Imaging was undertaken using a Siemens Medical Solutions Acuson Cypress Portable Colorflow Ultrasound System with a 2.5 MHz cardiac probe. The NEDU scale was used for grading VGE. The two dive profiles each had 174 minutes of total decompression stop times but differed in the distribution of time among stop depths. One dive profile had a traditional distribution of stop time and resulted in three DCS cases in 192 man-dives (3/192); 38 divers performed a total of 159 repeated dives on this dive profile. The other dive profile had a 'deep stops' distribution of stop time and resulted in 10 DCS cases in 198 man-dives (10/198); 49 divers performed a total of 172 repeated dives on this dive profile.

Two dive profiles were nitrogen-oxygen dives to 113 fsw (34.4 msw, 448 kPa; 132 fsw [40.2 msw] equivalent air depth) for 155 minutes time at bottom. Divers were at rest and dry throughout the dive and the mean chamber atmosphere temperatures ranged from 20.9°C to 26.7°C. Examinations for VGE were at around 29, 66, and 103 minutes post-dive. The two dive profiles each had 251 minutes of oxygen decompression stops but differed in the total oxygen time and air break time. One dive profile had 30-minute oxygen periods followed by six-minute air breaks and resulted in two DCS cases in 96 man-dives (2/96); 24 divers performed a total of 69 repeated dives on this dive profile. The other dive profile had either 12-minute oxygen periods followed by six-minute air breaks or 24-minute oxygen periods followed by 12-minute air breaks (same totals of oxygen and air break times) and resulted in 8 DCS cases in 136 man-dives (8/136). These slightly different air break schedules had similar probability of DCS and were considered equivalent and treated as one dive profile; 34 divers performed a total of 119 repeated dives on this dive profile. There were some variations in the VGE monitoring during this dive trial. Examinations were made by four different ultrasound operators. For the first 45 man-dives, VGE were detected using the same equipment described in the preceding paragraph after which this machine was replaced with a Sonosite M-Turbo ultrasound with a p21 5-1 MHz cardiac probe and VGE were detected using harmonic imaging. Fourteen divers had VGE measurements from different ultrasound machines on repeated dives. For the first 81 man-dives VGE were graded according to the original Eftedal and Brubbak scale with the 4a and 4b grades collapsed into a single grade 4. Remaining dives were graded with the modified Eftedal-Brubakk scale. The use of both the original and modified scales had trivial effect on overall within-diver variability: potentially increasing VGE grade variability between repeated dives for five diver-profile groups and decreasing variability for two diver-profile groups.

One dive profile was an air decompression dive to 132 fsw (40.2 msw, 506 kPa) for 20 minutes bottom time with a 9-minute decompression stop at 20 fsw (6.1 msw, 163 kPa).[11,12] Divers were immersed throughout the dive and the mean water temperatures ranged from 29.8°C to 29.9°C. Divers performed approximately 75 W of continuous work on an electrical-hysteresis-braked cycle ergometer during the time at bottom. Divers rested in a seated position during decompression. Examinations for VGE commenced at approximately 15 minutes after surfacing and continued at 20-minute intervals throughout the two-hour post-dive period. In this study and the one described in the next paragraph, VGE were imaged using a GE LOGIQ e R7 with a 3SC-R7 1.7–4.0 MHz phased array cardiac probe and tissue harmonic imaging. Venous gas emboli were graded using the modified Eftedal-Brubakk scale. This dive profile resulted in no DCS cases in 96 man-dives (0/96); 32 divers performed a total of 71 repeated dives on this dive profile.

One set of dives were 5–8 hour duration, closed-circuit rebreather dives.[10] This was a test of six decompression schedules that were computed with the same decompression algorithm, and this algorithm was designed to produce schedules with the same probability of DCS. Consequently, these six schedules were treated as the same dive profile for the present analysis. Dives ranged from 160 fsw (48.8 msw, 592 kPa ) to 200 fsw (60.9 msw, 714 kPa) for bottom times of 82 to 150 minutes. Divers were immersed throughout the bottom time and initial ascent and were breathing 1.3 atm $PO_2$ helium-oxygen. Divers performed weightlifting and treadmill work during the time at bottom. Decompression stops were in the dry with divers seated at rest and breathing 1.3 atm $PO_2$ nitrogen-oxygen with air breaks. The mean water temperatures ranged from 27.0°C to 27.5°C and the mean chamber air temperatures ranged from 24.0°C to 25.4°C. Examinations for VGE commenced 20 minutes after surfacing and continued at 30-minute intervals throughout the two-hour post dive period. Venous gas emboli were graded using the modified Eftedal-Brubakk scale. These dives resulted in one DCS case in 120 man-dives (1/120); 28 divers performed a total of 103 repeated dives on these schedules.

VGE VARIABILITY ASSESSMENT

In total, 244 divers performed the 838 man-dives resulting in 24 DCS cases. All these data were used to rank the profiles according to cumulative incidence of DCS. Venous gas emboli grades were not available for four man-dives: two cases of DCS and one other medical incident onset before VGE measurements, and VGE grades were lost for one man-dive. The remaining 834 dives will be referred to as the pooled data. In the pooled data there were 141 single dives (i.e., divers only performed one dive on a dive profile) and there were 151 divers who performed 202 groups of repeated dives (total of 693 repeated man-dives). There are fewer divers than diver-profile groups because some divers performed repeated dives on two dive profiles and are therefore represented by two diver-profile groups.

Ordinal logistic mixed-effect models of the form:

$$log\left(\frac{P(y_{ij}\leq k)}{P(y_{ij}>k)}\right) = \beta_0 + \beta_1 x + \alpha_i + \varepsilon_{ij}$$

Equation 1

were fit to the VGE grades in the pooled data where $y_{ij}$ is the VGE grade for the $i^{th}$ individual on the $j^{th}$ occasion, $k = 0,1,2,3$, $\beta_0$ and $\beta_1$ are the fixed effects (population intercept and dive profile), *x* indicates the dive profile, $\alpha_i$ is the random effect (diver) assumed to have a normal distribution with a mean of zero and variance $\sigma_\alpha^2$, and $\varepsilon_{ij}$ is a random error assumed to have the standard logistic distribution with a mean of zero and a variance $\sigma_\varepsilon^2 = \pi^2/3$.[15] Variability in VGE grades between diver-profile groups was assessed using the intraclass correlation coefficient calculated from the residual variances of the models as:[15]

$$\frac{\sigma_\alpha^2}{\sigma_\alpha^2 + \sigma_\varepsilon^2}$$

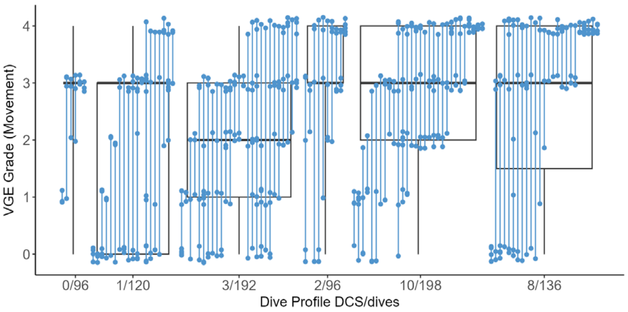
Equation 2

where $\sigma_\alpha^2$ were the group variances taken from the model output. Variability in VGE grades within diver-profile groups was one minus the intraclass correlation coefficient. Models were fit to the VGE grades using the ordinal package (v 2019.12-10. Christensen, RHB. Ordinal – regression models for ordinal data. 2022. URL: https://CRAN.R-project.org/package=ordinal) in R (v 4.2.2. R Core Team. R: A language and environment for statistical computing. Vienna, Austria R: Foundation for Statistical Computing; 2022. URL: https://www.R-project.org/).

Two analyses were performed to assess the impact of treating equivalent decompression schedules as the same in two of the dive profiles. The intraclass correlation coefficient was calculated for a variant of the pooled data, in which the two equivalent air break schedules in the 8/136 dive profile were separated. This approach was unsuitable for the 1/120 dive profile because there were relatively few repeated dives in each of the six equivalent schedules; instead, the intraclass correlation coefficient was calculated for a subset of the pooled data that excluded the 1/120 dive profile.

**Figure 1**

Venous gas emboli (VGE) grades for the six dive profiles; Y-axis is peak post-dive movement VGE grade. Dive profiles are identified on the x-axis by the number of DCS and number of all man-dives as given in the methods. Box and whisker plots show the median, interquartile range, and range of VGE grades for the pooled data (*n* = 834). The corresponding subsets of three or more repeated dives (*n* = 517) are illustrated with blue points and lines. Blue points are VGE grades for individual man-dives. Points are jittered vertically (random shift of up to ± 0.15 grade) to reduce overlap of points of the same grade. Blue lines connect VGE grades for repeated dives by the same diver



## Results

Figure 1 shows the movement VGE grades for the six dive profiles. The dive profiles are shown from left to right in increasing order of DCS cumulative incidence for all man-dives (pooled data plus the four missing VGE grades). The box and whisker plots show the median, interquartile range, and range of VGE grades for the pooled data. Although there is large variability in peak VGE grades following the same dive profile, there is a general shift of interquartile range to higher grades with increasing DCS cumulative incidence. Vertical blue lines connect groups of VGE grades for repeated dives by the same diver and the blue points illustrate the individual movement VGE grades for a subset of three or more repeated dives (single dives and two repeated dives are excluded to reduce clutter). Post-dive VGE for repeated dives by the same diver are highly variable.

Figure 2 shows individual movement VGE grades for a similar subset of dives as in Figure 1. In addition to three or more repeated dives, Figure 2 includes the five diver-profile groups of two repeated dives in which DCS occurred. In Figure 2, diver-profile groups of repeated dives are ordered by maximum then minimum VGE grade within the group, irrespective of the dive profile. This ordering clusters together diver-profile groups of similar variability. In this subset, 21 divers (22% of divers, 17% of diver-profile groups) had the same VGE grade after repeated dives. Of these consistent bubblers, the majority are divers who routinely produced grade 4 VGE, and this may be partly a ceiling effect since this is the highest discernable grade.

**Figure 2**

Movement VGE grades and DCS for three or more repeated dives and the five diver-profile groups of two repeated dives in which DCS occurred ($n = 527$). The Y-axis is peak post-dive movement VGE grade. Points are VGE grades for individual man-dives (points are jittered vertically – random shift of up to ± 0.15 grade to reduce overlap of points of the same grade). Blue points are VGE grades for dives that did not result in DCS; red points are VGE grades for dives that resulted in DCS. Blue lines connect VGE grades for repeated dives. The 122 diver-profile groups are ordered along the x-axis by increasing VGE grades

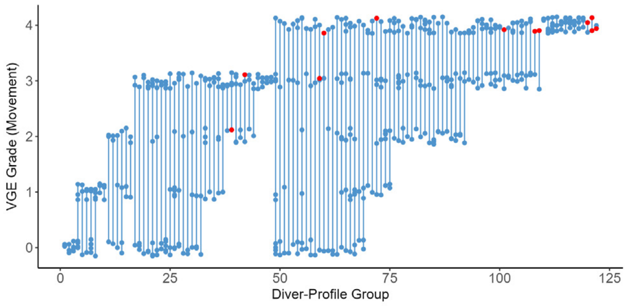
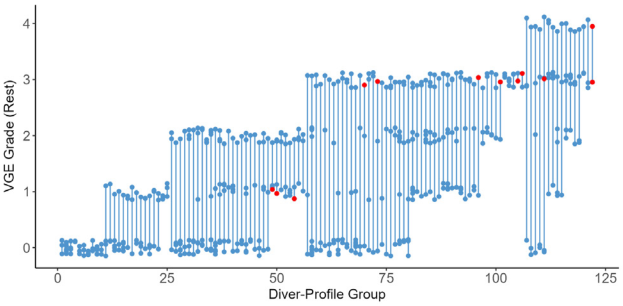


**Figure 3**

Resting VGE grades and DCS for three or more repeated dives and the five diver-profile groups of two repeated dives in which DCS occurred ($n = 527$). The Y-axis is peak post-dive resting VGE grade. Points are VGE grades for individual man-dives (points are jittered vertically – a random shift of up to ± 0.15 grade to reduce overlap of points of the same grade). Blue points are VGE grades for dives that did not result in DCS; red points are VGE grades for dives that resulted in DCS. Blue lines connect VGE grades for repeated dives. The 122 diver-profile groups are ordered along the x-axis by increasing VGE grades



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There were fewer consistent bubblers (2–4 divers) at any of the lower movement VGE grades. Figure 3 shows the resting VGE grades for the same subset of repeated dives as in Figure 2; there are few consistent bubblers, but the majority of these are divers who routinely produced no resting VGE.

For movement VGE grades in the pooled data set, intraclass correlation calculated from the ordinal logistic model was 0.33. The pooled intraclass correlation indicates that 33% of the variability in VGE grades is between-diver variability; correspondingly, 67% of variability in VGE grades is within-diver variability. For the resting VGE grades in the pooled data set, intraclass correlation for the grades was 0.37. For movement VGE grades in the pooled data, separating the two equivalent air break schedules in the 8/136 dive profile or leaving them combined resulted in no difference in intraclass correlation coefficient (0.33 in both cases). For movement VGE grades in the subset of the pooled data excluding the 1/120 dive profile, the intraclass correlation coefficient was 0.30, slightly lower than for the pooled data set. These results indicate that combining the equivalent schedules into single dive profiles did not increase within-diver variability in VGE grades in the pooled data.

Figure 2 shows the movement VGE grade in red for those repeated dives that resulted in DCS. There are only 12 cases DCS in this subset of repeated dives; nevertheless, it is striking that DCS was not confined to divers who routinely produce high bubble grades. Instead, DCS mostly occurred in divers with variability in VGE grades, but occurred in association with diver's highest or second highest VGE grade. Eleven of these DCS cases manifested as joint pain in the knees or shoulders. One diver had pruritic, mottled skin rash in association with grade 4 VGE after both repeated dives. Figure 3 shows three of the 12 DCS cases are associated with a diver's lowest peak post-dive resting VGE grade after repeated dives. This apparently degraded association of DCS cases with resting VGE grade compared with movement VGE grade is interesting but is not significant ($\chi^2$ test of proportions of DCS cases associated with maximum VGE grade, $P = 0.816$).

