Diving and Hyperbaric Medicine

The Journal of the South Pacific Underwater Medicine Society and the European Underwater and Baromedical Society©

SPUMS

Volume 56 No. 2 June 2026

EUBS

Does prior exposure facilitate recognition of hypercapnia?

Technical diving accidents in France

Measuring uptake and elimination of inert gas

Transoesophageal vs transthoracic echo for PFO detection

Accuracy of oxygen flowmeters in hyperbaric conditions

Recommendations for long term care in spinal DCS

Diving with bipolar spectrum disorder

Diving after otologic surgery

Rebreather diving fatalities

Four case reports

E-ISSN 2209-1491

ABN 29 299 823 713

THE JOURNAL OF DIVING AND HYPERBARIC MEDICINE
***Diving and Hyperbaric Medicine* is published online jointly by the South Pacific Underwater Medicine Society and the European Underwater and Baromedical Society**
https://www.dhmjournal.com

EDITORIAL OFFICE

PURPOSES OF THE SOCIETIES

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

E-ISSN 2209-1491; ABN 29 299 823 713

CONTENTS

Diving and Hyperbaric Medicine Volume 56 No. 2 June 2026

Diving and Hyperbaric Medicine is indexed on MEDLINE, Web of Science® and Embase/Scopus
Articles from 2017 are deposited in PubMed Central®

The Editor's offering

As the June issue of DHM 'goes to print' we are still bathing in the afterglow of a massively successful SPUMS meeting at Palau. Palau is one of those venues where there is a tacit understanding that the society will need to accept lower delegate numbers in order to access fantastic diving in a more remote location. Not this time. SPUMS 2026 at Palau was attended by an almost record number of delegates and accompanying partners and family. The meeting had a massive energetic vibe, lots of young attendees, and a fantastic scientific program themed around breath-hold diving and headlined by Peter Lindholm and Erika Shagatay.

This issue of DHM is strongly diving-aligned. It kicks off with a randomised study from my own group investigating whether an open label exposure to hypercapnia would make divers better at recognising hypercapnia in a subsequent blinded exposure. There was a non-significant trend to better recognition in divers who had experienced hypercapnia. The study may have been underpowered to demonstrate a real training effect, and/or it may be that hypercapnia is so unpleasant that it is recognised by most divers, even if they had not experienced it previously.

Emmanuel Gouin and colleagues extend their work thematically aligned with technical diving in a study describing diving accidents in French technical divers. They report that a significant proportion of this cohort suffering decompression sickness exhibited musculoskeletal symptoms.

Oscar Plogmark and colleagues describe a novel method for measuring uptake and elimination of inert gas during a dive and subsequent decompression. The uptake of inert gas is an under-studied aspect of vulnerability to decompression sickness, and Oscar's work is an exciting initiative.

Peter Wilmshurst and colleagues address the widely held belief that transoesophageal echocardiography (TOE) is the gold standard for cardiac imaging to detect a persistent foramen ovale (PFO). They showed that a substantial proportion of patients who had a PFO detected using transthoracic echocardiography (TTE) were negative for PFO with TOE. These findings reinforce the advocacy in the recent joint UKDMC – SPUMS consensus for use of TTE and bubble contrast when investigating a diver for PFO.[1]

In a technical report, Yoav Aufgang and the Alfred (Melbourne) group document the discrepancy between indicated and actual flow from commonly used oxygen flowmeters under hyperbaric conditions. This issue became important in supplying ECMO oxygenator sweep gas in what has become the famous world-first hyperbaric ECMO case reported in the December 2025 issue of DHM.[2]

In the first of three reviews, Camille Dubois and colleagues went looking for published guidance on the long term care of divers who have suffered spinal decompression sickness and found… nothing. They have provided such guidance, drawing partly on literature pertaining to other spinal cord injury, and partly based on their own extensive experience. Bram Querido and Thijs Wingelaar review the issue of diving by candidates with bipolar disorder. Perhaps not surprisingly they advocate a cautious individualised approach to assessment. The article is extremely useful in providing the framework for such an assessment.

In another review with a fitness for diving theme, Juan Riestra-Ayora and colleagues consider the issue of diving after otologic surgery. This has been a controversial matter, and this review is particularly useful in rationally challenging some of long-standing dogma around the subject.

In an article we have chosen to publish under the 'World as it is banner', Frauke Tillmans and colleagues have attempted to bring at least some objectivity to the debate about the safety or otherwise of rebreather diving. By the authors own admission, such a goal is hampered by incomplete data both in respect of a numerator (e.g., related fatalities) and a denominator (e.g., number of rebreather divers or number of dives). Nevertheless, the likely interest value of this paper to the rebreather diving community cannot be overstated.

There are reports describing cases of pyomyositis and choroidal neovascular membrane treated with hyperbaric oxygen. There is a report describing what I believe to be only the third published case of arterial gas embolism in a patient undergoing hyperbaric oxygen treatment, and only the second in which the event occurred on the first exposure. The authors highlight the dilemma in decision making where the primary treatment of a problem is the thing that caused the problem in the first place. There is a case report describing return to diving and hyperbaric exposure after pulmonary vein isolation for atrial fibrillation.

References

1 Smart D, Wilmshurst P, Banham N, Turner M, Mitchell SJ. Joint position statement on atrial shunts (persistent [patent] foramen ovale and atrial septal defects) and diving: 2025 update. South Pacific Underwater Medicine Society (SPUMS) and United Kingdom Diving Medical Committee (UKDMC). Diving Hyperb Med. 2025;55:51–5. doi: 10.28920/dhm55.1.51-55. PMID: 40090026. PMCID: PMC12043516.

2 Devaney B, Mathew J, Ferris S, Roberts L, Covelli C, Orosz J, et al. Novel use of hyperbaric oxygen treatment for treatment-resistant disseminated Saksenaea and Fusarium in a patient on extracorporeal membrane oxygenation (ECMO): a case report. Diving Hyperb Med. 2025;55:309–14. doi: 10.28920/dhm55.4.309-314. PMID: 41364853. PMCID: PMC12823155.

Professor Simon Mitchell
Editor, Diving and Hyperbaric Medicine

Cover photo: SPUMS delegates diving he Blueholes, Palau, May 2026. **Photo:** Simon Mitchell

Original articles

Effect of a prior hypercapnia experience on recognition of hypercapnia in divers: a randomised controlled study

Thalia Babbage[1], Hanna van Waart[1], Charlotte JW Connell[2], Nicholas Gant[2], Simon J Mitchell[1,3,4], Xavier CE Vrijdag[1]

[1] *Department of Anaesthesiology, University of Auckland, Auckland, New Zealand*
[2] *Department of Exercise Sciences, University of Auckland, Auckland, New Zealand*
[3] *Department of Anaesthesia, Auckland City Hospital, Auckland, New Zealand*
[4] *Slark Hyperbaric Unit, North Shore Hospital, Auckland, New Zealand*

Corresponding author: *Dr Xavier Vrijdag, Department of Anaesthesiology, School of Medicine, University of Auckland, Private bag 92019, Auckland 1142, New Zealand*
ORCiD: *0000-0001-5907-6083*
x.vrijdag@auckland.ac.nz

Keywords
Diving medicine; Diving research; Carbon dioxide; Rebreathers – closed circuit; Rebreathing; Technical diving

Abstract
(Babbage T, van Waart H, Connell CJW, Gant N, Mitchell SJ, Vrijdag XCE. Effect of a prior hypercapnia experience on recognition of hypercapnia in divers: a randomised controlled study. Diving and Hyperbaric Medicine. 2026 30 June;56(2):115–124. doi: 10.28920/dhm56.2.115-124. PMID: 42290571.)

Introduction: Rebreather diving carries an increased risk of hypercapnia. Hypercapnia can cause impaired cognition, breathlessness, and increase the risk of oxygen toxicity. We investigated whether a prior unblinded hypercapnia experience, compared to reading about hypercapnia symptoms, would improve divers' ability to recognise hypercapnia and initiate self-rescue.

