# Diving and Hyperbaric Medicine

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## Hypoxia signatures in rebreather divers

Accelerated decompression from saturation

Hyperoxia effects on the microcirculation

Delayed recompression for decompression illness

Decompression illness in Thailand

Proprietary Doppler vs TTE for detecting venous gas emboli

Underwater electric shock causing aspiration

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# CONTENTS

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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**
**To publish a journal and to convene members of each Society annually at a scientific conference**



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## DIVING AND HYPERBARIC MEDICINE

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# The Editor's offering

Welcome to the final issue of Diving and Hyperbaric Medicine Journal for 2022.

Inside, Daniel Popa and colleagues describe the effect of intentionally exposing divers to hypoxia as a training strategy to improve recognition of symptoms should hypoxia occur in a real dive. Like others studying hypoxia exposure in aviators, they found that the symptoms of hypoxia remained relatively consistent for each individual from one exposure to the next. Despite this, and despite having experienced hypoxia only hours earlier, when exposed to a second blinded hypoxia event only 9/20 subjects performed the bailout procedure prescribed as a response to recognition of hypoxia without prompting. Although the study design does not allow definitive conclusions about the value of hypoxia training, this result suggests that the risk of failing to respond appropriately to hypoxia during a dive would not be materially reduced by hypoxia training.

Jean Pierre Imbert and colleagues have collected accounts of the known instances where decompression from saturation dives has been accelerated because of an emergency. Such events are rare and when they occur, the diving and medical supervisors involved have to scramble around looking for guidance and precedent for their decisions. Such information can be hard to find. I am delighted to publish this paper which will be immediately available as a resource for medical supervisors who find themselves in such situations in future. The real-world scenarios recounted are fascinating from a human-interest perspective, and also for the almost invariable success reported in getting divers safely back to one atmosphere pressure more quickly than usual.

For those interested in the physiological effects of hyperbaric oxygen (HBO), Nicholas Cousin and colleagues offer a detailed study of the effects of normobaric and hyperbaric hyperoxia on human haemodynamics and the microcirculation. Hyperbaric hyperoxia produced bradycardia while cardiac output remained constant and arterial blood pressure increased. Additionally, the rise in tissue oxygenation during HBO exposure promoted an adaptative vasoconstrictive response, though microvascular reactivity remained unaltered. I believe this is the most comprehensive study of its type published to date.

Sofia Sokolowski and colleagues considered outcomes in Finnish divers treated for decompression illness (DCI) between 1999 and 2018. Evacuation distances and consequently delays to recompression are commonly long in Finland. Delayed recompression remained effective in most cases, though as the authors acknowledge, most of their cases were milder forms of DCI whose natural history is toward spontaneous resolution even without recompression. Shorter delays were associated with fewer recompression treatments to achieve full resolution or plateau in recovery. Vestibulocochlear symptoms were associated with the lowest rates of resolution on completion of all recompression treatment.

Pitchaya Chevasutho and colleagues provided a descriptive study of DCI cases treated at a major centre in Bangkok Thailand between 2015 and 2021. Perhaps not surprisingly the cases were similar in nature to those reported in other series, but interesting features of the series were the infrequent use of first aid oxygen, and (like the Sokolowski paper) long delays to recompression.

Oscar Plogmark and colleagues compared post-dive venous gas emboli (VGE) grades obtained using 2D echocardiography or the O'Dive™ device (which uses an automated Doppler algorithm to measure subclavian VGE signals). They found that the O'Dive was less sensitive in VGE detection than 2D echocardiography.

As the world opens up I have been fortunate to recently attend three superb professionally-relevant events. The first was the 'Diving Talks' meeting in Lisbon, Portugal; a terrific meeting with a relatively unique format of short talks and long periods of audience discussion and questions. This was the second iteration of this event, and a third is planned late 2023. It is highly recommended for those whose primary interest is diving. The second was the famous Oztek technical diving show in Melbourne. This show is a must for divers in the Antipodes but it won't occur again until 2024. Finally, I had the great pleasure of speaking at the British Hyperbaric Association ASM in Plymouth, hosted by the outstanding team at the Diving Diseases Research Centre. This was a fantastic meeting with many interesting speakers, and the usual casual and collegial atmosphere; a highlight of my year, and my thanks to Gary Smerdon and the team for inviting me.

As the year closes, I must thank those who support the journal. Members of our societies must remember that the journal is largely a labour of love kept afloat through the efforts of volunteers. I thank the societal presidents (Jean-Eric Blatteau and Neil Banham) and executive committees for their support. On the SPUMS side, special thanks to Soon Teoh, the SPUMS treasurer, who entirely voluntarily acts as the journal accountant. Similarly, my sincere thanks to our deputy editor Lesley Blogg, the editorial board, and the journal governance committee. I am deeply indebted to the colleagues from all corners of the world who perform reviews for us. Finally, as always, much of the journal's success depends on the skill and professionalism of our editorial manager Nicky Telles without whom I could certainly not function in this role.

*Professor Simon Mitchell*
*Editor, Diving and Hyperbaric Medicine Journal*

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**Front cover:** Pete Mesley ascending stairs from the hospital area on USS Saratoga at Bikini Atoll. Note the 'SICK BAY' sign just above his rebreather. Photo by Simon Mitchell.

# Original articles

## Hypoxia signatures in closed-circuit rebreather divers

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**Keywords**
Physiology; Rescue; Safety; Technical diving; Training

**Abstract**
(Popa D, Kutz C, Carlile M, Brett K, Moya EA, Powell F, Witucki P, Sadler R, Sadler C. Hypoxia signatures in closed-circuit rebreather divers. Diving and Hyperbaric Medicine. 2022 December 20;52(4):237–244. doi: 10.28920/dhm52.4.237-244. PMID: 36525681.)
**Introduction:** Faults or errors during use of closed-circuit rebreathers (CCRs) can cause hypoxia. Military aviators face a similar risk of hypoxia and undergo awareness training to determine their 'hypoxia signature', a personalised, reproducible set of symptoms. We aimed to establish a hypoxia signature among divers, and to investigate their ability to detect hypoxia and self-rescue while cognitively overloaded.
**Methods:** Eight CCR divers and 12 scuba divers underwent an initial unblinded hypoxia exposure followed by three trials; a second hypoxic trial and two normoxic trials in randomised order. Hypoxia was induced by breathing on a CCR with no oxygen supply. Subjects pedalled on a cycle ergometer while playing a neurocognitive computer game to simulate real world task loading. Subjects identified hypoxia symptoms by pointing to a board listing common hypoxia symptoms, and were instructed to perform a 'bailout' procedure to mimic self-rescue if they perceived hypoxia. Divers were prompted to bailout if peripheral oxygen saturation fell to 75%, or after six minutes during normoxic trials. Subsequently we interviewed subjects to determine their ability to distinguish hypoxia from normoxia.
**Results:** Ninety-five percent of subjects (19/20) showed agreement between unblinded and blinded hypoxia symptoms. Subjects correctly identified the gas mixture in 85% of the trials. During unblinded hypoxia, only 25% (5/20) of subjects performed unprompted bailout. Fifty-five percent of subjects (11/20) correctly performed the bailout but only when prompted, while 15% (3/20) were unable to bailout despite prompting. During blinded hypoxia 45% of subjects (9/20) performed the bailout unprompted while 15% (3/20) remained unable to bailout despite prompting.
**Conclusions:** Although our data support a normobaric hypoxia signature among both CCR and scuba divers under experimental conditions, most subjects were unable to recognise hypoxia in real time and perform a self-rescue unprompted, although this improved in the second hypoxia trial. These results do not support hypoxia exposure training for CCR divers.

### Introduction

A closed-circuit rebreather (CCR) is a self-contained diving unit that allows a diver to recycle or conserve the oxygen in their exhaled breath while removing the carbon dioxide with a chemical scrubber. CCRs have proliferated over the past several years in part due to increased commercial availability and advantages for certain applications over open-circuit scuba. For recreational and scientific divers, CCRs offer the advantages of longer dive times, deeper dives, increased wildlife encounters, and the ability to explore more remote locations. CCRs also offer increased stealth by minimising exhaled bubbles which provides a distinct tactical advantage in military applications. CCR diving also carries an estimated mortality risk of approximately 10 times that of recreational scuba diving, with hypoxia as one of the leading causes of reported CCR diving injuries or fatalities.[1–3] One study reported that of the recreational CCR deaths between 1998 and 2010 with a known cause, 17% were due to hypoxia.[2]

Like CCR divers, aviators can experience hypoxia with potentially fatal consequences if their cockpit depressurises while in flight or their supplemental oxygen systems

fail. Among these aviators, acute hypobaric hypoxia can present with a variety of symptoms including psychomotor (incoordination, tremors), cognitive (concentration, confusion, memory loss), visual impairment (blurred vision, colour/light intensity changes), psychological (anxiety, depression, euphoria), dyspnoea, paraesthesia, headache, dizziness, tachycardia, and loss of consciousness.[4–6] Significant interpersonal variation in the order, severity, and speed of onset of hypobaric hypoxia symptoms occurs. Interestingly, the intrapersonal manifestation of hypobaric hypoxia symptoms on repeated exposures appears reproducible and serves as a 'hypoxia signature'. Most aircrew experience a high level of agreement between the dominant symptoms experienced during acute hypoxia and those they recall from previous hypoxia exposures (training or real events).[4,5,7,8] Among military aircrew, this forms the basis for hypoxia awareness training at fixed intervals (typically 3–6 years) in a hypobaric chamber.

To our knowledge, using hypoxia signatures to train divers to recognise their symptoms and perform a self-rescue bailout procedure has not been fully investigated. We sought to investigate these questions more thoroughly while mimicking diving conditions with concomitant exercise and mental distraction among groups of subjects who dive with CCRs or only scuba. We hypothesised the following:

1. During gradual onset hypoxia, the majority of cognitively distracted subjects will recognise their hypoxia signature and then perform a bailout procedure without any prompting or alarm when blinded to the breathing gas mixture.
2. Subjects trained as CCR divers will perform a bailout procedure without prompting significantly more often than subjects trained as scuba divers only.
3. Performance of a bailout procedure without prompting improves with a second exposure to gradual onset hypoxia while using a rebreather.

If hypoxia signature training were to prove effective, CCR divers may be able to decrease their risk of hypoxia-associated accidents and fatalities, increasing the safety of CCR diving.

## Methods

The study protocol as approved by the institutional review board at the University of California, San Diego (Protocol #161414).

### EXPERIMENTAL PROTOCOL

Using posted fliers at dive shops, and announcements at diving clubs and professional diving organisations, we recruited experienced, healthy male and female scuba and CCR divers, with ages between 18–60 years old. We obtained informed consent from all participants. We aimed to recruit 30 subjects split evenly between scuba and CCR divers, but we were only able to recruit 21 subjects before the COVID-19 pandemic began and delayed experimental trials indefinitely.

Subjects underwent a total of four experimental trials in a single day. The first trial was an unblinded trial of the experimental set-up with hypoxia. We induced gradual onset hypoxia by starting with a normoxic oxygen mix (air) and then shut off the addition of oxygen to the breathing loop, mimicking a real life CCR malfunction. Each subject gradually consumed the oxygen in the breathing loop, eventually leading to a hypoxic inhaled gas mixture. This trial served as an unblinded training trial where subjects experienced hypoxia in a safe, controlled environment supervised by practicing emergency medicine physicians with rescue airway equipment and supplemental oxygen immediately available.

We used a Scubaforce (Mönchengladbach, Germany) SF2 rebreather, regularly maintained and serviced, equipped with three Analytic Industries Model PSR 11-39-XD oxygen sensors (Pomona, CA, USA). Soda lime scrubber (Sofnolime 797, Molecular Products Inc., Louisville, CO, USA) was used to remove carbon dioxide ($CO_2$) from inhaled gas. Subjects breathed from a standard diving mouthpiece and used a nose clip. A gas analyser (MediPines AGM100 Innovative Respiratory Monitor, Yorba Linda, CA, USA) sampled $CO_2$ and oxygen ($O_2$) levels from a port drilled into the CCR mouthpiece. After the unblinded hypoxia trial, subjects performed three additional trials; two normoxic control trials and one hypoxic experimental trial. In normoxic trials the fraction of $O_2$ in the rebreather loop was maintained at 21% by the investigators. In hypoxia trials the $O_2$ supply to the CCR was isolated. The order of the three additional trials was randomised, and subjects were blinded to the gas they were breathing (normoxic vs hypoxic). All trials ended when the subjects desaturated to 75%, six minutes elapsed (for normoxic trials), or if the subjects felt that they were experiencing an emergency and performed the self-rescue protocol. The self-rescue protocol (a 'bailout') required the subjects to turn a ball valve by pulling on a lever, simulating switching to a bailout gas on a CCR. If the subjects desaturated to 75% (hypoxic trials) or 6 minutes elapsed (normoxic trials), investigators prompted the subjects to bailout with a written sign.

After performing the bailout, investigators removed the mouthpiece and allowed the subjects to recover at least 10 minutes between trials. If subjects failed to perform the bailout, investigators rapidly removed the mouthpiece to prevent loss of consciousness or motor control. This decision was based on investigators' clinical assessment of each subject's reaction to the written prompt to bailout. For example, if a subject's eyes were not moving to read the written bailout sign or the subject made no purposeful hand movement, investigators immediately removed the mouthpiece and encouraged the subject to take deep breaths of room air. No subject lost consciousness.

**Figure 1**

Study protocol; CCR – closed circuit rebreather; $EtCO_2$ – end tidal carbon dioxide; $EtO_2$ – end tidal oxygen; $SpO_2$ – peripheral oxygen saturation

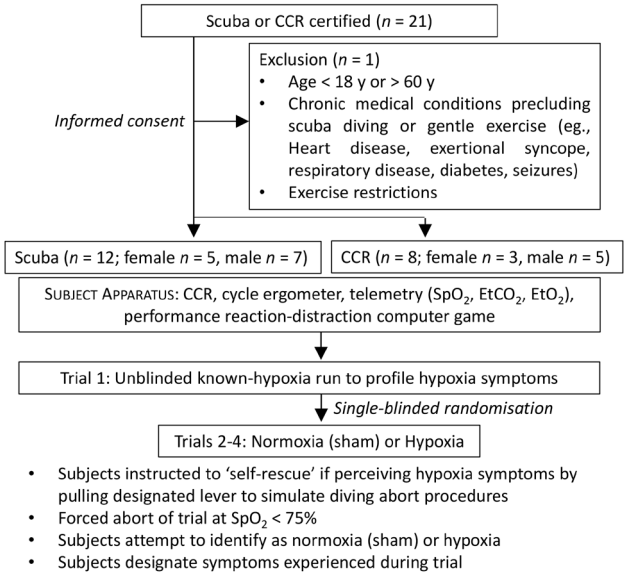
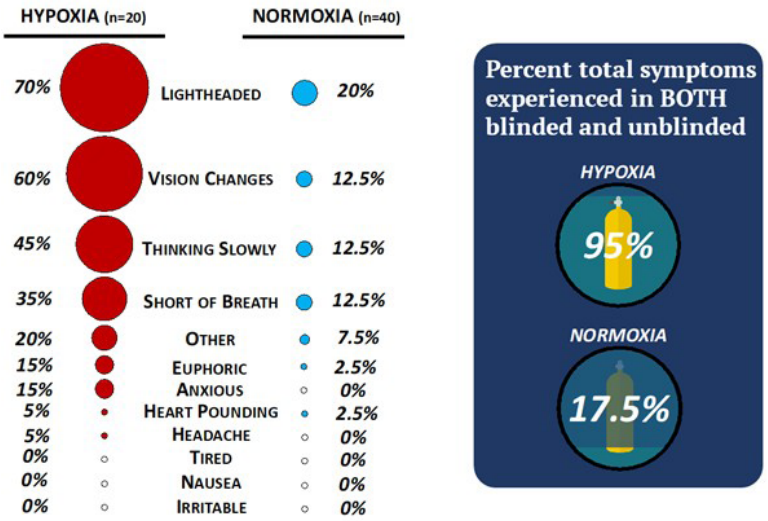



**Figure 2**

Reported hypoxia symptoms in descending order of frequency of occurrence at left. Percentage of blinded trials with recurrence of the symptoms reported during the unblinded hypoxia trial at right




During each trial, subjects pedaled a cycle ergometer set to 5W to simulate the attention needed for underwater finning without producing a large increase in metabolic rate. They were monitored with a finger pulse oximeter. While pedaling, each subject also played a distracting computer-based neurocognitive test ('Go/No-Go', Automated Neuropsychological Assessment Metrics (ANAM), Vista LifeSciences Inc., Parker, CO, USA) to simulate cognitive task loading underwater. Investigators also instructed the subjects to point to symptoms they were experiencing on a board listing common hypoxia symptoms. After each trial finished, investigators interviewed subjects to determine their perception of which gas mix they breathed (normoxic vs hypoxic), their recall of symptoms, and how the blinded trial compared to the unblinded hypoxia trial. The experimental protocol is summarised in Figure 1.

## OUTCOMES AND ANALYSES

The presence of a hypoxia signature under the gradual onset hypoxia condition was investigated. We compared each subject's symptoms which they identified in real-time between their unblinded and blinded hypoxia trials. We reported the frequency of these symptoms as well as

**Figure 3**
Blinded gas identification among scuba and closed circuit rebreather (CCR) divers after completion of each experimental trial

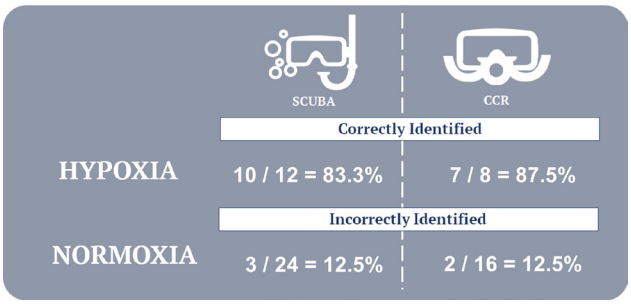
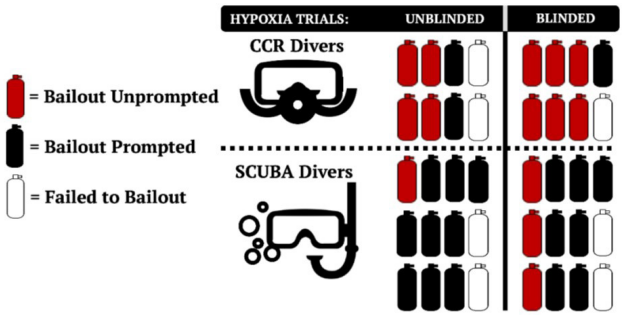



**Figure 4**
Performance of the bailout procedure with and without prompting during both hypoxia trials. Prompting occurred only when oxygen saturation reached 75%; CCR – closed circuit rebreather




the percentage of blinded hypoxia trials where subjects reported the same symptoms that they experienced during their unblinded hypoxia trial.

The subjects' ability to identify their breathing gas mixture (normoxia vs hypoxia) in interviews after the blinded experimental trials was recorded. We also compared the percentage of correct and incorrect identification of hypoxic and normoxic gases between CCR trained divers and scuba divers.

The subjects' ability to recognise their hypoxia signature symptoms and then perform the bailout procedure without any external prompting or alarm was measured. We reported the number of subjects who performed the bailout without prompting, those who required prompting and performed the bailout afterward, and those who were unable to perform the bailout despite prompting. As part of the analysis for these data, we performed the following:

- A comparison of the CCR trained divers with scuba divers in hypoxia signature recognition and then bailout without any prompting or alarm.
- A comparison of oxygen saturations at the time of bailout between subjects who correctly performed the bailout without prompting and those who required prompting or were unable to perform the bailout. We performed two-sided *t*-tests comparing the saturations of those who performed the unprompted bailout and those who did not (either required prompting to bailout or were unable to bailout) with significance defined as $P < 0.05$.

Lastly, the existence of a training effect with repeated exposure to hypoxia in a single day was investigated by comparing the number of subjects who correctly performed the bailout procedure during the blinded experimental hypoxic trial versus during the unblinded initial introductory hypoxic trial.

## Results

We recruited 21 subjects and excluded one due to age. All 20 subjects included in the study completed all trials and none suffered any complication such as loss of consciousness. The CCR group ($n = 8$) consisted of five males and three females while the scuba group ($n = 12$) consisted of seven males and five females. Neither group contained subjects who reported any chronic medical condition or prior hypoxia training, and no subject in either group reported a history of decompression illness.

During the experimental trials, the most commonly reported symptoms, regardless of gas mixture, were lightheadedness, vision changes, thinking slowly, and shortness of breath (Figure 2). Following blinded hypoxia trials, nearly all subjects (19/20, 95%) reported recurrence of symptoms experienced during unblinded hypoxia trials. The one subject who did not report the recurrence was a scuba diver. Among the blinded normoxia control trials, some subjects reported similar symptoms compared to the earlier unblinded hypoxia trial in 7/40 trials (17.5%, seven unique subjects) (Figure 2).

During the debriefing interview following each blinded trial, subjects correctly identified the gas mixture in 51/60 (85%) trials. Of those 60 blinded trials, subjects correctly identified 17/20 (85%) hypoxia trials and 34/40 (85%) normoxia trials (Figure 3). Grouped according to their diving history, 10/12 (83.3%) scuba divers correctly identified the blinded hypoxia trial, and 7/8 (87.5%) CCR divers also correctly identified the blinded hypoxia trial. Scuba divers incorrectly identified normoxia as hypoxia in 3/24 trials (12.5%, three unique subjects), while CCR divers incorrectly identified 2/16 normoxia trials as hypoxia (12.5%, two unique subjects).

Among all divers during the unblinded hypoxia trial, only 5/20 (25%) subjects performed the bailout unprompted based on the perception of hypoxia symptoms, and four of these subjects were CCR divers (Figure 4). Of the remaining subjects, 11/20 (55%) correctly performed the bailout only when prompted, while 3/20 (15%) were unable to bailout despite prompting. One CCR diver performed the bailout procedure unprompted but incorrectly during their unblinded hypoxia trial. For subjects who correctly performed the unprompted bailout during the unblinded hypoxia trial, the peripheral oxygen saturation ($SpO_2$) averaged 80% (SD 5.2),

at the time of bailout (81.3% (5.12) for CCR vs 75% for the 1 scuba subject). This mean $SpO_2$ of 80% (5.2) was not significantly different than the $SpO_2$ = 75% endpoint we used in our protocol for those subjects who required prompting to bailout or were unable to bailout (*t*-test, $n = 5$, $P = 0.10$).

In the blinded hypoxia trial, 17/20 (85%) subjects performed the bailout procedure, but only 9/20 (45%) subjects did so unprompted (Figure 4). This represented an increase of four subjects in comparison with the unblinded hypoxia trial where only 5/20 (25%) subjects correctly performed the bailout procedure unprompted. One scuba diver stated in their post-trial interview that they were aware of the need to bailout but continued the trial and then forgot to pull the lever. Of the three subjects who failed to perform the bailout procedure despite prompting, only one was a CCR diver. Among the CCR subjects, 6/8 (75%) correctly performed the bailout procedure unprompted during the blinded hypoxia trial. They represented 6/9 (66.7%) subjects that correctly performed the bailout procedure unprompted and based solely on their recognition of symptoms during the blinded hypoxia trial. Seventy-five percent of CCR subjects correctly performed the bailout procedure unprompted compared to 3/12 (25%) scuba diver subjects. During the blinded hypoxia trial, the average $SpO_2$ of the subjects who correctly performed the unprompted bailout was 78.6% (SD 4.1) at the time of bailout (78.8% (4.4) for CCR vs 78% (4.0) for scuba). The $SpO_2$ for these subjects who correctly performed the unprompted bailout during the blinded hypoxia trial was 78.6% (4.1); significantly different to the 75% endpoint we used in our protocol for those subjects who required prompting to bailout or were unable to bailout (*t*-test, $n = 9$, $P = 0.03$).

During the blinded normoxia trials that served as sham controls, subjects performed the bailout procedure unnecessarily in 3/40 trials (two unique subjects), misidentifying normoxia as hypoxia. None of these trials involved CCR subjects. These subjects, believing themselves to be dangerously hypoxic, had an average $SpO_2$ of 94.3% (3.3). The typical reason subjects gave in the post-trial interview was that they performed the bailout procedure due to perceived changes in the breathing resistance of the experimental CCR that we attributed to the counter-lung. The remaining subjects terminated their trials, after 6 minutes according to our protocol, with an average $SpO_2$ of 97.6% (1.5), consistent with normoxia.

Additionally, we measured end tidal $CO_2$ throughout the trials to demonstrate isocapnia. $CO_2$ levels in subjects undergoing the hypoxia trials averaged 37.94 (3.4) mmHg, while those undergoing the normoxia trials averaged 39.2 (2.8) mmHg.

## Discussion

Hypoxia can have an insidious and deleterious effect on CCR divers, sapping them of both motor function and cognitive ability. The development of hypoxia while using a CCR is particularly dangerous since the onset of symptoms is gradual as the diver consumes the available oxygen in the breathing circuit. Divers may overlook subtle symptoms of hypoxia due to a lack of awareness leading to a lack of problem recognition and failure to correct the problem. This may be compounded by underwater tasks or nitrogen narcosis.[1,9] Even if the hypoxia is recognised by the diver, as symptoms progress, the diver may quickly be incapacitated and unable to correct it. If left uncorrected, hypoxia will lead to a rapid loss of consciousness under water and subsequent drowning or death.

A potentially fatal hypoxic breathing loop may result from numerous causes such as the breathing mixture becoming hypoxic by over-dilution with hypoxic diluent gas, failure of the fuel cells to sense hypoxic gas levels, mechanical failure of the solenoid valve controlling the gas mixture, forgetting to open the oxygen tank valve or turn on the electronics prior to diving, and improper diluent gas selection.[1,10–12] In a CCR, oxygen is mixed with a diluent gas to maintain a constant partial pressure of inhaled oxygen ($PO_2$) regardless of depth. In order to maintain a constant $PO_2$, galvanic fuel cells measure the breathing loop $PO_2$, which is reported to the diver on their display. Unfortunately, these fuel cells have a finite lifespan, and failure may be difficult to predict. Due to this fact, most CCRs utilise three cells to measure $PO_2$, which are interpreted by the computer's algorithm. Many of these algorithms use voting logic where the computer averages the three cells' $PO_2$ readings, unless one of the three cells varies significantly in which case the computer ignores it. In an electronic CCR as the $PO_2$ drops below a set point defined by the diver, oxygen is added into the loop through the opening of an electronic solenoid valve. Alternately, if the $PO_2$ becomes elevated, most CCR models will not add diluent but rather wait for the diver's metabolism to consume the excess oxygen. In a CCR without electronic controls, the diver must perform these gas changes manually. CCR systems are typically set up to include an alarm to prompt the diver to look at their display if the $PO_2$ varies from the set point.[11,13]

With this study, we aimed to determine if subjects had a reproducible set of symptoms, the 'hypoxia signature', during gradual onset hypoxia as well as the ability to detect hypoxia during a simulated dive and then perform a self-rescue bailout procedure. With 95% agreement between unblinded and blinded hypoxia trials, the data support the presence of hypoxia signatures under our experimental conditions. The subjects also exhibited isocapnia with measured end tidal $CO_2$ levels all within normal limits across all subjects and trials.