## Discussion

The large variability in pooled peak VGE grades following identical dive profiles has been previously reported.[2,16] However, the present study is the first to show that this variability is principally due to within-diver variability in VGE grades. This within-diver variability in VGE grades is not attributable to differences in monitoring techniques because measurements were typically done with the same equipment, by the same ultrasound operator, and graded by the same investigator. The high within-diver variability in VGE grades occurs despite no practical variation in the diving and post-dive VGE monitoring period (dive profile, work, thermal status). Therefore, this variability in VGE grades must be caused by variability in some intrinsic host factor or pre-dive environmental factor that was not, or possibly cannot be, controlled.

The correlation of cumulative incidence of DCS with peak VGE grades has previously been reported for pooled data.[1,2] The present study is the first to indicate that DCS is associated with an individual diver's highest VGE grades and not with their lower VGE grades after repeated dives. It is noteworthy that most DCS were joint pain. Whereas some manifestations of DCS are thought to result from VGE or right-left shunt of VGE, DCS joint pain is thought to result from bubbles in the tissues.[17,18] The association of DCS joint pain with a diver's VGE grades suggests an individual diver's risk of DCS, irrespective of the pathophysiology, varies with VGE grades. The large variability in peak VGE grades after repeated dives might be interpreted as evidence of substantial day-to-day (within-diver) variability in DCS susceptibility.

The most obvious implication of the present findings is that monitoring of VGE following uncontrolled field dives is not useful for evaluating and recommending decompression practice for individual divers. Since an individual diver manifests widely varying peak VGE grades following carefully controlled repeated dives that are identical for all practical purposes, different VGE grades following successive uncontrolled field dives cannot be attributed to differences in decompression practice. The present findings do suggest that an individual diver's greatest risk of DCS coincides with high post-dive VGE grade, but this information has limited operational application. Post-dive intervention to mitigate the risk of DCS (such as surface oxygen breathing or recompression) implemented because of high VGE grade would usually be wasted because even the highest VGE grade has low positive predictive value for DCS (5–13%).[1,2] Moreover, by the time peak post-dive VGE occur it may often be too late to intervene to prevent DCS: about half of DCS cases onset by the time VGE grades typically peak following long bounce dives.[19–21]

On the other hand, the apparent association of risk of DCS with an individual's post-dive VGE grades is promising for individualized control of decompression, because it suggests VGE detected *during* decompression may also be usefully associated with risk of DCS. Early laboratory chamber experiments with animals show VGE can be detected during decompression, and VGE numbers altered by changing the decompression profile.[22] These observations suggest VGE could be a target for real-time control during decompression if VGE detected during decompression could be shown to be reliably associated with risk of DCS. However, practical methods for evaluating VGE in real-time during actual diving are yet to be developed.

When designing and analysing experimental decompression trials using DCS as the endpoint we have previously interpreted our own observation of within-diver variability in DCS outcomes after repeated dives, along with similar observations during altitude exposures,[23] as evidence of day-to-day (within-subject) variability in DCS susceptibility.[10,11] We have used this evidence to justify relaxing the typical definition of statistical independence and considered the experimental unit as the man-dive and not the subject. This is expedient because it is impractical to conduct hundreds of man-dives without repeated use of the same volunteers. The large within-diver variability in peak VGE grades and the possible association with variability in DCS susceptibility further supports the use of the man-dive as the experimental unit in studies with DCS as the endpoint. There are similar implications for studies that use peak VGE grade as the primary endpoint. Such studies are frequently designed as paired comparison of subjects[16] and the large within-diver variability in VGE grade suggest that this design is not necessarily better than an unpaired design for interventions to the dive profile, work, and thermal status.

The present data have the limitation of being assembled from dive trials not designed for the present retrospective analysis. As identified in the methods, for some dive profiles VGE examinations were less frequent than current recommendations.[14,19] Also, the number of DCS cases after repeated dives was small so must be interpreted cautiously. However, a principal strength is analysis of a VGE data set of a size that is unlikely to ever be produced for a prospective study of variability in VGE grades.

## Conclusions

These data demonstrate large within-diver variability in peak VGE grades following repeated dives on the same dive profile and suggest there is substantial within-diver variability in susceptibility to DCS. The well-known association of DCS with VGE grade in pooled data, and the low positive predictive value of that association, are apparent in individual divers. Post-dive VGE grades are not useful for evaluating decompression practice for individual divers.

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# Short communication

## Pulmonary oxygen toxicity breath markers after heliox diving to 81 metres

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### Abstract

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Pulmonary oxygen toxicity (POT), an adverse reaction to an elevated partial pressure of oxygen in the lungs, can develop as a result of prolonged hyperbaric hyperoxic conditions. Initially starting with tracheal discomfort, it results in pulmonary symptoms and ultimately lung fibrosis. Previous studies identified several volatile organic compounds (VOCs) in exhaled breath indicative of POT after various wet and dry hyperbaric hypoxic exposures, predominantly in laboratory settings. This study examined VOCs after exposures to 81 metres of seawater by three navy divers during operational heliox diving. Univariate testing did not yield significant results. However, targeted multivariate analysis of POT-associated VOCs identified significant ($P = 0.004$) changes of dodecane, tetradecane, octane, methylcyclohexane, and butyl acetate during the 4 h post-dive sampling period. No airway symptoms or discomfort were reported. This study demonstrates that breath sampling can be performed in the field, and VOCs indicative of oxygen toxicity are exhaled without clinical symptoms of POT, strengthening the belief that POT develops on a subclinical-to-symptomatic spectrum. However, this study was performed during an actual diving operation and therefore various confounders were introduced, which were excluded in previous laboratory studies. Future studies could focus on optimising sampling protocols for field use to ensure uniformity and reproducibility, and on establishing dose-response relationships.

### Introduction

Pulmonary oxygen toxicity (POT) is a significant risk in oxygen diving and prolonged hyperbaric oxygen treatment, and has damaging effects on the alveolar epithelium caused by reactive oxygen species. First described in 1899, it may lead to destruction of cellular membrane lipid bilayers and DNA damage in nuclei.[1,2] This initiates immune responses, apoptosis, and subsequent tracheobronchitis characterised by coughing and retrosternal discomfort.[3,4] In more severe cases, acute respiratory distress syndrome can develop, and prolonged exposure may result in pulmonary fibrosis.[3,4] A partial pressure of oxygen ($PO_2$) of ≥ 51 kPa (0.5 atmospheres absolute [atm abs]) can cause POT after several hours of continuous exposure, and symptoms tend to manifest more rapidly with higher $PO_2$ levels.[5] Special operations forces using rebreathers with 100% oxygen are susceptible to POT during long-range diving missions, which can last for several hours. Deep diving missions performed by mine-clearing divers also pose risks due to increased $PO_2$ levels in gas mixtures. To quantify hyperoxic exposure and stratify the risk of POT, the 'units of pulmonary toxic dose' (UPTD) measure has been developed (1 UPTD = 1 minute breathing 100% oxygen at 1 atm abs). Limits are based on the reduction in pulmonary vital capacity following excessive exposure to oxygen during hyperbaric chamber dives.[6] Currently, 615 UPTD is considered the maximum exposure for a single dive, corresponding with a 2% reduction in vital capacity in 50% of the population.[4]

The Royal Netherlands Navy has investigated volatile organic compounds (VOCs) in exhaled breath to identify potential biomarkers of POT.[7–12] These laboratory studies conducted after shallow hyperoxic in-water dives and hyperbaric oxygen treatment exposures identified predominantly increased levels of alkanes and aldehydes. The current study aimed to collect breath samples after operational deep 16/84 heliox dives (16% oxygen and 84% helium gas mixture) to investigate if changes in VOCs under these specific diving circumstances align with the compounds identified in laboratory dives.

## Methods

Three divers of the Royal Netherlands Navy made heliox dives to 81 metres of seawater (msw; equal to 816 kPa) in a Norwegian fjord using surface oxygen decompression. The dive profile was based on the Defence R&D Canada (DRDC) Helium-Oxygen Diving Table 8, using nitrox 32.5% instead of air as decompression gas[13] (Figure 1). The total dive and decompression time was 62 min, with calculated oxidative stress equal to 119 UPTD. The participating divers were all healthy, non-smoking volunteers and fit-for-diving according to the Netherlands Ministry of Defense's fitness requirements for diving. Informed consent was obtained before sampling. The sampling protocol was approved by the Medical Ethical Committee of the Amsterdam Academic Medical Center (ref. W18_424 # 21.083) and, together with the gas chromatography-mass spectrometry (GC-MS) analysis, was identical to those used in previous studies.[7,8,10,11] After diving and during sampling, the subjects were questioned whether they had experienced any pulmonary or other physical symptoms.

Four breath samples were collected per subject per dive: one pre-dive sample just before submerging, and at 30 min, 2 h, and 4 h after completing the surface decompression. In short, the subjects breathed for 5 min through an inspiratory VOC filter (Honeywell, Charlotte, NC, USA) to minimise environmental contamination. Thereafter, the subjects exhaled into a non-elastic balloon (Globos Nordic, Naestved, Denmark), from which 500 mL air was pumped (Gastec, Kanagawa, Japan) through a sampling tube (Tenax GR 60/80; Camsco, Houston, TX, USA). After sampling, the tubes were stored for several days at ~8°C until GC-MS analysis (GC-MS QP2010; Shimadzu, Japan, TD100; Markes, Sacramento, CA, USA) was performed in the laboratory of Amsterdam UMC. All divers were required to have a surface interval of at least 24 h between dives to minimise inert gas build-up and reduce the chance of decompression sickness. To minimise contamination from food, the subjects had a daily uniform diet and were not allowed to eat or drink within 1 h before sampling, except for water. The samples were analysed using GC-MS and compounds were identified using the NIST library.[14]

### STATISTICAL ANALYSIS

The data were statistically processed using R statistical software (v4.1.2; R Core team 2021) together with R-packages pROC (v1.18.0), Skillings.Mack (v1.10), and MixOmics (v6.18.1). Wilcoxon signed-rank and Skillings-Mack tests were conducted to select and identify untargeted ion fragments that varied significantly and to test fluctuations of individual molecules over time. Targeted sparse partial least squares discriminant analysis (sPLSDA) modeling for two components and Kruskal-Wallis rank sum testing were employed to identify and test previously discovered VOCs linked to POT from the VAPOR library.[15]

## Results

Twenty-seven exhaled breath samples were collected in eight dives. Complete series of four samples were collected in five dives, while two dives lacked the 4 h post-dive measurement (Figure 2). One dive consisted only of the pre-dive sample and thus was discarded. No symptoms of POT were reported by the subjects.

To determine if the consecutive dive days influenced the measurements, partial least squares discriminant analysis was performed of the sampling series. This demonstrated no individual dependencies of the sampling series between successive days. Thus, consecutive days of diving for a single diver could be statistically regarded as independent dives instead of repetitive dives.

Untargeted analysis did not yield significant findings between pre-dive and post-dive measurements. However, sPLSDA modeling based on previously identified VOCs of interest from the VAPOR library[15] found a significant ($P = 0.004$) change of component 1, consisting of dodecane, tetradecane, octane, methylcyclohexane, and butyl acetate (Table 1). When plotted according to the sampling timepoint, component 1 demonstrated a rise of sPLSDA values from pre-dive to 2 h post-dive, followed by normalisation of the signal intensity at 4 h post-dive (Figure 3).

## Discussion

To our knowledge, this is the first study to analyse exhaled VOCs after an operational heliox dive to 81 msw followed by surface oxygen decompression. As can be seen in Figure 1, the largest oxygen exposure in diving using surface decompression procedures comes from breathing 100% oxygen in the hyperbaric chamber during the surface phase. The surface decompression results in 80 UPTD, whereas the in-water stage is 32 UPTD. However, the effect of submersion during an in-water dive on POT is not fully understood and the UPTD was developed using dry hyperbaric exposures; thus, it could over- or underestimate in-water hyperoxic stresses. Nevertheless, it seems reasonable to assume that the surface decompression had the largest impact on the lungs.