Methods: Forty divers were recruited and randomised to receive either an unblinded hypercapnia experience (partial pressure of end-tidal carbon dioxide [$P_{ET}CO_2$] of 8.5 kPa) or an information leaflet explaining hypercapnia symptoms. At least one month later, participants in each group were further randomised to undergo blinded exposure to hypercapnia or normocapnia, allocated at 3:1. The primary outcome was the proportion of participants who self-initiated bailout prior to reaching $P_{ET}CO_2$ 8.5 kPa. Continuous cardiorespiratory data ($P_{ET}CO_2$ and $P_{ET}O_2$, tidal volume, respiratory rate, minute ventilation, heart rate, and blood pressure) were also recorded. Subjective symptoms associated with hypercapnia were assessed with a visual analogue scale.

Results: Thirteen of 15 participants (87%) who received the unblinded hypercapnia-experience self-initiated bailout compared to 10/15 information leaflet participants (67%) (P = 0.149). There was no difference in cardiorespiratory physiology parameters at bailout between the groups. Shortness of breath, light-headedness, and disorientation were the most intensely reported symptoms. Approximately half (47%) of participants who received a hypercapnia training experience had a correlated symptom response during their subsequent hypercapnia testing session.

Conclusions: Although no significant training benefit was shown, becoming familiar with the sensations associated with hypercapnia under appropriate supervision could be useful to rebreather divers both recreationally and within occupational settings.

Introduction

Rebreather diving, where gas is recirculated with carbon dioxide (CO_2) removed and oxygen (O_2) added to maintain a constant partial pressure of O_2 'setpoint', is common in recreational and occupational settings (e.g., military divers). Advantages include reduced gas usage, especially at greater depths, prolonged dive time, and staying undetected.[1] However, rebreather faults or user error can result in CO_2 rebreathing and hypercapnia.[2] Hypercapnia may also be provoked in diving by perturbation of respiratory control (reduced ventilatory responsiveness to rising CO_2 levels) caused by a combination of increased work of breathing and exercise.[3] Moreover, there is considerable inter-individual variability in this tendency, with some individuals more prone to allowing CO_2 levels to rise without increasing ventilation; often deemed 'CO_2 retainers'.[4]

Symptoms of hypercapnia include impaired cognitive function[5] and breathlessness.[6] Moreover, an elevated partial pressure of arterial CO_2 ($PaCO_2$) can also increase the risk of oxygen toxicity via cerebral vasodilation and consequent

enhanced oxygen delivery to the brain,[7] potentially causing seizures and death.[8] The narcotic effects of CO_2 can occur independently of nitrogen narcosis or in an additive or synergistic manner.[9] It can be difficult to differentiate the symptoms of hypercapnia from normal dive phenomena, e.g., increased breathing resistance or breathlessness on exertion.[10,11] Hence, symptoms alone are typically not reliable for detecting hypercapnia.[12]

Despite the well-established dangers of a hypercapnic episode at depth, there continues to be a technical difficulty in developing a reliable method of detecting causative hazards or hypercapnia itself while diving.[13] So-called temperature sticks in rebreather scrubbers only detect a potential failure of the scrubber.[14] Similarly, inhaled CO_2 monitors can only detect the presence of CO_2 in the inspired gas.[15] Neither technology can detect hypercapnia arising from dysregulation of respiratory control. Underwater capnography has not yet been developed,[13] and is challenged by confounding of near-infrared CO_2 sensors in the 100% humidity environment of a rebreather loop.[16]

The aim of this study was to investigate whether an open-label (unblinded) experience of hypercapnia symptoms would improve divers' recognition of hypercapnia and ability to perform a self-rescue in a subsequent blinded hypercapnia exposure.

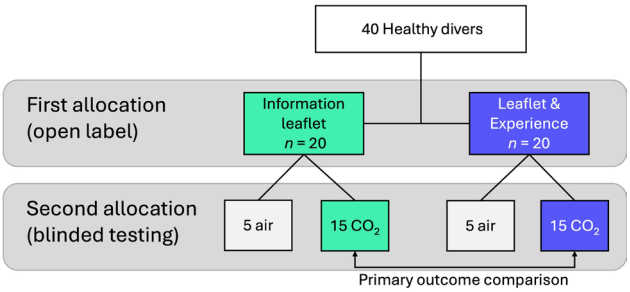
Methods

The study protocol was approved by the Health and Disability Ethics Committee, New Zealand (21/NTB/102) and was registered with the Australian New Zealand Clinical Trials Registry (U1111-1266-1320, http://www.anzctr.org.au/, RRID:SCR_002967). All participant data, informed consent, randomisation, and questionnaires were managed in a local secure database in RedCap.[17]

STUDY DESIGN

This single-blind randomised study was performed at the Exercise Physiology Laboratory at the University of Auckland, from April to August 2024. All participants received an information leaflet detailing the basic physiology of hypercapnia and frequent hypercapnic symptoms (*Appendix 1). In addition, they were randomly allocated into two groups: either receiving an unblinded hypercapnia experience (hypercapnia experience group) or not (information leaflet group). At least one month later, participants attended a blinded exposure where they underwent either a hypercapnic rebreathing protocol or breathed room air, allocated 3:1 (thus 15 from each group were exposed to hypercapnia, while five breathed room air) (Figure 1).

Figure 1
Study design; all participants were blinded to the intervention (CO_2 / hypercapnia or air exposure) during blinded testing visits



PARTICIPANTS

Forty healthy divers aged 18–55 years were recruited. Participants were deemed eligible following screening with the Diver Medical Screening Committee Diver Medical Participant Questionnaire.[18] All participants provided written informed consent. Study participants were excluded if they: were currently engaging in recreational or psychoactive drug use, had a history of mental illness, consumed greater than 21 alcoholic drinks per week, consumed more than five cups of coffee per day or equivalent caffeine consumption, were currently smoking or vaping, had previously participated in a hypercapnia study, or had significant freediving experience.

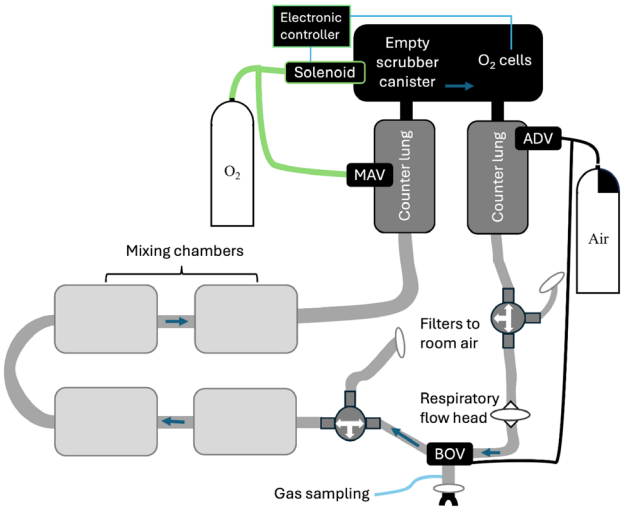
EQUIPMENT CONFIGURATION

A closed-loop breathing circuit was built from an O_2ptima closed-circuit rebreather (DiveRite, Lake City, USA), a Sentinel rebreather bailout valve (VR Technology, Poole, UK), and ADInstruments parts (ADInstruments, Dunedin, New Zealand) (Figure 2). The CO_2 scrubber cartridge was removed from the scrubber canister. A Petrel 2 rebreather controller (Shearwater Research, Richmond, Canada) continuously measured PO_2 within the circuit to maintain an inspired PO_2 of 30 kPa, thereby preventing a hypoxic inspired gas. Participants breathed through a mouthpiece connected to a bacterial filter (Elliptical filter, Alerkan Healthcare, Ankara, Turkey). The automatic diluent valve and bailout valve were connected to an air cylinder. Respiratory tubing (MLA1011A, ADInstruments) connected the rebreather parts through SP0143 3-way stopcocks (ADInstruments), which allowed for switching between room air and the rebreathing circuit. Respiratory filters increased the breathing resistance in room air mode to match the resistance in rebreather mode. During the blinded hypercapnia exposure, a series of four MLA246 4.7 L mixing chambers (ADInstruments) was added, so that rebreathed levels of CO_2 would not increase too precipitously.