A recent study reported a cohort of subjects exposed to hypoxia twice, approximately five weeks apart, and found no differences between the severity of various hypoxia symptoms during each trial using a visual analog scale (VAS) in interviews five minutes after the hypoxia exposure.[14]

These results support the idea of a hypoxia signature. However, the comparisons made in the VAS score were made between trials five weeks apart with the subjects grouped together versus a comparison between VAS scores on an individual subject basis. Furthermore, the purpose of that study was to determine if hypoxia training could affect the time of useful cognitive function as well as to characterise physiological parameters in the subjects who breathed a hypoxic gas mixture of 5.5% $O_2$ while performing a card recognition protocol. Our study provides additional insights into the presence of a hypoxia signature in divers and begins to examine its usefulness as a potential training mechanism. Our protocol consisted of a more gradual hypoxic stress where each subject breathed down the CCR loop from room air to a hypoxic concentration all while maintaining isocapnia. This approach mimics the insidious, real-world scenario where a CCR malfunctions and fails to add additional oxygen into the breathing loop. This gradual onset also distinguishes hypoxia in CCR diving from hypoxia seen in aviation which is typically more abrupt in onset in both reality and training.

Additionally, our study sought to examine if a diver could not only recognise their hypoxia signature but perform self-rescue. Recognition of the hypoxia signature provides no safety benefit if the diver is unable or unwilling to perform self-rescue by a bailout procedure. We had hypothesised that with its more gradual onset, our hypoxia protocol would allow the subjects more time for recognition of symptoms and then more time for corrective action, leading to a majority of subjects able to perform the bailout procedure unprompted. However, our results do not support this. While the large majority of subjects correctly differentiated the blinded hypoxia trial from sham trials during debriefing interviews, 55% of all subjects still did not bailout unprompted during the blinded hypoxia trial, contrary to what we expected. We anticipate that these subjects would have had serious adverse effects or died under analogous diving conditions. In fact, we suspect that our findings overestimate divers' ability to bailout due to the artificial nature of a laboratory setting and that our subjects knew we were studying hypoxia. This is an alarming finding given that current CCR equipment may not effectively alert the diver if multiple oxygen sensors fail. This failure is distinct from improper calibration. Dive time, humidity, high temperature, and life cycle can produce inaccurate millivolt potentials in oxygen sensors, which can lead to a 'false high' partial pressure calculation. Thus, oxygen is not injected, and hypoxia can result despite 'normal' readings. This has significant implications for checklist development and implementation.

Furthermore, among those subjects who performed the bailout unprompted, the $SpO_2$ levels were still quite low, even though the $SpO_2$ levels for the blinded hypoxia trial were significantly elevated in comparison to those of subjects who did not perform the bailout without prompting. These levels correspond to the steep portion of the oxyhaemoglobin dissociation curve, where small decreases in the partial pressure of $O_2$ correspond to large decreases in $SpO_2$, indicating a narrow time frame in which a subject could correct their hypoxia before becoming incapacitated. We predict that such low oxygen saturations would lead to cognitive deficits and impaired divers, risking both their lives and the lives of their dive buddies under real world conditions.

When analysing the two subject groups, the CCR divers outperformed scuba divers at correctly performing the bailout procedure unprompted during the blinded hypoxia trial (75% vs 25%). The improved performance by the CCR divers would seem to support our initial hypotheses regarding the gradual onset of hypoxia allowing for increased recognition and increased bailout but the small number of subjects in the CCR group may represent a sampling bias that is not generalisable to the greater population of CCR divers. The improved bailout performance in this group may be due to increased familiarity with the experimental equipment, increased awareness of their responses to breathing from an external device, increased ability to manage cognitive distractions, or some other effect from their dive training or other prior experience.

Comparing the unblinded hypoxia trial and the blinded hypoxia trial, we observed a nearly two-fold increase in the number of subjects that recognised their hypoxia signatures and performed the bailout unprompted. This increase may represent a training effect stemming from the first, unblinded trial which many of the subjects were keenly interested in experiencing. Aerospace researchers have shown that individuals without hypoxia awareness training are unlikely to recognise these symptoms and appreciate their insidious onset. One study reviewed 656 incidents of in-flight hypoxia within the US Air Force from 1976 to 1990 and found a large difference in the number of aircrew who experienced a loss of consciousness based on whether or not they had received hypoxia training, suggesting a beneficial hypoxia training effect.[15] This retrospective review however did not formally test the efficacy of the training protocol, and other differences between the two groups could explain the observation.

Although a training effect is certainly possible among the divers in our study, our experimental protocol did not test for this effect and other factors may explain the increase in subjects performing the bailout unprompted. For example, during their debriefing interviews, many of the subjects reported that they suppressed the desire to perform the bailout procedure during our unblinded hypoxia trial. They wanted to deliberately push their physiological limits to experience as profound a level of hypoxia as they could. Since the unblinded hypoxia trial served as a baseline of the performance of the bailout without prompting, this desire to push physiological limits artificially worsened the subjects' baseline and may account for the difference in performance between the unblinded and blinded hypoxia trials, negating

any evidence of a true training effect. Other possible factors such as a better understanding of the experimental protocol with repeated trials, increased familiarity with the experimental equipment, or a combination of multiple factors could explain the improvement we observed between hypoxia trials. Importantly, we do not feel that our results support a single day of hypoxia exposure training for CCR divers or that this protocol will benefit their ability to perform self-rescue. Whether a training benefit will occur during a repeat bout of testing of the ability to recognise one's hypoxia signature and then perform the bailout procedure without prompting remains unanswered. Mitchell et al recently took the first steps investigating these problems, finding that a "*hypoxic experience did not improve cognitive performance or subject insight into performance in a second exposure five weeks later*".[14]

Nonetheless, hypoxia exposure during CCR training may still be useful in demonstrating to the CCR diver the insidious and life-threatening danger of the condition. If a diver can recognise their hypoxia signature but cannot perform a bailout self-rescue, the diver should dedicate their efforts to reduce the likelihood of developing hypoxia in the first place. Furthermore, our finding that 3/20 (15%) subjects were unable to perform the simple self-rescue intervention during the blinded hypoxia trial, despite receiving a written command to bailout upon reaching 75% oxygen saturation, is alarming. This underscores the paramount importance of preventing hypoxia as well as the need for detection and prompting to bailout at a much less severe degree of hypoxia. Efforts to decrease the risk of hypoxia include properly maintaining gear, formulating clear dive plans, adhering to the buddy system, and using robust pre-dive checklists.

Future efforts will aim to repeat these trials in the same subjects after one year or more to determine if this first set of trials has provided a training benefit and whether the hypoxia signature remains reproducible. Additionally, we hope to perform this experimental protocol under hyperbaric conditions to investigate whether a normobaric hypoxia signature is reproducible and can act as a surrogate for the underwater environment.

## LIMITATIONS

This study was performed in a laboratory under normobaric conditions and may not completely mimic real world diving activity. The subjects' upright posture, lack of a wet suit or dry suit, lack of face mask, and absence of thermal stressors all may have effects in actual dives for which we did not account in this study. Real world scenarios likely will induce even worse performance than what we observed in our study. The monitoring we performed using a fingertip pulse oximeter, furthermore, represents a delayed measure of tissue hypoxia, particularly in the brain. Fingertip pulse oximeters can additionally be negatively affected by factors such as vasoconstriction, fingernail polish, and skin tone. In addition, we were unable to complete our original plan for 30 subjects due to the COVID-19 pandemic to ensure sufficient diversity, especially regarding gender among our subject groups. Nonetheless, the data presented here are rather provocative with a clear difference in the performance of the unprompted bailout by CCR divers versus scuba divers as well as in the performance improvement between the unblinded and blinded trials. Thus, we suspect an additional five subjects in each group would not significantly change the study conclusions regarding our hypotheses.

## Conclusions

Although our data support a normobaric hypoxia signature among divers under our experimental conditions, 55% of our diver subjects (11/20) were unable to recognise hypoxia in real time and perform self-rescue only hours after an unblinded demonstration of hypoxia symptoms. Further study is needed to determine the intrapersonal reproducibility of the hypoxia signature over time and under hyperbaric conditions. Additionally, further investigation is required to determine the ability to train the recognition of one's hypoxia signature and, in turn, if that recognition can lead to higher self-rescue rates among divers.

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# A review of accelerated decompression from heliox saturation in commercial diving emergencies

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**Abstract**
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**Introduction:** Saturation diving is a specialised method of intervention in offshore commercial diving. Emergencies may require the crew to be evacuated from the diving support vessel. Because saturation divers generally need several days to reach surface, the emergency evacuation of divers is based on dedicated hyperbaric rescue systems. There are still potential situations for which these systems cannot be used or deployed, and where an emergency decompression provides an alternative solution.
**Methods:** Our objective was to describe historical cases and assess the benefit of emergency decompressions, with the collection of data from the authors' direct experience and networks, providing witness or first-hand information.
**Results:** We documented three cases of emergency decompression following bell evacuations, and six cases of accelerated decompression performed in the chamber or hyperbaric rescue chamber. Review of these cases showed: 1) the complicated nature of such emergencies that make decisions difficult; 2) the variety of solutions implemented; and 3) the surprisingly safe and successful outcomes of several operations. Analysis of the accelerated decompression occurrences allowed derivation of the options used; upward initial excursion, increased chamber partial pressure of oxygen associated to increased ascent rates, and inert gas switching. We identified four published procedures for accelerated decompression.
**Conclusions:** Despite modern hyperbaric rescue systems, accelerated decompression remains an essential tool in case of emergency. The diving industry needs clear guidance on what can be achieved, depending on the saturation depth and the level of emergency.

## Introduction

Saturation diving is a specialised but common method in commercial diving. While working at sea, undesired events may occur requiring crew evacuation. In such situations, it is impossible for saturation divers to be evacuated at atmospheric pressure. They must perform a decompression that will take several hours to days before reaching surface pressure.

Equipment for hyperbaric evacuation has evolved; initially, the diving bell was the only option. In the late 1970s, diving companies in the North Sea developed hyperbaric rescue chambers (HRCs) i.e., floating chambers to be deployed overboard. Later, the concept evolved into a self-propelled lifeboat containing a chamber capable of accommodating the full dive team with support crew and gas reserve, until recovery and connection to a specific life support package (LSP) could be achieved.

Although some saturation diving projects still proceed without meeting state-of-the-art criteria, a modern hyperbaric evacuation system is based on the following:

1. Transfer of the divers from the endangered saturation chamber system to the connected self-propelled hyperbaric lifeboat (SPHL).
2. Disconnection and launch of the SPHL.
3. Recovery and transport of the SPHL on a nominated rescue vessel carrying a LSP to be connected to the SPHL for additional breathing gas, control equipment, thermal balance capabilities, power supply, etc.
4. Final connection of the SPHL to the hyperbaric reception facility (HRF); located either onshore or offshore, on board a suitable vessel or facility.

5. Decompression in controlled conditions inside the HRF where medical care can be provided.

The framework for the design of the equipment was provided by guidance notes from international organisations and industry trade associations. The first guidelines were published in 1998 by the International Maritime Organisation (IMO).[1] In 2013, the International Marine Contractors Association (IMCA) published the D052 guidance note that became the reference for the offshore oil and gas industry (now available as rev 2018-08).[2] Subsequently, in 2014, the International Oil and Gas Producers (IOGP) issued requirements for hyperbaric evacuation.[3]

However, events still occur where an accelerated decompression offers the only option for bringing the divers back to surface pressure. For example (see later case studies), the wave height did not allow the launching of the Resolute's HRC, while fire rendered the Samudra Suraksha's SPHL unusable, so accelerated decompression became the only realistic alternative.

In 2011, the Diving Medical Advisory Committee (DMAC) organised a workshop on accelerated emergency decompression from saturation in commercial diving.[4] The consensus reached provided the basis of DMAC 31, a guidance note that covers risk assessment, oxygen levels, hydration and thermal balance, though not the rates of ascent.[5] Recently, the Petroleum Safety Authority (PSA) in Norway has commissioned and published a report on emergency decompression from heliox saturation.[6]

The availability of an accelerated decompression procedure should not become an excuse for poor planning and ignoring duty of care. It should never be a substitute for a specific and comprehensive hyperbaric evacuation plan. Accelerated decompression exposes divers to a high oxygen dose and a greater risk of decompression sickness (DCS).

To preserve the knowledge learned from past incidents, we have reviewed the known cases of emergency accelerated decompression. In addition, we detail published accelerated decompression procedures. The objective of this review is to recall the contextual operational justifications made during these events, to document those decompression profiles, and to assess their respective risks and benefits.

## Methods

### INCLUSION CRITERIA

This review includes historical cases where decompression was required in an emergency, for example, cases where the situation required a combination of intermediate pressurisations/depressurisations, and cases where decompressions ended with a normal decompression once the situation was stabilised. The cases are categorised into two groups:

- Bell evacuation followed by an emergency decompression, not necessarily accelerated, but differing from standard conditions.
- Emergency decompression with accelerated ascent in the chamber system, HRC or SPHL.

### EXCLUSION CRITERIA

The review excludes:

- Cases where the divers were transferred into a bell, HRC or SPHL but not actually decompressed, as the event improved quickly after a transfer back to the main chamber system.
- Cases of emergency decompression for medical evacuation. Such cases involve different decision pathways and responsibilities.

### SOURCES

Data were drawn from the literature, books and reports, or use of our network to contact direct or indirect witnesses.[7] These data are not exhaustive, as there may be cases that we are unaware of. All the contributors have reviewed the manuscript and approved the inclusion of their data and the use of their names.

### UNITS

By convention, the pressure unit most used in saturation diving is metres of seawater (msw). The original data for these case studies have been reported in msw and to facilitate comparison we have retained this throughout the description and converted feet of seawater (fsw) to msw using the USN Navy conversion factor of 1 fsw = 0.30643 msw.

## Case studies

### REVIEW OF BELL EVACUATIONS WITH EMERGENCY DECOMPRESSION

*1975, Discovery one, Comex, Nigeria*

**Source:** Internal Comex account archived by the '*Club des Anciens de Comex'*. Reviewed by author (JPI).

The drill ship 'Discovery One' was working offshore Nigeria. Two divers had returned from a bell bounce dive to 90 msw and were finishing their decompression in the deck chamber. A drilling blowout occurred. All power sources were shut down to avoid fire. Because the seawater/gas emulsion threatened to sink the ship, everyone on board evacuated except for the dive team. They managed to attach a cable from a supply boat to the diving bell. The divers then transferred into the bell, the cables and umbilical were cut,

and the supply boat pulled until the bell was finally torn off the deck.

The bell remained hanging below the supply boat for one day. Finally, a crane was found that put the bell onto the supply boat deck allowing a decompression to be planned and then completed in Port Harcourt. The divers controlled the oxygen level in the bell using the manual metabolic oxygen make-up system. Two days were spent decompressing in the full heat on the deck, with fire pumps spraying sea water over the bell to reduce its temperature. As there was no bell emergency lock at the time, divers were fed with soup served through a hose and a set of skin valves. They finally reached surface without DCS. The Comex team used the 1974 standard decompression protocol after an initial 'pull up' of 10 msw.

*1982, Taipan one, Comex, Gabon*

**Source:** JPI interview with Michel Plutarque, '*Club des Anciens de Comex*'.

In September 1982, the Comex barge 'Taipan One' was working in Cameroon alongside a single point mooring buoy. Diving operations were in progress at around 30 msw. Welders were working on the deck and a fire started from an oil leak. The crew managed to cut the anchor lines and a supply boat pulled the barge away from the buoy. In the process, the bell was dropped to the bottom and lost.

A rescue diver from a nearby diving support vessel (DSV) found the bell half submerged in the mud and after cleaning the porthole, saw the divers were alive. The bell was recovered on to the deck of a supply boat that sailed to Douala. In the meantime, a saturation chamber was mobilised in the harbour. After 24 h, the bell was clamped to the chamber and the two divers finished their decompression using normal saturation procedures and without any symptoms of DCS.

This accident is the first that we are aware of to illustrate the chain of hyperbaric evacuation, onshore reception facility and decompression.

*1985, Garupa PGP-1 Platform, Comex - Marsat, Brazil*

**Source:** JPI interview with Jean Francois Irrmann, Brazil Comex diving manager at the time, '*Club des Anciens de Comex*'.

The PGP 1 platform on the Garoupa field, offshore Campos in Brazil, had a saturation system with four Comex divers at 126 msw when a gas leak occurred. The platform was abandoned, and only key personnel remained on site. The divers' evacuation was organised by wet transfer from bell to bell with the nearby DSV Stena Workhorse, which had a Marsat team in saturation at around the same depth. The vessel came alongside the platform, but the captain was reluctant to get too close. Fortunately, at that time, diving bells in Brazil used 120 m long umbilicals. The two bells were lowered to 120 msw and a Stena diver installed a swim line in between them. The four Comex divers were transferred in a single dive and the six divers found themselves squeezed into the very small Stena bell.

Once back on deck and clamped to the Stena system, the opening of the bell door took over 25 minutes because all divers were standing on it. Some divers had to climb into the upper part of the bell before the door could be opened. The team was finally decompressed according to Marsat saturation procedures, which at the time used an adaptation of the US Navy diving manual procedure.

## REVIEW OF EMERGENCY EVACUATIONS WITH ACCELERATED DECOMPRESSIONS

*1981, Norjarl Semi Sub, Oceaneering, North Sea*

**Source:** JPI interview with Dr Philip James, who was directly involved in the emergency management.

In February 1981, the semi-submersible 'Norjarl' barge, operated by Oceaneering, had four divers in saturation at 87 msw. The barge collided with a supply boat. One of its hulls was damaged below the water line. The barge began to list. She was then ballasted, and it was decided to attempt to tow her to Norway for repair. Due to the risk of capsizing, Dr James started an upward excursion according to the US Navy tables (87 msw to 63 msw). He then initiated an accelerated decompression using an elevated chamber $PO_2$ of 75 kPa and an ascent rate three times faster than the standard Oceaneering ascent at that time. He specified that the divers should drink one litre of water per hour.

During the transfer to Norway, a storm threatened the safety of the barge. Dr James's plan was to reach 18 msw and finish the saturation decompression with a US Navy Table 6. Fortunately, 24 h later the weather improved, the situation stabilised, and the end of the saturation was conducted without having to switch to Table 6 (see Table 1). There were no symptoms of DCS in any of the divers.

**Table 1**

Summary of the Norjarl emergency decompression; $FO_2$ – inspired fraction of oxygen; msw – metres of seawater; $PO_2$ – inspired pressure of oxygen

| Depth (msw) | Breathing gas | Ascent rate (msw·h$^{-1}$) |
|---|---|---|
| 63.0–49.5 | Heliox $PO_2$ = 80 kPa | 4.5 |
| 49.5–18 | $PO_2$ = 80 kPa | 3.6 |
| 18–0 | $FO_2$ = 23% | 1.8 |

**Table 2**
Summary of the DLB 269 emergency decompression; BIBS – built in breathing system; BIBS 20/5 – BIBS 20 min, chamber gas 5 min; DCS – decompression sickness; $FO_2$ – inspired fraction of oxygen; msw – metres of seawater; $PO_2$ – inspired pressure of oxygen

| Depth (msw) | Breathing gas | Ascent rate (msw·h$^{-1}$) | Comments |
|---|---|---|---|
| 30–20 | Chamber gas (heliox) $PO_2$ = 60 kPa | 1.2 | Normal decompression 16 h ascent per day |
| 20–10 | BIBS 20/5 BIBS heliox, $FO_2$ = 50% | 1.5 | Start of accelerated decompression |
| 10–3 | BIBS 20/5 BIBS $FO_2$ = 100% | 1.5 | |
| 3 | Chamber gas | Hold | 110 min stop |
| 3–0 | BIBS BIBS $FO_2$ = 100% | Unknown | Described as a slow ascent |
| Surface | BIBS 10 min, air 20 min for 6 h BIBS $FO_2$ = 100% | Hold | No DCS symptoms reported |

*1981, Sedco Phillips Semi Sub, Oceaneering, Ekofisk Field, North Sea*

**Source:** JPI interview with Dr Philip James, who was directly involved in the emergency management.

This incident was related to one of the worst storms recorded in the North Sea. In November 1981, the semi-sub barge 'Sedco Phillips' was operating with Oceaneering in the Ekofisk field when she was hit by the storm. The situation became critical. The barge had eight divers in saturation at a depth of 70 msw. The decision was made to transfer the divers into the HRC and to disconnect from the system. However, the HRC was not launched as the waves were breaking over the crane. Dr James directed an accelerated saturation decompression on the same principle as for the Norjarl event described previously. The divers reached surface with no DCS symptoms.

*1981, Transworld 58 Semi Sub, Argyll Field, North Sea*

**Source:** JPI interview with Dr Philip James, who was directly involved in the emergency management.

During the same November 1981 storm, the Transworld Rig 58 broke all anchor lines and drifted for several hours in hurricane winds. Four divers were in saturation on-board at 30 msw. Dr James initiated an upward excursion to 18 msw at 6 msw·min$^{-1}$. Decompression then proceeded at 1.2 msw·h$^{-1}$ to surface with a progressive gas switch from heliox to air. Divers were instructed to drink 1 L of liquid per hour. The divers reached surface with no DCS symptoms.

*1995, DLB 269, McDermott, Mexico*

**Source:** the book by Michael Krieger "*All the men in the sea*"[8] and author (PB) personal communication with Tim Cheshire and Tony Greenwood.

The McDermott derrick lay barge 'DLB 269' was finishing a tie-in offshore the Bay of Campeche at 48 msw, when a tropical storm turned into hurricane 'Roxanne'. The barge master decided to face the storm with two tugs pulling the barge to maintain position. The divers' decompression was initiated with normal procedures, as they thought they had three or four days before the storm would arrive. However, onshore support was contacted to obtain an accelerated decompression profile and a procedure was faxed back with input from Dr Russ Petersen and Dr Bill Hamilton. The hurricane moved faster, and six hours before Roxanne was due to reach the DLB 269, the divers agreed to be decompressed via this emergency procedure. The most likely profile for the DLB 269 decompression is presented in Table 2. The divers surfaced in the middle of the storm without any symptoms. The following day, Roxanne moved away to the North.

Two days later, Hurricane Roxanne turned back and hit DLB 269 again. The hull developed several leaks and water filled compartments; tow lines parted one after the other. Anchors were dropped but did not hold. The bow slowly went into the water, swept by giant waves. The crew had to abandon the barge before it sank. Six people lost their lives.

*2005, S. Suraksha, Bombay High Field, India*

**Source:** Dr Ajit Kulkarni who was directly involved in the emergency management.[9] This is an updated report following the discovery of further information.

A cook cut his finger onboard the 'S. Suraksha' diving support vessel working on the Bombay high field in India. It

was decided to evacuate the patient with a crane basket to the nearby Bombay high north platform. During manoeuvring, the vessel struck a gas riser. Both the platform and the vessel caught fire.

The S. Suraksha had six divers in saturation at two levels; the deepest storage depth being 42 msw. The deepest operating depth in the area is 85 msw and the SPHL was kept pressurised at that depth. As the vessel was on fire, the diving superintendent asked the divers to be pressurised to 85 msw to enter the SPHL. However, the first diver to enter could see the flames through the port hole and the bulkhead was hot; the SPHL was on fire. The divers returned to the living chamber. The trunking had heated considerably and two of the divers sustained burn injuries. Inside the chamber, the internal depth gauge indicated 70 msw. The vessel was abandoned. The emergency power supply in the saturation control failed. Left alone, the divers managed to decompress themselves to 54 msw using the bilge valve.

During the night, the diving superintendent of another vessel, the 'S. Prabha', which had been fighting the fire, boarded the S. Suraksha. All divers reported that they were OK. Communication was established using a sound-powered telephone. The diving superintendent and the life support technicians (LSTs) who had been rescued by a supply boat, came back on board the S. Suraksha. After flushing through the system and passing fruits and fluids in, they started decompression.

When Dr Kulkarni arrived on-board in the morning, the LSTs had decompressed the divers from 54 to 34 msw using gas mixtures available on board. The fire had not been extinguished completely, however the vessel did not appear to be in imminent danger. An 8 h hold was decided because the divers had undergone severe pressure variations in the previous 24 h. After the hold, the decompression resumed according to standard procedures without stops.

During the night, the fire erupted again at which time the system was at 23 msw pressure. The LSTs raised the chamber $PO_2$ to 60 kPa and abandoned the vessel. The divers were instructed to decompress at 3 msw·h$^{-1}$. The next morning, when Dr Kulkarni and the LSTs could board the vessel, the chambers were pressurised at 11 msw. The situation was deteriorating rapidly; the list of the vessel had increased, probably from ingress of firefighting water. It was then decided to carry out an abort decompression and transfer the divers to the nearby S. Prabha that was engaged in firefighting but also had divers in saturation. These divers had been decompressing for the past two days and were at shallow depth. The abort decompression was delayed for 45 min to allow the S. Prabha to recompress its divers to 30 msw. The S. Suraksha divers were rapidly decompressed to surface, jumped in a lightweight inflatable boat and arrived on-board the S. Prabha where they were immediately pressurised to 30 msw in the saturation system where they met with the other S. Prabha divers. One diver complained of pain in knee which relieved on reaching 30 msw. Later, all the divers surfaced safely.

The information collected from Dr Kulakarni's report permits reconstruction of the emergency decompression which is presented in Table 3.

*2013, Barge Resolute, East Java, Indonesia*

**Source:** Dr Phil Bryson and Dr Jean Yves Massimelli who were directly involved in the management of the emergency.[10]

In January 2013, the 'Resolute', a pipelay barge equipped with a mobile saturation diving system, lost anchors in bad weather offshore Jakarta. Six divers were in saturation being held in the main chamber at 45 msw, while three other divers were passing 28 msw during their decompression from saturation. These three divers were in the HRC that was being used as a living chamber. Containers and heavy gas cylinders had been wiped out by the waves and were crushing other deck equipment. The dive control station was flooded. While the rest of the barge's crew were already at the muster station preparing themselves to abandon ship, all members of the diving team were present on deck, to protect the saturation diving system. The diving superintendent noted the seriousness of the weather with the winds and massive waves slamming into the barge. He felt that there was a significant risk of the HRC losing its seal with the rest of the saturation system as well as the risk of capsizing.