A recent study on deep heliox dives with surface decompression showed a temporary decrease in spirometric parameters after diving to 80 msw, normalising within 24

**Figure 1**

Dive profile of the 81 msw heliox 16/84 (16% oxygen, 84% helium) dive, followed in-water decompression with enriched air nitrox (EANx) containing 32.5% oxygen) and 100% oxygen, and surface oxygen decompression to 121 kPa in a hyperbaric chamber

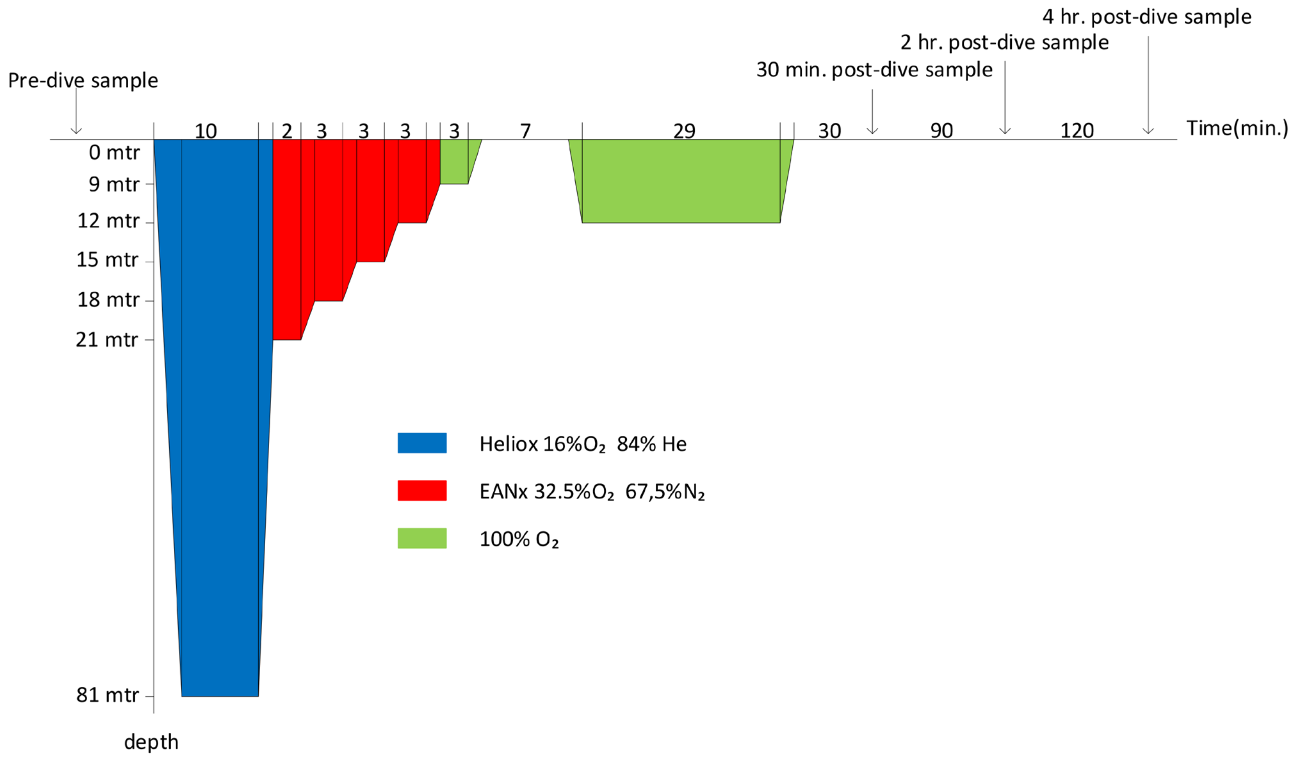


**Figure 2**

Overview of the sampling results

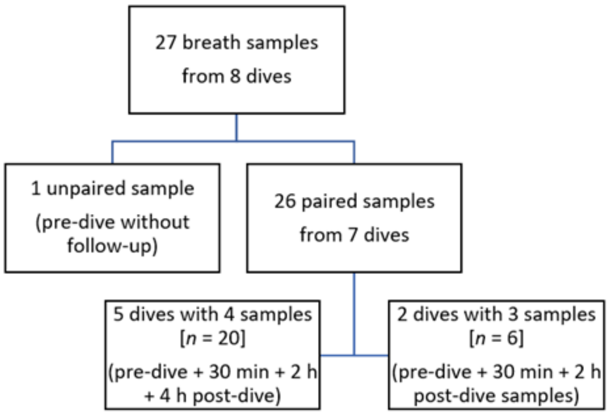


**Table 1**

Compounds identified by targeted analysis of known POT-associated VOCs from the VAPOR library;[15] CAS-no. – Chemical Abstract Services identification number[16]

| Compound | CAS-no. | Molecular weight | Ref |
|---|---|---|---|
| Dodecane | 112-40-3 | 170 | [6,7,9] |
| Tetradecane | 629-59-4 | 198 | [6,7,9] |
| Octane | 111-65-9 | 114 | [6,7,9] |
| Methylcyclohexane | 108-87-2 | 98 | [6,7,9–11] |
| Butyl acetate | 123-86-4 | 116 | [6,7,9,10] |

hours.[17] Although spirometry and exhaled breath analysis results showed little correlation in our previous studies, we feel it is important to note that these two modalities both show a transient response to deep helium diving and a full recovery afterwards.

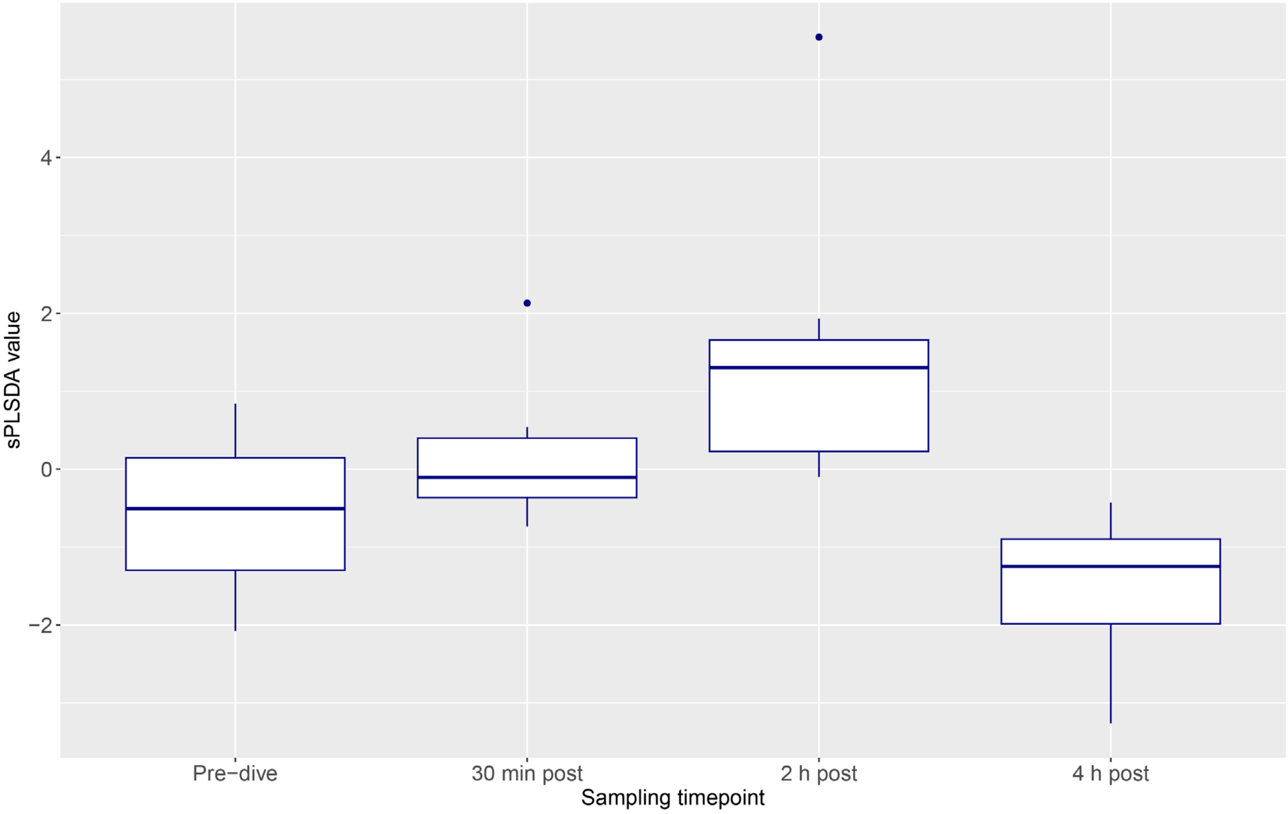
Similar to previous studies from our group, the change in VOC intensity was greatest in the 2 h post-dive samples, followed by a drop in intensity at the last measurement. However, this was only ascertained by targeted multivariate analysis of previously identified compounds from the VAPOR library, while untargeted testing was performed after previous hyperbaric exposures in laboratory settings.

## LIMITATIONS

This was a field study in an operational setting and thus has several limitations. Available resources and time management options were restricted. The full series of four samples per dive could not be collected in all cases due to

**Figure 3**

The sparse partial least squares discriminant analysis (sPLSDA) values at each sampling timepoint for component 1, consisting of the POT-associated VOCs dodecane, tetradecane, octane, methylcyclohexane, and butyl acetate; the boxes summarize the central tendency (median) and spread (interquartile range) of the sPLSDA values, the whiskers extend to values within 1.5 times the interquartile range, with two individual outliers at the second and third sampling points. Kruskal-Wallis Chi$^2$ = 13.317, df = 3, $P$ = 0.004



last-minute changes in plans and activities that had priority over this study, such as surface tending the next dive sortie. Another limitation is the small number of participating subjects. Due to operational dive team restrictions and smoking habits, only three divers were included in this study.

The dives were carried out by two divers per sortie, with subsequent dives starting after the surface decompression procedure of the previous pair of divers was completed. Consequently, samples were not collected at the same time of day for all divers, as in previous laboratory dives; one series of samples was collected in the morning and the next series was collected in the afternoon. This may have influenced VOC intensities because they fluctuate during the day.[18] This variability also applies to food ingestion. Although no food was consumed within 1 h before sampling, the morning divers had lunch in the 1 h food window between the third and fourth sampling timepoints, whereas the afternoon divers had lunch before the pre-dive sampling. Therefore, minimal influences of food cannot be excluded.

The small sample size ($n$ = 26) and relatively short hyperoxic exposures (62 min) with a relatively low (119) UPTD may explain why the VOC changes were smaller compared to previous studies. Additionally, the exact role of helium in a hyperbaric environment is not fully understood, but several studies suggest that helium plays a protective role at the cellular level.[19,20]

## Conclusions

The findings of this study indicate a limited and reversible reaction to hyperoxia occurs after deep heliox diving using surface oxygen decompression, but no signs of prolonged pulmonary damage were observed. This strengthens the theorem that POT develops sub-clinically before first symptoms are experienced. This study also demonstrates that it is feasible to capture VOCs in operational settings for further analysis. Further studies should focus on optimising sampling protocols for field use to ensure uniformity and reproducibility, and on establishing dose-response relationships of POT biomarkers in breath after various hyperbaric hyperoxic exposures.

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# Case reports

## Cerebral arterial gas embolism (CAGE) during open water scuba certification training whilst practising a controlled emergency swimming ascent

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**Abstract**

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We report the case of a 23-year-old male novice diver who sustained cerebral arterial gas embolism (CAGE) during his open water certification training whilst practising a free ascent as part of the course. He developed immediate but transient neurological symptoms that had resolved on arrival to hospital. Radiological imaging of his chest showed small bilateral pneumothoraces, pneumopericardium and pneumomediastinum. In view of this he was treated with high flow normobaric oxygen rather than recompression, because of the risk of development of tension pneumothorax upon chamber decompression. There was no relapse of his neurological symptoms with this regimen. The utility and safety of free ascent training for recreational divers is discussed, as is whether a pneumothorax should be vented prior to recompression, as well as return to diving following pulmonary barotrauma.

### Introduction

We report the case of a 23-year-old male novice diver who sustained a cerebral arterial gas embolism (CAGE) during his open water certification training whilst practising an emergency swimming ascent as part of his course.

### Case report

Written informed consent was obtained from the patient for presentation of the details of his clinical history and de-identified imaging.

The adult male diver had a history of childhood asthma for which he had not used any bronchodilator inhaler since about seven years of age and was otherwise well. His other hobbies included flying, without previous issues.

He had two unremarkable pool training dives the week before his CAGE episode during personal tuition by a certified diving instructor of a recognised training agency.

The incident dive was at a well-known recreational shore dive located approximately 30 kilometres south of Fiona Stanley Hospital (FSH), near Perth, Western Australia (WA). Fiona Stanley is the state referral centre for diving and hyperbaric medicine in WA.

He reported surface swimming from the shore, then descending to a depth of about six metres of sea water (msw) to practise emergency swimming ascent training with his instructor. The instructor demonstrated ascending to the surface with the regulator out of his mouth, breathing out and then exhaling into his buoyancy control device (BCD) upon reaching the surface. The diver then repeated this but upon reaching the surface noticed blackness of his vision and then briefly lost consciousness after exhaling his remaining breath into his BCD. His BCD was further inflated by the dive instructor who towed him back to shore. At that time the diver stated that he could not see but only hear his instructor speaking. He reported that he was unable to speak and that he had right-sided weakness with ‘pins and needles’ sensation. He also described sharp left-sided pleuritic chest pain and mild shortness of breath that had started to improve prior to arrival of the ambulance, as well as mild headache.

On arrival of the ambulance, some 20 minutes later, his weakness and altered sensation had resolved. Paramedics noted that he was mildly confused and vague with a Glasgow Coma Score (GCS) of 14 which rapidly improved to GCS 15 with oxygen ($O_2$). His vital signs were documented as peripheral oxygen saturation 92% on air, respiratory rate 16 breaths·min$^{-1}$, blood pressure 125/69 mmHg and heart rate 104 beats·min$^{-1}$ regular. An electrocardiogram (ECG) performed by paramedics on the scene apparently showed ST elevation and hence aspirin 300 mg was given, as per ambulance protocol. On arrival to the emergency department some 30 minutes later, the diver reported that he was asymptomatic except for mild left-sided pleuritic chest pain. A repeat ECG was normal.

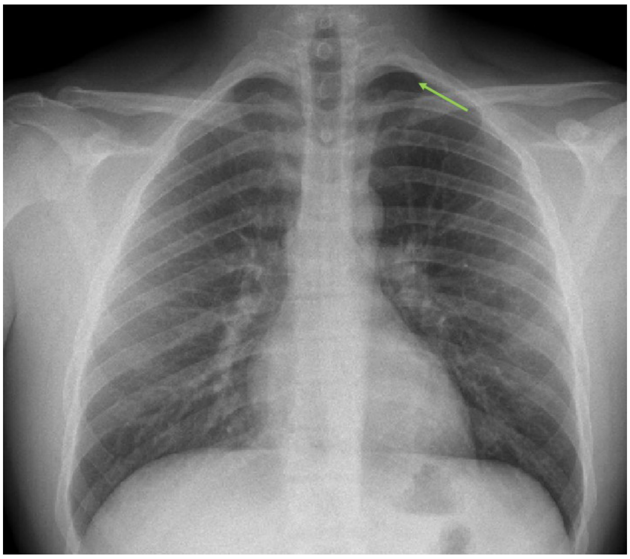
A chest X-ray (CXR) was performed soon after arrival was reported as showing "*features suspicious for a left apical pneumothorax measuring up to 11.7 mm in depth. Equivocal appearance of the right lung apex may be projectional or represent a further pneumothorax*" (Figure 1). The FSH Hyperbaric Medicine Unit (HMU) was contacted and reviewed the CXR prior to it being reported, with a similar interpretation but the possibility of an associated pneumopericardium was also noted. Our advice was to immediately obtain high resolution chest computed tomography (HRCT) and then send the patient directly to the HMU while continuing high flow oxygen via non-rebreather reservoir mask. The first author went to the CT scanner with the patient and interpreted the images in real time with the radiologist.