A sampling line, positioned just distal to the mouthpiece filter, continuously sampled inspired and expired gas,

***Footnote:** Appendix 1 is available online on our website https://www.dhmjournal.com/index.php/journals?id=416

Figure 2
Experimental rebreathing circuit set-up; the three-headed arrows represent the three-way manual stopcocks that allowed the switch between rebreathing (depicted) and breathing room air (turn counterclockwise). Mixing chambers in series were added for the blinded hypercapnia exposures only. ADV – automatic diluent addition valve; BOV – bailout valve; MAV – manual addition valve



via a desiccant cartridge (MLA604), Nafion drying tube (MLA0343), and in-line filter (MLA0110), to a ML206 respiratory gas analyser (all ADInstruments) set to 200 ml·min^{-1} for measurement of CO_2 and O_2. The gas analyser was calibrated with room air and a hypoxic/hypercapnic gas mixture prior to each measurement. Ventilation was measured with an inline MLT1000L respiratory flow head and FE141 spirometer (both ADInstruments). The flow head was zeroed and calibrated with a MLA5530 3L syringe (ADInstruments) filled with room air prior to each measurement. Participants were instrumented for continuous measurement of heart rate (electrocardiogram, lead II ECG, BioAmp, FE321, ADInstruments), ear clip pulse oximetry (MLT320/E, ADInstruments), and non-invasive blood pressure (the latter in the unblinded experience only) with finger photoplethysmography (Finapres Nova, Finapres Medical Systems, Enschede, Netherlands), all sampled continuously at 1 kHz using Powerlab 16/35 and acquired via LabChart Pro 8.1.24 (ADInstruments).

EXPERIMENTAL PROCEDURE

All participants wore a nose clip to ensure closed-loop integrity. The closed-circuit rebreather was initially filled with an enriched oxygen mixture with PO_2 around 30 kPa. Each exposure began with room air breathing via the circuit, after which the stop-cocks were switched to the closed-circuit rebreather without the participants' knowledge. At the end of each exposure, the stopcocks were returned to open-circuit, while participants recovered for at least two minutes or until heart rate and the end-tidal pressure of carbon dioxide ($P_{ET}CO_2$) returned to baseline.

Unblinded hypercapnia experience

Twenty participants performed a visual attention task[19] while seated, breathing from the experimental setup. A baseline two minutes of room air breathing was observed followed by rebreathing in rebreather mode. Rebreathing was performed until either $P_{ET}CO_2$ reached 8.5 kPa, or the participant's symptoms became intolerable and they requested to cease the experience.

Blinded hypercapnia or air exposure

Cycle ergometry (Velotron Dynafit Pro, Seattle, WA, USA) was included to simulate the mild respiratory effort that is associated with normal underwater finning. The cycle ergometer was set to a workload estimated to elicit 30% of each participant's expected maximal exercise capacity, with the capacity calculated based on height, weight, age, and sex.

Participants were seated on the cycle ergometer and fitted with a virtual reality (VR) headset (HTC Vive Pro Eye, Taoyuan, Taiwan). This was programmed with a virtual reality dive to provide a level of task focus/distraction with some relevance to diving. The task was to count orcas swimming past using a manual click-counter.

After one minute stationary and two minutes of cycling while breathing room air and without the subjects' knowledge, the circuit was either switched to the closed-circuit rebreather mode to induce hypercapnia or remained on room air. Participants were instructed to 'bailout' by operating the bailout valve if they perceived symptoms of hypercapnia or if the heads-up display (HUD) turned from green to red in the VR environment. This was activated if a hypercapnic participant reached a $P_{ET}CO_2$ of 8.5 kPa without bailing out, or if breathing room air, after six minutes.

OUTCOME MEASURES

The primary outcome measure was the proportion of self-initiated versus HUD-prompted bailout among hypercapnic participants from the information leaflet and hypercapnia experience groups. Secondary outcomes included $P_{ET}CO_2$, inspired O_2 pressure, tidal volume, respiratory rate, minute ventilation (all breath-by-breath in 10-second averages), heart rate, blood pressure (unblinded experience only) (all beat-to-beat in 10-second averages), and time from the switch to breathing on the closed loop to rescue (during the unblinded training experience) or bail-out (during the blinded exposure). Self-reported hypercapnia symptoms experienced on a 0–100 visual analogue scale (VAS), as well as the first recognised symptom, were recorded within five minutes after each exposure.

STATISTICAL ANALYSIS

Descriptive statistics were reported as mean and standard deviation (SD) or median (range) where appropriate.

Normality of data was evaluated with the Shapiro-Wilk test. The difference in the proportion of participants in the information leaflet versus the hypercapnia experience group who performed a self-initiated bailout was analysed with a Chi-square test, with Cohen's w to calculate an effect size. Differences in cardiorespiratory outcome measures between the information leaflet and unblinded hypercapnia experience groups were analysed with independent *t*-tests and reported as mean difference with 95% confidence intervals (95% CI). These parameters were compared for a 10-second interval at the end of both the baseline and the hypercapnia periods. For the 15 participants who completed both the unblinded hypercapnia experience and the hypercapnia exposure, symptom consistency between both exposures was assessed with Pearson correlation. All data were analysed with SPSS Statistics version 27.0 (IBM, Armonk, NY, USA), with α set at 5%.

Results

Forty participants completed the study. Table 1 describes participant characteristics. The information leaflet group appeared to be more experienced (greater number of dives, years of diving experience, and more rebreather divers), but this was considered unlikely to be a confounding influence following analysis of the ventilatory response to hypercapnia stratified by diving experience. The mean time interval between the unblinded hypercapnia experience and the blinded hypercapnia exposure was 46 days (range 28–76).

None of the participants from either the information leaflet or unblinded hypercapnia experience groups who were subsequently randomised to receive room air in the blinded exposure performed a self-initiated bailout; thus, there were no false positives.

In the assessment of the primary outcome, 13/15 (87%) participants in the unblinded hypercapnia experience group performed a self-initiated bailout compared to 10/15 (67%) participants in the information leaflet group ($P = 0.149$, effect size = 0.264, Figure 3). Two of 15 (13%) participants in the unblinded hypercapnia experience group and 5/15 (33%) participants in the information leaflet group required a HUD prompt to bailout when the $P_{ET}CO_2$ reached 8.5 kPa. All participants bailed out appropriately in response to this prompt.

Cardiorespiratory responses during the unblinded hypercapnia experience and the blinded hypercapnia exposure are shown in Table 2. One participant was excluded

Table 1
Characteristics of the study participants; SD – standard deviation

Parameter	Experience group *n* = 20	Information leaflet group *n* = 20	Total *n* = 40
Age (mean years, range)	32 (18–53)	36 (23–54)	34 (18–54)
Female *n* (%)	9 (45)	6 (30)	15 (38)
Body Mass Index, kg.m^{-2} (mean, SD)	25.8 (4.1)	28.1 (5.7)	27.0 (5.0)
Ethnicity *n* (%)			
NZ European	9 (45)	10 (50)	19 (48)
Māori	1 (5)	1 (5)	2 (5)
Chinese	2 (10)	1 (5)	3 (8)
Other	8 (40)	8 (40)	16 (40)
Highest level of education *n* (%)			
Secondary school	5 (25)	4 (20)	9 (23)
Bachelors	9 (45)	5 (25)	14 (35)
Masters	5 (25)	6 (30)	11 (28)
PhD or other doctorate	1 (5)	5 (25)	6 (15)
Diving history			
Number of dives (median, range)	98 (3–600)	150 (5–2,000)	102 (3–2,000)
Years of diving experience (median, range)	7 (< 1–15)	12 (1–34)	8.5 (< 1–34)
Diving certification *n* (%)			
Open-circuit recreational	17 (85)	16 (80)	33 (83)
Open-circuit technical	2 (10)	1 (5)	3 (8)
Closed-circuit rebreather	1 (5)	3 (15)	4 (10)

Figure 3

Bailout outcome based on prior hypercapnia experience ('Training') or information leaflet allocation; the 10 participants who received room air rather than hypercapnia are shown in the figure to demonstrate that no false positives (i.e., a diver performing a bailout when they were not hypercapnic) occurred; HUD – head-up display