He decided to recompress the three divers in the HRC and then transfer the six divers from the main chamber into the HRC. The HRC was then compressed with all the divers to 80 msw (seabed depth + 20 msw) to secure the seal. The HRC was disconnected from the system. However, launching the HRC in such a sea state would have led to the HRC being crushed against the hull. A decision was made to delay the decompression and to wait for the anchor-handling tug to hook up a tow line which, eventually, was successfully completed. Thereafter, the barge came back to level and could keep a more stable position. With the immediate danger of capsizing removed, the diving superintendent instructed the HRC to be re-connected to the surface supply and the divers to remain in the HRC.

The circumstances remained perilous and unpredictable with the safety of the barge still at risk. An accelerated decompression was initiated under the shore guidance provided by the company medical advisors and by Dr Bryson. The situation was continuously monitored by the offshore and onshore teams who had acknowledged that, following surfacing, it would have been practically impossible to re-compress the divers as the diving system was damaged. Communication was difficult due to the weather and on-site conditions. Near the end of the decompression, these concerns and the improved barge stability were conducive to reducing the decompression rate and enforcing a hold

**Table 3**
Summary of the S. Suraksha emergency decompression; BIBS – built in breathing system; $FO_2$ – inspired fraction of oxygen; LST – life support technician; msw – metres of seawater; $PO_2$ – inspired pressure of oxygen; USN – United States Navy

| Depth | Breathing gas | Ascent rate | Comments |
|---|---|---|---|
| Initially 28 and 42, compressed to 85 msw | | | Two separate teams of divers compressed to deepest operating depth in the area |
| 85–54 msw | Chamber gas (heliox) $FO_2$ = 6% (uncertain) | ~4–5 msw·h$^{-1}$ | Empirical decompression carried out by the divers |
| 54–34 msw | Chamber gas $FO_2$ = 8–12% | 2.50 msw·h$^{-1}$ | Decompression during the night, under the control of the LSTs on site<br>No power, no scrubber, divers on emergency rebreather |
| 34 msw | Chamber gas $FO_2$ = 12% | Hold | Eight hour hold decided by Dr Kulkarni |
| 34–23 msw | Chamber gas $FO_2$ = 16% | 1.20 msw·h$^{-1}$ | Standard decompression under the control of LSTs |
| 23–11 msw | Chamber gas $PO_2$ = 60 kPa | 3.00 msw·h$^{-1}$ | Decompression performed by the divers |
| 11 msw | Chamber gas $FO_2$ = 20% | Hold | Decision to transfer<br>Stop for 45 min waiting on the S. Prabha to prepare for divers' reception |
| 11–2.4 msw | BIBS $FO_2$ = 100% | 1.00 msw·min$^{-1}$ | 8.6 min from 11 to 2.4 msw |
| 2.4–1 msw | BIBS $FO_2$ = 100% | 0.16 msw·min$^{-1}$ | 10 min from 2.4 to 1 msw |
| 1 msw to surface | BIBS $FO_2$ = 100% | 0.08 msw·min$^{-1}$ | 12 min from 1 msw to surface |
| Surface | | | Divers transferred to the S. Prabha |
| Recompression to 30 msw in less than 30 min | | | One case of knee pain in one diver relieved on arrival at 30 msw |
| 30 msw to surface | | USN heliox saturation diving decompression schedule | No DCS symptoms reported |

at 10 msw to help reduce the risk of DCS. This event illustrates the need for a reliable communication capability, continuous monitoring and assessment, and flexibility in these situations.

**In summary:** After 4–5 hours hold (maintaining chamber pressure unchanged), the decompression was initiated from 76 msw (the depth after cooling of the chamber following a fast compression to 80 msw).

The divers took aspirin and fluids (initially 1 L·h$^{-1}$) and managed to 'exercise' during the decompression as far as possible in a full nine-man HRC. The doses of aspirin and the quantity of water were not accurately recorded. The decompression is presented in Table 4.

Medical examinations were conducted by the barge's medical officer following surfacing, and then by the diving medicine specialist in Singapore, one week later. No signs or symptoms of DCS or pulmonary oxygen toxicity were seen. All divers resumed their commercial diving careers.

Immediately after the initial incident notification, the medical assistance provider had mobilised an airplane able of maintaining a 1 atmosphere cabin pressure in flight. If a medical evacuation to a recompression facility had been required, it would have been carried out in optimum conditions.

Table 4 and Figure 1 display the $PO_2$ breathed by the divers along the ascent. The overall UPTD (units of pulmonary toxicity dose) exposure was 1265 UPTD during the decompression.

**Table 4**

Summary of the Resolute emergency decompression; 20/5 – BIBS 20 min, chamber gas 5 min; 25/5 – BIBS 25 min, chamber gas 5 min; BIBS – built in breathing system; $FO_2$ – inspired fraction of oxygen; msw – metres of seawater; $PO_2$ – inspired pressure of oxygen

| Depth (msw) | Breathing gas | Ascent rate (msw·h$^{-1}$) | Comments |
|---|---|---|---|
| 76–55 | Chamber gas (heliox) $PO_2$ = 60 kPa | 7.8 (average) | Ascent of 21 msw performed in 2 h 42 min |
| 55–43 | Chamber gas $PO_2$ = 60 kPa BIBS 20/5, 5 sessions heliox, $FO_2$ = 20% | 5 | Decompression |
| 43–20 | Chamber gas $PO_2$ = 60 kPa BIBS 20/5, 2 sessions BIBS 25/5, 7 sessions heliox $FO_2$ = 35% | 5 | Decompression |
| 20 | Chamber gas $PO_2$ = 60 kPa BIBS 25/5, 2 sessions heliox, $FO_2$ = 50% | Hold | 3 h 35 min hold |
| 20–16 | Chamber gas $PO_2$ = 60 kPa | 1 | Decompression |
| 16–10 | Chamber gas $FO_2$ = 23% | 1 | Decompression |
| 10 | Chamber gas $FO_2$ = 23% BIBS 25/5, 3 sessions $FO_2$ = 100% | Hold | 5 h hold |
| 10–0 | Chamber gas $FO_2$ = 23% After 4 h chamber gas BIBS 20 mins every 2 h to surface. $FO_2$ = 100% | 0.5 | Decompression No DCS symptoms reported |

**Figure 1**

Depth (left axis) and inspired $PO_2$ (right axis) time profile of the Resolute emergency decompression

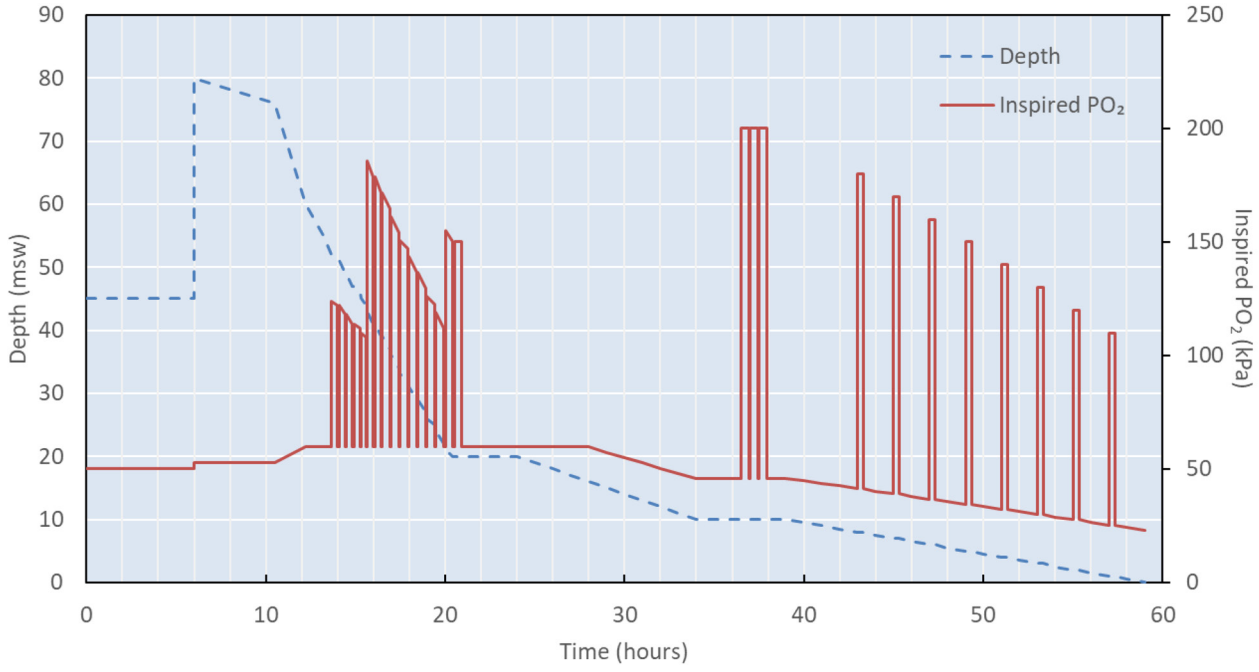


**Table 5**
Comex 1974 heliox saturation decompression; $FO_2$ – inspired fraction of oxygen; msw – metres of seawater; $PO_2$ – inspired pressure of oxygen

| Depth (msw) | Chamber gas | Ascent rate (min·msw$^{-1}$) | Ascent rate (msw·h$^{-1}$) |
|---|---|---|---|
| 280–240 | $PO_2$ 60 kPa | 20 | 3.0 |
| 240–160 | | 25 | 2.4 |
| 160–80 | | 30 | 2.0 |
| 80–20 | | 35 | 1.7 |
| 20–15 | | 40 | 1.5 |
| 15–10 | $FO_2$ 24% | 40 | 1.5 |
| 10–5 | | 45 | 1.3 |
| 5–0 | | 50 | 1.2 |

**Table 6**
US Navy diving manual Rev 7, 2016, emergency abort decompression; $FO_2$ – inspired fraction of oxygen; msw – metres of seawater; $PO_2$ – inspired pressure of oxygen

| Depth (msw) | Chamber gas | Ascent rate or duration |
|---|---|---|
| Decompression from 306.4–83.7 msw with 60 kPa chamber $PO_2$ | | |
| 306.4–61.3 | $PO_2$ = 60 kPa | 1.53 msw·h$^{-1}$ |
| 61.3–16.1 | | 0.88 msw·h$^{-1}$ |
| 16.1–1.2 | $FO_2$ = 23% | 0.88 msw·h$^{-1}$ |
| 1.2–0 | | 4 min |
| Decompression from 83.4–62.5 msw with 70 kPa chamber $PO_2$ | | |
| 83.4–61.3 | $PO_2$ = 70 kPa | 1.67 msw·h$^{-1}$ |
| 61.3–20.4 | | 0.97 msw·h$^{-1}$ |
| 20.4–1.2 | $FO_2$ = 23% | 0.97 msw·h$^{-1}$ |
| 1.2–0 | | 4 min |
| Decompression from ≤ 62.2 msw with 80 kPa chamber $PO_2$ | | |
| 62.2–61.3 | $PO_2$ = 80 kPa | 1.67 msw·h$^{-1}$ |
| 61.3–24.8 | | 1.02 msw·h$^{-1}$ |
| 24.8–1.2 | $FO_2$ = 23% | 1.02 msw·h$^{-1}$ |
| 1.2–0 | | 4 min |

**Table 7**
Italian accelerated decompression procedure; $FO_2$ – inspired fraction of oxygen; msw – metres of seawater; $PO_2$ – inspired pressure of oxygen

| Depth (msw) | Chamber gas | Ascent rate (msw·h$^{-1}$) |
|---|---|---|
| 180–90 | $PO_2$ = 65 kPa | 3.0 |
| 90–30 | | 2.4 |
| 30–18 | | 1.2 |
| 18–0 | Air flushing to never exceed an $FO_2$ of 23.5% | 0.6 |

**Table 8**
Comex accelerated saturation decompression procedures; $FO_2$ – inspired fraction of oxygen; msw – metres of seawater; $PO_2$ – inspired pressure of oxygen

| Depth (msw) | Chamber gas | Ascent rate (msw·h$^{-1}$) |
|---|---|---|
| Decompression from not deeper than 130 msw | | |
| 130–16 | $PO_2$ = 60 kPa | 1.4 |
| 16–0 | $FO_2$ = 23% | 0.6 |
| Decompression from not deeper than 90 msw | | |
| 90–20 | $PO_2$ = 70 kPa | 1.6 |
| 20–15 | $FO_2$ = 23% | 1.2 |
| 15–0 | $FO_2$ = 23% | 0.6 |
| Decompression from not deeper than 70 msw | | |
| 70–25 | $PO_2$ = 80 kPa | 1.7 |
| 25–15 | $FO_2$ = 23% | 1.2 |

## REVIEW OF AVAILABLE ACCELERATED DECOMPRESSION PROCEDURES

*Early Comex saturation decompression procedures*

In the early 1970s, decompressions that were considered as standard procedures appear today as excessively fast ascents. Unfortunately, the procedures at the time were a mixture of bounce and saturation diving and cannot be directly translated into modern practice. However, some profiles provide useful references to what can be done in terms of rapid decompression.

In 1974, Comex published their first set of original heliox saturation procedures that were used until 1979. The ascent could be initiated by a 10 msw upward excursion depending on the last dive interval. Decompression was continuous over 24 hours. Chamber oxygen was controlled to a $PO_2$ of 60 kPa when deeper than 15 msw, and then adjusted to a $FO_2$ of 24% when shallower. It took five days and 16 hours to decompress from 280 msw storage depth to surface (Table 5). The overall safety performance based on data from the Comex database indicated a DCS risk of 5 to 10%; all symptoms were related to joint pain occurring in the last 10 msw of ascent.[11]

*US Navy 2016 emergency abort procedures*

Revision 7 of the US Navy diving manual,[12] paragraph 13.23.7.2, provides a specific procedure for emergency abort decompression, defined for serious life-threatening emergency, however, no information is provided on its validation. The emergency ascent includes several phases: an initial upward excursion, a hold, and an accelerated decompression (Table 6).

The ascent rates are defined (Table 6) according to the starting depth, which decides the chamber $PO_2$. These ascent rates appear very slow compared to the emergency situations studied and seem of little practical use. We could not find any instance when these procedures were used.

*Italian accelerated decompression procedures*

An accelerated decompression procedure can be found in the Italian UNI 11366 diving regulations.[13] The procedure has continuous decompression varying with depth and constant chamber $PO_2$ until 18 msw when the chamber is flushed with air to change from helium to nitrogen (Table 7). We could not find any instance when these procedures were used.

*Comex emergency decompression procedure*

In the 1994 revision of its diving manual, Comex introduced an accelerated decompression procedure that provided three options depending on the starting depth. These procedures were based on a higher level of chamber $PO_2$ and thus allowed faster ascent rates. Considering pulmonary oxygen toxicity as the limiting factor, the $PO_2$ selected controlled the maximum decompression time, and therefore the depth of use. Three depth ranges were proposed: 70 msw, 90 msw and 130 msw, with their respective chamber $PO_2$. For an emergency deeper than 130 msw, the only possibility was to decompress the divers to 130 msw using standard saturation decompression and then consider the possibility of using an accelerated decompression to the surface (Table 8).

An option was available where decompression could be further accelerated by putting the divers on a higher $FO_2$ via the built-in breathing system (BIBS) during the last 10 msw of the ascent to the surface. The ascent rate could be increased to 60 min per msw. To our knowledge, these procedures have never been used by Comex.

## Discussion

### THE EVENTS

Weather was clearly a critical factor in four out of the six incidents discussed. It prevented the evacuation via an HRC in the Sedco Phillips SS, the Transworld 58, the DLB 269 and the Resolute cases. Accurate planning and preparedness are critical in risk management.

It is notable today that HRC's are not accepted in the UK or Norwegian sectors of the North Sea and other regions due to their limitations of life support and seaworthiness.

### THE OPTIONS

Faced with an event requiring an emergency decompression, a commercial diving company will mobilise its safety response network and involve the diving medical advisor in the decision-making process. The decisions will be

made on information received via telecommunication systems, generally with limited real time knowledge of the actual situation and its evolution. The circumstances are often dramatic and changeable, with emotional pressure to manage. History has shown that decisions often must be revised promptly according to the development of the situation.

Upon deciding whether to use an emergency decompression, the first consideration will be the depth of the divers. An accelerated decompression is only useful if the divers are close enough to the surface and the time scale allows them to be brought to safety. If these criteria are fulfilled, then methodological options for the rescue would be:

- To decide on a starting depth. The situation may require the recompression of a team in decompression or at a different storage depth to a deeper depth.
- To perform a rapid large excursion to get the divers closer to the surface. However, too great an excursion might cause DCS and impair further decompression.
- Decompress with increased ascent rates. However, too rapid an ascent rate might cause DCS.
- Decompress with an increased $PO_2$ to allow faster ascent rates. However, too high an oxygen exposure might induce oxygen toxicity.
- Possibly store the divers at a depth close to the surface waiting for the best time to evacuate.
- A combination of the above.

The decision is therefore a balance between the time left to decompress to surface and the accepted risk of DCS and/or oxygen toxicity. This may lead to a graded response where two levels of emergency could be considered:

- A 'level one emergency' where time is available and a fast, but still reasonable ascent rate could be employed to minimise the DCS risk.
- A 'level two emergency' where the immediate integrity of the system is at risk and a life-threatening situation involves the whole saturation team. This could justify an aggressive ascent protocol and the acceptance of a higher risk of DCS and oxygen toxicity.

Finally, operational constraints must be evaluated:

- Feasibility:
    - Are communications reliable enough to direct the decompression?
    - Is the diving support vessel a safe place to decompress, and for how long?
    - Are LSTs present?
- Acceptability:
    - Can the divers be informed of the options and involved in the decision?
- Control of decompression:
    - Is the chamber atmosphere breathable?
    - Can a breathing mix be supplied on BIBS?
    - Is the chamber temperature within limits?
- Treatment options:
    - In case of DCS, would it be possible to treat a diver during the emergency decompression or would the diver have to wait until he is evacuated to a hyperbaric facility?
    - How long would it take to take the divers to a nearby vessel of opportunity or a shore-based facility equipped with a saturation diving system?

## INITIAL EXCURSION

In several recorded instances, the immediate strategy was to perform a rapid upward ascent or excursion to bring the divers closer to surface. This protocol is described in the US Navy diving manual (paragraph 13–23, revision 7) that allows the start of a final decompression to begin with an upward excursion. The excursion amplitude can be quite significant, for example, a 30 msw ascent from 120 msw to 90 msw.

Diving companies have become more cautious about upward excursions. This is because the data from the Comex diving database, the Hades database from Seaways, and the US Navy have all shown that too great an excursion may induce vestibular DCS symptoms, which could have a dramatic impact on the rest of the emergency management.[11,14,15]

One way of controlling the risk of DCS is to perform this initial ascent at a slower rate, as during the Resolute case (approximately 7.8 msw·h$^{-1}$). Alternatively, the divers may be kept at constant depth for a while after the excursion, as per the US Navy abort decompression procedure, which requires a two hour hold before any further ascent.

## FINAL EXCURSION

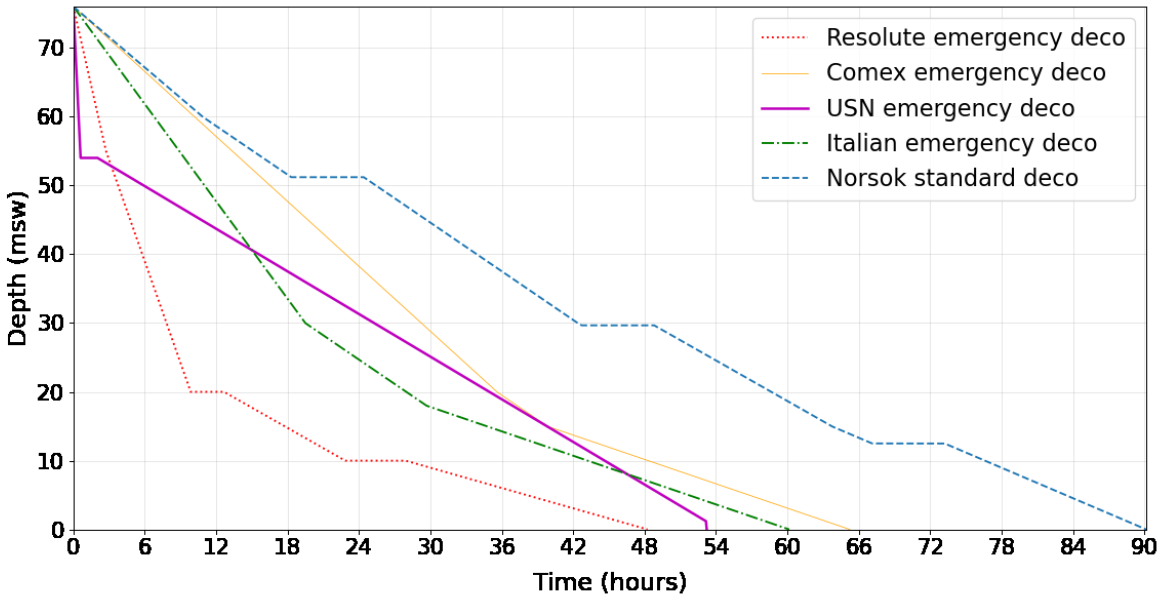
Another documented emergency decompression strategy consists of decompressing the divers to a depth close to surface and keeping the divers at this depth until the situation is controlled. The 'holding' depth was 10 msw during the Resolute case, 3 msw during the DLB 269 case, and 11 msw during the S. Suraksha case. This hold has the advantage of stabilising the divers in terms of decompression, providing a higher $PO_2$ on BIBS (if required for a DCS treatment) and still permitting a rapid escape to surface if needed. The S. Suraksha case showed that divers could ascend from 11 msw to surface in 30 minutes and then be recompressed to 30 msw in a nearby vessel system, with only one case of DCS (pain only) among six saturated divers.

## FASTER ASCENT RATES

During decompression, the ascent rate and the inhaled $PO_2$ are closely related. This relationship is linear, according to Vann's model.[16] With the use of data from commercial saturation decompressions, a regression line has been

**Figure 2**

The depth/time profile of the Resolute emergency decompression compared to the Comex, US Navy and Italian emergency decompression procedures for the same starting depth. The Norsok standard saturation profile is added to allow a comparison to a standard saturation decompression. One profile includes an initial upward excursion to initiate the ascent (24 msw for the US Navy Procedures)




established between the safe rate of ascent and chamber $PO_2$ in the deeper part (> 60 msw).[17] This is the design principle of the US Navy and Comex emergency procedures that propose three values of chamber $PO_2$ associated with three different ascent protocols. To control oxygen toxicity, each decompression $PO_2$ is associated with a time limit, translated into a limitation in starting depth.

To compare emergency protocols, we first considered the Resolute case and displayed its actual depth/time profile (Figure 2). We then added the profiles of the US Navy, Italian and Comex emergency decompressions, for the same starting depth. The Norsok profile was also added to provide a reference associated with a standard and conservative saturation decompression.[18]

Two strategies emerge from this figure. The US Navy and Comex procedures have relatively slow decompression rates (1.5 to 1.8 msw·h$^{-1}$) and are adapted to the evacuation of a diver with an injury or an illness, where the risk of DCS must be controlled. These situations we class as Level 1 emergencies. The figure shows that ascent rates can be significantly increased in a life-threatening situation. On board the Resolute, the decompression was initiated with an upward excursion at approximately 7.8 msw·h$^{-1}$ from 76 msw to 55 msw and then continued at 5 msw·h$^{-1}$ from 55 msw to 20 msw. This situation represents a Level 2 emergency, and these are imbued with a higher risk of DCS and oxygen toxicity, which are accepted given the circumstances.

Estimation of DCS risk is a key decision factor. For standard saturation decompressions not exceeding 200 msw, a study using data from the Comex database, based on 60 kPa chamber $PO_2$, showed that DCS cases were associated with pain symptoms alone, which occurred in the last part of the ascent.[19] Therefore, with Level 1 emergency decompression, the risk seems to be limited to mild DCS. For deeper dives, three cases of vestibular symptoms have been reported during historical deep experimental dives with an initial rapid decompression. These included: a Comex PLC I dive made in 1968, from 335 msw, with an initial ascent rate at 3.5 msw·h$^{-1}$; in 1971, a Royal Navy RNPL 457 msw (1500 feet of seawater) dive, varying ascent rates starting at 12 msw·h$^{-1}$;[20] and in 1974, a Comex Physalie VI dive, 610 msw, initial ascent rate at 2.4 msw·h$^{-1}$.

With Level 2 emergency decompressions, a tangible risk is vestibular symptoms associated with DCS. Current experience and algorithms do not allow the control of this risk.

## CENTRAL NERVOUS SYSTEM (CNS) OXYGEN TOXICITY

Increasing the $PO_2$ allows the ascent rate to be accelerated. However, oxygen toxicity may lead to convulsions, which are dangerous due to their sudden onset and limited warning signs that are either difficult to recognise or absent. The simplest way of managing CNS toxicity is to consider it as a matter of threshold and set limit values to the $PO_2$. During

immersion, the limit for pure oxygen breathing is set to 175 kPa.[21] In the dry environment of a deck decompression chamber, the $PO_2$ is set to 220 kPa during normal bounce diving and can reach up to 280 kPa during treatment (US Navy table 6 for instance).

Data from animal studies have documented that oxygen breathing interruptions delay CNS oxygen toxicity.[22,23] In practice, BIBS sessions are associated with interruptions, generally five minutes 'off BIBS', then 25 minutes 'on BIBS'. These breaks in oxygen breathing provide divers with the possibility to rest, talk and drink. It is believed that they also allow a recovery from CNS toxicity. Arieli's oxygen toxicity model suggests that a five-minute break after a 25-minute exposure can reduce the CNS toxicity dose by 67%, for this range of $PO_2$ breathed.[24] If Arieli's model is applied to the Resolute case scenario, a detailed $PO_2$ profile can be derived, whereby the index computed for CNS toxicity reaches a score of 80 during the BIBS sessions, but is almost zero by the end of the decompression due to recovery. The computed index remained below the threshold score of 196, which is associated with a 4% risk of CNS oxygen toxicity.

Our review has shown that in several instances, the people managing the emergency did not hesitate to provide the divers with high $PO_2$ in the BIBS breathing mix, but with interruptions to allow a safe, rapid decompression. Based on the Resolute case, it seems that sessions of 200 kPa $PO_2$ on BIBS can be managed over a two–three day decompression. It all depends on the interruptions and the expected recovery process, which is difficult to estimate. Interruptions also assume that the chamber atmosphere remains breathable, and this might not always be the case (as in the S. Suraksha event). Finally, we note that during the DLB 269 case, the BIBS sessions were continued out at surface pressure for six hours after the end of the decompression. This may be operationally difficult in some circumstances but certainly helps to protect the divers from developing DCS symptoms, especially if the divers omitted significant decompression.