The HRCT was reported as showing "*bilateral pneumothoraces measuring 7.2 mm in maximal depth on the right and 4.7 mm on the left. There is soft tissue surgical emphysema seen within the pericardium and throughout the mediastinum extending along the paraesophageal, paratracheal and parabronchial regions*" (Figures 2 and 3). Interestingly, the CT scan showed a larger pneumothorax on the right side compared to the left which was poorly visible if at all on the CXR.

On examination in the HMU he had normal vital signs and was alert with GCS 15. He had coherent and fluent speech, without evidence of dysphonia. Cranial nerve and limb motor and sensory function were normal, and reflexes were symmetrically brisk. Coordination via finger-nose testing was normal. The patient was kept supine and hence gait was not tested. Cardiovascular examination was unremarkable; of note Hamman's crunch was not heard on cardiac auscultation and subcutaneous emphysema was not evident.

In view of the presence of small bilateral pneumothoraces and given that the patient was asymptomatic with a normal neurological exam, the decision as to whether or not to recompress him (with United States Navy Treatment Table 6 (USN TT6)) was discussed by three senior hyperbaric physicians. The invasive nature of inserting bilateral chest drains was considered, as was the risk of a tension pneumothorax during the decompression phases of the table should the pneumothorax(es) continue to expand without placement of chest drains. It was noted that untreated pneumothorax is listed as an absolute contraindication to hyperbaric oxygen treatment (HBOT) in many references (see **Discussion**) although, theoretically, if there is no longer a gas leak into the pleural space, HBOT should accelerate resolution of a pneumothorax,[1] as it does for surgical emphysema and pneumomediastinum.

**Figure 1**
Chest radiograph showing small left apical pneumothorax (green arrow); the right apical pneumothorax is occult



Based on the principle of 'first do no harm', it was decided to observe the patient overnight with frequent neurologic observations and continuous vital signs monitoring while continuing high flow $O_2$ (via a non-rebreather mask) with a plan to insert bilateral chest drains and proceed to USN TT6 should neurological symptoms recur.

Upon review the next morning, he remained well. Cardiorespiratory and neurological examinations were normal. He was able to perform a Sharpened Romberg test to 60 seconds on the third attempt. Bedside ultrasound scanning showed no evidence of either pneumothorax or pneumopericardium. The patient was discharged with planned follow-up in the HMU in four weeks with advice to rest and not dive or fly until then.

Upon review at four weeks, he reported no problems except for extreme tiredness; sleeping up to 12 hours nightly instead of his usual seven hours. He had recently attended the emergency department of the adjacent private hospital for this where a repeat chest HRCT was performed. This was reported as being entirely normal.

He was advised not to scuba dive until he was reviewed again at six months post event, during which time formal pulmonary function would be performed and he had time to consider whether he wished to continue diving. Pulmonary function testing performed two months post event was

**Figure 2**

Coronal chest computed tomography (CT) slice showing bilateral apical pneumothoraces (green arrows) and pneumomediastinum (red arrow)

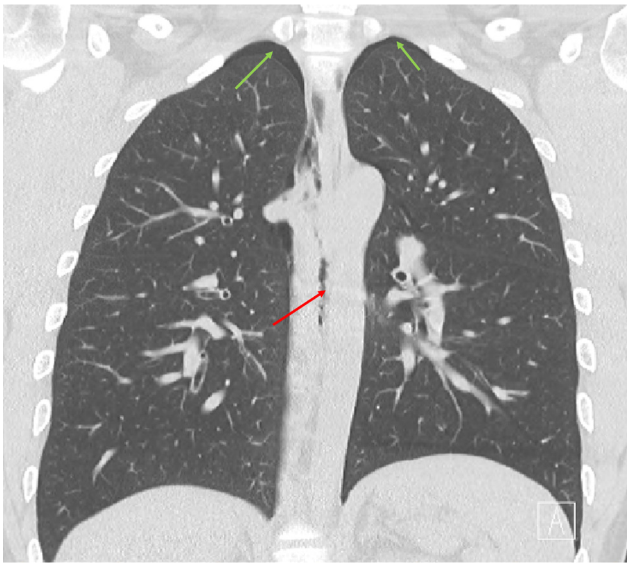
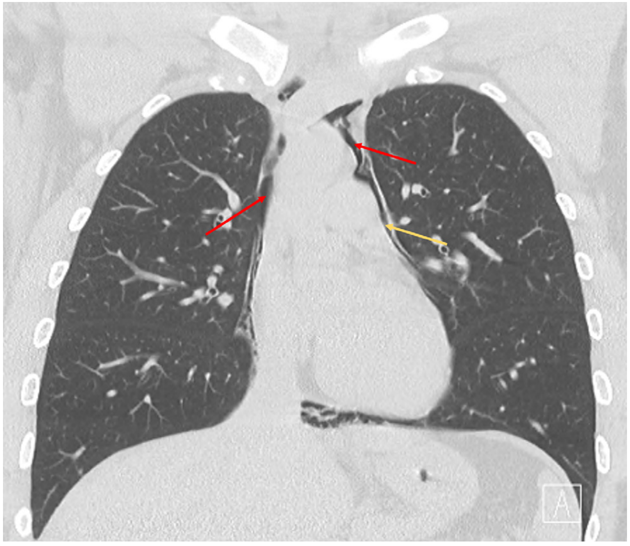


**Figure 3**

Coronal chest computed tomography (CT) slice showing pneumopericardium (yellow arrow) and pneumomediastinum (red arrows)



normal with no evidence of small airways disease or gas trapping.

## Discussion

This case highlights several issues. The first being the utility and safety of controlled emergency swimming ascent training for recreational divers.

Controlled emergency swimming ascent (CESA) training and 'buddy breathing' were introduced in the early days of diving when out-of-air events were commonplace, reportedly contributing to half of scuba fatalities in Australia and New Zealand in the 1980s.[2] Since then, pressure gauges have become ubiquitous, training more focused and breathing gas exhaustion less common.[3,4] However, some occasionally still find themselves out of gas and distant from a buddy and have no alternative but to free ascend with the associated risk of pulmonary barotrauma (PBt), arterial gas embolism, hypoxic blackout and/or drowning.

Previous data from the Professional Association of Diving Instructors (PADI) revealed that from 1989 to 1992, there were four cases of CAGE or PBt in a calculated 1,251,568 CESAs, an incidence of 0.31 per 100,000 ascents.[5] Periodically, there is discussion about whether there is sufficient benefit in continuing to teach a training practice that can potentially cause harm. For example, a report from Belgium found that in a cohort of 34 cases of PBt treated at a particular hospital from 1995 to 2005, 16 had occurred during emergency ascent training.[6] As a result, the Belgium Underwater Federation discontinued this training and a subsequent review revealed a substantially decreased incidence of PBt.[7]

More current Australian data from PADI indicate that from 2001 to July 2023, there was one reported case (the present case) of PBt or CAGE associated with ascent training during approximately 550,000 open water certifications (each including at least one CESA) (D Dwyer, personal communication, 2023 July 13). Although there might be some shortcomings in reporting, it is obvious that the risk of such an incident is very low.

Some certification agencies have abandoned teaching free ascent, others teach it as a simulation in a horizontal orientation, while others require divers to demonstrate the procedure under strict guidelines and control. PADI requires that open water diver students perform a CESA from a depth of 6 to 9 m in open water. The instructor maintains control through contact with both a secure ascent line and the candidate. The latter keeps their regulator in their mouth, looks upwards and continuously exhales throughout the ascent while maintaining contact with their BCD deflator to control ascent, the rate of which should not exceed 18 m.min$^{-1}$. The student is advised to resume normal breathing if they stop the ascent or have any difficulty. At the surface, they orally inflate their BCD or drop their weights.[8] Scuba Schools International (SSI) training standards mandate that students perform an emergency ascent in a pool or open water with a maximum depth of 9 m.[9]

In this case, other than the free ascent itself, which was reportedly not overly fast, there appears to be no obvious precipitating or risk factors. Although the diver did not have his demand valve in his mouth as required, he believes that he did not aspirate any water and, despite feeling a bit low on breath over the last couple of metres, was not aware of breath-holding, blew bubbles throughout (confirmed by the instructor) and had sufficient residual air on surfacing to exhale some into his BCD.

The second issue is how best to manage a diver who has confirmed CAGE with neurological symptoms that have resolved, but who has a pneumothorax. Recompression is the recommended treatment for CAGE, even where symptoms have resolved, because of the tendency of patients to deteriorate after apparent recovery.[10,11]

Pneumothorax is uncommon in diving, occurring in fewer than 10% of cases of PBt.[12] Recompression without drainage of a pneumothorax is often characterised as contraindicated, particularly in monoplace chambers,[1] although recommendations vary as shown below.

The widely accepted practice is: "*...a diver with pneumothorax should always have a chest drain inserted before any air evacuation, or before recompression if there is another problem such as CAGE or DCS that justifies recompression in the presence of a pneumothorax*".[13] However, Neuman, in Bove and Davis' Diving Medicine textbook states: "*Theoretically, recompression and then subsequent decompression can convert a simple pneumothorax to a tension pneumothorax; however, this has not been reported as a frequent problem, and standard therapy is appropriate in such cases*".[14]

Moon, in Hyperbaric Oxygen Therapy Indications, states: "*In patients with AGE caused by pulmonary barotrauma there may be a coexisting pneumothorax, which could develop into tension pneumothorax during chamber decompression. Therefore, placement of a chest tube in patients with pneumothorax prior to* $HBO_2$ *should be considered and is recommended for patients treated in a monoplace chamber. For multiplace chamber treatment, careful monitoring is a feasible option*".[10] He provides similar advice elsewhere.[11]

In Neuman and Thom's Physiology and Medicine of Hyperbaric Oxygen Therapy, it is advised that "*Although an untreated pneumothorax is almost universally considered an absolute contraindication to hyperbaric treatment, if a practitioner is so equipped, a tension pneumothorax can be treated by simple venting within a multiplace chamber*".[15]

In the US Navy Diving Manual Revision 7 it is advised: "*Divers recompressed for treatment of arterial gas embolism or decompression sickness, who also have a pneumothorax, will experience relief upon recompression. A chest tube or other device with a one-way relief valve may need to be inserted at depth to prevent expansion of the trapped gas during subsequent ascent. A tension pneumothorax should always be suspected if the diver's condition deteriorates rapidly during ascent, especially if the symptoms are respiratory. If a tension pneumothorax is found, recompress to depth of relief until the thoracic cavity can be properly vented. Pneumothorax, if present in combination with arterial gas embolism or decompression sickness, should not prevent immediate recompression therapy. However, a pneumothorax may need to be vented as described before ascent from treatment depth. In cases of tension pneumothorax, this procedure may be lifesaving*".[16]

The relatively recent availability of the expertise and equipment to perform in-chamber ultrasound allows detection of a pneumothorax under pressure, rather than other causes of deterioration, and hence confirms the need for thoracostomy.[17]

Other cases of CAGE with PBt have been reported where an expectant approach was decided and recompression withheld, after balancing the risk of recurrent CAGE upon decompression, in divers with neurological symptoms that had resolved.[18]

From the above, it would seem prudent to drain a pneumothorax prior to recompression unless there is equipment and expertise to diagnose a pneumothorax in chamber and perform tube thoracostomy where required. This option was considered by three senior hyperbaric physicians versus normobaric $O_2$ and careful observation for deterioration, which was the eventual management. Certainly, if this patient did not have pneumothoraces, they would have been recompressed with USN TT6. A similar conservative approach for the management of a CAGE case in a remote area with difficult and delayed access to recompression could also be considered.

A third issue is return to diving after PBt / CAGE. This has evolved from the traditional absolute contraindication to further scuba diving, to informed decision making by the diver in consultation with a diving medicine specialist, occasionally with input from a respiratory medicine specialist.[13] This risk assessment approach may not be unreasonable, particularly if HRCT and pulmonary function testing after recovery are normal, and a suitable period of time has elapsed to allow tissue healing. The evidence for the question 'when?' returning to dive after a PBT is lacking, and it can only be inferred from other recommendations for other conditions. The European Diving Technology Committee imposes an absolute contraindication for diving during the first three months after any pneumothorax. Return to diving could be considered in some situations, after 3 months, if pulmonary function testing is normal, with a $FEV_1/FVC$ at least 0.7. The same is suggested for returning to dive after thoracic surgery.[19]

The medical manual of the U.S. Navy, in its "*Examination and Standards*", differentiates, for disqualifying purposes, spontaneous from traumatic pneumothoraces. Spontaneous events are an absolute contra-indication for diving duties, while traumatic (but non-barotrauma) related events could be accepted, even for a diver candidate, after a recovery period of six months, with favourable lung function tests, thoracic radiology and expert opinion. For diving related PBt (i.e., pneumothorax, mediastinal or subcutaneous emphysema, or CAGE), a diver candidate will be excluded, but a "*designated diver*" may receive a waiver to return to duties after a 30-day period if predisposing respiratory conditions are excluded.[20]

From the above it is evident that there is a different approach in the case of a spontaneous compared to traumatic pneumothorax, the main difference being related to the risk of recurrence based on an underlying lung condition. Even though the risk of recurrence of a spontaneous pneumothorax is highest in the first two years, incidences as high as 18% were reported more than six years after the initial episode.[21] Reasonably, traumatic pneumothorax is not considered such a problem as it is associated with low rates of recurrence.

The Undersea and Hyperbaric Medical Society (UHMS) in its Diving Medical Guidance to the Physician and the British Thoracic Society have a similar recommendation of ceasing diving after a spontaneous pneumothorax, unless there are no lung parenchymal abnormalities on HRCT and the diver underwent bilateral surgical correction.[21,22] However, a clear discussion should take place with the diver as even after surgery the risk of another episode could be as high as 10%.[21] A recent systematic review on returning to diving following a first episode of primary spontaneous pneumothorax concluded that *"Given that a pneumothorax at depth can result in a fatality, the current practice of generally advising against further diving is probably sound"*.[23]

Returning to dive after an episode of CAGE is more consensual, with commercial and military standards allowing it provided the diver is symptom free.[19,20] The US Navy Medical Manual also requires a normal magnetic resonance imaging scan of the brain performed within seven days of the event.[20] For our case, considering that no abnormalities were found on HCRT and pulmonary function testing, return to diving after a period of six months seemed a reasonable approach, with a low risk of recurrence.