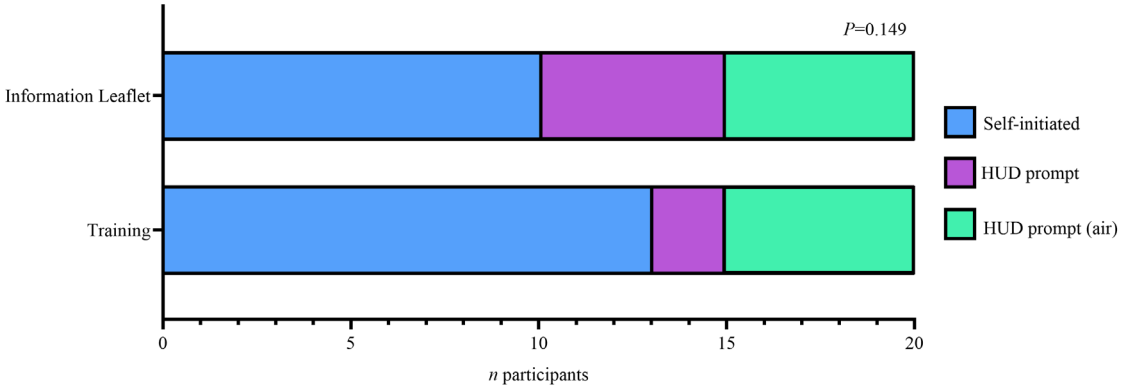


Table 2

Cardiorespiratory responses to hypercapnia during baseline and final 10 s of the unblinded hypercapnia experience (n = 19) and the blinded hypercapnia exposure for the hypercapnia experience group (n = 13) and the information leaflet (n = 15) group; room-air breathing controls and data from two subjects were excluded – see text. Data are presented as mean (standard deviation) and mean differences with 95% confidence intervals (CI). Mean difference is between groups during the final 10 s of the blinded hypercapnia exposure. $P_{ET}CO_2$ – end-tidal partial pressure of carbon dioxide; P_IO_2 – inspired partial pressure of oxygen

Group / parameter	Unblinded experience		Blinded hypercapnia exposure			
	Baseline n = 19	Hypercapnia n = 19	Baseline stationary n = 20	Baseline cycling n = 20	Hypercapnia n = 13	Mean difference (95% CI)
$P_{ET}CO_2$ (kPa)						
Experience group	5.1 (1.0)	8.2 (0.5)	5.2 (0.6)	5.8 (0.7)	8.0 (0.5)	0.1 (-0.3 to 0.5)
Information leaflet group			5.3 (0.6)	5.8 (0.5)	8.1 (0.6)	
P_IO_2 (kPa)						
Experience group	21.0 (0.2)	31.1 (1.1)	20.8 (0.3)	20.8 (0.3)	29.5 (0.7)	0.5 (-0.1 to 1.0)
Information leaflet group			21.1 (0.2)	21.3 (0.7)	29.9 (0.6)	
Tidal volume (L)						
Experience group	0.9 (0.4)	2.2 (0.5)	0.9 (0.2)	1.6 (0.4)	2.2 (0.6)	0.2 (-0.3 to 0.6)
Information leaflet group			1.1 (0.3)	1.7 (0.3)	2.3 (0.6)	
Respiratory rate (breaths.min^{-1})						
Experience group	12 (6)	19 (7)	13 (3)	14 (3)	19 (6)	3 (-8 to 1)
Information leaflet group			13 (4)	13 (4)	15 (6)	
Minute ventilation (L.min^{-1})						
Experience group	11.2 (8.9)	41.3 (17.6)	12.4 (4.1)	20.7 (5.7)	37.4 (7.3)	1.7 (-12.6 to 9.1)
Information leaflet group			13.9 (2.7)	22.2 (8.7)	35.7 (17.8)	
Heart rate (beats.min^{-1})						
Experience group	77 (13)	91 (20)	74 (26)	100 (33)	111 (38)	2 (-27 to 31)
Information leaflet group			85 (17)	107 (17)	113 (36)	
Blood pressure (mmHg)						
Systolic blood pressure	120 (35)	163 (18)				
Diastolic blood pressure	68 (22)	93 (12)				
Mean arterial pressure	86 (26)	117 (13)				

Figure 4
Matchstick figure showing individual ventilation (V_E) responses from stationary baseline to cycling baseline, and final 10 s of hypercapnia in the 15 participants from each initial group allocated to hypercapnia in the blinded exposures; the topmost line (a hypercapnia experience group participant) was classified as an outlier due to an extreme psychological response

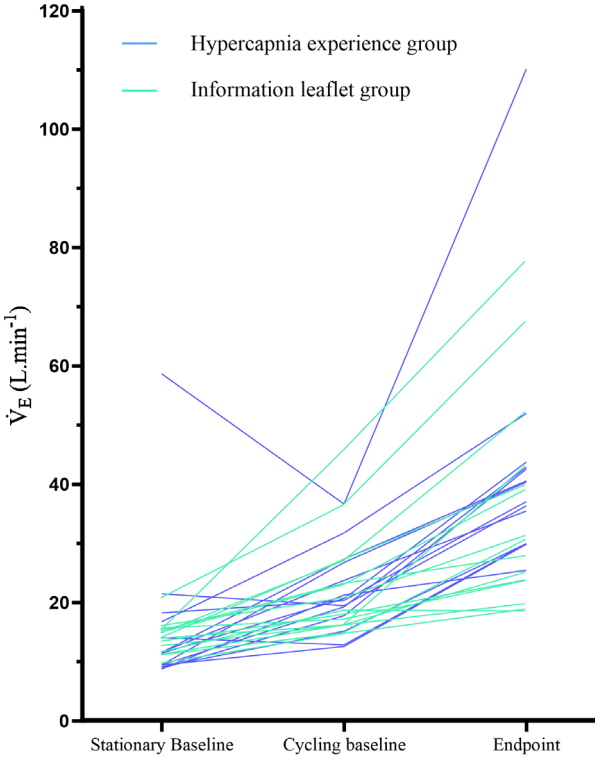
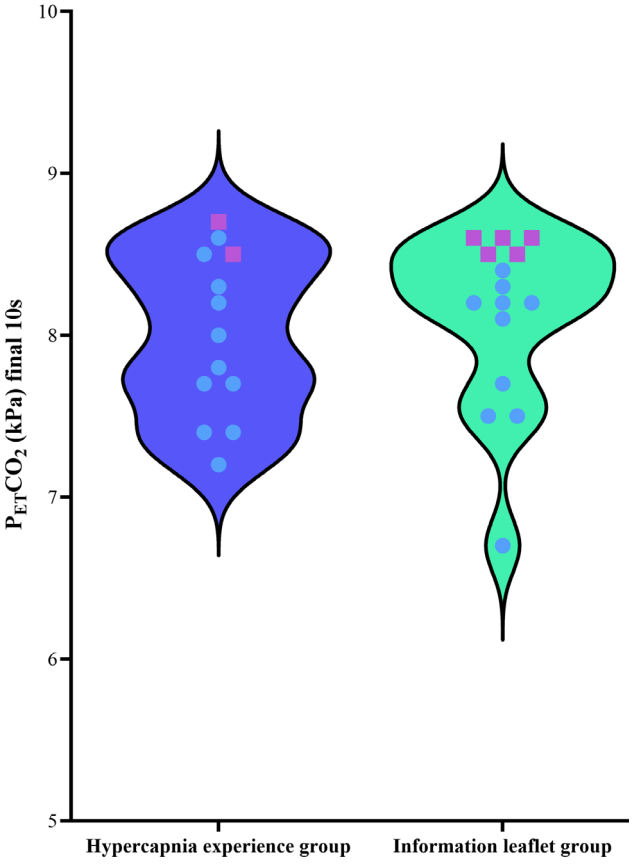


Figure 5
Violin plot of the end tidal CO_2 ($P_{ET}CO_2$) during the final 10 s of the blinded hypercapnia exposure, for the hypercapnia experience and information leaflet groups; blue circles represent self-initiated bailouts, while purple squares indicate those that required a head-up display prompt



from the physiological data (for both exposures) due to a disproportionate psychological response at baseline and during hypercapnia (top blue line in Figure 4). Hypercapnia was associated with an increase in all parameters. Divers in the hypercapnia experience and information leaflet groups had similar cardiorespiratory responses at bailout (Figure 5, Table 2).

During the unblinded hypercapnia experience, one experiment was terminated after breathing from the circuit for 10 minutes due to technical difficulties (a crack in the tube, causing a plateau in the accumulation of CO_2), while four participants requested to stop the unblinded hypercapnia experience early due to symptom intolerance.

In the blinded hypercapnia exposure, those who performed a self-initiated bail-out had a lower $P_{ET}CO_2$ than those needing a HUD-prompt (mean difference 0.7 kPa, 95%CI: -1.1 to -0.2, Table 3). Participants perceived hypercapnia and bailed out at $P_{ET}CO_2$ as low as 6.7 kPa, with a mean of 7.9 ± 0.5 kPa for those who self-initiated the bailout (Table 3). However, there was no difference between the groups.