In relation to CNS oxygen toxicity, benzodiazepines could, in theory, be used as secondary prevention agents. However, their prophylactic effect remains unknown. In fact, the respiratory depressant effects of these drugs could potentially lead to $CO_2$ retention,[25] which would increase the risk of CNS oxygen toxicity.[26] They would also introduce sedation into an unfolding emergency, which could have disastrous consequences. For these reasons, pre-emptive use of such drugs during emergency decompression to mitigate the risk of CNS oxygen toxicity is not justified.

## PULMONARY OXYGEN TOXICITY

Another recognised type of oxygen toxicity affects the lung (pulmonary oxygen toxicity). The symptoms include coughing, chest pain and dyspnoea. Extreme exposures may lead to pulmonary oedema.

The difficulty is setting the upper $PO_2$ limit to avoid severe pulmonary toxicity. One study exposed 12 subjects for 48 h at $PO_2$ = 105 kPa during a simulated air saturation dive.[27] Pulmonary oxygen toxicity symptoms occurred, and pulmonary function changes consisted of significant decrements in vital capacity, flow rates and diffusing capacity for carbon monoxide. Subjects showed a complete recovery in both symptoms and pulmonary function in about eight days.[27] In 1979, Comex conducted a deep saturation dive with eight divers to 450 msw. Decompression lasted 10 days and 5 h (corresponding to an average 44.1 msw per day), using 70 kPa chamber $PO_2$ from 314 msw to surface pressure. No DCS or pulmonary oxygen toxicity of note was reported (Imbert JP, personal communication 2022). These data suggest that $PO_2$ may be raised significantly in the event of an emergency, but a mathematical tool is required to evaluate this limit.

Several mathematical models can be used to estimate the pulmonary toxicity dose: the unit pulmonary toxic dose (UPTD) calculation from Clark and Lambertsen;[28] the oxygen tolerance model from Harabin;[29] and the more recent oxygen toxicity index from Arieli.[24] However, these models do not translate well to data drawn from conditions different from their validation.[30] Their weakness is multiple injury pathways and the obvious individual variability that may confound models.

The simplest model is the UPTD, which provides an immediate dose evaluation in an emergency. However, it has well-known limitations. First, it was validated with a $PO_2$ higher than 152 kPa and its prediction curves were extrapolated to the lower range of $PO_2$; it tends to overestimate toxicity in saturation diving. Second and more importantly, it does not account for any recovery. The computation of UPTD on emergency dive profiles generally leads to doses higher than 1,000 UPTD that far exceed the daily limit of 625 UPTD set for a 5% decrement in vital capacity. Arieli's toxicity index offers a new alternative, accounting for recovery.[31] It provides a more relevant dose/limit indication, but its calculation might not be practical during an emergency. We applied both models over the Resolute $PO_2$ profile and obtained a dose of 1,265 UPTD and a cumulative value of 36 with the Arieli's pulmonary index.

This overall 1,265 UPTD dose is not regarded as excessive; in the early Comex experimental dives it was documented that a dose of 1,300 UPTD was acceptable during saturation based on vital capacity measurements.[32] The index computed with Arieli's model for pulmonary toxicity reached a maximum value of 566 during the BIBS sessions but was very low by the end of the decompression. This would indicate that divers' vital capacity decrement reached 7.5% but a recovery took place.

Pulmonary oxygen toxicity remains the limitation of accelerated decompression. A high chamber $PO_2$ accelerates the decompression but can only be tolerated for a few days.

Therefore, efficient accelerated decompressions can only be carried out from depths shallower than 100 msw.

## DIVERS' HYDRATION

There is a considerable literature suggesting the importance of hydration during or after immersion. Immersion exposes the diver to heat and cold, exercise, dry gas breathing and modifies cardiac function. In particular, it has been shown that hydration before immersion reduces the level of circulating venous gas emboli post-dive.[33] However, these situations are not pertinent to saturation decompression, where the divers are in a dry environment with controlled humidity and temperature. We could not find studies on divers' hydration during saturation decompression. However, one study showed a diminution of the plasma volume and haemoconcentration between pre- and post-saturation measurements.[34]

There is a general assumption that if vascular volume is maintained, it will optimise perfusion and help to eliminate dissolved gases during decompression, thus reducing bubble formation. The DMAC report on emergency decompression from saturation recommends encouraging divers to drink as much as they can.[5] Plain water or oral rehydration mixtures are preferred.

DMAC guidance note 31 mentions possible additional treatments, such as analgesics and non-steroidal anti-inflammatory agents but acknowledges that there is no human evidence that such drugs would offer benefits.[5]

## INERT GAS SWITCHING

Inert gas sequencing (helium, nitrogen and argon) was developed in the sixties by Dr Bühlmann to accelerate gas exchange during deep bounce decompressions.[35] He reported decompression time of 22 h after a 6 h bottom time at 100 msw and 40 h decompression time after 6 h at 150 msw.[36] Another study reported 62–64 h decompression time from 220 msw with 66–68 h bottom time using an inert gas switch from 30 msw.[37]

Based on the same principle, chambers were flushed with air at around 10 msw by the end of the heliox decompression during the Predictive Study experimental dives at the University of Pennsylvania.[38] A gas switch was introduced by slowly venting the chamber with air during the 1981 Transworld 58 incident. An air switch is also prescribed in the Italian accelerated decompression procedures.

The difficulty with an inert gas switch is the control of the dynamics of the gas exchange, which depends on the physical properties of the gas and the depth of switch. When the technique is performed under controlled conditions and the decompression is previously validated, inert gas sequencing allows the design of efficient bounce tables (as for instance, historical Comex Cx 70 or Oceaneering bell bounce tables with transfer to an air-filled deck chamber). In case of an emergency, if the divers have already been subjected to an accelerated decompression, it is difficult to assess the gas kinetics without a complex mathematical model. In fact, the University of Pennsylvania stopped using inert gas switches because of the occurrence of specific DCS symptoms that were difficult to treat. In practice, inert gas switching should not be recommended in an emergency as it would add complexity to an already difficult situation, for example, at which depth should the change occur, what decompression rate after the change, and how to treat associated DCS?

## EMERGENCY RESPONSE AND RESPONSIBILITY FOR DECISIONS

Diving companies have based their emergency response on a supportive network, that includes all their departments in addition to their medical advisor. In an ideal case, all parties involved cooperate and share the decision. In real cases, the operational personnel are often in the front line before reliable communication can be established with shore-based resources. In most of the cases reviewed, the medical advisor, once contacted, had to take the decision on the emergency decompression. The authors believe that the duty of the medical advisor is too often perceived as exclusively focussed on the responsibility of making therapeutic decisions as an event is unfolding. Ideally, medical advisors should be involved from the earliest stage of project design and elaboration of diving procedures, until project completion. We noted, however, that in several cases, the divers were instructed on the available options and shared the decision on the accelerated decompression (DBL 269) or took the decision themselves (S. Suraksha). The diving industry needs optimised guidance on what can be achieved, depending on the saturation depth and the level of emergency. This guidance must be developed with the involvement of the diving teams themselves.

## Conclusions

The use of emergency decompressions procedures to substitute for appropriate resourcing, planning and the provision of reliable hyperbaric evacuation systems is not justifiable.

The present review of the literature and case studies shows that emergency decompressions have saved lives over the years and suggests that further investigations of methods to accelerate saturation decompression are of definite worth. The review includes 37 divers involved in six emergency decompression profiles with one case of articular pain. No meaningful DCS risk value can be attributed to emergency decompressions from this review considering the variety of scenarios.

Emergency decompression protocols known in the industry are derived from a limited number of original procedures. These procedures propose the following options for accelerating the decompression:

- An initial excursion
- Increased ascent rates
- Increased respired $PO_2$
- A combination of the above

The existing procedures for accelerated decompression remain conservative and could be considered for controlled situations, like the evacuation of a diver with an injury or an illness, where the risk of DCS must remain controlled. We defined these situations as Level 1 emergencies where time is of the essence but the life support system (the integrity of the diving support vessel, of the saturation diving system and of the surface-support team) has not been impaired.

We defined Level 2 emergencies as disaster situations where the life support system is compromised and there is an imminent threat to saturation divers' lives. There is a lack of available procedures for these Level 2 emergencies. In the dramatic cases reviewed, accelerated decompressions were generated and carried out during the management of the emergency.

We believe that advances in decompression algorithms and oxygen toxicity models could allow the design of accelerated procedures, and that databases containing historical rapid decompression data should allow the validation of these procedures.

Emergency or accelerated decompression procedures should be:

- Simple in their description to ease communications.
- Flexible during their execution, to account for the situation evolutions.
- Published in the public domain and endorsed by industrial and professional associations.
- Supported by: medical resources, i.e., specialised medical teams, and adequate medical equipment; the life support team and the divers themselves; and highly reliable communication systems.

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# Effects of high oxygen tension on healthy volunteer microcirculation

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Hyperbaric oxygen treatment; Hyperoxia; Laser Doppler flowmetry; Near-infrared spectroscopy; Perfusion

**Abstract**
(Cousin N, Goutay J, Girardie P, Favory R, Drumez E, Mathieu D, Poissy J, Parmentier E, Duburcq. Effects of high oxygen tension on healthy volunteer microcirculation. Diving and Hyperbaric Medicine. 2022 December 20;52(4):260–270. doi: 10.28920/dhm52.4.260-270. PMID: 36525683.)
**Introduction:** Previous studies have highlighted hyperoxia-induced microcirculation modifications, but few have focused on hyperbaric oxygen (HBO) effects. Our primary objective was to explore hyperbaric hyperoxia effects on the microcirculation of healthy volunteers and investigate whether these modifications are adaptative or not.
**Methods:** This single centre, open-label study included 15 healthy volunteers. Measurements were performed under five conditions: T0) baseline value (normobaric normoxia); T1) hyperbaric normoxia; T2) hyperbaric hyperoxia; T3) normobaric hyperoxia; T4) return to normobaric normoxia. Microcirculatory data were gathered via laser Doppler, near-infrared spectroscopy and transcutaneous oximetry ($PtcO_2$). Vascular-occlusion tests were performed at each step. We used transthoracic echocardiography and standard monitoring for haemodynamic investigation.
**Results:** Maximal alterations were observed under hyperbaric hyperoxia which led, in comparison with baseline, to arterial hypertension (mean arterial pressure 105 (SD 12) mmHg vs 95 (11), $P < 0.001$) and bradycardia (55 (7) beats·min$^{-1}$ vs 66 (8), $P < 0.001$) while cardiac output remained unchanged. Hyperbaric hyperoxia also led to microcirculatory vasoconstriction (rest flow 63 (74) vs 143 (73) perfusion units, $P < 0.05$) in response to increased $PtcO_2$ (104.0 (45.9) kPa vs 6.3 (2.4), $P < 0.0001$); and a decrease in laser Doppler parameters indicating vascular reserve (peak flow 125 (89) vs 233 (79) perfusion units, $P < 0.05$). Microvascular reactivity was preserved in every condition.
**Conclusions:** Hyperoxia significantly modifies healthy volunteer microcirculation especially during HBO exposure. The rise in $PtcO_2$ promotes an adaptative vasoconstrictive response to protect cellular integrity. Microvascular reactivity remains unaltered and vascular reserve is mobilised in proportion to the extent of the ischaemic stimulus.

## Introduction

Inhalation of high oxygen concentrations is a standard therapy in many medical situations. To ensure sufficient oxygenation, supraphysiological levels are commonly used. During the nineteenth century, Paul Bert and J Lorrain Smith discovered that high oxygen tensions may lead to toxicity. Mechanisms involved include reactive oxygen species production, pulmonary oedema, altered endothelial function, activation of coagulation and reduced cardiac output. Recent meta-analyses of the potential benefits of hyperoxia in critically-ill patients have been controversial.[1,2] However, some subgroup analyses, supported by animal experiments, have shown a potential benefit of hyperoxia in highly selected patients (e.g., focal cerebral ischaemia).[3–5] Hyperbaric oxygen treatment (HBOT), where trancutaneously measured tissue oxygen partial pressure ($PtcO_2$) is up to ten times higher than normal, has proved to be beneficial in diverse conditions.[6]

Microcirculatory perfusion is a key component of tissue oxygen delivery. A recent study used side-stream dark-field (SDF) imaging to provide anatomic based information on the human microcirculation in healthy volunteers exposed to normobaric (NB) hyperoxia. Its two main findings were a reversible, significant decrease in perfused microvascular density and an increased heterogeneity in microcirculatory perfusion.[7] Those results raised concerns about oxygen therapy safety. Indeed, microvascular alterations (impairment of nitric oxide dysregulation-induced arteriolar vasodilation, functional impairment of many cell types found in the microcirculation and increased venular leukocyte-endothelial interaction) have been independently associated with poor outcomes in critically-ill septic patients.[8] However, while these alterations in critically-ill patients' microcirculation resulted in tissue hypoxia (microcirculatory abnormalities contribute to a decreased functional capillary density with less perfused areas), it is not the case in healthy subjects.[9–11] These microvascular alterations might be an adaptive phenomenon protecting cellular integrity from a drastic

rise in oxygen partial pressure ($PO_2$). The pathophysiology causing microcirculatory changes in sepsis is different than the physiologic 'adaptive' response seen with HBO.

In HBO exposure, hyperbaric (HB) hyperoxia further increases oxygen doses, thus may lead to greater microcirculation modification with a potential risk of hypoxia even in healthy subjects. Relevant data are scarce. The fact remains that HBO has been in use for over a century, and HBO-induced hypoxic injury has not been seen. One study exposed rabbits to both NB and HB hyperoxia and highlighted a significant decrease in microvascular density in both conditions.[12] Nevertheless, while the microcirculation may change, the significant elevation in arterial $PO_2$ during HBOT allows for adequate $O_2$ delivery to tissue/mitochondria despite the vasoconstriction. A recent study of HBO exposure in human subjects found that microcirculatory vasoconstriction did not inhibit the development of increased tissue oxygen partial pressure.[13]

Hence, to better understand the effects of hyperoxia on the microcirculation, we designed a study to explore microvascular function of healthy subjects exposed to both NB and HB hyperoxia. Our primary objective was to assess the impact of HB hyperoxia on a surrogate of microvascular function: the microvascular reactivity to an ischaemic stimulus. The other objectives were to study the impact of HB hyperoxia on healthy human microcirculatory perfusion, the impact of NB hyperoxia on microcirculatory perfusion and reactivity, and the haemodynamic response during the various oxygen exposures.

## Methods

The study received ethics (Comité de Protection des Personnes Ile de France V) and institutional (Agence Nationale de Sécurité des Médicaments et des produits de santé) approvals, and was registered on Clinicaltrial.gov under NCT03980210.

This was a single centre open label study conducted in the hyperbaric oxygen facility of a teaching hospital (CHU Lille), between June and July 2019. Non-professional divers between 18 and 64 years old who had received medical clearance to practice scuba-diving in the past year were able to participate to the study. People 65 years of age and more were considered more likely to exhibit age-linked microvascular modifications, and were not included in our study. After obtaining written informed consent, inclusion criteria were checked by an independent physician from the Clinical Investigation Centre of our institution. Patients with disease known to alter microcirculation (i.e., arterial hypertension, smoking, diabetes, arteriopathy, systemic sclerosis, Raynaud's disease) or a contraindication to HBOT (e.g., heart failure, pneumothorax, unstable asthma, perilymph fistula, vestibular disorders, vascular proliferation in the eye) were excluded. General and anthropometric data such as age, gender, weight and height were recorded.

### PROTOCOL

Subjects lay on a bed inside a hyperbaric chamber thoughout study and were exposed to five consecutive conditions: T0) baseline value (normobaric normoxia) with the subject breathing air at atmospheric pressure; T1) hyperbaric normoxia with a tightly fitting aviator style mask delivering a hypoxic gas mix (8% oxygen, 92% nitrogen) at 253.3 kPa (2.5 atmospheres absolute [atm abs]) ambient pressure; T2) hyperbaric hyperoxia with oxygen ($FiO_2$ = 1) delivered via the same mask at 253.3 kPa ambient pressure; T3) normobaric hyperoxia with oxygen ($FiO_2$ = 1) delivered via the same mask at 101.3 kPa ambient pressure; T4) a final set of measurements after return to normobaric normoxia conditions to detect residual effects of hyperoxia.

Measurements were performed after a 30-minute period in each condition to let the microcircualtion and any haemodynamic changes reach an equilibrium (Figure 1).

At each step of the study protocol, vascular occlusion tests (VOTs) were performed with a cuff positioned over the brachial artery. It was inflated to at least 50 mmHg over systolic arterial pressure for 3 min as longer durations tend to compromise the subject's comfort, and shorter ones provide insufficient post-occlusive reactive hyperaemia (PORH).[14] On completion of the ischaemic period, the cuff was quickly deflated to zero.

Two investigators present in the hyperbaric chamber and a trained certified hyperbaric technologist ensured safety during the whole protocol.

### MEASUREMENTS

#### *Microcirculatory parameters*

Subclavian artery transcutaneous pressures of $O_2$ ($PtcO_2$) and carbon dioxide ($CO_2$) ($PtcCO_2$) were continuously recorded (PERIFLUX® PF5040, Perimed, Jiirfalla, Sweden).

Near infrared spectroscopy (NIRS) was used to detect changes in muscle perfusion. NIRS data were acquired with an INSPECTRA® 850 monitor (Hutchinson Technology, Hutchinson, Minnesota, USA) and 25 mm-probe attached to the thenar eminence of the right hand. The thenar eminence is an anatomical region with no subcutaneous fat layer, thus providing VOT-parameters highly consistent among subjects.[15,16] When tissue oxygen saturation ($StO_2$) values remained stable for three minutes, a VOT was performed. Baseline, minimal and maximal $StO_2$ (respectively $StO_{2basal}$, $StO_{2min}$, $StO_{2max}$), total haemoglobin index before ($THI_{basal}$) and during VOT ($THI_{occlu}$), $StO_2$ descending (desc) and ascending (asc) slopes, time to $StO_2$max ($Time_{max}$), time to baseline ($Time_{base}$), and area under the curve of hyperaemia (AUC) were computed (Figure 2). Muscle oxygen consumption ($VO_2$) was calculated according to the following formula: NIRS $VO_2$ = ($THI_{basal}$ + $THI_{occlu}$) /

**Figure 1**
Illustration of the study protocol showing the five consecutive conditions (normobaric normoxia, hyperbaric normoxia, hyperbaric hyperoxia, normobaric hyperoxia and normobaric normoxia); near infrared spectroscopy (NIRS), laser Doppler, echocardiography measurements were taken after 30 min of exposure in each condition

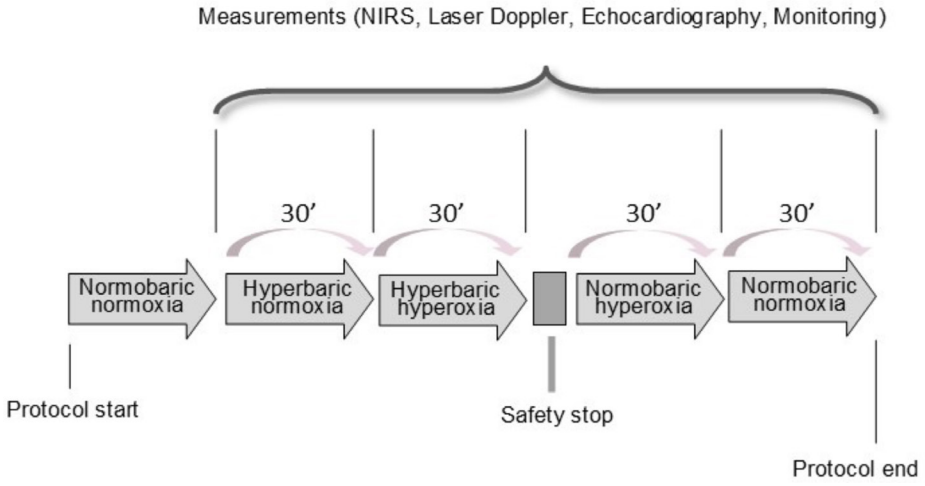
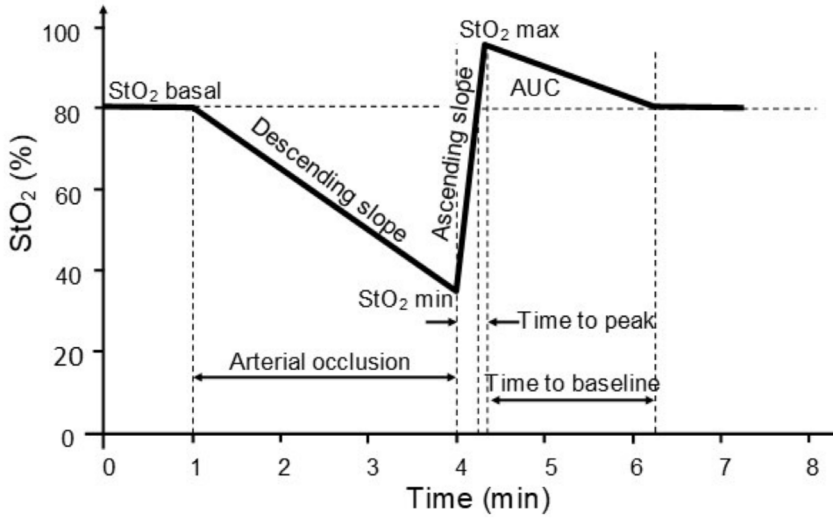



**Figure 2**
Schematic representation of tissue oxygen saturation ($StO_2$) evolution during near infrared spectroscopy monitoring of a vascular occlusion test; AUC – area under curve

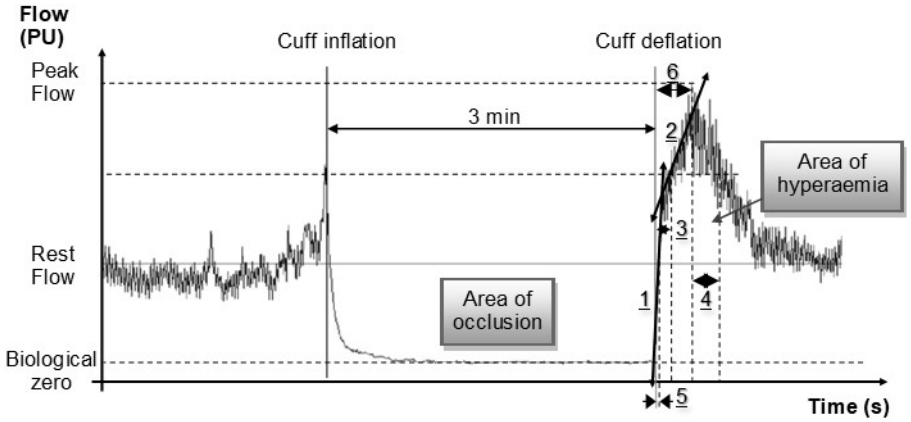



2 × (–Desc slope).[17] Tissue oxygen saturation variations during ($\Delta StO_{2min}$) and after ($\Delta StO_{2max}$) VOT were also calculated. Schematically, $StO_{2max}$ and AUC represent oxygen delivery; $Time_{base}$, $Time_{max}$, Asc slope and $\Delta StO_{2max}$ explore microvascular reactivity; $\Delta StO_{2min}$ reflects the extent of ischaemia and is known to influence Asc slope.[15]

Laser Doppler flowmetry recorded cutaneous blood flow (expressed in perfusion units [PU]) continuously throughout the procedure by using the laser Doppler shift principle to measure velocity and concentration of moving blood cells. The laser Doppler flowmeter probe (PERIFLUX® PF407 (Perimed, Jiirfalla, Sweden) was placed on the right index fingertip. The gain was adjusted to 1, the cut-off frequency to 12 Hz, and the time constant to 0.2 seconds. Recordings were later analysed with Perisoft for Windows 2.5.5 software. Microvascular reactivity was tested with VOTs. The signal obtained during arterial occlusion is flux-independent and was taken as biological zero (BZ). Rest flow (RF) was measured during the 5 min before VOT. The first ascending slope (Slope 1), a reflection of the myogenic phase of hyperaemia, was measured during the first 3 s of the hyperaemic peak. Given that peak flow (PF) occurred later than 3 s, a second ascending slope (Slope 2), a reflection of the hyperaemia metabolic phase, was measured during the interval between the end of the first 3 s and peak flow.[18] Parameters exploring microvascular reactivity including time to recovery (TR), time to half of the difference between rest and peak flow during onset of hyperaemia (TH1), time to half of the difference between rest and peak flow during

**Figure 3**

Example of a laser Doppler recording during post-occlusion hyperaemia; 1 – first upward slope; 2 – second upward slope; 3 – time to half of the difference between rest and peak flow during onset of hyperaemia; 4 – time to half of the difference between rest and peak flow during offset of hyperaemia; 5 – time to recovery; 6 – time to max; PU – perfusion units




offset of hyperaemia (TH2), time to max (TM), and the ratio of the hyperaemia area over the occlusion area (AH/AO), were also computed (Figure 3).[19]

*Haemodynamic parameters*

Non-invasive monitoring (heart rate, arterial pressure) was obtained with a HAUX-MEDICAL-MONITORING® system (Haux-Life-SupportGmbH, Karlsbad-Ittersbach, Germany). Systolic arterial pressure – heart rate product (SAP*HR) was calculated as a reflection of myocardial oxygen consumption.[20]

Transthoracic echocardiography (TTE) was performed with a VIVID-I® echocardiograph (GE Medical Systems, Milwaukee, WI, USA). The left ventricle was studied via volumetric analyses (left ventricular end-diastolic volume [LVEDV], left ventricular end-systolic volume [LVESV]), systolic function parameters (left ventricular ejection fraction [LVEF] [modified Simpson method], subaortic velocity-time integral [AoVTI]) and diastolic function parameters (mitral inflow doppler assessment including E- and A-wave velocity [respectively E and A] and E-wave deceleration time [EDT], tissue Doppler of the mitral annulus e'-wave [e]). The right ventricle function was assessed through tricuspid annular plane systolic excursion (TAPSE) and tissue Doppler of the tricuspid annulus S-wave (S). We then calculated E/A and E/e ratios, left ventricle stroke volume (SV-TTE) and cardiac output (CO-TTE). We used the left ventricular outflow tract Doppler method as it has been previously validated and has shown an acceptable agreement with the thermodilution method.[21] A single investigator, qualified in transthoracic echocardiography performed all measurements. To reduce intra-observer variability the average of three measures was used.