## Conclusions

We report a novice diver without apparent predisposing factors or significant violation of training procedures who developed PBt with bilateral pneumothoraces and CAGE during CESA training. The utility and safety of such training for recreational divers is debateable. Patients exhibiting complete spontaneous recovery after CAGE are typically recompressed but prolonged surface oxygen and careful observation for relapse is an option if there are obvious associated risks such as in the present case with bilateral pneumothoraces.

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# Hyperbaric oxygen treatment in delayed post-hypoxic encephalopathy following inhalation of liquefied petroleum gas: a case report

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## Abstract

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Delayed post-hypoxic encephalopathy can occur after an episode of anoxia or hypoxia. Symptoms include apathy, confusion, and neurological deficits. We describe a 47-year-old male patient who inhaled gas from a kitchen stove liquid petroleum gas cylinder. He was diagnosed with hypoxic ischaemic encephalopathy 12 hours after his emergency department admission. He received six sessions of hyperbaric oxygen treatment (HBOT) and was discharged in a healthy state after six days. Fifteen days later, he experienced weakness, loss of appetite, forgetfulness, depression, balance problems, and inability to perform self-care. One week later, he developed urinary and fecal incontinence and was diagnosed with post-hypoxic encephalopathy. After 45 days from the onset of symptoms, he was referred to the Underwater and Hyperbaric Medicine Department for HBOT. The patient exhibited poor self-care and slow speech rate, as well as ataxic gait and dysdiadochokinesia. Hyperbaric oxygen was administered for twenty-four sessions, which significantly improved the patient's neurological status with only hypoesthesia in the left hand remaining at the end of treatment. Hyperbaric oxygen has been reported as successful in treating some cases of delayed neurological sequelae following CO intoxication. It is possible that HBO therapy may also be effective in delayed post-hypoxic encephalopathy from other causes. This may be achieved through mechanisms such as transfer of functional mitochondria to the injury site, remyelination of damaged neurons, angiogenesis and neurogenesis, production of anti-inflammatory cytokines, and balancing of inflammatory and anti-inflammatory cytokines.

## Introduction

Delayed post-hypoxic encephalopathy has been mostly associated with carbon monoxide poisoning, but it has also been reported in other patients. The clinical presentation is characterised by apathy, confusion, agitation, or progressive neurological deficits developing after an initial period of apparent recovery following a brief episode of anoxia or hypoxia.[1–4]

There is currently no pharmacological treatment with proven efficacy for delayed post-hypoxic encephalopathy. It can progress rapidly and render the patient a dependent individual, posing a great challenge for clinicians both in terms of diagnosis and treatment planning. Reported cases of delayed neurologic sequelae occurring after liquified petrolium gas (LPG) inhalation are limited.[5] We describe a case of delayed neurological sequelae (DNS) after LPG poisoning treated with hyperbaric oxygen (HBO).

## Case report

The patient provided written consent for his case and MRI images to be reported.

A 47-year-old male patient attempted suicide by inhaling gas from the kitchen stove cylinder. He was found unconscious and vomiting after approximately 10 hours of gas exposure, and was taken to the emergency department. He was unconscious, and was intubated. Initial arterial blood gas test results (breathing air) showed a $PaO_2$ of 10.1 kPa (76 mmHg), pH 7.42, and $PaCO_2$ of 4.0 kPa (30 mmHg). The patient had decreased respiratory sounds and diffuse rales. He was diagnosed with hypoxic ischaemic encephalopathy and twelve hours after his admission, he received HBO (242.3 kPa for 120 minutes) followed by five further identical treatments once daily. At the time of discharge, he was in a good general condition, his consciousness was clear, but he still complained of numbness in his left hand.

Fifteen days after discharge, the patient started experiencing weakness, loss of appetite, forgetfulness, depression, balance problems, and inability to perform self-care. He was admitted to the psychiatry clinic in another medical centre. One week after admission he developed urinary and faecal incontinence. The patient was diagnosed with delayed post-hypoxic encephalopathy by the consulting neurologist. The cranial magnetic resonance imaging (MRI) findings were evaluated as consistent with this diagnosis. The patient was referred to the Underwater and Hyperbaric Medicine Department by the neurologist for HBO treatment as a delayed neuropsychiatric sequel of LPG intoxication. The time elapsed between the onset of symptoms and the referral to our clinic was approximately 45 days.

On evaluation it was observed that his self-care was poor. An apathetic facial expression was observed. The content and fluency of speech were normal, but the speech rate was significantly slow. Cranial nerve examination and global muscle strength and tone were normal. However, there was a broad-based ataxic gait, bilateral dysdiadochokinesia, and dysmetria. Hypoaesthesia was observed in the distal left upper extremity. Deep tendon reflexes were normal. The minimental state examination (MMSE) test revealed decrements in attention and executive functions, and the overall score was 11/30. The patient still had urinary and fecal incontinence.

In the initial cranial MR images, in the fluid attenuation inversion recovery (FLAIR) and T2 weighted (T2W) series, there were widespread, symmetrical hyperintensities with a tendency to coalesce in the vertex, corona radiata, centrum semiovale and the periventricular white matter. There was restricted diffusion in these areas on the diffusion-weighted imaging series. Symmetrical hypointensities were observed in the bilateral basal ganglia, more prominent in the globus pallidus (Figure 1).

We explained to the patient and his family that delayed post-hypoxic encephalopathy after LPG inhalation is not an accepted indication for HBO treatment and that there has been no scientific study about this subject. Nevertheless, given the resemblence of the underlying mechanism to carbon monoxide (CO) intoxication, it was possible that HBO treatment may have beneficial effects. After the approval of the patient and his family, a total of twenty-four

**Figure 1**

Bilateral lesions on FLAIR (fluid attenuation inversion recovery), T2W (T2 weighted) and DWI (diffusion weighted imaging) sequences at the level of basal ganglia

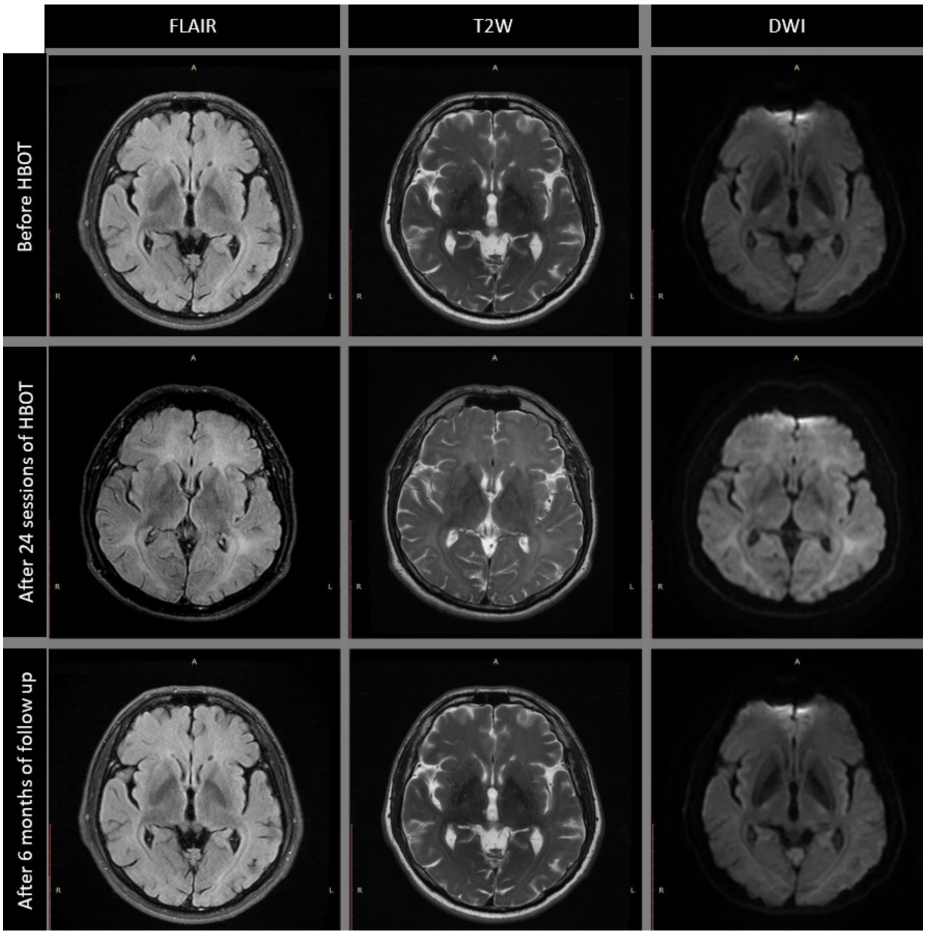
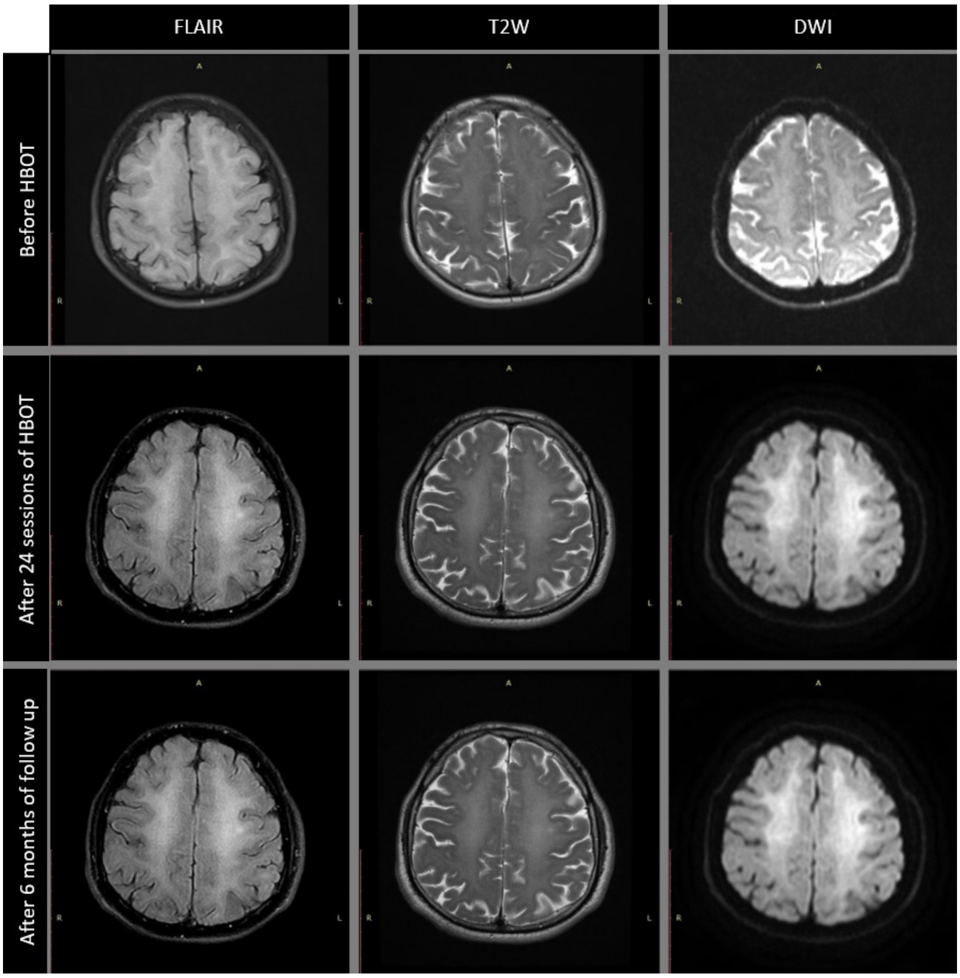


**Figure 2**

Bilateral lesions on FLAIR (fluid attenuation inversion recovery), T2W (T2 weighted) and DWI (diffusion weighted imaging) sequences at the level of the centrum semiovale



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HBO treatments were administered once daily, 120 minutes at 242.3 kPa, five days a week. The patient did not receive concurrent medical treatment during these treatments. After the 10th session, the bilateral dysdidiokinesia, dysmetria and ataxic gait were completely resolved, a significant improvement was observed in the MMSE (29/30). However, it was also noted that there was still hypoaesthesia in the distal part of the left upper extremity, and the patient continued with HBO treatment.

At the end of the twenty-fourth session, the patient once again scored 29/30 on the MMSE and showed no change in the hypoaesthesia in the left hand. He was able to perform self-care. On the MR images taken after 24 HBO treatments, the previously described signal changes had regressed (Figure 2). The patient's HBO treatment was discontinued as his symptoms and depressive mood had improved. After six months, the MRI findings had completely returned to normal (Figure 2). During a phone interview after five years follow-up, the patient stated that he continues with daily life activities without any problem. He has no health problems but the numbness in his left hand still persists.

## Discussion

This patient was initially admitted to the psychiatry service with a new onset of depressive mood, loss of balance, and dementia fifteen days after LPG poisoning and initial recovery following HBO treatment. He was evaluated by neurology because of the onset of urinary and fecal incontinence. The patient was diagnosed with late neuropsychiatric sequelae based on the MRI findings by the consulting neurologist. After twenty-four sessions of HBO treatment the MMSE score was 29/30 and the clinical symptoms completely resolved. No other treatment was given concomitantly.

Liquified petroleum gas is a flammable hydrocarbon gas mixture used as a fuel with propane as the main component and it additionally contains isobutane.These substances are lipophilic, so after inhalation and absorption into the bloodstream from the lungs, they distribute in high concentrations in lipid-rich tissues, especially the brain.[6] Following a case of Parkinsonism due to LPG inhalation it was suggested that LPG may have a direct toxic effect on the brain by creating histotoxic hypoxia similar to CO intoxication.[5] It was stated that ataxia, dystonia, bradykinesia, widespread plastic rigidity and dysarthria continued after one year follow up in the patient who was not treated with HBO.[5] Ours is the only reported case of apparently delayed neurological pathology (hypoxic-ischaemic encephalopathy) following LPG inhalation. Unlike the other case, HBO treatment was administered and the symptoms completely regressed, with only hypoaesthesia remaining in the left hand.