Shortness of breath was most frequently reported as the first and most intense symptom across all hypercapnia exposures (unblinded and blinded), regardless of initial group allocation (columns 1–3 Figure 6). The first symptom reported during the blinded exposure across both groups was shortness of breath (15/30, 50%), followed by a feeling of warmth (4/30, 15%), light-headedness (4/30, 15%), visual disturbance (1/30, 3%), and nausea (1/30, 3%). Of those who received hypercapnia in both the unblinded experience and the blinded exposure, 7/15 (47%) experienced similar symptoms (and intensity of symptoms) between both sessions ($P < 0.05$), while 8/15 (53%) did not.

Discussion

This study did not show a significant improvement in recognition of hypercapnia and self-initiated bailout after the unblinded hypercapnia experience compared to receiving the information leaflet only. There was a small difference between groups in the proportions of participants who self-initiated bailout during the blinded hypercapnia exposure (87% in the hypercapnia experience group versus 67% in

Table 3

Cardiorespiratory responses during the final 10 s of the blinded hypercapnic exposure (room-air breathing controls and data from two subjects were excluded – see text) comparing the hypercapnia experience group (HE) and the information leaflet group (IL), divided between those performing a self-initiated bailout and those needing a head-up display (HUD) prompt. Data are presented as mean (SD) and mean differences with 95% confidence intervals (CI). BPM – breaths per minute (respiratory rate) or beats per minute (heart rate); $P_{ET}CO_2$ – end-tidal partial pressure of carbon dioxide; P_IO_2 – inspired partial pressure of oxygen

Group	**Self-initiated** HE (n = 12) IL (n = 10)	**Mean difference (95% CI)**	**HUD** HE (n = 1) IL (n = 5)	**Mean difference (95% CI)**
$P_{ET}CO_2$ (kPa)				
Experience training	7.9 (0.5)	0.1 (0.5 to -0.4)	8.5	-0.1 (0.1 to -0.3)
Information leaflet	7.9 (0.5)		8.6 (0.1)	
P_IO_2 (kPa)				
Experience training	29.4 (0.8)	-0.4 (0.3 to -1.0)	29.9	-0.3 (1.4 to -1.9)
Information leaflet	29.8 (0.7)		30.2 (0.6)	
Tidal volume (L)				
Experience training	2.2 (0.6)	-0.2 (0.3 to -0.8)	2.2	0.0 (1.5 to -1.4)
Information leaflet	2.4 (0.6)		2.2 (0.5)	
Respiratory rate (BPM)				
Experience training	19.0 (6.6)	3.3 (8.9 to -2.4)	13.6	-0.5 (15.2 to -16.1)
Information leaflet	15.8 (6.0)		14.1 (5.1)	
Minute ventilation ($L.min^{-1}$)				
Experience training	38.1 (7.3)	-0.8 (12.3 to -13.9)	29.9	0.5 (31.0 to -30.1)
Information leaflet	38.8 (20.4)		29.4 (10.1)	
Heart rate (BPM)				
Experience training	109.6 (39.9)	-2.0 (35.2 to -39.2)	125.6	9.9 (54.7 to -34.9)
Information leaflet	111.6 (43.7)		115.7 (14.7)	
Time to 'rescue'/bail-out (min)				
Experience training	2.74 (0.67)	0.2 (0.8 to -0.3)	3.8	0.5 (3.4 to -2.4)
Information leaflet	2.51 (0.58)		3.4 (1.3)	

the information leaflet group), which might indicate that, for some divers, hypercapnia exposure training is useful. Nevertheless, the lack of a substantial training effect imparted by undertaking the unblinded experience may reflect the inherently unpleasant nature of hypercapnia, meaning that most participants will recognise it even if not previously exposed. This is also supported by the finding of no difference in the cardiorespiratory response between participants who self-initiated their bail-out. This contrasts with our recent finding in a similar study of a significant training effect for prior exposure to hypoxia, whose symptoms are more subtle and perhaps less appreciable on the basis of written material alone.[20]

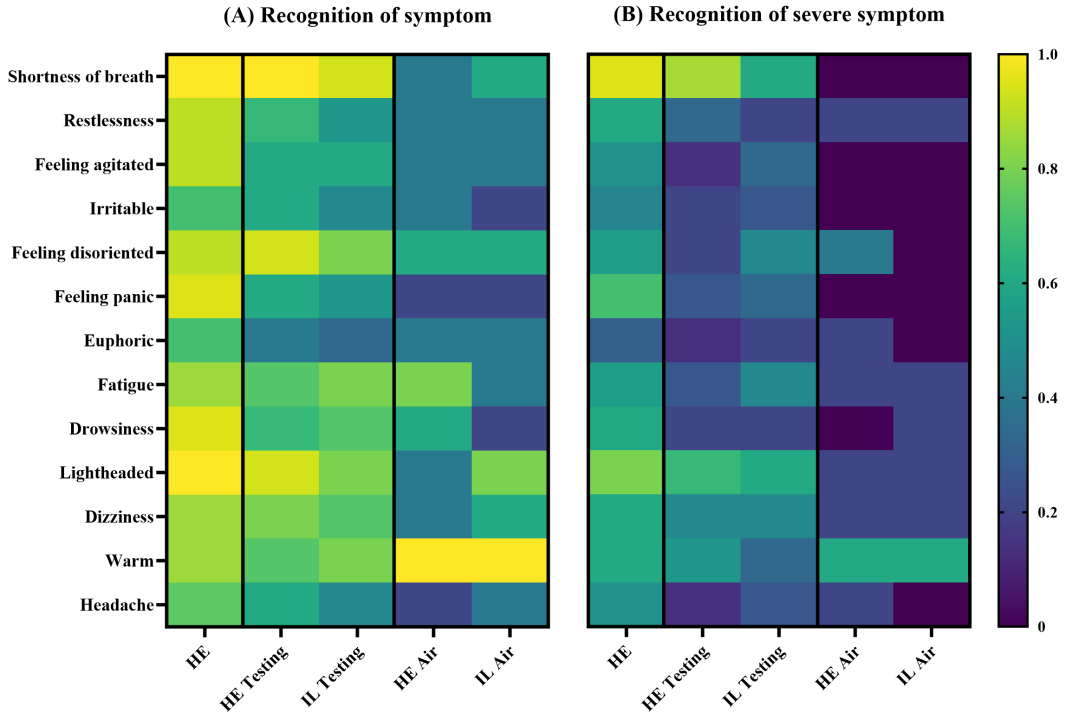
These findings align with previous work from our lab showing that during a five-minute 'pre-breathe', 15/20 divers were able to detect a fully absent scrubber in the rebreather apparatus.[11] However, 18/20 (90%) of divers were unable to detect a partially-failed scrubber in the rebreather set-up, as the inspired carbon dioxide levels were lower (but still elevated).

A previous study of hypercapnia exposure as a training strategy found that only 34% of 213 non-trained divers could detect a P_ICO_2 of < 3.6 kPa, while 90% of 186 trained divers could.[21] Moreover, hypercapnia-trained divers were able to detect CO_2 at lower levels ($P_{ET}CO_2$ 6.3 [SD 0.7] kPa) compared to non-trained divers (7.2 [SD 1.2] kPa). Those positive results could be due to the large sample size compared to our much smaller population, but more importantly, their unblinded hypercapnia exposure session took place immediately before the testing session.

Shortness of breath, light-headedness, and disorientation were the most intensely reported symptoms based on visual analogue scale results. Symptoms were more severe

Figure 6

Self-reported symptom heatmap; (A) shows the reporting of a symptom (visual analogue scale score ≥ 5/100), and (B) shows the reporting of severe symptoms (visual analogue scale score ≥ 50/100). Yellow indicates all participants recognised this symptom (A) or recognised it as severe (B), while dark blue indicates no participant recognised this symptom (A) or as severe (B). HE – unblinded hypercapnia experience; HE Testing – unblinded hypercapnia experience group exposed to hypercapnia during the blinded testing session; IL Testing – information leaflet group exposed to hypercapnia during the blinded testing session; HE or IL Air – participants from the hypercapnia experience or information leaflet groups breathing room air during the blinded testing session



in the unblinded experience session, potentially due to the masking by cycling in the blinded exposure. As expected, the symptom experience was more severe and specific to hypercapnia in the hypercapnia group versus those who were breathing room air, indicating that our blinding was effective. During the blinded exposure, the prior hypercapnia experience group more often recognised and experienced more severe symptoms compared to the information leaflet group, indicating a potential training effect, as the $P_{ET}CO_2$ range was similar in both groups.