STATISTICAL ANALYSIS

Statistical analysis was performed using GraphPad PRISM® (version 8.2.0, GraphPad Software, San Diego, CA, USA). Categorical variables were expressed as frequencies with percentages. Continuous variables with a normal distribution were expressed as mean (standard deviation); otherwise, data were presented as medians with percentiles [25; 75%]. For comparison at different times, a one-way analysis of variance (ANOVA) or Friedman's test was used as appropriate with Tukey's corrections for repeated measurements. A two-sided $P < 0.05$ was considered as significant.

## Results

Fifteen subjects were included; three (20%) females and 12 (80%) males, aged 48.4 (SD 11.1) years, with an average height and weight of 1.75 (0.08) m and 75.5 (7.7) kg respectively, representing a mean body mass index of 24.8 (2.5) $kg \cdot m^{-2}$.

There was no statistical difference between the beginning and end normobaric normoxia conditions (i.e., T0 vs T4) in either microcirculatory or haemodynamic measurements.

Haemodynamic data are presented in Table 1. Briefly, two significant changes occurred in hyperoxic conditions (i.e., T2 and T3); bradycardia and a rise in arterial pressure. They both reached a maximum with HB hyperoxia (T2). Of note, bradycardia also occurred during hyperbaric normoxia (T1). There were no changes in echocardiographic measurements in any conditions.

Microcirculatory data gathered with NIRS and laser-Doppler flowmetry are presented in Table 2 and Table 3 respectively.

**Table1**

Haemodynamic changes ($n$ = 15); data are mean (standard deviation); a – $P < 0.05$ vs baseline; b – $P < 0.05$ vs hyperbaric normoxia; c – $P < 0.05$ vs hyperbaric hyperoxia; d – $P < 0.05$ vs normobaric hyperoxia; e – $P < 0.05$ vs normobaric normoxia; HR*SAP – index of myocardial oxygen consumption; LVEDV – left ventricle end-diastolic volume; LVEF_SBP – modified Simpson method left ventricle ejection fraction; LVESV – left ventricle end-systolic volume; MA e-wave velocity – mitral annulus e-wave velocity; SV_VTI – stroke volume calculated with velocity-time integral of the left ventricular outflow track; VTI – velocity-time integral of the left ventricular outflow track; TAPSE – tricuspid annular plane systolic excursion; TA S-wave velocity – tricsuspid annulus S-wave velocity

| Condition | Normobaric normoxia | Hyperbaric normoxia | Hyperbaric hyperoxia | Normobaric hyperoxia | Normobaric normoxia |
|---|---|---|---|---|---|
| **Haemodynamic monitoring** | | | | | |
| Heart rate (beats·min$^{-1}$) | 66 (8)[b,c,d] | 61 (8)[a,c] | 55 (7)[a,b,d,e] | 60 (10)[a,b] | 62 (8)[c] |
| Systolic arterial pressure (mmHg) | 127 (14)[c,d] | 132 (13) | 136 (12)[a] | 135 (19)[a] | 130 (11) |
| Mean arterial pressure (mmHg) | 95 (11)[c] | 99 (8) | 105 (12)[a,e] | 100 (8) | 98 (8)[c] |
| Diastolic arterial pressure (mmHg) | 79 (8)[c] | 79 (8)[c] | 89 (8)[a,b,d,e] | 85 (9)[c] | 81 (8)[c] |
| HR*SAP (beats·min$^{-1}$.mmHg) | 8,167 (1,250) | 8,162 (1,613) | 7,490 (1,208) | 8,073 (1,282) | 8,150 (1,381) |
| **Transthoracic echocardiography** | | | | | |
| LVEDV (mL) | 144 (18) | 144 (23) | 137 (18) | 143 (23) | 141 (28) |
| LVESV (mL) | 67 (13) | 67 (14) | 66 (13) | 63 (14) | 62 (16) |
| LVEF_SBP (%) | 54 (4) | 54 (4) | 53 (5) | 55 (5) | 56 (6) |
| SV_VTI (mL) | 69 (10) | 70 (14) | 72 (15) | 71 (15) | 68 (14) |
| VTI (cm) | 19 (3) | 19 (3) | 19 (3) | 19 (3) | 18 (3) |
| TAPSE (mm) | 23 (4) | 24 (3) | 21 (3) | 21 (3) | 23 (3) |
| TA S-wave velocity (cm·s$^{-1}$) | 13 (1) | 13 (1) | 12 (1) | 12 (1) | 13 (1) |
| Mitral E-wave velocity (cm·s$^{-1}$) | 63 (12) | 64 (13) | 62 (8) | 61 (8) | 60 (12) |
| E-wave deceleration time (ms) | 165 (37) | 177 (44) | 170 (34) | 176 (34) | 176 (46) |
| Mitral A-wave velocity (cm·s$^{-1}$) | 50 (11) | 51 (13) | 47 (12) | 50 (9) | 48 (9) |
| MA e-wave velocity (cm·s$^{-1}$) | 14 (3) | 14 (4) | 13 (2) | 12 (3) | 13 (3) |
| E/A ratio | 1.32 (0.33) | 1.36 (0.58) | 1.42 (0.40) | 1.28 (0.34) | 1.27 (0.34) |
| E/e ratio | 4.72 (1.03) | 5.11 (1.76) | 5.00 (1.02) | 5.46 (1.44) | 4.84 (1.24) |
| Cardiac output (L·min$^{-1}$) | 4.43 (0.76) | 4.33 (0.96) | 3.95 (0.83) | 4.13 (1.08) | 4.20 (0.96) |

At rest, there was a significant decrease in perfusion (RF) associated with a significant rise in $StO_{2basal}$ during HB hyperoxia. Transcutaneous measurements showed no statistical difference in $PtcCO_2$ in any conditions. In contrast, $PtcO_2$ reached a median of 19.1 kPa [16.7; 21.2] in NB hyperoxia and 99.3 kPa [77.3; 137.3] in HB hyperoxia ($P < 0.0001$ vs T0, T1, T3 and T4) (Figure 4A).

During the ischaemic period, occlusion area ($P < 0.05$ vs T0, T1 and T4), $StO_{2min}$ ($P < 0.05$ vs T0, T1, T3 and T4) and $\Delta StO_{2min}$ ($P < 0.05$ vs T0, T1 and T4) were significantly lower in HB hyperoxia. In parallel, laser-Doppler PORH parameters (peak flow, hyperaemia area and Slope 1) decreased significantly during HB hyperoxia (Figure 4B).

No parameters relating to microvascular reactivity (i.e., ascending slope, TR, $\Delta StO_{2max}$, RF/PF and AH/AO ratios) showed any significant changes at any time of the experiment (Figure 4C).

## Discussion

This study confirmed previously published effects of normbaric hyperoxia on the microcirculation in healthy volunteers.[7] By increasing the oxygen dose, hyperbaric hyperoxia further increases the magnitude of these effects. Of note, microvascular reactivity remained unimpaired throughout the protocol, suggesting that these alterations are indeed an adaptative phenomenon.

**Table 2**

Near infrared spectroscopy variables ($n = 15$); data are mean (standard deviation) or median [25;75% percentiles]; a – $P < 0.05$ vs baseline; b – $P < 0.05$ vs hyperbaric normoxia; c – $P < 0.05$ vs hyperbaric hyperoxia; d – $P < 0.05$ vs normobaric hyperoxia; e – $P < 0.05$ vs normobaric normoxia; AU – arbitrary unit; AUC – area under curve of hyperaemia; NIRS $VO_2$ – muscle oxygen consumption; $StO_2$ – tissue oxygen saturation; $\Delta StO_{2max}$ – calculated from $(StO_{2max} - StO_{2basal})/StO_{2basal}$; $\Delta StO_{2min}$ – calculated from $(StO_{2min} - StO_{2basal})/StO_{2basal}$; THI – total haemoglobin index

| Condition | Normobaric normoxia | Hyperbaric normoxia | Hyperbaric hyperoxia | Normobaric hyperoxia | Normobaric normoxia |
|---|---|---|---|---|---|
| $StO_{2basal}$ (%) | 81 (3)[c] | 81 (2)[c] | 85 (4)[a,b,d,e] | 81 (3)[c] | 80 (3)[c] |
| $StO_{2min}$ (%) | 52 (7)[c] | 52 (6)[c,d] | 62 (8)[a,b,d,e] | 56 (4)[b,c] | 51 (5)[c] |
| $StO_{2max}$ (%) | 95 [93; 97][c] | 96 [94; 97][c] | 99 [97; 99][a,b,e] | 96 [96; 99][c,e] | 94 [91; 96][c,d] |
| $\Delta StO_{2max}$ (%) | 17 (3) | 18 (3) | 15 (4) | 19 (5) | 17 (6) |
| $\Delta StO_{2min}$ (%) | -38 [-43; -29][c] | -36 [-41; -33][c] | -28 [-35; -18][a,b,e] | -32 [-35; -28] | -35 [-39; -32][c] |
| $THI_{basal}$ (AU) | 15.4 (2.3)[c,d] | 14.4 (2.2) | 13.8 (2.7)[a] | 13.7 (2.4)[a] | 14.5 (2.0) |
| $THI_{occlu}$ (AU) | 15.3 (3.1)[c] | 14.7 (2.9)[c] | 13.0 (3.2)[a,b] | 14.2 (2.7) | 14.6 (1.9) |
| Descending slope (%·s$^{-1}$) | -0.15 (0.03)[c] | -0.15 (0.03) | -0.13 (0.04)[a] | -0.14 (0.03) | -0.15 (0.04) |
| Ascending slope (%·s$^{-1}$) | 2.53 [1.69; 6.81] | 5.67 [2.79; 8.25] | 2.42 [1.71; 4.33] | 2.17 [1.65; 4.75] | 2.82 [1.6; 5.16] |
| $Time_{max}$ (s) | 29 (11) | 26 (8) | 32 (10) | 37 (15) | 33 (15) |
| $Time_{base}$ (s) | 277 [222; 353] | 256 [201; 332] | 388 [235; 457] | 325 [166; 436] | 277 [138; 353] |
| AUC (%.s) | 1,475 (617)[c] | 1,706 (737) | 2,414 (1,143)[a] | 2,161 (1,262) | 1,680 (1,107) |
| NIRS $VO_2$ (AU) | 2.32 (0.58)[c] | 2.19 (0.58)[c,d] | 1.69 (0.68)[a,b,e] | 1.94 (0.55)[b] | 2.23 (0.63)[c] |

## HAEMODYNAMIC CHANGES

Haemodynamic change was investigated as interactions with the microcirculation have been previously described.[22] This phenomenon is known as haemodynamic coherence. It stipulates that hemodynamic alterations have a direct effect on regional and microcirculatory perfusion and oxygen delivery to the cells.

Hyperoxia-induced bradycardia is a well-known phenomenon described for the first time in 1897 and proven by others.[23] In that study, HBO induced a more significant bradycardia than normobaric hyperoxia (a reduction of 12–16 beats·min$^{-1}$ vs 4–7 beats·min$^{-1}$). Both oxygen-dependent (a direct effect of high oxygen tension on myocardium[24] and autonomic nervous system alterations[25,26]) and oxygen-independent mechanisms (diminution in sympathetic tone) were involved in the phenomenon. Bradycardia is known to reach a maximum under therapeutic barometric pressure ranges and to gradually disappear over time.[27]

In our study, HBO induced a hyperoxic vasoconstriction, and as a consequence, a rise of approximately 10 mmHg in systolic, diastolic and mean arterial blood pressures. This phenomenon occurs in healthy small diameter arterioles as soon as the $PO_2$ reaches 101.3 kPa.[28] A further increase in $PO_2$ (up to 202.7 kPa[29]) constricts larger vessels such as resistance arteries, leading to a rapid rise in systemic vascular resistance,[30] hence in arterial blood pressure.

As arterial hypertension originated from microcirculatory modifications (e.g., hyperoxic vasoconstriction) while cardiac output remained steady, haemodynamic changes did not impact the microcirculation. Hence, all microcirculatory modifications may be considered a direct consequence of the oxygen exposure.

## MICROCIRCULATORY CHANGES

This study showed that significant microcirculatory modifications occur during normobaric and hyperbaric hyperoxia. In comparison with baseline value, $PtcO_2$ increased during normobaric hyperoxia (18.8 [SD 5.2] vs 6.1 [2.4] kPa). In response, a fall in rest flow occurred (Figure 4A). Even though it does not reach statistical significance, probably due to insufficient number of subjects, these results are consistent with previously published data.[7,31] Hyperbaric oxygen, by leading to a critical rise in $PtcO_2$ (104.0 [45.9] kPa) significantly reduced microcirculatory flow. As hyperbaric normoxia has no effects on the microcirculation, it seems that the high oxygen pressure drives this phenomenon.

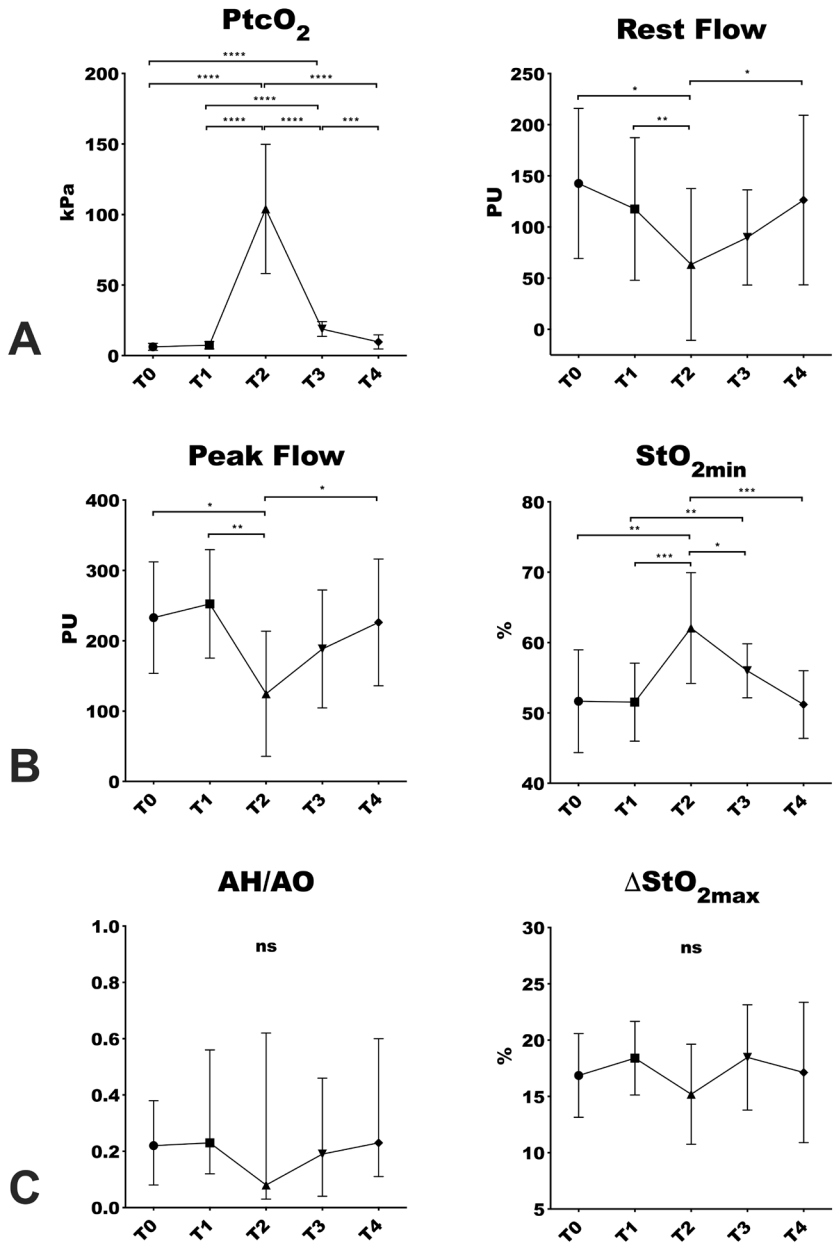
**Table 3**

Laser-Doppler flowmetry variables ($n$ = 15); data are mean (standard deviation) or median [25; 75% percentiles]; a – $P < 0.05$ vs baseline; b – $P < 0.05$ vs hyperbaric normoxia; c – $P < 0.05$ vs hyperbaric hyperoxia; d – $P < 0.05$ vs normobaric hyperoxia; e – $P < 0.05$ vs normobaric normoxia; AH/AO – hyperaemia area / occlusion area ratio; AU – arbitrary unit; PF – peak flow; PU – perfusion units; RF – rest flow; ΔRF_PF – calculated from ((peak flow – rest flow) / rest flow) x 100

| Condition | Normobaric normoxia | Hyperbaric normoxia | Hyperbaric hyperoxia | Normobaric hyperoxia | Normobaric normoxia |
|---|---|---|---|---|---|
| Rest flow (PU) | 143 (73)[c] | 118 (70)[c] | 63 (74)[a,b,e] | 90 (47) | 126 (83)[c] |
| Peak flow (PU) | 233 (79)[c] | 253 (77)[c] | 125 (89)[a,b,e] | 188 (84) | 226 (90)[c] |
| Δ RF_PF (%) | 72 [31; 173] | 127 [59; 172] | 93 [49; 274] | 154 [47; 218] | 111 [59; 191] |
| Occlusion area (PU.s) | 24,602 [16,808; 37,431][c] | 19,821 [13,007; 29,987][c] | 7,387 [5,524; 13,917][a,b,e] | 18,747 [7,553; 23,675] | 19,700 [9,986; 59,397][c] |
| Hyperaemia area (PU.s) | 4,442 [1,800; 8,249][c] | 4,237 [2,948; 7,467][c] | 0,519 [0,311; 1,678][a,b,e] | 1,920 [0,802; 6,701] | 6,923 [4,145; 10,488][c] |
| AH/AO ratio | 0.22 [0.08; 0.38] | 0.23 [0.12; 0.56] | 0.08 [0.03; 0.62] | 0.19 [0.04; 0.46] | 0.23 [0.11; 0.60] |
| Time to recovery (s) | 1.12 [0.96; 1.32] | 0.97 [0.56; 1.52] | 1.65 [0.73; 3.29] | 1.05 [0.40; 1.95] | 1.24 [0.53; 1.60] |
| Time to half before hyperaemia (s) | 0.95 [0.71; 1.76] | 1.02 [0.71; 4.82][c] | 3.13 [1.18; 9.65][b] | 1.08 [0.62; 4.40] | 0.93 [0.71; 1.84] |
| Time to max (s) | 39.75 (59.42) | 25.62 (14.94) | 41.24 (49.26) | 14.53 (12.07) | 36.00 (24.00) |
| Time to half after hyperaemia (s) | 34.15 [13.95; 82.53] | 42.44 [27.53; 53.81] | 26.32 [8.31; 73.13] | 27.93 [13.55; 41.17][e] | 59.67 [42.84; 71.49][d] |
| Slope 1 ($PU \cdot s^{-1}$) | 58.4 [38.0; 79.1][c] | 52.5 [33.9; 89.1][c] | 19.5 [10.1; 35.1][a,b] | 47.7 [28.0; 77.0] | 56.7 [32.0; 68.6] |
| Slope 2 ($PU \cdot s^{-1}$) | 5.3 [4.2; 7.3] | 3.6 [2.8; 4.9] | 4.3 [0.9; 5.0] | 5.3 [4.6; 9.2] | 2.7 [1.5; 5.5] |

**Figure 4**

A – microcirculatory parameters at rest; B – results of ischaemic stimulus and vascular reserve mobilisation; C – microvascular reactivity; *$P$ < 0.05; **$P$ < 0.01; ***$P$ < 0.001; ****$P$ < 0.0001; AH/AO – hyperaemic area / occlusion area ratio; ns – not significant; $PtcO_2$ – transcutaneous partial pressure of oxygen; PU – perfusion units; $StO_2$ – tissue oxygen saturation measured with near infrared spectroscopy; $\Delta StO_{2max}$ – calculated from $(StO_{2max} - StO_{2basal})/StO_{2basal}$; T0 – baseline normobaric normoxia; T1 – hyperbaric normoxia; T2 – hyperbaric hyperoxia; T3 – normobaric hyperoxia; T4 – final normobaric normoxia




The underlying mechanism highlighted is known as hyperoxic vasoconstriction.[29,32] It is an adaptative response to protect cellular integrity from high oxygen tension.[33,34] The mechanisms by which hyperoxia leads to systemic vasoconstriction are not fully elucidated. The elevated arterial content of oxygen ($CaO_2$) itself may contribute due to the pivotal role of the erythrocyte. Following a fall in haemoglobin-$O_2$ saturation, haemoglobin may act as an '$O_2$ sensor', releasing adenosine triphosphate (ATP) and nitric oxide. Conversely, plasma ATP concentrations are lower in hyperoxia, suggesting reduced vasodilator signalling. Moreover, the high $PaO_2$ may reduce the availability of other vasodilators such as prostaglandin PGI2.[35] Finally, hyperoxia causes an increase in reactive oxygen species (ROS), which in turn inhibit a range of vasodilators such as nitric oxide (e.g., superoxide reacts with nitric oxide to generate peroxynitrite).[13] Then, during prolonged HBO exposure, the activation of extracellular superoxide

dismutase removes superoxide and reduces nitric oxide antagonism. Thus, the hyperoxic vasoconstriction could be transient as recently reported elsewhere.[13]

Previous studies have proved that hyperoxic vasoconstriction only takes place in well-perfused territories while microvascular flow remains stable in ischaemic tissues.[31] It should result in microvascular flow redistribution toward poorly perfused territories. This phenomenon, known as the 'Robin-Hood effect', is supportive of the use of HBO in patients with heterogeneous microcirculation alterations such as ischaemia-reperfusion injuries.[31]

Performing VOTs in our study allowed us to explore vascular reserve, and the integrity and functionality of the microcirculation when exposed to elevated $PO_2$. Parameters exploring microvascular reactivity (i.e., NIRS $Time_{base}$, $Time_{max}$, Asc slope and $\Delta StO_{2max}$; and laser-Doppler AH/AO and RF/PF ratios) do not vary over time (Figure 4C). One study had already demonstrated similar results under normobaric hyperoxia.[7] Our study extends their findings to higher oxygen pressures. It emphasises the fact that hyperoxic vasoconstriction is an adaptative phenomenon that does not alter microvascular reactivity.

Laser Doppler parameters exploring vascular reserve (i.e., PF, Slope 1 and AH) decrease significantly under hyperbaric hyperoxia. However, the ischaemic stimulus (i.e., $StO_{2min}$, $\Delta StO_{2min}$ and occlusion area) is substantially lower during hyperbaric hyperoxia (Figure 4B). The predefined 3-min period of ischaemia produces a weaker ischaemic stimulus, probably because $StO_{2basal}$ is higher and dissolved oxygen must first be consumed before tissues react to hypoxia.[7] Hence, a smaller part of the available vascular reserve is mobilised in response to ischaemia.

In parallel, $StO_{2max}$ and AUC significantly increase. These phenomena also occur during normobaric hyperoxia (without reaching statistical significance) but are absent during hyperbaric normoxia. This highlights the fact that the rise in arterial oxygen content plays a decisive part in these modifications.

A marked but statistically non-significant drop in TH2 (20%) suggests a faster vascular reserve demobilisation during normobaric and hyperbaric hyperoxia. The rise in oxygen delivery (represented by $StO_{2max}$ and AUC) may have triggered protective mechanisms to limit the PORH duration. Further studies are needed to confirm or refute this hypothesis.

In our study, the reduction in $StO_2$ Desc slope and NIRS $VO_2$ during normobaric hyperoxia and hyperbaric hyperoxia raise important methodological issues. First, in hyperoxic conditions, the dissolved oxygen rises. In hyperbaric hyperoxia, it could reach up to 6 mL·100 $mL^{-1}$ of plasma at 304.0 kPa. This amount is sufficient to cover all biological needs.[36] Hence, during VOT, this supplement in dissolved oxygen (which can't be detected by NIRS-$StO_2$) must be consumed before the signal starts to decrease. This time-lapse between cuff inflation and the inflection point of the Desc slope alters the reliability of these parameters. Then, when multiple VOTs are repeated, a phenomenon called ischaemic preconditioning occurs. As highlighted in a previous study, ischaemic preconditioning results in a drop in Desc slope.[37] Thus, Desc slope and NIRS $VO_2$ interpretations in our study may be unreliable. Future studies would have to focus on oxygen-induced changes on a metabolic level to further investigate this phenomenon.

LIMITATIONS

Our study has several limitations. First, the small number of healthy volunteers limits the study power and its capacity to detect slight variations or different response patterns. Hence, the effect of age or gender on microcirculatory response to hyperoxia could not be investigated. Moreover, we excluded patients > 65 years, whereas many of the patients treated with HBO are > 65 years. This may limit the generalisability of our results. Second, the sex ratio, largely in favour of men (80% vs 20%) limits a wider application of our findings to the general population as gender is known to impact microcirculatory measurements. Third, we standardised the extent of the ischaemic insult with a predefined three minute ischaemic period. Alternatively, we could have used a predefined $StO_2$ threshold (as proposed by Bezemer et al.[15]) to release cuff inflation. Doing so, VOTs would have taken more than three minutes and might have become uncomfortable for healthy awake subjects.

Finally, our study was designed to explore oxygen-induced microcirculation alterations but is limited in its ability to explore their pathophysiological mechanisms. We decided to be as non-invasive as possible to make recruitment of volunteers easier. Nevertheless, invasive blood flow and $VO_2$ measurements, blood samples to investigate inflammatory pathways, or *in vivo* microdialysis may be of interest to further investigate this question.

## Conclusions

High oxygen tensions significantly alter haemodynamics and microcirculation in healthy subjects, with hyperbaric oxygen exposure further increasing those modifications. Bradycardia occurred while cardiac output remained constant and arterial blood pressure increased. The rise in tissue oxygen saturation and transcutaneous oxygen partial pressure promotes an adaptative vasoconstrictive response to protect cellular integrity. Indeed, microvascular reactivity remained unaltered and vascular reserve is mobilised in proportion to the magnitude of an ischaemic stimulus. Further experiments are require to understand the pathophysiological pathways involved in hyperoxia-induced microcirculation modifications and to explore its effects in pathological conditions such as ischaemia or sepsis.