The clinical course and MRI abnormalities in our patient can be associated with brain injury caused by histotoxic hypoxia, as well as being similar to DNS cases associated with CO poisoning. The brain regions affected by CO toxicity include the basal ganglia, especially the globus pallidus, substantia nigra, and hippocampus. These are areas with high metabolic rate and high oxygen demand. Laminar necrosis of the cerebellar cortex and Purkinje cell loss are frequently seen in CO intoxication.[7] Similar MRI findings were found in our case as shown in Figures 1 and 2. It has been reported that HBO treatment has sometimes been associated with positive results in patients with DNS associated with CO poisoning.[8,9]

Studies have suggested that HBO can transfer functional mitochondria to an injured area and reduce inflammation by decreasing cytokines that cross the blood-brain barrier, which play a role in secondary cell damage mechanisms.[10] After the development of DNS, HBO treatment may have aided recovery by transferring functional mitochondria to the injury site, re-myelination of damaged neurons, angiogenesis and neurogenesis, production of anti-inflammatory cytokines, and balancing of inflammatory and anti-inflammatory cytokines.[11–13]

## Conclusions

In the present case, we believe that LPG, which is considered to have low neurotoxicity, may have caused direct toxic damage to the brain through a histotoxic hypoxia mechanism, similar to CO poisoning. Hyperbaric oxygen treatment has been reported to have positive results in treating DNS following CO intoxication in some cases, and it is possible that HBO therapy may also be effective in delayed post-hypoxic encephalopathy related to LPG poisoning. There is a need for better understanding of the pathophysiology of DNS in these settings and for further investigation of potential benefit from HBO treatment.

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# Hypoxic loss of consciousness in air diving: two cases of mixtures made hypoxic by oxidation of the scuba diving cylinder

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## Abstract

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Without an adequate supply of oxygen from the scuba apparatus, humans would not be able to dive. The air normally contained in a scuba tank is dry and free of toxic gases. The presence of liquid in the tank can cause corrosion and change the composition of the gas mixture. Various chemical reactions consume oxygen, making the mixture hypoxic. We report two cases of internal corrosion of a scuba cylinder rendering the respired gas profoundly hypoxic and causing immediate hypoxic loss of consciousness in divers.

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## Introduction

There are multiple factors that could cause loss of consciousness (LOC) while diving. However, in most cases of LOC, the cause remains unknown. A wide variety of reasons can be mentioned, but many such events probably result from cardiac causes (such as myocardial ischaemia, arrythmias, pulmonary edema, vasovagal syncope) with immersion and exercise as likely provocation factors. However, when LOC occurs at the surface or early during descent the question arises as to whether it was coincidental or had a causality related to the gas mixture breathed by the diver. Death due to drowning is a major risk if unconsciousness occurs underwater.

Loss of consciousness while diving is rare. Causes other than cardiac events (mentioned above) are hypoxia, toxic gas effects and arterial gas embolism. Use of rebreathers is commonly described.[1] Hypoxic accidents are also well known in the breath-hold diving community.[2,3] Immediate LOC after breathing gas from a the scuba cylinder suggests possible inappropriate composition. Loss of consciousness caused by hypoxia due to corrosion of the tank was first (and only in our opinion) described in 2008 after a diving fatality.[4] Such cases are rare.

In these two reports we describe immediate LOC after breathing air on open-circuit scuba. Case 1 is older and it was not possible to obtain patient consent for publication. The patient in case 2 consented to publication of the case history.

## Case 1

The patients were a middle age adult male who was an experienced diver and his early adolescent son, also a diver. Both had no medical history.

While testing new diving equipment (a buoyancy control jacket) in his swimming-pool, the father suggested his son try it with his own scuba cylinder. The cylinder gas pressure was 60 bar, which was considered enough for the test. After a few breaths from the regulator, the son suddenly lost consciousness. He was immersed with the head above the water. After removal of the regulator he quickly regained consciousness without any resuscitation measures; he had no ongoing symptoms. Then, the father, partially immersed in the pool, decided to test the new jacket. He also lost consciousness in the minute that followed. As with his son, he regained consciousness after removal of the scuba regulator a few minutes later, without any resuscitation. The two divers were checked at the hospital. Medical assessment was normal. No complications were reported.

**Table 1**

Analysis of gas in the scuba cylinder case 1; ppm – parts per million; VOC – volatile organic compound

| Parameters | Measured values | Limit values | Measurement method |
|---|---|---|---|
| Oxygen | 0.9% | 20.5 to 21.5% | Electrochemical cell |
| Carbon dioxide | 5 ppm | 500 ppm | Infra red |
| Carbon monoxide | 21 ppm | 5 ppm | Infra red |
| Moisture | 1,283 $mg \cdot m^3$ | 100 $mg \cdot m^3$ | Hygrometry |
| VOCs | 607 $mg \cdot m^3$ | < 5 $mg \cdot m^3$ | Flame ionisation |
| Hydrogen | 0.5% | 0 | Gas chromatography |

Analysis of the gas contained in the tank was performed by a Navy specialised laboratory, LASEM (Laboratoire d'Analyse, de Surveillance et d'Expertise de la Marine), Toulon, France. Pressure in the cylinder was 25 bars and the oxygen level was almost zero. Hydrogen, carbon monoxide and volatile organic compounds were found in large quantities, outside acceptable tolerance limits. Moisture was also very elevated (Table 1).

After cutting the cylinder open, 300 ml of a brown and viscous liquid were recovered. Significant corrosion was covering the entire wall (Figure 1).

**Figure 1**

Internal condition of the scuba cylinder from case 1

## Case 2

The patient was a 51-year-old man; an experienced dive instructor (more than 1,000 dives). While preparing to dive he noted that the cylinder pressure was only 100 bars, not enough to dive as expected. So he changed the cylinder with the 'safety scuba tank' off the boat, which contained 140 bars. Immediately after initiating descent he remembered performing a Valsalva manoeuvre after which he lost consciousness. He was retrieved unconscious by another diver, from 14 msw, without the regulator in his mouth. His buddy performed a control lift to the surface where the rescued diver was not breathing and there was pink foam around his lips. The rescuer performed mouth-to-mouth resuscitation. The rescued diver regained consciousness, and breathed again. He was evacuated to a hyperbaric facility.

Initial assessment reported tachypnoea and crackling lung sounds, a facial barotrauma with a conjunctival hyperaemia, and bilateral barotrauma of the ears and sinuses; the patient was conscious with a complete amnesia of the event. Cerebral and chest computed tomography conducted in the following hour showed diffused sinus haemorrhage, right exophthalmos caused by orbital hemorrhage, and diffuse infiltrate of the alveolar and interstitial pulmonary tissue.

The cylinder gas was analysed by LASEM (Table 2). Cylinder pressure was 185 bar; initial pressure was about 140 bar and the tank has been refilled with air after the accident. Despite this, the oxygen content was only 8%. Hydrogen and volatile organic compounds were found in large quantities, outside usual tolerance limits. Moisture was also very elevated.

Before cutting the tank open, multiple gas analyses were repeated during eight weeks, and oxygen concentration was recorded. A progressive reduction in oxygen concentration was shown (Table 3). After cutting the tank open, 250 ml of a brown and viscous liquid (with a high salinity) was recovered. Significant corrosion covered the entire wall (Figure 2).

## Discussion

Aviators can experience hypoxia while flying (cockpit depressurisation, failure of supplemental oxygen systems). A variety of personal symptoms, referred to as the 'hypoxia signature' is detected by training in a hypobaric chamber among military aircrew. However, it seems very difficult for divers to detect suggestive symptoms of hypoxia in real time and perform self-rescue.[2] In these two cases, immediate loss of consciousness occurred without awareness.

**Table 2**

Analysis of gas in the scuba cylinder case 2; ppm – parts per million; VOC – volatile organic compound

| Parameters | Measured values | Limit values | Measurement method |
|---|---|---|---|
| Oxygen | 7.7% | 20.5 to 21.5% | Electrochemical cell |
| Carbon dioxide | < 2 ppm | 500 ppm | Infra red |
| Carbon monoxide | 1.7 ppm | 5 ppm | Infra red |
| Moisture | 248 mg·m$^3$ | 100 mg·m$^3$ | Hygrometry |
| VOCs | 31 mg·m$^3$ | < 5 mg·m$^3$ | Flame ionisation |
| Hydrogen | 1% | 0 | Gas chromatography |

**Figure 2**

Internal condition of the scuba cylinder from case 2

**Table 3**

Oxygen fractions in the scuba cylinder from case 2 over two months of sequential oxygen content analyses

| Analysis day | Oxygen fraction |
|---|---|
| 1 | 7.68% |
| 16 | 5.14% |
| 27 | 4.55% |
| 62 | 2.80% |

Medical assessments of these three victims were normal, eliminating any medical concerns, especially cardiac disability. Biochemical etiology, and more specifically hypoxia, was suspected, and confirmed by gas analysis of residual gas in the cylinders. Hypoxic gas is defined by an inspired $PO_2$ less than 0.16 bars; below 0.1 bar, loss of consciousness is likely. Underwater, drowning is the main risk.

Liquid and moisture were found in the two scuba tanks. This internal contamination is abnormal. Severe corrosion of the steel tank is caused by iron oxidation with water. Many chemical reactions likely occurred but some of them are prone to consume oxygen and progressively render the 'air' (initially containing 20.92% oxygen) hypoxic.

Oxygen in contact with iron, when water is present, can lead to oxidation by sequential reactions:

$$2H_2O + O_2 + 4e^- \rightarrow 4OH^-$$
$$Fe + 2OH^- \rightarrow Fe(OH)_2 + 2e^-$$
$$4Fe(OH)_2 + 2H_2O + O_2 \rightarrow 4Fe(OH)_3$$
$$2Fe(OH)_3 \rightarrow Fe_2O_3 + 3H_2O$$

The global reaction leading to iron oxide, also known as rust, is:

$$4\ Fe + 3O_2 + 6H_2O \rightarrow 4\ Fe(OH)_3$$

The process consumes oxygen so concentration eventually tends to zero. As described, the rate of oxygen consumption is high: within two months, the oxygen concentration decreased from 7.68% to 2.80% in the cylinder. Total lack of oxygen during the incident dive seems plausible.

The corrosion process may also include a redox reaction that forms iron oxide and hydrogen according to the chemical reaction:[5]

$$2Fe + 3H_2O \rightarrow Fe_2O_3 + 3H_2$$

Hydrogen was identified in the cylinders in both above cases. Based on the experience of LASEM, when hydrogen is present in a steel-made scuba bottle, without any history of intentional hydrogen use, it strongly suggests an internal corrosion process.

The presence of carbon monoxide (CO) in the cylinder in case one is unexplained. Carbon dioxide combined to hydrogen can produce CO and water ($CO_2 + H_2 \rightarrow CO + H_2O$). However, carbon dioxide levels in the cylinder should have been low. It is also possible the CO detected could also be a contaminant introduced during tank filling.

Multiple chemical reactions may occur in a scuba cylinder contaminated by liquids, but the most significant result for health is the generation of a hypoxic breathing gas. In these two cases, hypoxic gas was the trigger of unconsciousness. Luckily, both cases had a happy ending with the divers being rescued by their buddy. An advanced protocol of gas/diving material analysis, usually performed by our team, allows us to explain this situation and provide preventive advice: systematic opening and inspection after water ingress and, as far as possible, a pre-dive oxygen check with a low-cost analyser before diving in case of any doubt about the internal condition of a scuba cylinder.

## Conclusions

Consideration of the accident scenario plays an essential role in diving accident diagnosis. Immediate loss of consciousness without awareness at surface or during descent suggests a hypoxic breathing gas. To confirm this hypothesis, diving gas in the cylinder used by the victim must be analysed. The cylinder used by a buddy may constitute a useful reference.

In open-circuit scuba, regular maintenance and visual inspections of cylinders are key components of safe diving. Every cylinder must be carefully stored and maintained, especially those used intermittently such as safety tanks, to avoid a fatal outcome. Annual visual inspection is highly recommended.

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# Letter to the Editor

## University of Auckland Postgraduate Diploma in Diving and Hyperbaric Medicine

At the 2023 SPUMS Annual Scientific Meeting Professor David Smart presented an outline of current Australasian diving medicine education. This has prompted me to summarise an alternative that existed in the early 2000s that failed to establish permanently. In 2002, a Postgraduate Diploma in Medical Science – Diving and Hyperbaric Medicine (DHM) was established by Professor Des Gorman in the Department of Occupational Medicine, Auckland University Faculty of Medical and Health Sciences. This was a one-year-full-time or two-year-part-time distance learning programme which could be undertaken as a stand-alone diploma or incorporated into a Master's degree, for instance, in occupational medicine. I was appointed to deliver this course. The programme consisted of five papers which included an initial obligatory one on the physics and physiology of hyperbaric environments. In addition, candidates could choose which papers on diving medicine, hyperbaric medicine or a research project to undertake to meet the completion requirements of the course.

Each taught paper consisted of a series of weekly Power Point (PP) lectures as a pdf, combined with a background Word file, pdfs of key publications and other recommended reading. For some topics, there was a set of questions to answer with feedback provided. The university had limited face-to-face and videoed lecture capacity at that time, so regular interactive discussions were done largely by email Q&A. Each course was examined at the end of its semester. The project paper was assessed twice, the initial proposal and design and the final dissertation, by an external examiner and me. One of the elective papers had an experiential component at an approved hyperbaric medicine unit in Australasia.

About 16 doctors qualified, from NZ, Australia, the UK and Oman, whilst five did not complete the programme for various reasons. The course was recognised by the Australian and New Zealand College of Anaesthetists (ANZCA) for their Certificate in DHM as an alternative path to that of the SPUMS Diploma. Indeed, at the time Professor Gorman proposed the diploma's establishment, he envisaged that it would replace the SPUMS Diploma and provide a formal academic degree for DHM. The course was terminated by the university in 2007, along with most other medical postgraduate diplomas.

Several reasons contributed to this failure, but three in particular. Firstly, uptake was limited despite over 100 enquiries internationally. Whilst New Zealanders and Australians received government grants, candidates from other countries had to pay the full university fee as though they were 'in residence' and utilising all the facilities of the campus, which was a considerable sum. This put off the majority of international enquirers!