The present study has several limitations. Most importantly, it may have been underpowered to demonstrate significance for a real but small-to-medium training effect. In addition, the study environment contrasts with diving, where submergence, higher pressures, and dense gas affect breathing in ways that could reduce or exaggerate any benefit of prior hypercapnia training.[22] For example, hyperoxia (via a suppression of the carotid body's response to hypercapnia) or nitrogen narcosis might reduce symptom experience,[3,4] whereas immersion augments chemosensitivity,[23] and cold water can intensify symptom experience.[24]

In contrast, the study is strengthened by the inclusion of a control group, who did not perform a training exposure (but received related educational material), and the further inclusion of five participants from each group who continued to breathe room air during their blinded ('testing') session. There were no false positives in any of these ten exposures, suggesting that despite understanding the purpose of the study, the weight of expectation was appropriately calibrated among our participants. Moreover, rather than performing a step change in inspired CO_2, a progressive increase in CO_2 was imposed. This is a more ecologically valid model of elevation of inspired CO_2 when diving a rebreather. Our study had a one-month period between both sessions, allowing for some settlement of the experience before the blinded exposure. This contrasts with previous research that had both sessions on the same day.[21,25] Lastly, we mitigated some confounding factors that may influence the potential for premature detection of a hypercapnic stimulus outside of normal dive conditions by having the participants exercise on a cycle ergometer at an intensity similar to casual finning. This exercise was designed to elicit an increase in ventilation and a feeling of warmth that would mask these hallmark hypercapnia symptoms, as would be the case underwater.

Although the results from this study showed a minimal effect of prior hypercapnia exposure in improving recognition of a subsequent hypercapnic event, we remain open-minded about the concept of hypercapnia exposure as an educational strategy for rebreather divers. It remains possible that prior

experience of hypercapnia symptoms may help in symptom recognition during diving. In addition, experience of the unpleasant 'breathless' nature of hypercapnia would at least engender a respect for the problem, demonstrate the elevated gas consumption likely to accompany hypercapnic events after bailout onto open circuit scuba, and motivate attention to avoidance strategies such as proper replacement of CO_2 absorbents and avoiding hard work and dense gas at extreme depths. Importantly, unlike hypoxia which could result in loss of consciousness, controlled hypercapnia exposures in a dry environment are unlikely to be harmful in healthy individuals, especially if simple precautions are taken. If done, we would advise that hypercapnia exposures be performed under experienced supervision, for instance, as part of a rebreather diver training course. A small $P_{ET}CO_2$ sensor,[26] should also be included at the mouthpiece to reassure that the $P_{ET}CO_2$ remains within clearly safe limits (for example, < 8.5 kPa).

Currently, there is no reliable method of detecting rising arterial CO_2 during a dive.[27] The present research indicates that, if warned, divers are still capable of performing a self-initiated bail-out at $P_{ET}CO_2$ of 8.5 kPa (albeit in a dry lab environment), while Warkander et al. showed some divers could become incapacitated at levels marginally higher than this.[4] Therefore, if underwater capnography were developed, bailout warnings should be given well in advance of 8.5 kPa. Our results provide some reassurance that with warnings prior to this level, divers can successfully perform a self-rescue bailout procedure adequately. However, we should note that previous research has already shown an increase in reaction time at a $P_{ET}CO_2$ of 7.3 kPa.[5]

Conclusions

We report that divers who underwent an unblinded hypercapnia experience were not better at recognising hypercapnia and bailing out from a rebreather during a subsequent simulated dive compared to those who read about hypercapnia symptoms from an information leaflet. However, our study may have been underpowered to prove a small to medium training effect based on the predefined primary outcome. There was also no difference in the physiological response between the two groups at bail-out. We remain sympathetic to the idea that becoming familiar with the symptoms associated with hypercapnia, particularly the feeling of shortness of breath, through undergoing a hypercapnia experience with knowledgeable supervision in a dry, safe environment may be valuable in rebreather diver training.

References

1 Mitchell SJ, Doolette DJ. Recreational technical diving part 1: an introduction to technical diving methods and activities. Diving Hyperb Med. 2013;43:86–93. PMID: 23813462. [cited 2025 Jun 27]. Available from: https://dhmjournal.com/images/IndividArticles/43June/Mitchell_dhm.43.3.86-93.pdf.

2 Fock AW. Analysis of recreational closed-circuit rebreather deaths 1998–2010. Diving Hyperb Med. 2013;43:78–85. PMID: 23813461. [cited 2025 Jun 27]. Available from: https://www.dhmjournal.com/images/IndividArticles/43June/Fock_dhm.43.2.78-85.pdf.

3 Doolette DJ, Mitchell SJ. Hyperbaric Conditions. Compr Physiol. 2011;1:163–201. doi: 10.1002/cphy.c091004. PMID: 23737169.

4 Warkander DE, Norfleet WT, Nagasawa GK, Lundgren CEG. CO_2 retention with minimal symptoms but severe dysfunction during wet simulated dives to 6.8 atm abs. Undersea Biomed Res. 1990;17:515–23. PMID: 2288042.

5 Sayers JA, Smith RE, Holland RL, Keatinge WR. Effects of carbon dioxide on mental performance. J Appl Physiol (1985). 1987;63:25–30. doi: 10.1152/jappl.1987.63.1.25. PMID: 3114218.

6 Dunworth SA, Natoli MJ, Cooter M, Cherry AD, Peacher DF, Potter JF, et al. Hypercapnia in diving: a review of CO_2 retention in submersed exercise at depth. Undersea Hyperb Med. 2017;44:191–209. doi: 10.22462/5.6.2017.1. PMID: 28779577.

7 Dean JB, Mulkey DK, Garcia AJ 3rd, Putnam RW, Henderson RA. Neuronal sensitivity to hyperoxia, hypercapnia, and inert gases at hyperbaric pressures. J Appl Physiol (1985). 2003;95:883–909. doi: 10.1152/japplphysiol.00920.2002. PMID: 12909594.

8 Manning EP. Central nervous system oxygen toxicity and hyperbaric oxygen seizures. Aerosp Med Hum Perform. 2016;87:477–86. doi: 10.3357/AMHP.4463.2016. PMID: 27099087.

9 Hesser CM, Fagraeus L, Adolfson J. Roles of nitrogen, oxygen, and carbon dioxide in compressed-air narcosis. Undersea Biomed Res. 1978;5:391–400. PMID: 734806.

10 Shykoff BE, Warkander DE. Exercise carbon dioxide (CO_2) retention with inhaled CO_2 and breathing resistance. Undersea Hyperb Med. 2012;39:815–28. PMID: 22908838.

11 Deng C, Pollock NW, Gant N, Hannam JA, Dooley A, Mesley P, et al. The five-minute prebreathe in evaluating carbon dioxide absorption in a closed-circuit rebreather: a randomized single-blind study. Diving Hyperb Med. 2015;45:16–24. PMID: 25964034. [cited 2025 Jun 27]. Available from: https://dhmjournal.com/images/IndividArticles/45March/Deng_dhm.45.1.16-24.pdf.

12 Earing CMN, McKeon DJ, Kubis HP. Divers revisited: The ventilatory response to carbon dioxide in experienced scuba divers. Respir Med. 2014;108:758–65. doi: 10.1016/j.rmed.2014.02.010. PMID: 24612621.

13 Mitchell SJ. Developments in carbon dioxide monitoring. In: Pollock NW, ed. Rebreather Forum 4. Proceedings of the April 20-22 workshop. Valletta, Malta; 2024. p. 142–50. [cited 2025 Jun 27]. Available from: https://indepthmag.com/?sdm_process_download=1&download_id=52421.

14 Silvanius M, Mitchell SJ, Pollock NW, Frånberg O, Gennser M, Lindén J, et al. The performance of 'temperature stick' carbon dioxide absorbent monitors in diving rebreathers. Diving Hyperb Med. 2019;49:48–56. doi: 10.28920/dhm49.1.48-56. PMID: 30856667. PMCID: PMC6526050.