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# Delayed treatment for decompression illness: factors associated with long treatment delays and treatment outcome

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**Keywords**
Decompression sickness; Hyperbaric oxygen treatment; Epidemiology; First aid oxygen; Remote locations; Treatment sequelae

**Abstract**
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**Introduction:** Effectiveness of delayed hyperbaric oxygen treatment (HBOT) for decompression illness (DCI) and factors affecting treatment delays have not been studied in large groups of patients.
**Methods:** This retrospective study included 546 DCI patients treated in Finland in the years 1999–2018 and investigated factors associated with recompression delay and outcome. Treatment outcome was defined as fully recovered or presence of residual symptoms on completion of HBOT. The symptoms, use of first aid oxygen, number of recompression treatments needed and characteristics of the study cohort were also addressed.
**Results:** Delayed HBOT (> 48 h) remained effective with final outcomes similar to those treated within 48 h. Cardiopulmonary symptoms were associated with a shorter treatment delay (median 15 h vs 28 h without cardiopulmonary symptoms, $P < 0.001$), whereas mild sensory symptoms were associated with a longer delay (48 vs 24 h, $P < 0.001$). A shorter delay was also associated with only one required HBOT treatment (median 24 h vs 34 h for those requiring multiple recompressions) ($P = 0.002$). Tinnitus and hearing impairment were associated with a higher proportion of incomplete recoveries (78 and 73% respectively, $P < 0.001$), whereas a smaller proportion of cases with tingling/itching (15%, $P = 0.03$), nausea (27%, $P = 0.03$), motor weakness (33%, $P = 0.05$) and visual disturbances (36%, $P = 0.04$) exhibited residual symptoms. Patients with severe symptoms had a significantly shorter delay than those with mild symptoms (median 24 h vs 36 h respectively, $P < 0.001$), and a lower incidence of complete recovery.
**Conclusions:** Delayed HBOT remains an effective and useful intervention. A shorter delay to recompression is associated with fewer recompressions required to achieve recovery or recovery plateau.

## Introduction

Scuba diving is popular all around the world and at times practised in locations remote from hyperbaric treatment facilities. Therefore, in cases of diving-related injuries the time taken to reach medical facilities can be long. Furthermore, the symptoms of decompression illness are often mild, further increasing the delay. The causes of treatment delay and how they influence the treatment outcome remains a matter of interest to the diving medicine community.

Decompression illness (DCI) is a collective term which includes two pathophysiologically different syndromes: arterial gas embolism (AGE) following pulmonary barotrauma and decompression sickness (DCS) caused by bubble formation from dissolved gas.[1] In this study the term DCI is used as it can be difficult to differentiate between AGE and DCS in a clinical setting,[2] although it is likely that the vast majority of the cases were DCS. The gold standard intervention for DCI is hyperbaric oxygen treatment (HBOT), which can also be used as a treatment for non-diving related injuries, such as carbon monoxide poisoning, gas gangrene, delayed radiation injuries, necrotizing soft tissue infection and severe burns.[3]

The manifestations of DCI can vary greatly in severity. The agreed mild symptoms include constitutional symptoms such as fatigue, limb pain, some sensory changes such as tingling, skin rash and subcutaneous swelling as long as the

manifestations are static and neurological dysfunction is excluded by a diving medicine physician.[4] Therefore, other symptoms are classified as severe. These include dizziness/vertigo, motor weakness, mental, pulmonary, or coordinative disorders, decrease in the level of consciousness, auditory, bladder and cardiovascular symptoms.[1,4]

Whether or not the treatment outcome is influenced by a long delay from symptom onset to HBOT, is still a debated subject, as it is also profoundly affected by the severity of manifestations. There is evidence that a short treatment delay is beneficial in severe cases of DCI.[4] Some older research has also shown that a shorter time to recompression is associated with better treatment outcomes.[5–7] However, these studies did not stratify the presentations according to severity. Other recent studies have shown that although there is some evidence that treatment outcome is better with shorter delays, divers with a longer delay can still benefit from HBOT.[8–9] In addition, worse outcomes may be linked to specific symptoms, such as severe neurological symptoms, not so much to the delay.[10] There is a broad consensus that mild DCI can be adequately treated without HBOT[4,11] particularly, if recompression is logistically difficult or hazardous to access, as the symptoms tend to disappear with time.[12,16]

The aim of this study was to investigate the effect of delayed HBOT (> 48 h) and other factors on treatment outcome for DCI. Moreover, factors affecting the time to the chamber treatment were also evaluated.

## Methods

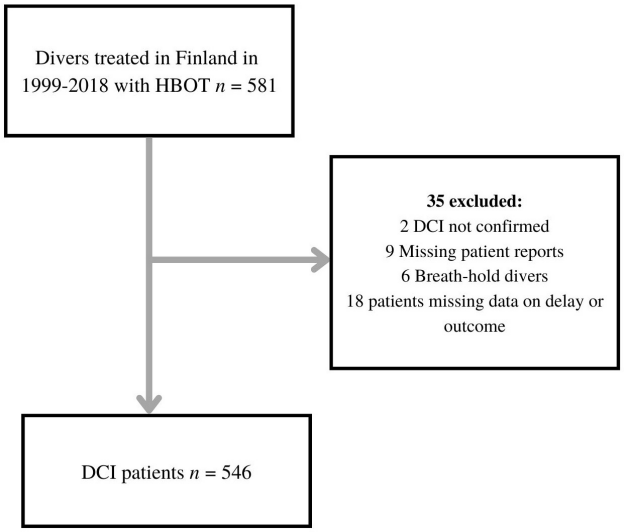
The study received ethics approval from the National Institute for Health and Welfare, Helsinki, Finland (THL/285/5.05.00/2016). The study adhered to the Declaration of Helsinki.

### PATIENT POPULATION AND DATA COLLECTION

The data for this retrospective study includes approximately 95% of all treated DCI cases in Finland from the years 1999–2018. The patients were treated in the Hyperbaric Medical Clinic Medioxygen in Helsinki or in the National Hyperbaric Unit of Turku University Hospital in Turku, Finland. Unfortunately, Medioxygen was closed in 2015 and Turku is the only treatment centre currently operational. Both of these were located in the South of Finland resulting in a long journey (up to 1,000 km) from other parts of the country.

Data were collected retrospectively from medical records of 546 patients treated at the two facilities. The flow chart for patient selection is shown in Figure 1. For the majority of cases the initial treatment was United States Navy (USN) TT6 with or without extensions (79%), however, a few milder cases received USNTT5. In the era 1999–2015 the follow-up treatments were usually USNTT9, later mainly USNTT6 or 5.[13,14] The HBOT treatments were continued as long as there was diminishing of the symptoms, until complete recovery or until there was no sustained improvement between two consecutive treatments. Patients were clinically evaluated directly after HBOT and at discharge. If patients left the treatment facility the day they were treated, the physician called the patient the next day to ensure that symptoms had not re-evolved.

**Figure 1**
Flow chart of patient selection and exclusion criteria for patients treated in Hyperbaric Medical Clinic Medioxygen in Helsinki or in the National Hyperbaric Unit of Turku University Hospital in years 1999–2018




Finland has challenging diving conditions leading divers to travel abroad looking for warmer and clearer waters. However, roughly 78% of the patient population was diving in cold water (4–10°C), whereas 22% were diving in warm water abroad.[13] This dataset includes divers from beginners to professional divers. The training level of the divers was defined as beginner, advanced, or expert. Beginner divers were open water divers (OWD) of any training organisation, advanced divers were advanced open water divers (AOWD) or nitrox divers, and expert divers were those who completed a higher course than AOWD including technical diving. This group also included professional divers. Additionally, the use of first aid oxygen ($FAO_2$) and any previous DCI treatment were recorded.

### TREATMENT DELAY, SYMPTOMS AND OUTCOME

Treatment delay was the number of hours from onset of DCI symptoms to recompression. Delayed treatment was defined as treatment delay greater than 48 h. This time point was arbitrary but has been used before,[9] and is long enough for the secondary symptoms to appear. The treatment outcome was defined as either fully recovered (no residual symptoms after HBOT) or not (presence of residual symptoms). Presenting symptoms were categorised as either mild (as defined by the 2018 consensus guideline[4]) or severe as explained in the introduction.

**Table 1**
Demographics of the patient population ($n = 546$); data are median (IQR) or $n$ (%); depth and dive time data missing for four and 57 patients respectively; DCI – decompression illness; $FAO_2$ – first aid oxygen; m – metres (distinction between seawater and freshwater depths not made); min – minutes

| Parameter | Data |
|---|---|
| Age (years) | 36 (30–42) |
| Sex, Male | 423 (78%) |
| Previous DCI | 119 (22%) |
| Depth (m) | 30 (21–42) |
| Dive time (min) | 45 (30–64) |
| $FAO_2$ provided | 145 (27%) |
| **Dive training level** | |
| Beginner | 92 (17%) |
| Advanced | 209 (38%) |
| Expert | 136 (25%) |
| Not recorded | 95 (17%) |

Treatment delay and outcome were evaluated among groups of symptoms including the following categories: subjective findings including musculoskeletal pain and neurosensorial symptoms (tingling, itching, subjective numbness), and objective findings including skin rash, neuromotor symptoms (motor weakness), vestibulocochlear symptoms (dizziness, vertigo, nausea, hearing impairment, tinnitus), central nervous system (CNS) symptoms (visual, coordination or verbal disturbances, rigidity, tremor, abnormal reflexes, numbness, bladder dysfunction) and cardiopulmonary symptoms. Some symptoms (bowel pain, subcutaneous swelling) were difficult to categorise and were left out of the analysis, as the number of these cases were small (bowel pain $n = 8$, swelling $n = 17$). However, they were taken into consideration in the mild vs severe classification as mild symptoms. If multiple symptoms were present, the patient was categorised based on the most severe symptom. It is important to note that the 'neurosensorial' category consists of only mild symptoms. Vestibulocochlear symptoms were considered severe. In addition to groups of symptoms, the outcome of treatment was evaluated for various individual symptoms and whether the patient recovered fully or had residual symptoms.

### STATISTICAL ANALYSIS

We describe the data using counts and percentages for categorical variables and median and interquartile ranges (IQRs) for continuous variables. Categorical variables were compared using Chi-squared tests or Fisher's exact test and continuous variables using Mann-Whitney U tests or Kruskall-Wallis tests depending on the number of categories compared. $P$-values < 0.05 were considered significant. The analyses were done using R version 4.1.0[15] and the plots were done with the ggplot2-package (open source url GitHub - tidyverse/ggplot2: An implementation of the Grammar of Graphics in R).

## Results

### PATIENT POPULATION

The demographics of the diver population are shown in Table 1.

### TREATMENT DELAY AND THE RELATIONSHIP BETWEEN OUTCOME AND DELAY

Patients with no residual symptoms had a median delay from symptom onset to recompression of 24 h (IQR 12–72) and the patients with residual symptoms had a median delay of 28 h (12–96); a statistically insignificant difference. Of the patients who fully recovered, 59% were treated within 48 h. Similarly, 53% of the patients with residual symptoms were treated within 48 h (also a non-significant difference).

### FACTORS ASSOCIATED WITH DELAY TO RECOMPRESSION

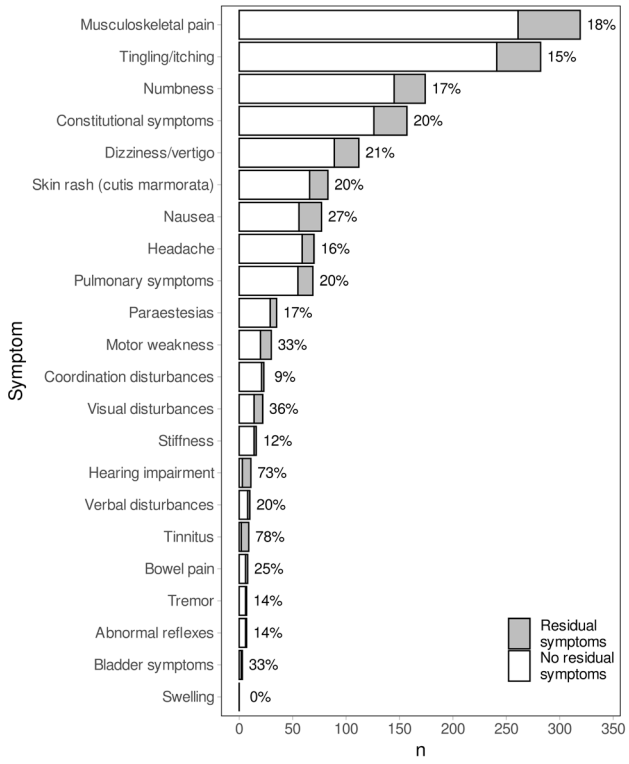
When DCI symptoms were categorised into symptom groups, mild neurosensorial and cardio-pulmonary symptoms had a significant association with treatment delay. Neurosensorial symptoms had a significantly longer delay than patients with no such symptoms (median 48 h vs 24 h, respectively). On the other hand, patients with cardio-pulmonary symptoms had a significantly shorter delay than patients with no such symptoms (15 h vs 28 h, respectively) (Table 2). Other symptom categories (pain only, skin, neuromotor, vestibulocochlear, CNS) did not show a statistically significant difference in terms of treatment delay or the groups were too small for statistical analysis (e.g., AGE, $n = 2$). Patients with severe symptoms ($n = 259$) had a significantly shorter delay than those with mild symptoms ($n = 287$) (24 h vs 36 h respectively, $P < 0.001$).

Patients who used $FAO_2$ had a significantly shorter delay to recompression; 14 h (5–27) vs 48 h (21–96) where $FAO_2$ was not used, ($P < 0.001$). There was also a significant difference in the delay between divers of different training levels. Beginners had the longest delays (72 h [21–144]) and the delay decreased as the training level improved as advanced divers had a median delay of 48 h (24–96) and expert divers had a delay of 24 h (7–48) ($P < 0.001$). Additionally, a pattern between the number of HBOT treatments needed and delay was observed. Patients who only needed one treatment had a median delay of 24 h (9–54) whereas those who needed two, three and four or more had delays of 37 h (24–96), 24 h (8–72), and 36 h (20–96) respectively. Therefore, patients who only needed one treatment had a shorter median delay than those who needed multiple treatments (34 h [20–96], $P = 0.002$).

**Table 2**
Treatment delay in different categories of symptoms; data are median (IQR) or n (%)

| Symptom | *n* (%) | Delay without symptom (h) | Delay with symptom (h) | *P*-value |
|---|---|---|---|---|
| Pain only | 76 (14%) | 26 (12–96) | 24 (20–48) | 0.09 |
| Neurosensorial | 214 (39%) | 24 (8–72) | 48 (24–108) | < 0.001 |
| Skin | 41 (8%) | 25 (14–90) | 24 (8–48) | 0.09 |
| Neuromotor | 21 (4%) | 25 (12–72) | 24 (11–72) | 0.43 |
| Vestibulocochlear | 90 (17%) | 24 (16–72) | 24 (6–96) | 0.36 |
| Central nervous system | 58 (11%) | 26 (12–72) | 24 (13–72) | 0.32 |
| Cardiopulmonary | 43 (8%) | 28 (17–96) | 15 (6–24) | < 0.001 |

**Figure 2**
The relationship between individual symptoms and treatment outcome. Percentages show the number of patients with residual symptoms after HBOT in different symptom categories




## ASSOCIATIONS WITH TREATMENT OUTCOME

There were both mild and severe symptoms that were associated with a better treatment outcome, i.e., no residual symptoms after treatment. These were tingling/itching (15% with residuals, $P = 0.03$), nausea (27%, $P = 0.03$), motor weakness (33%, $P < 0.05$) and visual disturbances (36%, $P = 0.04$). Among severe symptoms only tinnitus (78% residuals) and hearing impairment (73%) (both $P < 0.001$) were significantly associated with a worse outcome (residual symptoms). Other symptoms analysed (Figure 2) were not associated with a treatment outcome. However, 85% of patients with mild symptoms had no residual symptoms after treatment, whereas the corresponding number for patients with severe symptoms was 78%, $P = 0.03$.

There was no difference in the treatment outcome between sexes, nor did the use of $FAO_2$ influence the treatment outcome. A better treatment outcome was associated with younger patients. The median age for patients with no residual symptoms was 35 years (30–41) vs 39 years (32–44) for patients with residual symptoms, $P = 0.01$.

## Discussion

### DELAYED TREATMENT

In this large study, patients who underwent delayed recompression (> 48 h), had similar treatment outcomes compared to those who were treated with HBOT within 48 h. There are several possible explanations for this finding. Firstly, the effectiveness of HBOT remains good even with long delays until the recompression. This conclusion is also supported by a Chinese study, which emphasised that HBOT treatment should not be abandoned even after long delays, since its effectiveness decreased only minimally.[8] In another retrospective study, the time frame of 48 h for HBOT was used, and the findings were similar to our study.[9] Another possibility relates to the fact that in this cohort truly severe cases were rare, and patients exhibiting only mild symptoms (the majority in this cohort) can be expected to fully recover even without recompression. It follows that delay to recompression would be expected to make little or no difference to final outcome in mild cases. This underpins the expert consensus on the possibility for treating mild DCI without recompression, particularly when the treatment facility is far away.[11] Our data support this idea as Finnish diving is mostly done in relatively remote locations where the transportation to HBOT facility takes many hours and even though patients with mild symptoms had a median 12 h longer delay, they still recovered well.

Although spontaneous recovery in mild cases complicates interpretation, the fact remains that patients in our study who had symptoms and were recompressed even after a long delay mainly became asymptomatic when treated

with HBOT. The placebo effect must, of course, be taken into account, but it is possible that HBOT actually had an effect on DCI secondary changes, such as endothelial damage, impaired endothelial function, platelet activation and deposition, leukocyte-endothelial adhesion and possible consequences of vascular occlusion (ischaemia-reperfusion injury and apoptosis), and therefore contributed to healing the injury.[1]

### SYMPTOMS AND DELAY

Patients with certain severe symptoms, such as cardio-pulmonary symptoms, had a shorter delay to recompression. This is expected as someone who is very ill is more likely to seek medical attention. In contrast, mild neurosensorial symptoms were associated with a longer delay to recompression. With such mild symptoms, divers are less motivated to seek treatment or might not even realise they are experiencing symptoms of DCI. There is recent evidence of divers self-treating mild DCI with rest, fluids and normobaric oxygen.[16]

### OTHER FACTORS AND DELAY

In this study, the use of $FAO_2$ was related to a shorter delay, but not with a better treatment outcome. A shorter delay was also associated with a higher diver training level. Therefore, there is a possibility that these factors are linked, as better trained divers may have greater awareness of symptoms and more often have $FAO_2$ on the diving site. However, they also dive deeper, thus they risk developing more severe symptoms and a worse outcome. In other studies, the use of $FAO_2$ on the diving site has been associated with faster early recovery in DCI.[17] There is a possibility that the use of $FAO_2$ prevented more serious symptoms from developing, however any such conclusion would require comparison with a control group of patients with similar symptoms and dive history not receiving $FAO_2$. A shorter delay was associated with fewer required treatments, which is not only more comfortable for the patient, but also important in terms of cost-effectiveness and hospital resources.

### SYMPTOMS AND OUTCOME

Tinnitus and hearing impairment were associated with the lowest proportion of patients fully recovered after completion of all HBOT. Both are considered severe symptoms. Motor weakness and visual disturbances were associated with a higher proportion of patients fully recovered, even though they are also considered to be severe symptoms. Nevertheless, incomplete recovery from motor weakness remains a serious problem for the affected divers (33% in this study). In general, other studies report that severe symptoms are linked to a worse treatment outcome.[8,10,18] Mild symptoms such as tingling/itching were associated with a better treatment outcome, which supports the previous studies suggesting good prognosis for mild DCI symptoms.[11]

### OTHER FACTORS AND OUTCOME

There was a relationship between the patient's age and full recovery after HBOT, as patients with no residual symptoms were significantly younger although the median age difference was only four years (35 vs 39). It is often suggested that ageing increases the risks of diving.[19,20] Additionally, age has been associated with a worse outcome in multiple studies, even though the additional risk is not considered of great importance.[18,21,22]

### LIMITATIONS

As with many retrospective studies, the data collection in the two HBOT centres was not systematic, especially in the early years, which resulted in missing data in some cases. The majority of our patients had mild symptoms. There is a broad consensus that mild cases tend to get better even without recompression. Therefore, such a cohort is poorly suited to show a correlation between recompression delay and treatment outcome. In addition, very short delays to recompression were rare due to long distances to the remoteness of diving sites in Finland. Therefore, conclusions about the effect of very short delays to recompression cannot be drawn. Severe cases, such as dizziness and vertigo, occurred in only small numbers so the proportions of divers recovered (or not) from severe symptoms must be interpreted cautiously. Additionally, there was no long term follow up, thus no way of knowing if residual symptoms resolved later. However, this dataset was quite large and was gathered from only two HBOT centres, which adds to its strength.

### APPLICATIONS AND IMPROVEMENTS

Delay in recompression for DCI is still somewhat of a controversial topic in the diving medical community. Prospective data collection with structured methods would give a more robust database and results allowing stronger conclusions. In order to obtain enough data in a relatively short time period, the collection should be done from multiple HBOT centres with the same treatment protocols. When considering treatment delays and the treatment outcome, an inevitable question arises as to whether the efficacy of HBOT could be evaluated more precisely. This could provide guidance when patients can really benefit from HBOT, which in turn may provide a more cost-effective evacuation and treatment plan. An example of such work appeared in a recent study which found that a simple scoring system for spinal cord DCS helped define the urgency of evacuation of the injured diver.[23]

## Conclusions

Recompression and HBOT for DCI remains effective, even after a 48 h delay. Therefore, treatment should not automatically be discounted in the case of longer delays. A short delay to HBOT improves the efficacy of the

treatment in general, indicated by fewer required numbers of treatments. The overall efficiency of HBOT should be evaluated more systematically especially in cases of milder symptoms and delayed treatment.

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# Descriptive study of decompression illness in a hyperbaric medicine centre in Bangkok, Thailand from 2015 to 2021

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**Keywords**
Arterial gas embolism; Decompression sickness; Diving incidents; Hyperbaric oxygen treatment

**Abstract**
(Chevasutho P, Premmaneesakul H, Sujiratana A. Descriptive study of decompression illness in a hyperbaric medicine centre in Bangkok, Thailand from 2015 to 2021. Diving and Hyperbaric Medicine. 2022 December 20;52(4):277–280. doi: 10.28920/dhm52.4.277-280. PMID: 36525685.)
**Introduction:** This study aimed to determine the characteristics of decompression illness patients and their treatment outcomes, at the Center of Hyperbaric Medicine, Somdech Phra Pinklao Hospital, one of the largest centres in Thailand.
**Methods:** Past medical records of patients with decompression illness from 2015 to 2021 were retrieved and analysed.
**Results:** Ninety-eight records of diving-related illness from 97 divers were reviewed. Most of the divers were male ($n = 50$), Thai ($n = 86$), and were certified at least open water or equivalent ($n = 88$). On-site first aid oxygen inhalation was provided to 17 divers. Decompression sickness (DCS) cases were characterised according to organ systems involved. The most prominent organ system involved was neurological (57%), followed by mixed organs (28%), musculoskeletal (13%), and pulmonary (2%). There were three cases of arterial gas embolism (AGE). Median presentation delay was three days. Ninety patients were treated with US Navy Treatment Table 6. At the end of their hyperbaric oxygen treatment, most divers (65%) recovered completely.
**Conclusions:** Despite oxygen first aid being given infrequently and long delays before definitive treatment, treatment outcome was satisfactory. Basic knowledge and awareness of diving-related illnesses should be promoted among divers and related personnel in Thailand along with further studies.

## Introduction

Thailand is famous as a tourist destination for its beautiful maritime landscape, coastal scenery, and other oceanic natural resources. The World Tourism Organization reported that Thailand was the country with the highest revenue from tourism in the Asia-Pacific region in 2017.[1] Out of a total of 77 provinces, 24 are coastal and attract approximately 86 million visitors per year, contributing 53.35% of Thailand's gross domestic product.[2] Some of the recommended water activities are canoeing, kayaking, yachting, and open water diving. Scuba diving is a popular recreational activity in Thailand; however, related injuries occur in both recreational and occupational settings. Decompression illness (DCI) is one such injury that has scarcely been studied in Thailand, despite it being a potential cause of morbidity and mortality amongst scuba divers.[3] DCI (a collective term embracing decompression sickness [DCS] and arterial gas embolism [AGE]) may be an indication for urgent recompression and hyperbaric oxygen treatment (HBOT). One study published in 2007, reported 453 cases treated between 2001–2005 at both public and private hospitals with hyperbaric chambers in Thailand.[4] There are currently 112 hyperbaric chambers in Thailand, with 40 facilities being able to provide treatment for DCI. These are primarily located at navy hospitals, navy facilities, and commercial diving facilities.

To further understand the characteristics and outcome of DCI patients after recompression treatment in Thailand the authors analysed medical records from the Centre of Hyperbaric Medicine at Somdech Phra Pinklao Hospital, one of the largest of its kind in Thailand. This unit operates under the Naval Medical Department of the Royal Thai Navy. It was established in 2015, with the first patient being admitted on 1 October 2015. Since then, it has provided HBOT to hundreds of patients, including those with diving and non-diving indications, with its multiplace and monoplace hyperbaric oxygen chambers. The multiplace hyperbaric chamber at Somdech Phra Pinklao Hospital can accommodate up to 30 patients in three compartments.

The aim of this study was to determine the characteristics of DCI patients and their treatment outcomes, at the Centre of Hyperbaric Medicine, Somdech Phra Pinklao Hospital.

## Methods

This study was approved by the human research ethics committees of the Naval Medical Department (Case number: RP048/64).

Past medical records of patients with DCI were retrieved from 1 October 2015 to 30 June 2021. There were 97 patients with DCI. This study included only patients admitted to Somdech Phra Pinklao Hospital who declared a history of diving before a DCI incident.

The data collection form was divided into two parts: (1) personal data, including age, gender, nationality, weight, height, and history of past diving-related illness, types of divers and diving certificates, and dive site or location; and (2) data related to the incident, including date and time of symptom onset, time arrived at the Center of Hyperbaric Medicine, target organ of DCI, normobaric oxygen first aid received at scene, treatment table applied, total number of hyperbaric oxygen treatments given, and treatment outcome.

Data were analysed using descriptive statistics, via Microsoft Excel and STATA Version 14.0.

## Results

Ninety-seven patients were admitted for HBOT to treat DCI over the study period. Two divers each experienced two episodes of diving-related illness. The first episode in one of these patients occurred in 2015 before the establishment of the Center of Hyperbaric Medicine, so they were treated at the Underwater and Aviation Medicine Division, Naval Medical Department and this case was therefore excluded from the analysis. The other patient suffered from diving-related illness twice in 2020 with both treatments provided at the Center of Hyperbaric Medicine. It follows that this study pertains to 97 divers and 98 DCI incidents.