Secondly, after four years the course was reviewed by a professor of education from Auckland University of Technology and an academic DHM physician from Australia. They were only provided with the PP & Word files and not made aware of the numerous interactive components that had been used. Unsurprisingly, their comments were less than favourable! An important linked issue was that the university would only look at the numbers of Diploma students graduating. The number of these was small because most of the students were either doing the DHM papers as part of a Masters in Occupational Medicine or switched from a diploma-level degree to one of Master of Medical Science by dint of possessing a clinical post-graduate qualification such as a Fellowship of the ANZCA or the Australian College of Emergency Medicine (FACEM), etc. The assessing board refused to consider these students as course graduates so our output looked minimal!

Thirdly from my personal perspective, SPUMS failed to recognise the potential benefits of having a university degree in diving and hyperbaric medicine and, thus, as a professional organisation did not provided support, although individual members did so strongly as honorary faculty. In the long run, the ANZCA-based qualification appears to be providing the better option anyway, as described by David Smart in his presentation.

So what of the graduates? Six became hyperbaric medicine unit directors at some stage in their careers; another is regarded as one of the most experienced and respected physicians in primary diving medicine in Australasia, whilst readers of the *SPUMS Journal* and *Diving and Hyperbaric Medicine* have benefitted from the publication of articles based on five of the theses written for these degrees. For me, it was a wonderful academic experience and made me some good friends.

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# Notices and news

EUBS notices and news and all other society information can be found on:
http://www.eubs.org/

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## EUBS President's report

### Jean-Eric Blatteau

After two difficult years of the COVID-epidemic, we are now faced with numerous large-scale conflicts around the world. These conflicts are affecting the daily lives of many citizens. My thoughts are with all our colleagues who are enduring these difficult and painful situations.

Nevertheless, we were able to resume the normal course of our meetings at the international congresses in Prague and Porto, which were excellent in terms of organisation and thematic interest. I hope that in the years to come we'll be able to propose a joint congress with SPUMS, since the experience so far has been excellent.

While our discipline is regularly enriched by new clinical studies and basic research, there is a danger of parallel non-medical and purely profit-making activity, often using knowledge misappropriated from the field to justify itself. It is our duty as a learned society to strengthen the level of knowledge and evidence-based indications by proposing new consensus conferences, which I hope we will soon be able to do with the EUBS-ECHM, and also to fight against the development of backdoor practices by putting the society's official positions online, as we recently did on 'mild HBO'.

I wish you all the best for the festive season and hope to see you soon at the next EUBS in September 2024 in Brest, France.

*Jean-Eric Blatteau*
*EUBS President*

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The EUBS website is at
http://www.eubs.org/
Members are encouraged to log in and keep their personal details up to date.

The latest issues of *Diving and Hyperbaric Medicine* are via your society website login.

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## EUBS Notices and news

**EUBS 2024 Annual Scientific Meeting**

After the great success of the 47th Annual Scientific Meeting in Porto, EUBS will move to Brest (France) for its 48th meeting, which will take place from 16–20 September 2024. At the heart of a beautiful harbour, Brest is a city oriented towards the ocean. A military, commercial and yachting port with one of Europe's largest centres dedicated to the sea, the city has a centuries-old maritime tradition. The region has managed to preserve its beauty and authenticity, and its coastline, dotted with magnificent beaches and lighthouses, is well worth a visit.

We would be delighted to welcome you to this meeting and contribute to its success.

Keep monitoring the website https://eubs2024.sciencesconf.org/ for all the news and updates.

**EUBS Annual General Assembly**

Our annual EUBS General Assembly took place on 16 September 2023, in Porto, on the last day of the EUBS Annual Scientific Meeting. The report of the General Assembly, as well as the supporting documents (financial report, results of Executive Committee (ExCom) elections) are available for our members via the Members Area on the EUBS website.

It has been decided that the membership fees for the next year will be unchanged, but the price for the 'print option' for the DHM Journal will increase (after five years of stability) from €40 to €50 Euro, in response to both increased printing costs and a higher number of pages per issue. EUBS ExCom expresses their appreciation and thanks to our Corporate Members, as well as to our 10 'Affiliated Societies' – national scientific societies and organisations supporting and promoting EUBS among their members, who benefit from a 10% reduction in EUBS membership fee.

**EUBS Executive Committee**

To replace Oscar Camacho from Porto (Portugal), after serving a three-year term, we have elected a new Member-at-Large. There were three valuable candidates:

Pedro Coelho Barata (Portugal), Kurt Magri (Malta) and Michal Hajek (Czech Republic). Michal Hajek has been elected as Member-at-Large 2023. Because our 2022 M-a-L, Charles Azzopardi (Malta) has resigned from the ExCom, it was suggested that Oscar Camacho remain another year to complete the ExCom, effectively serving four years. This was accepted by the general assembly.

The ExCom wish to express their thanks to all candidates and Members-at-Large for their willingness to help our Society move forward and their contributions to the ExCom activities. The composition of the new ExCom can be found on the EUBS website, with contact information for each member.

**EUBS social media**

All EUBS members are reminded to bookmark and follow our Social Media channels:
Facebook: https://www.facebook.com/European-Underwater-and-Baromedical-Society-283981285037017/
Twitter: @eubsofficial
Instagram: @eubsofficial

While the "*EUBS website news*" email messages are a way to communicate important information directly to our EUBS members, Facebook, Twitter and Instagram will be used to keep also non-members updated and interested in our Society. The EUBS social media is managed by Bengusu Mirasoglu bengusu.mirasoglu@eubs.org.

**EUBS membership**

Do not forget to renew your EUBS membership and if your membership has expired, you will see a message when logging in on the EUBS website. You can then renew it online.

EUBS membership gives you significant advantages, such as immediate access to the most recent issues of the DHM journal, (if selected) a print copy of the e-journal for your convenience, reduced registration fee at our Annual Scientific Meeting (this alone already pays back your membership fee), reduced membership fees at selected Affiliate Societies, access to the GTUEM database of non-indexed scientific literature, searchable membership database, etc.

Members of Affiliate Societies benefit from a 10% discount on the EUBS membership fee. When applying for or renewing your membership, select your Affiliate Society from the drop-down list and the reduction in membership fee will be automatically applied.

If you have difficulties renewing or accessing your Membership Area, please contact us at secretary@eubs.org. Please do note that payment by PayPal is the easiest and also cheapest way to pay your membership fee. We hope to implement a way to make your membership renewal automatic, should you wish to do so – that way you will not have to remember to renew. This will be optional, not mandatory, and only with PayPal, hopefully from 1 January 2024. Stay tuned.

You can also pay by bank transfer, but this will include banking costs for international money transfers (EUBS is registered in the UK, which is now outside of Europe). Please ensure to select this ("*all banking costs carried by the sender*") when you make the transfer. Also, the money transfer may take up to one week, and may fail for some obscure reason. Therefore, unless you are in the UK, we do not recommend this payment option. Using Wise (formerly 'Transferwise') is another option to reduce or avoid banking costs and have a faster and secure transfer of your membership fee.

**EUBS website**

Visit our EUBS website to be informed of news, conferences and meetings, endorsed documents and courses. You can also find information on travel and research grants, employment opportunities, research projects looking for multicentric collaboration, and much more.

The OXYNET database, previously managed by the European Committee for Hyperbaric Medicine (ECHM) is now an integral part of the EUBS website, and can be viewed through a European (and world) map interface, accessed via the menu item 'OXYNET Map' or directly at www.eubs.org/oxynet (or http://www.eubs.org/?page_id=1366).

Have a look at the 'EUBS History' section which has been added under the menu item 'The Society'. There is still some information missing in the list of EUBS Meetings, Presidents and Members at Large – please dig into your memories and help us complete this list.

Please also have a look at our Corporate Members – societies and companies who support the EUBS by their membership. Their logos and contact information can be found at the Corporate Members page (http://www.eubs.org/?page_id=91).

If you have any suggestions or corrections for the information in our notices, please contact us at: webmaster@eubs.org.

South Pacific Underwater Medicine Society

# Notices and news

SPUMS notices and news and all other society information can be found on:
https://spums.org.au/

## SPUMS President's report

Neil Banham

The 52nd SPUMS Annual Scientific Meeting (ASM) will be held at the Pearl Resort, Pacific Harbour, Fiji from Sunday 12th – Friday 17th May 2024. Registrations have opened with good numbers so far. The early bird registration discount ends on 12 January 2024.

**Conference theme:** *A plunge into recreational diving and diver health*

**Convenors:** David Smart and Neil Banham (Scientific Convenor)

The Pearl Resort offers direct access to Beqa Lagoon with its famous soft corals and shark feeding dives. The diving will be organised by Diveplanit, who coordinated the diving at our 2023 Cairns ASM and who have experience in conference diving planning at the Pearl Resort.

Our Keynote Speaker will be Dr Peter Wilmshurst, a British cardiologist who some will remember from our highly successful 2014 Bali ASM, which culminated in the publication of the SPUMS and the United Kingdom Sports Diving Medical Committee (UKSDMC) Joint Position Statement (JPS) on persistent foramen ovale (PFO) and diving in 2015.[1] Peter is a world authority on PFO and diving as well as immersion pulmonary oedema (IPO), reporting the first case.

Workshops will be held with a view to update the JPS on PFO and diving and to develop one for return for diving (or not!) following an episode of IPO.

Supporting speakers include Dr John Lippmann (diving injuries/ deaths) and Professor Simon Mitchell (update on decompression illness), both very knowledgeable and engaging speakers.

Registration, the preliminary Programme and further information can be found at: https://spums.au/index.php/asm-registration

A form for submitting an Abstract is linked to the registration page. As the programme is filling fast, there are only a few presentation slots still available. The provisional programme is available on the SPUMS website.

On behalf of SPUMS, I again sincerely thank David Smart for again taking on the Convenor role. David and our Web Manager Nicky Telles have already spent countless hours working on the next ASM as well as updating the SPUMS website to make organisation of future ASMs much easier for future Convenors.

Deliberations are continuing as to the venue for our 2025 ASM, with the Maldives, the Philippines, Palau and Bali being considered. A decision on the venue will be made at our next ExCom face to face meeting on 23 February 2024. Those who wish to assist with convening this are welcome to contact me.

The positions of Editor (Professor Simon Mitchell) and Deputy Editor (Lesley Blogg) for *Diving and Hyperbaric Medicine* journal have been renewed for a 5-year term-congratulations and our sincere thanks Simon and Lesley!

The ANZHMG Introductory Course in Diving and Hyperbaric Medicine will be next held 19th February – 1st March 2024, again in Fremantle. The 2024 course is fully subscribed, so those considering attending in 2025 should apply as early as possible. Dates are likely to be similar (last week in February/first week of March).

https://spums.au/index.php/education/spums-approved-courses-for-doctors.

Scholarships for trainees to attend this course are available thanks to the generosity of the Australasian Diving Safety Foundation. Please contact John Lippmann at johnl@adsf.org.au for more information.

*Dr Neil Banham*
*SPUMS President*

### Reference

1 Smart D, Mitchell S, Wilmshurst P, Turner M, Banham N. Joint position statement on persistent foramen ovale (PFO) and diving. South Pacific Underwater Medicine Society (SPUMS) and the United Kingdom Sports Diving Medical Committee (UKSDMC). Diving Hyperb Med. 2015;45:129-31. PMID: 26165538. Available from: https://www.dhmjournal.com/images/IndividArticles/45June/Smart_dhm.45.2.129-131.pdf

## The Australian and New Zealand Hyperbaric Medicine Group

### Introductory course in diving and hyperbaric medicine

**Dates:** 19 February – 01 March 2024
**Venue:** Hougoumont Hotel, Fremantle, Western Australia
**Cost:** AUD$3,200.00 (inclusive of GST) for two weeks

Successful completion of this course will allow the doctor to perform Recreational and Occupational (as per AS/ NZS 2299.1) fitness for diving medicals and be listed for such on the SPUMS Diving Doctors list (provided that they continue to be a financial SPUMS member).

The course content includes:

- History of diving medicine and hyperbaric oxygen treatment
- Physics and physiology of diving and compressed gases
- Presentation, diagnosis and management of diving injuries
- Assessment of fitness to dive
- Visit to RFDS base for flying and diving workshop
- Accepted indications for hyperbaric oxygen treatment
- Hyperbaric oxygen evidence based medicine
- Wound management and transcutaneous oximetry
- In water rescue and management of a seriously ill diver
- Visit to HMAS Stirling
- Practical workshops
- Marine Envenomation

**Contact for information:**

Sam Swale, Course Administrator
**Phone:**+61-(0)8-6152-5222
**Fax:**+61-(0)8-6152-4943
**Email:** fsh.hyperbaric@health.wa.gov.au
Accommodation information can be provided on request.

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The

SPUMS
South Pacific Underwater Medicine Society

website is at
https://spums.org.au/

Members are encouraged to login and check it out!
Keep your personal details up-to-date.

The latest issues of *Diving and Hyperbaric Medicine* are via your society website login.

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HBOE
HBOEvidence

## HBOEvidence

HBO Evidence is seeking an interested person/group to continue the HBOEvidence site. The database of randomised controlled trials in diving and hyperbaric medicine: hboevidence.wikis.unsw.edu.au. The HBOEvidence site is planned to be integrated into the SPUMS website in the near future.

Those interested in participating in this project can contact Neil Banham president@spums.org.au

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## Royal Australian Navy Medical Officers' Underwater Medicine Course

**Date:** 18–29 March 2024
**Venue:** HMAS Penguin, Sydney

**Cost:** The course cost remains at AUD$1,355.00 (excl GST).

The MOUM course seeks to provide the medical practitioner with an understanding of the range of potential medical problems faced by divers. Emphasis is placed on the contraindications to diving and the diving medical assessment, together with the pathophysiology, diagnosis and management of common diving-related illnesses. The course includes scenario-based simulation focusing on the management of diving emergencies and workshops covering the key components of the diving medical.