15 Ineson A, Henderson K, Teubner D, Mitchell S. Analyser position for end-tidal carbon dioxide monitoring in a rebreather circuit. Diving Hyperb Med. 2010;40:206–9. PMID: 23111936. [cited 2025 Jun 27]. Available from: https://dhmjournal.com/images/IndividArticles/40Dec/Mitchell_dhm.40.4.206-209.pdf.

16 Ranu UB, Rahman MdA, Sriram S, Agarwal PB. Infrared non-invasive exhaled biomarker sensing: a review. Advanced Sensor Research. 2024;3. doi: 10.1002/adsr.202300085.
17 Harris PA, Taylor R, Minor BL, Elliott V, Fernandez M, O'Neal L, et al. The REDCap consortium: Building an international community of software platform partners. J Biomed Inform. 2019;95:103208. doi: 10.1016/j.jbi.2019.103208. PMID: 31078660. PMCID: PMC7254481.
18 Diver Medical Screening Committee. Recreational diving medical screening system. [cited 2025 Nov 19]. Available from: https://www.uhms.org/resources/featured-resources/recreational-diving-medical-screening-system.html.
19 Connell CJW, Thompson B, Kuhn G, Gant N. Exercise-induced fatigue and caffeine supplementation affect psychomotor performance but not covert visuo-spatial attention. PLOS ONE 2016;11(10), e0165318. doi: 10.1371/journal.pone.0165318. PMID: 27768747. PMCID: PMC5074788.
20 Allocco A, van Waart H, Connell CJ, Wong NY, Charukonda A, Gant N, et al. An unblinded training exposure to hypoxia enhances subsequent hypoxia awareness. Diving Hyperb Med. 2025;55:136–44. doi: 10.28920/dhm55.2.136-144. PMID: 40544141. PMCID: PMC12267069.
21 Eynan M, Daskalovic YI, Arieli Y, Arieli R, Shupak A, Eilender E, et al. Training improves divers' ability to detect increased CO_2. Aviat Space Environ Med. 2003;74:537–45. PMID: 12751583.
22 Anthony G, Mitchell SJ. Respiratory physiology of rebreather diving. In: Pollock NW, Sellers SH, Godfrey JM, eds. Rebreathers and scientific diving. Proceedings of NPS/NOAA/DAN/AAUS June 16-19, 2015 Workshop. Wrigley Marine Science Center, Catalina Island, CA; 2016; p. 66–79.
23 Sackett JR, Schlader ZJ, O'Leary MC, Chapman CL, Johnson BD. Central chemosensitivity is augmented during 2 h of thermoneutral head-out water immersion in healthy men and women. Exp Physiol. 2018;103:714–27. doi: 10.1113/EP086870. PMID: 29527752.
24 Fothergill DM, Taylor WF, Hyde DE. Physiologic and perceptual responses to hypercarbia during warm- and cold-water immersion. Undersea Hyperb Med. 1998;25:1–12. PMID: 9566081.
25 Bugelli NC. Can U.S. Navy divers be trained to improve their recognition of carbon dioxide in their breathing mixture while exercising? [cited 2025 Jun 27]. Available from: https://www.proquest.com/docview/2552997168/.
26 Vrijdag XCE, van Waart H, Sames C, Sleigh JW, Mitchell SJ. Comparing the EMMA capnograph with sidestream capnography and arterial carbon dioxide pressure at 284 kPa. Diving Hyperb Med. 2023;53:327–32. doi: 10.28920/dhm53.4.327-332. PMID: 38091592. PMCID: PMC10735710.
27 Mitchell SJ, Pollock NW. Rebreather forum four consensus statements. Diving Hyperb Med. 2023;53:142–6. doi: 10.28920/dhm53.2.142-146. PMID: 37365132. PMCID: PMC10584388.

Acknowledgements

The authors would like to thank the participants who were involved in the study. Additionally, thanks to the rebreather manufacturers (Dive Rite and VR Technology) for the generous donation of parts to construct the breathing circuit. We thank Shearwater Research for the modified firmware for the Petrel2 rebreather controller. Additionally, we would like to thank Axel Busch for creating the VR orca counting environment.

Conflicts of interest and funding

This work has been supported by funding from the Office of Naval Research Global (ONRG), United States Navy (N62909-22-1-2003), and the ANZCA Foundation, Australian and New Zealand College of Anaesthetists (AEG22/002). Professor Mitchell is the editor of *Diving and Hyperbaric Medicine* journal but played no role in the peer review process or decision to publish this manuscript. The manuscript was managed by the deputy editor Dr Lesley Blogg. No other conflicts of interest were declared.

Submitted: 21 September 2025
Accepted after revision: 20 March 2026

Technical diving accidents in France: a 15-year retrospective study reporting a high prevalence of musculoskeletal decompression sickness

Emmanuel Gouin[1,2,3], Anaïs Caillard[2,3], Colin Veses[4], Marc Luciani[5], Charlotte Lecomte[6], Emmanuel Dugrenot[1,2,7,8], François Guerrero[2], Jean-Éric Blatteau[9]

[1] *Divers Alert Network, Durham, NC, United States*
[2] *University Brest, Laboratory ORPHY EA 4324, IBSAM, Brest, France*
[3] *CHRU La Cavale Blanche. Anesthesiology, perioperative and intensive care Units, Brest, France*
[4] *Pasteur University Hospital, Hyperbaric Oxygen Therapy Department, Nice, France*
[5] *Notre-Dame de la Miséricorde Hospital, Department of Hyperbaric Medicine, Ajaccio, France*
[6] *Sainte Marguerite Hospital, APHM, Department of Hyperbaric Medicine, Marseille, France*
[7] *Joint Department of Biomedical Engineering, The University of North Carolina and North Carolina State University, Chapel Hill, NC, USA*
[8] *Subaquatic Operational Research Team (ERRSO), Military Institute of Biomedical Research (IRBA), Toulon, France*
[9] *Military Teaching Hospital Sainte-Anne, Department of Hyperbaric Medicine and Diving Expertise, Toulon, France*

Corresponding author: *Dr Emmanuel Gouin, Laboratoire ORPHY, EA 4324, Université de Bretagne Occidentale, 6 Av. Le Gorgeu, 29200 BREST, France*
ORCiD: *0000-0003-3691-5870*
emmanuel.gouin@univ-brest.fr

Keywords
Diving incidents; Gas-toxicity; Helium; Mixed-gas; Pulmonary oedema; Rebreather; Sport injuries

Abstract
(Gouin E, Caillard A, Veses C, Luciani M, Lecomte C, Dugrenot E, Guerrero F, Blatteau J-É. Technical diving accidents in France: a 15-year retrospective study reporting a high prevalence of musculoskeletal decompression sickness. Diving and Hyperbaric Medicine. 2026 30 June;56(2):125–136. doi: 10.28920/dhm56.2.125-136. PMID: 42290572.)
Introduction: Technical diving, involving rebreathers and/or helium-based gas mixtures for deeper and longer dives, may influence risk and clinical presentation of injuries due to helium's properties, equipment constraints, or exposure conditions. This study aims to describe the specific characteristics of this accidentology.
Methods: A retrospective study was conducted across five French coastline hyperbaric units. Medical records of technical divers presenting with decompression sickness (DCS), immersion pulmonary oedema (IPO), or gas-toxicity between 2010 and 2024 were reviewed.
Results: 127 technical divers were included, three declined participation, leaving 124 cases for analysis. DCS was the most frequent condition (n = 105) followed by IPO (n = 16) and gas toxicity (n = 3). Median age was 45 [IQR 37–53] years, and 113 (91%) were male. Rebreathers were used in 94 (75.8%) cases and helium-based mixtures in 77 (62%). Previous diving-related accidents were reported in 36 (29%) cases. IPO occurred mainly after shallower dives in wetsuits and was frequently associated with procedural errors. Among DCS cases isolated musculoskeletal DCS predominated (n = 36), whereas spinal involvement was less frequent. When indicated, median recompression delay was 238 [IQR 135–555] minutes. Unfavourable outcomes occurred in 26 (25%) DCS cases, primarily with bone or inner-ear involvement.
Conclusions: Technical diving accidents exhibit distinct patterns from recreational diving, notably greater musculoskeletal involvement and a possible increased risk of dysbaric osteonecrosis (DON). Current evidence does not support different management, but the risk of potential initially silent bone lesions should not be overlooked. Further research on helium-related risks and hyperbaric treatment's role in DON prevention is needed.