Fifty divers were male and 47 were female. The mean age was 35.7 years with a range of 19–63 years. The body mass index (BMI) of the divers was calculated and categorised according to the Thai Department of Disease Control, Ministry of Public Health.[5] The mean BMI for this group of divers was 23.9 $kg \cdot m^{-2}$ (range 17.0–44.8 $kg \cdot m^{-2}$). Most of the divers (45%) were within normal BMI limits (18.5–22.9 $kg \cdot m^{-2}$), 15% were overweight (BMI 23–24.9 $kg \cdot m^{-2}$), 37% were obese (BMI ≥ 25 $kg \cdot m^{-2}$) and 3% (BMI < 18.5 $kg \cdot m^{-2}$) were underweight.

Not surprisingly, the majority of divers (86) were Thai, while 11 were foreigners, including one each of American, Belgian, English, Guatemalan, Israeli, Japanese, Dutch, Spanish, Swedish, and two of Austrian nationality.

The majority of divers (80 cases, 82%) were reported to have dived in Thailand while 18 had dived overseas, including Indonesia (8, 8%), Maldives (3, 3%), Philippines (3, 3%), Japan (1, 1%), Malaysia (1, 1%), Palau (1, 1%), and one overseas dive site not stated.

Of 79 recreational dives, 62 were local dives in Thailand. Twenty-one (34%) occurred in provinces with Andaman Sea coastlines including Krabi, Phangnga, Phuket, and Satun. The other 36 dives (58%) took place in provinces with the coastal zoning of the Gulf of Thailand. Dive site data for the remaining five divers were either not properly recorded or missing. The province with the highest incidence of DCI was Surat Thani, especially in the Koh Tao area, a popular site among Thai and foreign divers, followed closely by Chonburi province in the Sattahip district, both of which are parts of the Gulf of Thailand coastlines. For Andaman coastlines, the most prominent provinces were Phangnga and Phuket.

Indonesia, being a popular dive destination among Thais, comprised the most common (8) site for diving-related illnesses of the 18 overseas dive sites. The others were three incidents each from the Maldives and the Philippines, one each from Japan, Malaysia, Palau, and one overseas dive site was not stated.

On-site first aid with normobaric oxygen was provided to only 17 divers (17%). In Thailand there were 13 incidents out of 80 (16%) in which divers received first aid oxygen, compared with four out of 18 overseas dives (22%). One Thai diver who had been diving in Indonesia received treatment with HBOT before arriving at the Center of Hyperbaric Medicine.

There were 16 divers with diving insurance, nine of which were Thai, while the other seven were foreigners. The rest of the divers did not have diving insurance or did not provide such information.

Regarding diving certification, 19 divers were certified to 'open water' level or its equivalent, while 69 divers were qualified to more advanced levels. There were nine divers without any diving certification, or their level of certification was not clearly specified.

Defining the duration between the first day that symptoms appeared and the day that the divers arrived at the Center of Hyperbaric Medicine as presentation latency, after excluding 14 outliers, the range of presentation latency was 0 to seven days, and the median was three days.

The most prominent organ system diagnosis was neurological DCS with 56 cases (57%). Twenty-three cases (23%) presented with mixed signs and symptoms (more than one involved organ system). Musculoskeletal DCS comprised 13 cases, while two cases most prominently showed pulmonary symptoms. There were three cases of AGE as shown in Figure 1.

**Figure 1**
Number of reported arterial gas embolism and decompression sickness cases by organ systems and year; AGE – arterial gas embolism

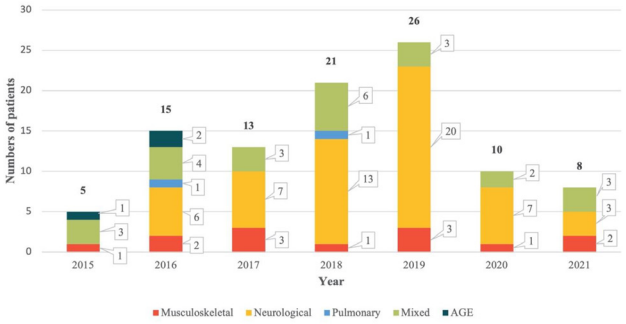



**Table 1**
Initial hyperbaric oxygen treatment administered in relation to clinical diagnosis; AGE – arterial gas embolism; TT5 – United States Navy Treatment Table 5; TT6 – United States Navy Treatment Table 6

| Diagnosis | TT5 | TT6 | Kindwall | Total |
|---|---|---|---|---|
| Musculoskeletal | 3 | 10 | 0 | 13 |
| Neurological | 2 | 51 | 3 | 56 |
| Pulmonary | 0 | 2 | 0 | 2 |
| Mixed | 0 | 24 | 0 | 24 |
| AGE | 0 | 3 | 0 | 3 |

The recompression treatment table utilised for cases grouped by organ system involvement is shown in Table 1. Ninety out of 98 cases (92%) were initially treated with US Navy Treatment Table 6 (USNTT6).[6] US Navy Treatment Table 5 (USNTT5) was used in five cases, and the remaining three cases were treated with Kindwall's monoplace treatment table, which can be utilised to treat both mild and severe DCI with no air breaks.[7]

Nearly half of the divers (48) underwent two to five hyperbaric oxygen treatments, another 48 divers had only one treatment, while the remaining two underwent more than five treatments. By completion of all recompression treatment, 64 cases (65%) had completely recovered, while 34 cases had residual symptoms.

## Discussion

The mean age of divers in this study, at 35.7 years, was lower than the 45 years reported in the DAN Diving Incidence Reporting System (DIRS). Gender results also differed, with 48% male divers in this study and 68% in the DIRS study.[8]

A total of 79 divers were reported to have dived in Thailand, while 18 dived overseas. The median presentation latency did not differ between divers whose incidents occurred in Thailand and divers who dived overseas, even though transportation time from local dive sites would suggest it should be shorter. While the median presentation latency between the two groups of divers were similar, the treatment outcome differed markedly. Full recovery status was noted in 70% of divers in Thailand compared with 47% in those who had been diving abroad. The reason for this difference in recovery rates is unknown, and but it may simply arise from 'statistical noise' associated with small samples. Also, it is interesting to note that similar to a study from Geneva, a delay to definitive treatment was not associated with a worse outcome, even though the median presentation latency in the Geneva was six hours, compared to our finding of three days.[9] This is also consistent with an Israeli study that showed that treatment delayed more than 48 hours is still effective in reducing DCI symptoms.[10]

First aid oxygen at scene was infrequently given to divers both in Thailand and overseas. This is comparable to a study from the Canary Islands, which also reported a low rate of provision of on-site first aid.[11] A contributing factor might be lack of awareness regarding symptoms and proper management for DCI among divers and even some physicians.[12] Of the 17 divers who received first aid oxygen on-site, 12 (71%) completely recovered from their symptoms after HBOT, compared with 53 of 82 divers (64%) without first aid oxygen (Figure 2). Accepted best practice is to administer high-concentration oxygen along with hydration as first aid for divers suspected of having DCI.[13] Apparently, either this notion is still not widely recognised, or there was not enough proper first aid kit provided at dive sites, both local and abroad, considering the low proportion of divers given oxygen first aid.

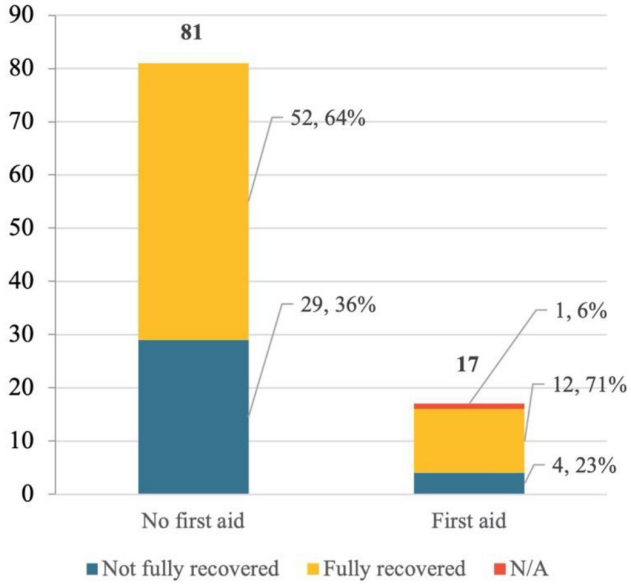
In this study, almost half of the patients received only one session of HBOT while the other half required two to five sessions. As reported elsewhere, most cases of DCS respond satisfactorily to a single session of HBOT, however, subsequent sessions are also encouraged if stepwise improvement is evident.[14]

Most dives were recreational (79 dives out of 98). There were also 14 professional dives and five dives by informal workers. The recovery rate was independent of the purpose of the diving, with 65% of both recreational and professional divers showing full recovery.

Most of the divers in this study were qualified with at least open-water certificates or equivalent. A study conducted in New Zealand, reported a declining incidence in DCI in recent years partly due to increased diving safety together with a decreased number of entry-level diving certifications.[15] A study with a larger number of cases is indicated in order to further investigate any trends in case numbers in Thailand.

The study had some limitations. The medical records were retrieved from only one treatment centre, despite many other governmental and private organisations providing

**Figure 2**
Number of reported decompression illness cases by first aid oxygen treatment received and recovery status; N/A – not available




HBOT in Thailand. Hence, the sample size was small, and no inferential statistical analyses could be performed.

## Conclusions

Decompression illness is regularly encountered in Thailand. It appears to occur in males and females almost equally. First aid treatment with normobaric oxygen is given quite infrequently. Two-thirds of the divers who received HBOT reported full recovery after treatment. The median presentation latency of three days before receiving recompression was longer than ideal, and was similar in those who dived locally and abroad. This suggested that more knowledge and awareness of diving-related illnesses should be promoted in divers, diving schools, and those involved in the tourism industry. Additionally, so as to better understand the associated factors affecting the disease and treatment outcome, more comprehensive and thorough research is needed.

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# Agreement between ultrasonic bubble grades using a handheld self-positioning Doppler product and 2D cardiac ultrasound

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**Keywords**
Decompression; Decompression illness; Decompression sickness; Diving research; Echocardiography; Ultrasound; Venous gas emboli

**Abstract**
(Plogmark O, Hjelte C, Ekström M, Frånberg O. Agreement between ultrasonic bubble grades using a handheld self-positioning Doppler product and 2D cardiac ultrasound. Diving and Hyperbaric Medicine. 2022 December 20;52(4):281–285. doi: 10.28920/dhm52.4.281-285. PMID: 36525686.)

**Introduction:** Intravascular bubble load after decompression can be detected and scored using ultrasound techniques that measure venous gas emboli (VGE). The aim of this study was to analyse the agreement between ultrasonic bubble grades from a handheld self-positioning product, the O'Dive™, and cardiac 2D ultrasound after decompression.

**Methods:** VGE were graded with both bilateral subclavian vein Doppler ultrasound (modified Spencer scale) and 2D cardiac images (Eftedal Brubakk scale). Agreement was analysed using weighted kappa ($K_w$). Analysis with $K_w$ was made for all paired grades, including measurements with and without zero grades, and for each method's highest grades after each dive.

**Results:** A total of 152 dives yielded 1,113 paired measurements. The $K_w$ agreement between ultrasound VGE grades produced by cardiac 2D images and those from the O'Dive was 'fair'; when zero grades were excluded the agreement was 'poor'. The O'Dive was found to have a lower sensitivity to detect VGE compared to 2D cardiac image scoring.

**Conclusions:** Compared to 2D cardiac image ultrasound, the O'Dive yielded generally lower VGE grades, which resulted in a low level of agreement (fair to poor) with $K_w$.

## Introduction

Bubbles in the bloodstream and tissue can form when the surrounding pressure decreases below the pressure of dissolved inert gas in the body, as during decompression from a dive. This is generally accepted to be a potential instigator for decompression sickness (DCS).[1] Intravascular bubble load after decompression can be detected and graded using ultrasound techniques that measure venous gas emboli (VGE). Two different ultrasound techniques are used to quantify VGE; Doppler audio and two dimensional (2D) echocardiography.[1] Doppler was the first of the techniques used, and VGE grades have been correlated to the risk of DCS.[2] In recent years, a device called the O'Dive™ has been developed by Azoth Systems (Ollioules, France), which is designed for use in the field to perform subclavian ultrasonic Doppler detection of bubbles.

When using this device as a lay person/diver, Azoth System's method integrates the bubble grade with depth and time information to calculate a 'severity index' for the dive. With this severity index, the manufacturer has attempted to let the diver simulate what changes in time and depth would have done for that score and what it might have done for the risk of DCS.[3] Ultrasonic 2D echocardiography grades have also been proven to be related to DCS,[4] and a good agreement with audio Doppler grades has been reported.[5] Two dimensional cardiac image grading is easier to perform for an untrained rater compared to audio Doppler grading.[6] However, it can prove significantly more challenging to get an apical four-chamber cardiac view, than collecting subclavian Doppler audio data, so both methods are challenging in order to collect reliably secure high-quality data.

The key element contributing to the O'Dive's severity index is the Doppler grade assigned by the device and therefore, we aimed to study the comparability of this score to a previously established method. We evaluated the level of agreement between bubble grades from the O'Dive and 2D ultrasonic cardiac images after wet chamber dives.

## Methods

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

### DESIGN AND SUBJECTS

This study investigated a cohort of divers performing experimental air dives (ValTKLHN2021) as per the EL-DCM Thalman dive table (SWEN 21B, unpublished), which was developed to yield an overall risk of DCS of approximately 1%. All the dives were performed in a hyperbaric wet chamber (water temperature 10°C ± 1 degree). Two divers wearing dry suits performed each dive, with several dives at each time/depth combination.

Inclusion criteria were healthy subjects that were eligible for diving in the Swedish Armed Forces, which meant they had passed the fitness-to-dive standard. Exclusion criteria were diving within the previous 48 hours, and any ongoing infection.

### ASSESSMENTS

Two dimensional cardiac images were obtained using an UltraSound EDGE II ultrasound machine (Fujifilm SonoSite, Bothell WA, USA) using a cardiac probe (rP19x5-1MHz) with the subject lying on the left side (left lateral decubitus position) giving an apical four-chamber view; in one case, the subject was shifted to the supine position and the probe positioned in the subcostal position in order to attain a view that had otherwise been unattainable. Images were graded using the Eftedal Brubakk (EB) scale[6] by two physicians in real time. All 2D cardiac recordings were preserved for review. The grading system was as follows: 0 = no bubbles; 1 = occasional bubbles; 2 = at least one bubble every 4th cycle; 3 = at least one bubble every cycle; 4 = continuous bubbling at least one bubble /cm$^2$; 5 = chamber white-out.

Doppler measurements over the left and right subclavian veins were obtained using O'Dive's Doppler transducer, VISION (2 MHz), with the recommended interface and the subject in a sitting position. Doppler assessments were graded blindly with Azoth Systems' proprietary algorithm based on a modified Spencer scale:[7] 0 = a complete lack of bubbles; 1 = an occasional bubble signal discernible with the cardiac motion signal with the great majority of cardiac periods free of bubbles; 2 = many, but less than half, of the cardiac periods containing bubble signals, singly or in groups; 3 = most of the cardiac periods contain showers of single-bubble signals but not dominating or overriding the cardiac motion signals; 4 = the maximum detectable bubble signals sounding continuously throughout systole and diastole of every cardiac period and over-riding the amplitude of the normal cardiac signals. Proprietary software (Azoth Systems) automatically determined 0, 1, 2 grades and 'high grades, 3–4' from the frequency, bubbles over time (with the assumption of a heart rate of 60). The high grades were then manually graded by a technician at Azoth System and differentiated to grade 3 or 4. We chose to use Azoth Systems grades, not grading the sound files ourselves, as we wanted to see if the semiautomatic grading system agreed with 2D ultrasound grades. The highest grade from the right or left subclavian registration was used.

The dives were conducted in pairs, and the diver who removed his diving suit first was taken for 2D cardiac imaging grading, while the second diver was then sent for evaluation with the O'Dive device. The initial measurement for all divers was made within five to 15 minutes after surfacing, and every 15 minutes thereafter. The period between the two measurements was three to ten minutes. Between four and nine paired measurements were performed after each individual dive.

### STATISTICAL ANALYSES

Agreement between bubble grades from the O'Dive and cardiac 2D images was analysed using weighted kappa ($K_w$) and reported with standard error (SE).[2,5,8] Weighted kappa is used to evaluate agreement between two grading methods when the scale is categorical and has more than two categories.[9] It ranges between 0 (worst agreement, equal to chance) and 1 (perfect agreement). In accordance with earlier studies,[5,8] we weighted deviations so complete agreement gave 1.0 credit, 0.75 credit for one category disagreement, 0.5 credit for two category disagreements and so on, down to 0 credit for four categories of disagreement (B-E category 5 were never used). Weighted deviation for the analysis with no zeros gives 1.0 credit for complete agreement, 0.67 credit for one category disagreement and so on, down to 0 credit for three categories of disagreement. O'Dive grades were paired chronologically (within ± 10 minutes) to the 2D image grades. O'Dive grades with no 2D image score within ± 10 minutes were excluded (less than 1%). Weighted kappa was calculated for the highest grades from each dive. The level of agreement (based on $K_w$) was evaluated by the following categories: poor = < 0.2; fair = 0.21–0.40; moderate = 0.41–0.60; good = 0.6–0.80; and very good = 0.81–1.00.[5,6]

Because Azoth Systems refers to an article[10] using a binary scoring system to characterise the amplification of the risk of precordial measurement compared to subclavian measurements, we also performed a complementary binary agreement analysis with Cohen's kappa.[9] Per the referenced article,[10] the adopted categories were high bubble score (3–4) or low bubble score (0–2).

To evaluate if any methodological disagreements between the two methods could be explained by scattered grades

**Table 1**
Agreement between paired bubble grades from the O'Dive and 2D cardiac imaging including zero grades; weighted Kappa 0.24 (SE 0.017)

| Grades O'Dive | Paired 2D cardiac imaging grades | | | | | Total |
|---|---|---|---|---|---|---|
| | 0 | 1 | 2 | 3 | 4 | |
| **0** | **256** | 141 | 107 | 132 | 17 | 653 |
| **1** | 36 | **44** | 35 | 103 | 13 | 231 |
| **2** | 11 | 13 | **19** | 69 | 13 | 125 |
| **3** | 1 | 6 | 6 | **54** | 12 | 79 |
| **4** | 0 | 0 | 0 | 15 | **10** | 25 |
| **Total** | 304 | 204 | 167 | 373 | 65 | **1,113** |

**Table 2**
Agreement between paired bubble grades from the O'Dive and 2D cardiac imaging excluding zero grades; weighted Kappa 0.16 (SE: 0.026)

| Grades O'Dive | Paired 2D cardiac imaging grades | | | | Total |
|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | |
| **1** | **44** | 35 | 103 | 13 | 195 |
| **2** | 13 | **19** | 69 | 13 | 114 |
| **3** | 6 | 6 | **54** | 12 | 78 |
| **4** | 0 | 0 | 15 | **10** | 25 |
| **Total** | 63 | 60 | 241 | 48 | **412** |

**Table 3**
Agreement between highest bubble grades from the O'Dive and 2D cardiac imaging; weighted Kappa: 0.30 (SE 0.045)

| Highest grades O'Dive | Highest grades 2D cardiac imaging | | | | | Total |
|---|---|---|---|---|---|---|
| | 0 | 1 | 2 | 3 | 4 | |
| **0** | **8** | 13 | 11 | 12 | 2 | 46 |
| **1** | 4 | **9** | 5 | 16 | 3 | 37 |
| **2** | 2 | 1 | **5** | 16 | 5 | 29 |
| **3** | 0 | 0 | 3 | **16** | 9 | 28 |
| **4** | 0 | 0 | 0 | 3 | **9** | 12 |
| **Total** | 14 | 23 | 24 | 63 | 28 | 152 |

**Table 4**
Binary categorical agreement analysis of low bubble grades (0–2) and high bubble grades (3–4); Cohen's Kappa 0.31 (SE: 0.064)

| Highest grades O'Dive | Highest grades 2D cardiac image | | Total |
|---|---|---|---|
| | Low | High | |
| Low | **58** | 54 | 112 |
| High | 3 | **37** | 40 |
| Total | 61 | 91 | 152 |

or systematically biased grades, the Wilcoxon signed rank test was used, with $P < 0.05$ indicating a clearly biased disagreement with lower grades for one of the methods.

## Results

A total of 162 individual dives were performed by 48 divers with eight depth/time combinations. In 152 dives, we were able to grade bubbles with both 2D cardiac ultrasound and bilateral Doppler over the subclavian veins. The mean period between each measurement was 4.6 min (SD 1.9, range 3–10). The number of dives made by each diver varied from one to 12 (11 divers with one dive, 22 divers with two dives, two divers with three dives, four divers with four dives, two divers with six dives, two divers with seven dives, one diver with 10 dives, one diver with 11 dives and one diver with 12 dives). Three different divers were diagnosed with DCS and received hyperbaric oxygen treatment once. Nine divers had minor cutaneous stress, three of whom were treated with normobaric oxygen.

Agreement between all 1,113 paired grades was $K_w$ 0.24 (SE 0.017), which was equal to a fair level of agreement (Table 1). Perfect agreement was found in 383 (34%) measurements. The O'Dive had 642 grades (58%) that were lower than the 2D image grades, and only 88 grades (8%) that were higher (Table 1). When analysing the agreement between 850 paired grades that had no more than 5 min between the measurements, the same level of agreement was found $K_w$ 0.22 ( 0.019) (without zero grades $K_w$ 0.14 (0.030); a poor level of agreement).

Agreement between all 412 paired grades without the inclusion of zero grades was $K_w$ 0.16 (0.026); a poor level of agreement (Table 2). Perfect agreement was found in 127 (31%) measurements. The O'Dive had 245 grades (59%) that were lower than the 2D cardiac ultrasound grades, and 40 grades (10%) that were higher (Table 2).

The agreement between all 152 paired highest grades was $K_w$ 0.30 (0.045); a fair level of agreement (Table 3). In 47 cases (31%), the highest grades were the same. In 92 cases (61%) the O'Dive's highest Doppler grades were lower compared to 2D image highest grades, and in 13 cases (9%), it was higher (Table 3). In 30% (14 of 46) of the O'Dive's zero grades, the 2D image grade was 3 or 4. However, none (0 of 14) of the 2D ultrasound zeros resulted in a high grade (3 or 4) from the O'Dive.

The binary categorical agreement analysis of low bubble grades (0–2) and high bubble grades (3–4) Cohen's kappa was 0.31 (0.064) (Table 4). In 95 cases (63%), both methods

produced the same category. In 54 cases (36%), the O'Dive's category was lower compared to the 2D image category, and in only 3 cases (2%) was it higher (Table 4).

All agreement analyses clearly indicated a bias, with generally lower grades given by the O'Dive, which was also shown by the Wilcoxon signed-rank test, which gave significant results in all cases.

## Discussion

The main finding of the present study was that the O'Dive's subclavian Doppler bubble grades had a fair to poor agreement with 2D cardiac ultrasound images.

An earlier study comparing subclavian Doppler grades collected manually by a trained Doppler probe operator, with 2D cardiac grades showed an agreement of good to very good.[5] In contrast, in the present study the O'Dive generally yielded lower bubble grades, which means that it has a lower sensitivity to intravascular bubbles transported to the right heart. Another recently published study[11] involving 173 paired measurements reported a similarly weak correlation between the O'Dive and 2D image categorical assessment scales. That study also found a poor sensitivity to VGE for the O'Dive in comparison to 2D images made using a Vivid q™ device (GE Healthcare, Chicago IL, USA). As the O'Dive had only a fair to poor level of agreement with the established methodology, the implication of this study is that the grades from one method cannot be directly translated to the grades of the other; the degree of difference between the two clearly indicates bias.

A strength of this study is the large number of controlled dives ($n = 162$). In 152 dives, we were able to assess bubbles in four to nine post-dive measurements with both Doppler and 2D cardiac ultrasound, giving a total number of 1,113 paired measurements to include in the analysis. This method of measuring bubbles after dives with 2D ultrasound grades complies with published guidelines.[12] Furthermore, our dives had a relatively high frequency and range of bubbles; this diversity is important when analysing agreement and correlations related to bubble load across the range of the scales.

Limitations of this analysis include the two different anatomical locations. Intravascular bubbles coming from the lower body and/or the neck and head cannot be detected in the subclavian veins, as they drain blood from the arms and shoulders only. However, one large study[2] did not report that the subclavian Doppler signals had a decreased sensitivity to detect intravascular bubbles in general, compared to signals from the chest.

Another limitation is that the frequency of the O'Dive's ultrasonic Doppler device is 2 MHz, while that of the 2D cardiac images we obtained using the UltraSound EDGE II device was 5-1 MHz. This difference in probe frequencies and detection techniques could theoretically lead to different sensitivity to bubbles. The smallest bubble detectable using 2D cardiac images is thought to be between 10–20 μm[8,13] and for Doppler audio ultrasound no smaller than 30 μm.[8,14]

A third limitation is the time taken between the measurements (mean 4.6 min [SD 1.9, range 3–10 min]). This can influence agreement due to the dynamic character of bubble evolution, especially as there was no restriction to the participants in terms of movement between the measurements. A fourth limitation is that many divers did more than one dive. For example, three divers did 33 of the 152 dives. Therefore, these divers will influence the agreement between the two methods more than the other individuals. A fifth limitation is the method by which the O'Dive measurements are graded, combining an automatic and manual grading of bubbles by Azoth Systems (see methods). In this process, the heart rate is approximated to 60 beats per minute, which may certainly influence the result as the Spencer scale categorises bubbles by heart period. The fact that Azoth Systems choose to assess grades 3 and 4 manually is probably because of software limitations, making the results harder to interpret.

## Conclusions

The O'Dive's grades yielded a low level of agreement compared to 2D ultrasound cardiac image grades. Generally, the grades were lower with O'Dive and the level of agreement was fair to poor.

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**Conflicts of interest and funding**

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No conflicts of interest were declared.

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# Case report

## Electric shock leading to acute lung injury in a scuba diver

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**Keywords**
Burns; Diving; Electric injuries; Salt water aspiration

**Abstract**
(Johnson-Arbor K. Electric shock leading to acute lung injury in a scuba diver. Diving and Hyperbaric Medicine. 2022 December 20;52(4):286–288. doi: 10.28920/dhm52.4.286-288. PMID: 36525687.)
**Introduction:** Electrical injuries are a rarely reported complication of scuba diving.
**Case report:** A 33-year-old woman wore a 12-volt heated shirt designed for motorcycling, powered by a canister light battery, while scuba diving. A leak in her drysuit allowed water to make contact with an electrified connector from the heated shirt, and she experienced painful electrical shocks. She was able to disconnect the power source and finish the dive, but she developed progressive fevers and dyspnoea several hours later. She was diagnosed with acute lung injury and treated with bronchodilators. Her symptoms resolved over subsequent weeks.
**Discussion:** Acute lung injury is rarely reported after low voltage electrical injury. In this case, the use of a heated shirt that was not intended for underwater activities heightened the patient's risk for electric shock that likely resulted in aspiration of sea water and subsequent acute lung injury. To reduce risk of injury, divers should use equipment that is designed for underwater submersion. Medical professionals who treat the diving population should be aware that divers may use modified equipment that increases the risk of diving-related complications.