***For information and application forms contact:***

*Rajeev Karekar, for Officer in Charge*
*Submarine and Underwater Medicine Unit*

*HMAS Penguin*
*Middle Head Rd, Mosman*
*NSW 2088, Australia*
***Phone:** +61 (0)2-9647-5572*
***Fax:** +61 (0)2-9647-511*
***Email:** rajeev.karekar@defence.gov.au*

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SPUMS Facebook page
Like us at:
SPUMS on Facebook

South Pacific Underwater Medicine Society

# 52nd Annual Scientific Meeting
# REGISTRATION NOW OPEN

**Sunday May 12th to Friday May 17th 2024**

## The Pearl Resort, Pacific Harbour Fiji

## THEME:

***A plunge into recreational diving and diver health***

## KEYNOTE SPEAKER:

***Dr Peter Wilmshurst***

*UK Cardiologist and world authority on patent foramen ovale and diving and immersion pulmonary oedema*

## SUPPORTING SPEAKERS

**Dr John Lippmann and Professor Simon Mitchell**

**Register at:**

**https://spums.au/index.php/asm-registration**

Diveplanit Travel
Your personal dive travel agency

# SPUMS Diploma in Diving and Hyperbaric Medicine

## Requirements for candidates (May 2014)

In order for the Diploma of Diving and Hyperbaric Medicine to be awarded by the Society, the candidate must comply with the following conditions: They must

1 be medically qualified, and remain a current financial member of the Society at least until they have completed all requirements of the Diploma;

2 supply evidence of satisfactory completion of an examined two-week full-time course in diving and hyperbaric medicine at an approved facility. The list of such approved facilities may be found on the SPUMS website;

3 have completed the equivalent (as determined by the Education Officer) of at least six months' full-time clinical training in an approved Hyperbaric Medicine Unit;

4 submit a written proposal for research in a relevant area of underwater or hyperbaric medicine, in a standard format, for approval before commencing the research project;

5 produce, to the satisfaction of the Academic Board, a written report on the approved research project, in the form of a scientific paper suitable for publication. Accompanying this report should be a request to be considered for the SPUMS Diploma and supporting documentation for 1–4 above.

In the absence of other documentation, it will be assumed that the paper is to be submitted for publication in *Diving and Hyperbaric Medicine*. As such, the structure of the paper needs to broadly comply with the 'Instructions for authors' available on the SPUMS website https://spums.org.au/ or at https://www.dhmjournal.com/.

The paper may be submitted to journals other than *Diving and Hyperbaric Medicine*; however, even if published in another journal, the completed paper must be submitted to the Education Officer (EO) for assessment as a diploma paper. If the paper has been accepted for publication or published in another journal, then evidence of this should be provided.

The diploma paper will be assessed, and changes may be requested, before it is regarded to be of the standard required for award of the Diploma. Once completed to the reviewers' satisfaction, papers not already submitted to, or accepted by, other journals should be forwarded to the Editor of *Diving and Hyperbaric Medicine* for consideration. At this point the Diploma will be awarded, provided all other requirements are satisfied. Diploma projects submitted to *Diving and Hyperbaric Medicine* for consideration of publication will be subject to the Journal's own peer review process.

### Additional information – prospective approval of projects is required

The candidate must contact the EO in writing (or email) to advise of their intended candidacy and to discuss the proposed topic of their research. A written research proposal must be submitted before commencement of the research project.

All research reports must clearly test a hypothesis. Original basic and clinical research are acceptable. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis and if the subject is extensively researched in detail. Reports of a single case are insufficient. Review articles may be acceptable if the world literature is thoroughly analysed and discussed and the subject has not recently been similarly reviewed. Previously published material will not be considered. It is expected that the research project and the written report will be primarily the work of the candidate, and that the candidate is the first author where there are more than one.

It is expected that all research will be conducted in accordance with the joint NHMRC/AVCC statement and guidelines on research practice, available at: https://www.nhmrc.gov.au/about-us/publications/australian-code-responsible-conduct-research-2018, or the equivalent requirement of the country in which the research is conducted. All research involving humans, including case series, or animals must be accompanied by documentary evidence of approval by an appropriate research ethics committee. Human studies must comply with the Declaration of Helsinki (1975, revised 2013). Clinical trials commenced after 2011 must have been registered at a recognised trial registry site such as the Australia and New Zealand Clinical Trials Registry http://www.anzctr.org.au/ and details of the registration provided in the accompanying letter. Studies using animals must comply with National Health and Medical Research Council Guidelines or their equivalent in the country in which the work was conducted.

The SPUMS Diploma will not be awarded until all requirements are completed. The individual components do not necessarily need to be completed in the order outlined above. However, it is mandatory that the research proposal is approved prior to commencing research.

Projects will be deemed to have lapsed if:

- the project is inactive for a period of three years, or
- the candidate fails to renew SPUMS Membership in any year after their Diploma project is registered (but not completed).

For unforeseen delays where the project will exceed three years, candidates must explain to the EO by email why they wish their diploma project to remain active, and a three-year extension may be approved. If there are extenuating circumstances why a candidate is unable to maintain financial membership, then these must be advised by email to the EO for consideration by the SPUMS Executive. If a project has lapsed, and the candidate wishes to continue with their DipDHM, then they must submit a new application as per these guidelines.

The Academic Board reserves the right to modify any of these requirements from time to time. As of October 2020, the SPUMS Academic Board consists of:

Associate Professor David Cooper, Education Officer, Hobart
Professor Simon Mitchell, Auckland

***All enquiries and applications should be addressed to:***
*Associate Professor David Cooper*
*education@spums.org.au*

**Keywords**
Qualifications; Underwater medicine; Hyperbaric oxygen; Research; Medical society

# Courses and meetings

## BIS_on_DHM_2024

The third edition of the Baltic International Symposium on Diving and Hyperbaric Medicine (BIS_on_DHM) is taking place in Gdynia, Poland, from 6–8 June 2024.

This conference aims to exchange knowledge between scientists and clinical practitioners on diving and hyperbaric medicine. Lectures in this symposium will be by invitation only, and speakers are cherry-picked to verify that they bring the latest scientific and medical perspectives to the forum. There will also be vivid sessions on emergency scenarios to discuss the real cases in diving and hyperbaric practice.

**Visit the Symposium website at:**
http://www.BISDHM.events

Register now for an early registration discount.

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## Publications database of the German Diving and Hyperbaric Medical Society (GTÜM)

EUBS and SPUMS members are able to access the German Society's large database of publications in diving and hyperbaric medicine. EUBS members have had this access for many years. SPUMS members should log into the SPUMS website, click on 'Resources' then on 'GTÜM database' in the pull-down menu. In the new window, click on the link provided and enter the user name and password listed on the page that appears in order to access the database.

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## The Science of Diving

Support EUBS by buying the PHYPODE book '*The science of diving*'. Written for anyone with an interest in the latest research in diving physiology and pathology. The royalties from this book are being donated to the EUBS.

**Available from:**
Morebooks
https://www.morebooks.de/store/gb/book/the-science-of-diving/isbn/978-3-659-66233-1

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## DHM Journal Facebook

Find us at:
https://www.facebook.com/divingandhyperbaricmedicine

## Scott Haldane Foundation

As an institute dedicated to education in diving medicine, the Scott Haldane Foundation has organized more than 300 courses all over the world, over the past 31 years. SHF is targeting on an international audience with courses world wide. Below the schedule of upcoming SHF-courses in the first half of 2024.

The courses Medical Examiner of Divers (part 1 and 2) and SHF in-depth courses, as modules of the level 2d Diving Medicine Physician course, fully comply with the ECHM/EDTC curriculum for Level 1 and 2d respectively and are accredited by the European College of Baromedicine (ECB).

| **2024** | |
|---|---|
| **27 January** | Refresher course Diving Medical in Practice Zeist, The Netherlands |
| **5–6 April** | Medical Examiner of Divers part 1 (level 1) Zeist, The Netherlands |
| **11–13 April** | Medical Examiner of Divers part 2 (level 1) Amersfoort, The Netherlands |
| **11–18 May** | Medical Examiner of Divers part 2 (level 1) Bonaire, Dutch Caribbean |
| **14–15 June** | In-depth course Brain under pressure (level 2d) Putten, The Netherlands |
| **On request** | Internship HBOt (level 2d) NL/Belgium |

The course calendar will be supplemented regularly. For the latest information see: www.scotthaldane.org.

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P O Box 347, Dingley Village Victoria, 3172, Australia
**Email:** info@historicaldivingsociety.com.au
**Website:** https://www.historicaldivingsociety.com.au/

# Diving and Hyperbaric Medicine: Instructions for Authors

(updated February 2023)

*Diving and Hyperbaric Medicine* (DHM) is the combined journal of the South Pacific Underwater Medicine Society (SPUMS) and the European Underwater and Baromedical Society (EUBS). It seeks to publish papers of high quality on all aspects of diving and hyperbaric medicine of interest to diving medical professionals, physicians of all specialties, scientists, members of the diving and hyperbaric industries, and divers. Manuscripts must be offered exclusively to *Diving and Hyperbaric Medicine* unless clearly authenticated copyright exemption accompanies the manuscript. All manuscripts will be subject to peer review. Accepted contributions will also be subject to editing.

***Address***: The Editor, Diving and Hyperbaric Medicine, Department of Anaesthesiology, University of Auckland, Private Bag 92019, Auckland 1142, New Zealand
***Email***: editor@dhmjournal.com
***Phone: (mobile)***: +64 (0)27 4141 212
***European Editor***: euroeditor@dhmjournal.com
***Editorial Manager***: editorialassist@dhmjournal.com
***Journal information***: info@dhmjournal.com

Contributions should be submitted electronically by following the link:
http://www.manuscriptmanager.net/dhm

There is on-screen help on the platform to assist authors as they assemble their submission. In order to submit, the corresponding author needs to create an 'account' with a username and password (keep a record of these for subsequent use). The process of uploading the files related to the submission is simple and well described in the on-screen help provided the instructions are followed carefully. The submitting author must remain the same throughout the peer review process.

## Types of articles

DHM welcomes contributions of the following types:

**Original articles, Technical reports and Case series**: up to 3,000 words is preferred, and no more than 30 references (excluded from word count). Longer articles will be considered. These articles should be subdivided into the following sections: an **Abstract** (subdivided into Introduction, Methods, Results and Conclusions) of no more than 250 words (excluded from word count), **Introduction, Methods, Results, Discussion, Conclusions, References, Acknowledgements**, **Funding** sources and any **Conflicts of interest**. **Legends/captions** for illustrations, figures and tables should be placed at the end of the text file.

**Review articles**: up to 5,000 words is preferred and a maximum of 50 references (excluded from the word count); include an informative **Abstract** of no more than 300 words (excluded from the total word count); structure of the article and abstract is at the author(s)' discretion.

**Case reports, Short communications** and **Work in progress** reports: maximum 1,500 words, and 20 references (excluded from the word count); include an informative **Abstract** (structure at author's discretion) of no more than 200 words (excluded from the word count).

**Educational articles, Commentaries** and **Consensus reports** for occasional sections may vary in format and length but should generally be a maximum of 2,000 words and 15 references (excluded from word count); include an informative **Abstract** of no more than 200 words (excluded from word count).

**Letters to the Editor**: maximum 600 words, plus one figure or table and five references.

The journal occasionally runs **'World as it is'** articles; a category into which articles of general interest, perhaps to divers rather than (or in addition to) physicians or scientists, may fall. This is particularly so if the article reports an investigation that is semi-scientific; that is, based on methodology that would not necessarily justify publication as an original study. Such articles should follow the length and reference count recommendations for an original article. The structure of such articles is flexible. The submission of an abstract is encouraged.

## Formatting of manuscripts

All submissions must comply with the following requirements. **Manuscripts not complying with these instructions will be suspended** and returned to the author for correction before consideration. Guidance on structure for the different types of articles is given above.

**Documents on DHM website** https://www.dhmjournal.com/index.php/author-instructions

The following pdf files are available on the DHM website to assist authors in preparing their submission:

Instructions for Authors 2023 (this document)
DHM Keywords 2021
DHM Mandatory Submission Form 2020
Trial design analysis and presentation
English as a second language
Guideline to authorship in DHM 2015
Helsinki Declaration revised 2013
Is ethics approval needed?

# DIVER EMERGENCY SERVICES PHONE NUMBERS

**AUSTRALIA – DAN**
**1800-088200 (in Australia toll free)**
**+61-8-8212-9242 User pays**
**(outside Australia)**

**NEW ZEALAND – DAN Emergency Service**
**0800-4DES-111 (in New Zealand toll free)**
**+64-9-445-8454 (International)**

**ASIA, PACIFIC ISLANDS – DAN World**
**+618-8212-9242**

**EUROPE – DAN**
**+39-06-4211-8685 (24-hour hotline)**

**SOUTHERN AFRICA – DAN**
**+27-10-209-8112 (International call collect)**

**USA – DAN**
**+1-919-684-9111**

**JAPAN – DAN**
**+81-3-3812-4999 (Japan)**

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## Scholarships for Diving Medical Training for Doctors

The Australasian Diving Safety Foundation is proud to offer a series of annual Diving Medical Training scholarships. We are offering these scholarships to qualified medical doctors to increase their knowledge of diving medicine by participating in an approved diving medicine training programme. These scholarships are mainly available to doctors who reside in Australia. However, exceptions may be considered for regional overseas residents, especially in places frequented by Australian divers. The awarding of such a scholarship will be at the sole discretion of the ADSF. It will be based on a variety of criteria such as the location of the applicant, their working environment, financial need and the perception of where and how the training would likely be utilised to reduce diving morbidity and mortality. Each scholarship is to the value of AUD5,000.00.

There are two categories of scholarships:

1. ADSF scholarships for any approved diving medical training program such as the annual ANZHMG course at Fiona Stanley Hospital in Perth, Western Australia.
2. The Carl Edmonds Memorial Diving Medicine Scholarship specifically for training at the Royal Australian Navy Medical Officers' Underwater Medicine Course, HMAS Penguin, Sydney, Australia.

Interested persons should first enrol in the chosen course, then complete the relevant ADSF Scholarship application form available at: https://www.adsf.org.au/r/diving-medical-training-scholarships and send it by email to John Lippmann at johnl@adsf.org.au.

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### DISCLAIMER