### Introduction

Electrical injuries are rare, accounting for approximately 5% of admissions to major burn centres.[1] Although significant exposures to electrical current are rarely encountered during underwater diving, electrical injury can cause significant disease or death if it occurs while submerged underwater. This report describes a case of low voltage electrical shock in a scuba diver that indirectly resulted in acute lung injury. Written permission for case report publication was obtained from the patient.

### Case report

A 33-year-old woman was participating in technical scuba diving using a closed-circuit rebreather apparatus. To prevent cold exposure, she wore a thermal base layer and a 12 volt (V), 2.8 ampere (Amp) heated shirt marketed for motorcyclists, underneath her drysuit. The heated shirt was long-sleeved, with electrical wires throughout the trunk and arms, and was powered by an 18.5 V diving canister light battery attached to the exterior of her drysuit. On occasion, she wore heated motorcycle gloves. These gloves had wires that connected to the heated shirt and were also powered by the battery.

On an excursion to a depth of 45 meters (150 feet) of seawater, she chose to not wear the heated motorcycle gloves due to a need for increased manual dexterity. The distal left wrist connector on the heated shirt, which was normally connected to the left glove wire, remained exposed within the drysuit (Figure 1). A leak in one of her drysuit wrist seals allowed water to enter the suit. When she turned on power to the heated shirt, she immediately felt the sensation of electrical shocks along her left wrist, adjacent to the exposed connector. She experienced pain, muscular contractions of her upper extremities, and blurred vision, but did not lose consciousness. She screamed in pain, but a mouthpiece retaining strap prevented her from expelling her dive surface valve (DSV) from her mouth. She was unable to communicate what had happened to her dive partner, but she was able to manually disconnect power to the heated shirt and stop the electrical shocks. As her dive profile had been otherwise uneventful, she then finished her dive, completed a normal ascent and decompression, and drove home.

Later that evening, she developed a low-grade fever. The following day, her temperature rose to 39.2°C and she experienced chills as well as dyspnoea. She took rapid COVID-19 antigen tests which were negative. Due to worsening dyspnoea, she sought medical care three days later.

**Figure 1**
Wrist connector that was exposed to electrical current and salt water

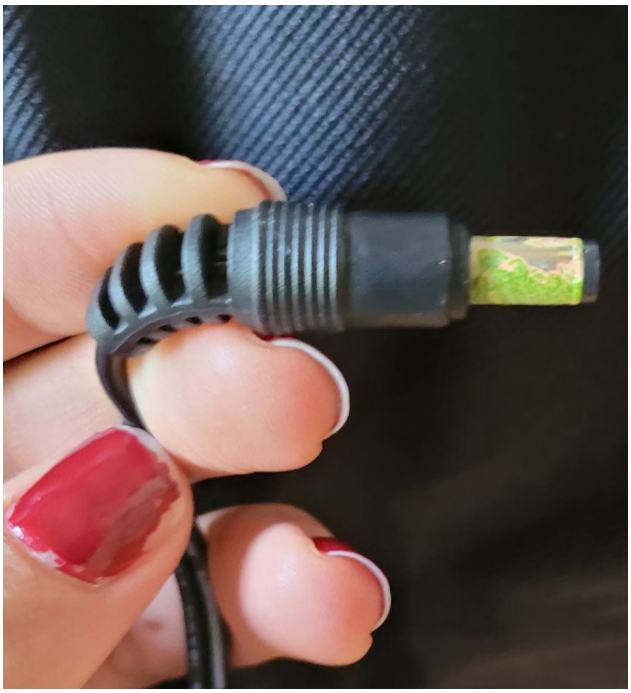
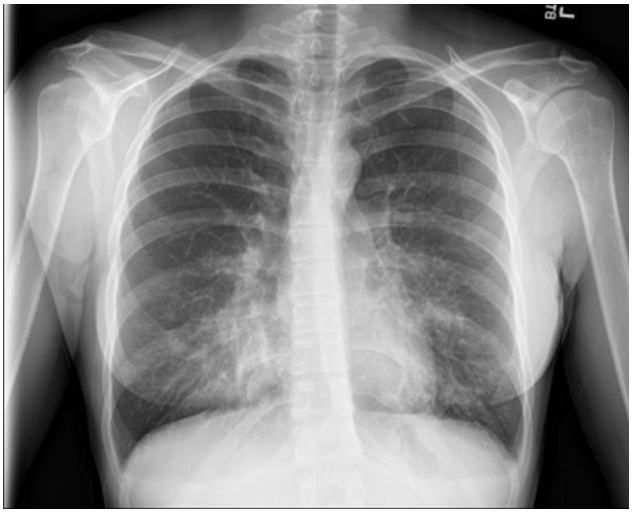


**Figure 2**
Bilateral airspace disease involving the right middle lobe and lingula on chest radiograph



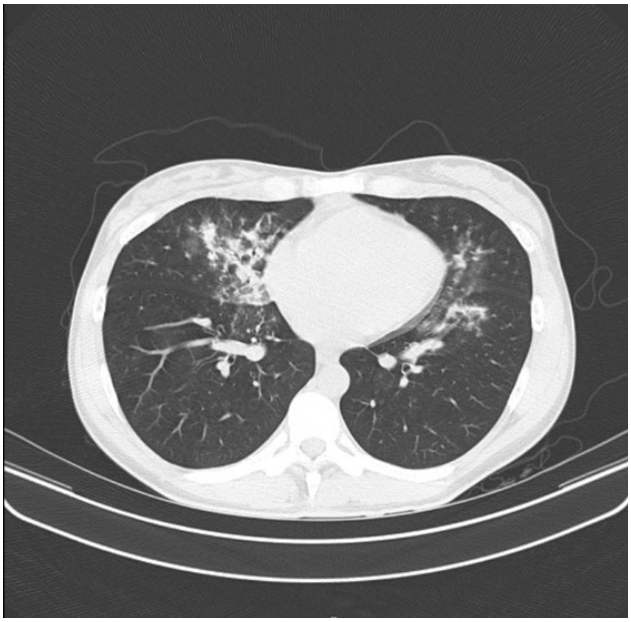
At that time, her vital signs were unremarkable (temperature 36.7°C, respiratory rate 18 per minute, oxygen saturation 99% on room air). Physical examination revealed diminished sounds at both lung bases. An electrocardiogram revealed normal sinus rhythm, normal QRS and QTc intervals, and no acute ischaemic changes. A chest X-ray showed bilateral airspace changes (Figure 2), and computed tomography (CT) of the chest performed two days later revealed bilateral anterior nodular and ground glass opacities involving all five lung lobes (Figure 3). Laboratory evaluation, including renal function, urinalysis, creatinine kinase concentration, and complete blood count, was unremarkable (the white blood cell count was 7.5 1,000·uL$^{-1}$ [normal range 3.4–10.8 1,000·uL$^{-1}$]. A diagnosis of acute lung injury was established, and an albuterol metered-dose inhaler was prescribed. The patient's dyspnoea and fevers improved over the next few days, although she reported experiencing intermittent palpitations with exertion. Six weeks after the dive, she remained asymptomatic. Subsequent evaluations, including pulmonary function testing, cardiac echocardiography, and exercise stress testing were within normal limits. A repeat chest CT scan showed resolution of the previously noted pulmonary abnormalities. The diver was advised that she could safely resume diving, although use of the heated motorcycle shirt while underwater was strongly discouraged.

## Discussion

This diver experienced acute lung injury after sustaining electrical shocks, but the relationship between the electrical injury and her acute lung injury was initially unclear. Electrical injuries result from the passage of heat and electrical current through tissues, which causes cellular damage through muscle fibrillation and coagulation necrosis.[2] Electrical injuries are often divided into those resulting from low voltage (< 1,000 V) and high voltage (> 1,000 V) exposures, but the current and duration of shock are also factors that determine the extent of injury. Acute lung injury is rarely described in the context of low voltage electrical injury but has been reported to occur after exposures to 110-380 V, much higher than the 12 V exposure experienced by the patient described above.[3-6] As a 12 V shock is unlikely to directly cause acute lung injury, it is unlikely that the electrical shocks experienced by this diver were a direct cause of her pulmonary injury, and thus alternative causes were explored. Both immersion pulmonary edema and a 'caustic cocktail' exposure were considered as potential diagnoses, but were ultimately dismissed as the timing and characteristics of the signs and symptoms were inconsistent with these diagnoses. Seawater aspiration can cause acute lung injury, and the anterior location of the infiltrates noted on this patient's chest CT imaging were consistent with aspiration occurring in the prone (e.g., diving) position. Although the patient did not recall aspirating oral contents or sea water during the dive, it is possible that this occurred while she screamed after initially receiving electrical shocks, and that this caused her subsequent pulmonary injury.

After seawater aspiration was established as the most likely cause of acute lung injury, the events that contributed to the aspiration event were investigated. As described, the diver wore a heated shirt under her drysuit. A leak in the drysuit allowed water to contact an exposed electrical connector on the shirt, which created an electrical current. Heated undergarments are used by divers for thermal protection and to potentially reduce the risk of decompression sickness.

**Figure 3**
Chest computed tomography scan image with bilateral opacities present



The use of a heated undergarment while diving introduces a risk of electric shock (and possibly thermal burns), as demonstrated in this case report. Divers may also experience unexpected injuries when using batteries with a different voltage than the heated garment (e.g., an 18 V battery on a 12 V heated shirt). While there are heated undergarments that are marketed as being safe for use while diving, some divers use retrofitted shirts intended for motorcycle riding, hunting, or other outdoor activities. These garments may be advertised as being water-resistant, but they are not intended for use underwater. Modification of these garments may result in complications including electrical injury, especially in the event of a leaking drysuit. The diver was fortunate in that she wore a mouthpiece retainer strap that prevented her from expectorating her DSV during the initial electric shock. The location of the battery exterior to her drysuit also made it easier for her to disconnect the power to the heated shirt. Had the battery been secured on the inside of the drysuit, the painful muscular contractions she experienced as a result of the electrical shocks would have complicated her ability to quickly or effectively disconnect the battery from the shirt. Divers who wear drysuits with interior batteries are at increased risk for electrical injury as well as thermal burns and should remain cognizant of these potential complications when purchasing and using these devices. Additionally, to optimise diver safety, drysuit system manufacturers should avoid producing garments with interior batteries.

## Conclusions

Seawater aspiration precipitated by electric shock represents an extremely rare but potentially life-threatening complication of underwater diving. To reduce the risk of electric shock and acute lung injury related to seawater aspiration, divers should only use equipment that is designed for underwater use. When wearing heated garments underwater, divers must ensure that their drysuits and other equipment are functioning properly, without significant leaks that might allow water to make contact with electrified wires. A mouthpiece retainer strap may be lifesaving in the event of underwater electric shock or any other event that causes distress or loss of consciousness, and use of such a device should be considered by all divers who wear heated equipment. Divers should be aware of the potential for adverse events, including direct electrical injury and/or seawater aspiration, when using battery-powered equipment underwater and should be encouraged to promptly seek medical attention if electrical injury occurs while submerged.

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**Conflicts of interest and funding:** nil

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# Obituary

## Professor Peter Bennett, PhD, DSc

It is with great sadness that we mark the passing of yet another iconic figure in our field in 2022. Professor Peter Bennett died on 9 August 2022.

Peter was originally from the UK (born in Portsmouth in 1931). He completed his undergraduate degree in London before working at the Royal Navy Physiological Laboratory (RNPL) during the 1950s. The RNPL was a true centre of excellence in diving medicine research over this period. He completed his doctoral degree through the University of Southampton in 1964, and contributed immensely to knowledge of the effects of gases respired under pressure during this 'heyday' of diving medicine research.

Peter moved to the USA in 1972 and initially worked at Duke. In 1981 he conducted the Atlantis III dive lasting 43 days with a maximum depth of 690 m (2,250 feet). Around the same time he began the work that I believe will form his most enduring legacy; the creation of a diving emergency hotline service that evolved into the Divers Alert Network (DAN) that we recognise today. It was during this process, and in his transformation from Duke scientist to President and CEO of DAN, that it became clear Peter was not only a talented scientist but also a visionary and skilled entrepreneur. His vision was for DAN to become not only a hotline, but also an institution with the resources to sustain a program of research targeted at improving diving safety. DAN developed and sold oxygen provision equipment, provided related training, and marketed diving insurance (all being substantial contributions to safety in their own right), but the best part was that the income from these ventures was ploughed back into DAN-funded research. The research itself was invariably targeted at answering questions of high practical relevance to the diving community. Absolutely brilliant! Peter retired from DAN in 2003.

Peter's service to the diving community didn't end with DAN. He was a founding member of the Undersea and Hyperbaric Medical Society, served as President for two years, and was Editor of Undersea Biomedical Research (later Undersea and Hyperbaric Medicine) for three years. He also came on board as the Executive Director in 2007 at time of significant financial stress for the society. This was an opportunity for Peter to once again demonstrate his entrepreneurial credentials, and he did not disappoint. His careful stewardship of the operational side of the society's activities saw it grow into a position of financial stability that has persisted long after his departure.

I will confess, as a junior doctor in the field in the early 90s, to having found Peter an intimidating figure. He was always immaculately dressed, unfailingly dignified in bearing and demeanour and just a little bit scary. My understanding of him completely changed when, as part of the selection process for his successor at DAN in 2003, I was invited to go out for a one-to-one dinner with him. I truly had no idea what to expect, but what it turned into was a magical evening of convivial insights and anecdotes from all corners of the diving medicine world. I probably learned more about our field that evening than in any other single encounter in my career. Then, around 2010 my wife Siân and I were in Durham where I was speaking at a meeting. Siân had a desire to experience southern Creole style food at an authentic restaurant and the one night when we might be able to do it we had arranged to have dinner with Peter and Margaret. Despite predictions by some that this was not the sort of place Peter would normally choose for entertaining guests, we will never forget Peter and Margaret whisking us off to a basic but ultra-authentic eatery in Chapel Hill. A terrific evening with the best laughs. Siân's verdict: a truly delightful English gentleman! Perhaps an odd anecdote for an obituary, but it's a very fond personal memory of a friend and colleague often perceived as 'formal' but in reality, often witty, warm and 'casual'. I am very grateful for having gotten to know Peter properly.

Peter's legacy cannot be overstated. The founding of DAN and the stewardship of its growth into by far the most recognisable organisation contributing to diving safety worldwide was an immense achievement, as was his collaboration with David Elliott (who we also lost this year) to produce a book that remains the field's most famous even 20 years after it was last published. He was indisputably a giant in diving medicine.

*Professor Simon Mitchell*
*Editor, Diving and Hyperbaric Medicine Journal*

# Notices and news

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

## EUBS President's report

Jean-Eric Blatteau

Dear friends and colleagues,

I take advantage of this page to pay tribute to the epic story of human diving at great depths with the use of synthetic breathing mixtures based on hydrogen.

Thirty years ago, on 20 November 1992, a diver from the Comex company in France, Theo Mavrostomos, succeeded in making the deepest simulated dive in the world at 701 metres. The "Hydra X" project was the culmination of several decades of work in the field of saturation diving and deep incursions conducted in collaboration with the French Navy. The media objective of "Hydra X" was to exceed 683 meters (the depth record at that time, held by the Americans) by testing an innovative mixture composed of hydrogen, helium and oxygen ('hydreliox').

The team was made up of three men for this attempt at the record but at 675 metres, two of them presented symptoms and did not feel they were in a condition to continue. The attempt seemed to fail and they ascended back to the pressure of 650 metres, but after many discussions and consultation with the medical team, Theo continued alone with the attempt to beat the record. He stayed for three hours at 701 metres and even carried out a physical exercise program. It took 13 days of compression to live these three short hours at depth and 23 days to come back to the surface pressure.

This extreme dive represents only the visible part of the vast Hydra program, which over 17 years has allowed identification of the psycho-sensory and behavioral manifestations of 'hydrogen narcosis' which only appears beyond a partial pressure of hydrogen of 2 MPa. By respecting this limit, the hydreliox breathing mixture has made it possible to limit the effects of HPNS and to increase respiratory comfort, thus allowing human work at very great depths. The Hydra program was completed in 1999 with the 'Hydra Ludion' which showed the possibility of achieving rapid variations in pressure between the saturation depth of the diver (life level) and that of the underwater work site (work level), thanks to hydrogen. Compared to the helium ludions currently used, hydrogen allows pressure variations that are twice as important.

Finally, we must not forget the precursory work of the engineer Arne Zetterström of the Swedish Navy, pioneer of hydrox diving (hydrogen-oxygen mixture), who succeeded between 1943 and 1944 to reach a depth of 160 metres at sea. He set up a technique of substitution of the breathing mixtures in diving in order to limit the risk of flammability of hydrogen by maintaining a percentage of oxygen lower than 4%. Zetterström tragically died during a dive on 7 August 1945 after a miscommunication on the surface resulting in a rapid ascent without decompression stops nor gas switch, resulting in severe hypoxia and decompression sickness.

The memory of Arne Zetterström is celebrated every year by the EUBS by delivering the 'Zetterström Award' for best poster presentation at the EUBS Annual Meeting.

*Jean-Eric Blatteau*
*EUBS President*

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.

# EUBS news

**EUBS 2023 Annual Scientific Meeting**

The 46th EUBS Annual Scientific Meeting in Prague was a great success, and it is now time to start preparing for the 47th EUBS Annual Scientific Meeting. Our EUBS 2023 meeting will take place from 13–16 September 2023, in Porto, Portugal. Porto ('Oporto') was elected Europe's best city destination at the World Travel Awards 2022.

EUBS hopes to welcome its members and many friends and scientists from around the globe for this four day conference, to gather in person and renew/strengthen our professional and personal friendship and relationships. Keep monitoring the website www.eubs2023.com for all the news and updates. Our next annual meetings will be scheduled as: 2024 – Brest, France; 2025 – Turku, Finland.

**EUBS Annual General Assembly**

Our annual EUBS General Assembly took place on 3 September 2022, in Prague, on the last day of the EUBS Annual Scientific Meeting.

The report of the General Assembly and supporting documents (financial report, results of 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. 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.

The collaboration between EUBS and ECHM is ongoing and growing, with the final goal of achieving a fully integrated collaboration/merger to benefit the goals and objectives of both organisations to mutual satisfaction. Examples of this already strong bond are the joint Position Statements on COVID-19 related matters in diving and hyperbaric medicine, and a new Position Statement on Mild Hyperbaric Therapy, which will be published by ECHM as a joint document, should be publicly available on both societies' websites by the time this issue of DHM is published.

**EUBS Executive Committee**

To replace Gerardo Bosco from Padua (Italy), after serving a three year term, we have elected two new Members-at-Large. According to the GA2021 decision, the position of Member-at-Large will, from now on, have a four-year tenure instead of three. Both candidates for the position have been elected: Charles P Azzopardi (Valetta, Malta) will serve a three-year term and Anne Räisänen-Sokolowski (Helsinki, Finland) will serve a four-year term.

The Executive Committee wish to express their gratitude for Gerardo's (Dino) contributions to the ExCom activities, and we are happy that he will continue to support ExCom as a member of the research and education committee. 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 are managed by Bengusu Mirasoglu (bengusu.mirasoglu@eubs.org).

**EUBS membership**

Do not forget to renew your EUBS membership. In case your membership has expired, you will see a message when trying to log in on the EUBS website. You can then renew it immediately online.

EUBS membership gives you significant advantages, such as immediate access to the most recent issues of the DHM Journal and (if selected) a print copy of the eJournal 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.

In case you have difficulties renewing or accessing your membership area, please contact us at secretary@eubs.org. Please do note that payment by PayPal is by far the easiest and cheapest way to pay your membership fee. You can also pay by bank transfer, but this will incur banking costs for international money transfers (EUBS is registered in the UK, which is now outside of Europe). You have to make sure to select this ("*all banking costs carried by the sender*")

when you make the transfer. Also, a money transfer may take up to one week, and may fail for some obscure reason. Therefore, unless you are in the UK, we cannot 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 and keep updated on 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 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 consulted through a European (and World) map interface, through the Menu item 'OXYNET map' (sounds logical) 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.

Also please 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).

In case you have any suggestions for additional info or corrections, please contact us at webmaster@eubs.org.

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SPUMS
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

I write this report having just returned from a liveaboard diving trip to the Rowley Shoals with our Immediate Past President, David Smart and friends.

The Rowley Shoals is a group of three coral reef atolls (Clerke, Mermaid and Imperieuse) approximately 300 kilometres to the west of Broome, Western Australia and only accessible by boat. The reefs have amazing diving (wall and bommie) with pristine hard and soft corals and abundant fish life, sadly, unless we rafted up the few live-a-boards that go there, it is not suitable for a SPUMS Annual Scientific Meeting (ASM). We do, however, have a confirmed ASM venue and theme in Australia in 2023 and we are looking at overseas options for 2024 and any suggestions regarding a suitable location would be gratefully received.

The SPUMS 2023 ASM will be held in Cairns and will include diving with the programme and registration available now on the (new!) SPUMS website. South Pacific Underwater Medicine Society – SPUMS-ASM.

> ***Conference theme:*** *Diver health and ocean health amidst the storm clouds of climate change. A shared vision for underwater medicine and marine science.*
>
> ***Convenors:*** *David Smart and Cathy Meehan*
> ***Date:*** *Sunday 4 June to Friday 9 June 2023*
> ***Venue:*** *Crystal Brook Riley Hotel, Cairns, Australia*

There will also be a workshop to develop a SPUMS Position Statement on paediatric diving.

If you are yet to register, I strongly encourage you to do so soon before the 'early bird discount' expires on 4 January 2023. Pre- and post-conference liveaboard trips to the Great Barrier Reef are available for registrants which feature diving with minke whales.

The beginning of 2023 will see the retirements of two giants in the field of diving and hyperbaric medicine, former SPUMS Presidents Clinical Professor David Smart, AM and Professor Mike Bennett, AM. Both Mike and David have published extensively and have made enormous contributions to our Society. On behalf of SPUMS I offer our sincere thanks to David and Mike for their hard work over many years. We wish you a long and happy (and not too busy) retirement. David will continue as Immediate Past President until I assume that role when I make way for our next President.

Life seems to be returning to some semblance of normalcy as we continue to 'live' with COVID-19. Members are reminded that there is useful information on our website under the 'Resources' tab for assessment of fitness to return to diving post COVID-19. South Pacific Underwater Medicine Society – COVID-19 Updates (spums.au).

The next Introductory Course in Diving and Hyperbaric Medicine will again be held in Fremantle from 27 February – 10 March 2023, with strong interest already shown. This course is only held yearly and is always fully subscribed early, so if you want to register, don't delay. South Pacific Underwater Medicine Society - SPUMS-Approved Courses.

Finally, I would like to wish all our members a safe and enjoyable festive season and a happy new year. I look forward to seeing you in Cairns in June next year.

*Neil Banham*
*SPUMS President*

SPUMS Facebook page
Like us at:
SPUMS on Facebook

Government of **Western Australia**
**South Metropolitan Health Service**
**Fiona Stanley Fremantle Hospitals Group**


## The Australian and New Zealand Hyperbaric Medicine Group

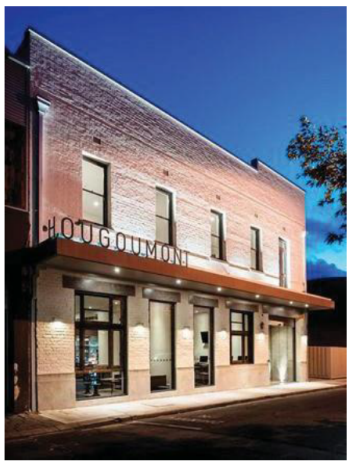
# Introductory Course in Diving and Hyperbaric Medicine

**Dates:** 27th February – 10th March 2023
**Venue:** Hougoumont Hotel, Fremantle, Western Australia
**Cost:** AUD 2,900 for 2 weeks

The course is for medical graduates with an interest in diving and hyperbaric medicine. It is designed both for those wishing to pursue a career in this specialised field and those whose primary interest lies in related areas. The course will be held in Fremantle with excursions to the Fiona Stanley Hyperbaric Medicine Unit, HMAS Stirling and the local Royal Flying Doctor base. The course is accredited with the South Pacific Underwater Medicine Society and ANZCA for the Diploma of Diving and Hyperbaric Medicine.

**The Course content includes:**

- History of diving medicine and hyperbaric oxygen
- 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 Ovens, Course Administrator
Phone: +61-(0)8-6152-5222
Fax: +61-(0)8-6152-4943
E-mail: fsh.hyperbaric@health.wa.gov.au
Accommodation information can be provided on request

FSHM20220322004

## Royal Australian Navy Medical Officers' Underwater Medicine Course

**Date:** 13–24 March 2023

**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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The **NEW**

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.

# 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

## 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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Historical Diving Society Australia - Pacific

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

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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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## 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 30 years. SHF is targeting on an international audience with courses worldwide.

We are happy that the world has reopened after the COVID-19 pandemic and we can announce courses around the world again. Below the schedule of upcoming SHF-courses in 2023.

The courses Medical Examiner of Diver (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).

### 2023 (first half)

| | |
|---|---|
| **25 February** | Diving Medical in Practice<br>The Netherlands |
| **24–25 March** | Medical Examiner of Divers part 1 (level 1)<br>The Netherlands |
| **30 March – 1 April** | Medical Examiner of Divers part 2 (level 1)<br>The Netherlands |
| **13–20 May** | Medical Examiner of Divers-part 2 (level 1)<br>Bonaire, Dutch Caribbean |
| **9–10 June** | In-depth course Nightmares for the Diving Doc (level 2d)<br>The Netherlands |
| **In planning** | Decompression,recompression and HBOT (level 2d), tbd |
| **On request** | Internship HBOT (level 2d certification), NL/Belgium |

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

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## DHM Journal Facebook

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

# Diving and Hyperbaric Medicine: Instructions for authors (summary)

(updated August 2021)

*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 accompaniesthe 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 user name 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 word count); include an informative **Abstract** of no more than 300 words (excluded from 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 word count); include an informative **Abstract** (structure at author's discretion) of no more than 200 words (excluded from word count).

**Educational articles, Commentaries** and C**onsensus 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 requirements outlined in the full version of the Instructions for authors. 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 (Full version)
DHM Key words 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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ADSF
AUSTRALASIAN DIVING SAFETY FOUNDATION

## 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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