Introduction

Although scuba diving is generally considered to be a relatively safe activity, it still carries inherent risks. The most frequently reported injuries are barotrauma of the ears and sinuses which predominantly affect entry-level divers. However, diving-related accidentology encompasses a much broader spectrum of injuries, some of which may result long-term complications or even fatalities.[1,2]

Advances in specialised equipment, such as closed-circuit rebreathers (CCRs) and helium-based mixed-gases, have significantly expanded the possibilities for deeper and longer dives, referred to as technical diving.[3,4] Following prolonged time at depth, the reduction in ambient pressure during ascent leads to the elimination of inert gases from saturated tissues, thereby linked with the risk of decompression sickness (DCS). The clinical manifestations of DCS range from mild to life-threatening and may involve multiple organ systems.[5]

Immersion pulmonary oedema (IPO) is another condition requiring particular attention, especially as the breathing resistance imposed by diving equipment is suspected to promote its onset.[6,7] Furthermore, CCRs and gas mixtures increase the likelihood of technical and specific issues, such as gas toxicity.[8]

The physiological effect of different gas mixtures (e.g., helium-based mixtures referred to as 'trimix') and the expanded time and pressure of exposure could modify inherent diving-related accident risk. There are few data regarding specific accidentology in this area, though clinical presentation may differ from recreational air diving.[3,9,10]

Advancing our understanding of diving accidentology is essential for enhancing both medical care and preventive strategies. The aim of this study was to describe the technical diving-related accidents referred to the French hyperbaric units and to investigate the influence of potential risk factors associated with the different pathological entities.

Methods

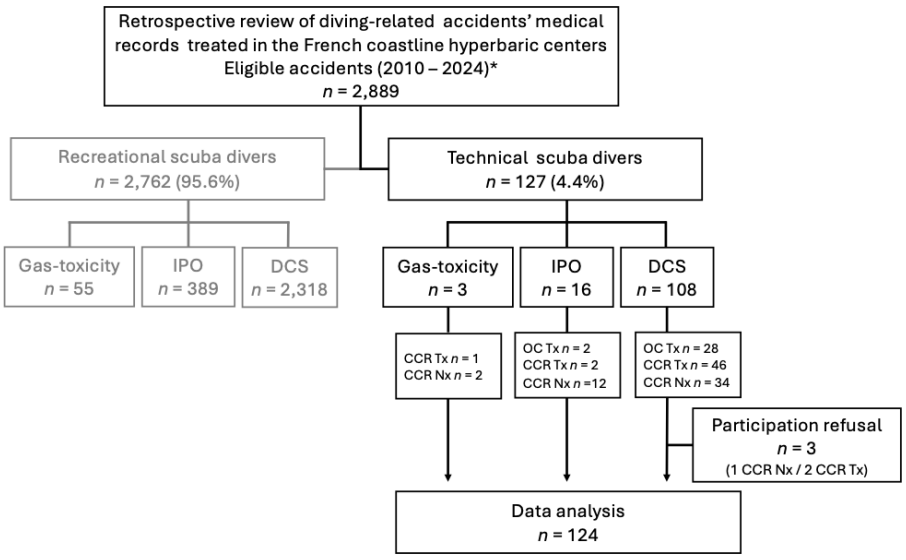
This multicentre, observational retrospective study was approved by the Regional Ethical Committee of Brest (B2024CE.31) and was prospectively registered on ClinicalTrial (NCT06627153). A letter of non-objection to the use of their data for the purposes of this research was sent to each eligible patient.

Technical diving was defined as the use of rebreathers regardless of the breathing gas (i.e., nitrox or trimix) and open-circuit (OC) apparatus with trimix. Diving injuries resulting from military operations were excluded due to the significantly different context of practices. Sixteen hyperbaric chambers in mainland France treat diving-related injuries, though many are inland and remote from the most active diving areas. Given their proximity to dive sites, coastal hospitals are more likely to manage most diving incidents. Therefore, hyperbaric units located along the French mainland coastline were invited to participate in this study. One declined, resulting in the enrolment of five units (with number of contributed cases shown): Ajaccio Hospital (n = 5), Brest University Hospital (n = 7), Nice University Hospital (n = 17), Marseille University Hospital (n = 44), and Toulon Military Teaching Hospital (n = 54). The non-participating hospital reported no diving accidents involving technical divers in its unit over the past 10 years.

All medical records of patients diagnosed with DCS, IPO, or gas-toxicity following a technical dive between 1 January 2010 and 31 December 2024 were reviewed (Figure 1). All collected data were de-identified. Data collection was divided into three sections: 1) Demographics and diving experience, including age, sex, body mass index (BMI), medical history, diving experience and any prior diving-related injuries. 2) Incident-related data including breathing apparatus, gas mixture, dive plan and profile, symptoms, and onset delay. Given the complex profiles of some technical dives and frequent absence of dive computer records, only maximum depth and total dive time were reported. The initial DCS severity was classified as 'mild' or 'severe' presentations, following recent published clinical consensus.[5] When available, the MEDSUBHYP neurological prognostic score

Figure 1

Flowchart of diving-related accidents among technical and recreational divers; recreational diver data (in grey) are shown for contextual comparison but detailed analysis of this group is beyond the scope of the present study. *All data covered the study period, except for data from the Hyperbaric Unit of Marseille (2013–2024), due to the unavailability of earlier medical records. CCR – closed-circuit rebreather; DCS – decompression sickness; IPO – immersion pulmonary oedema; Nx – nitrox; OC – open-circuit scuba; Tx – trimix (helium-based mixed gas)



was recorded.[11] 3) Pre-hospital and in-hospital management, delay for the first recompression treatment after symptom onset (or surfacing in case of underwater onset), total number of follow-up sessions, and the final diagnosis pronounced by physicians. Hospital stay duration and discharge outcomes were recorded. Incomplete recovery at discharge was defined by the presence of residual symptoms or magnetic resonance imaging (MRI) findings suggestive of dysbaric osteonecrosis (DON). In cases of combined diagnoses involving DCS, patients were categorised under DCS, as it primarily guided management.

STATISTICAL ANALYSIS

Statistical analysis was performed with GraphPad Prism v10.4.1 (GraphPad Software Inc., San Diego, CA, USA). Continuous variables are reported as median [interquartile range (IQR), Q1–Q3], and categorical variables as counts and percentages. Normality was assessed using the Shapiro-Wilk test. Group comparisons were conducted using unpaired *t*-test for normally distributed continuous variables and the Mann-Whitney U test otherwise. Categorical variables were compared using the Chi-square test or Fisher's exact test in case of small sample sizes. Logistic regression analyses were performed to identify factors associated with the risk of IPO versus DCS, including all variables with *P*-value < 0.2 in univariate analysis. Given the predominance of musculoskeletal (MSK) involvement, specific risk factors for this subgroup were further evaluated among the overall DCS population. A small proportion of divers suffered recurrent DCS episodes, introducing a partial dependency in the data. In the context of a descriptive study, which is event-based rather than individual-based, each episode was treated as an independent observation. Statistical significance was defined as a *P*-value < 0.05. Due to missing data in medical records, the reported **n* throughout the results reflects the number of cases with available information whenever it differs from the total study population.

Results

DESCRIPTION OF POPULATION

During the study period, 127 technical divers (4.4% of the entire injured-diver population) were admitted to the hyperbaric facilities for diving-related accidents. Three individuals (2.4%) declined participation, resulting in the analysis of 124 cases (Figure 1). The temporal distribution of accidents is shown in Figure 2, illustrating the gradual increase in CCR use relative to OC in technical diving. The median rate of admissions was nine [IQR 6–12] patients per year. Demographic data are summarised in Table 1. During the study period, six (4.8%) divers had two admissions (five for recurrent DCS and one for separate episodes of IPO

Figure 2

Repartition of injured technical divers referred to the French coastline hyperbaric units by primary diagnosis, breathing apparatus, and gas used, reported annually (2010–2024); CCR – closed-circuit rebreather; DCS – decompression sickness; IPO – immersion pulmonary oedema; Nx – nitrox; OC – open-circuit scuba; Tx – trimix (helium-based mixed gas)

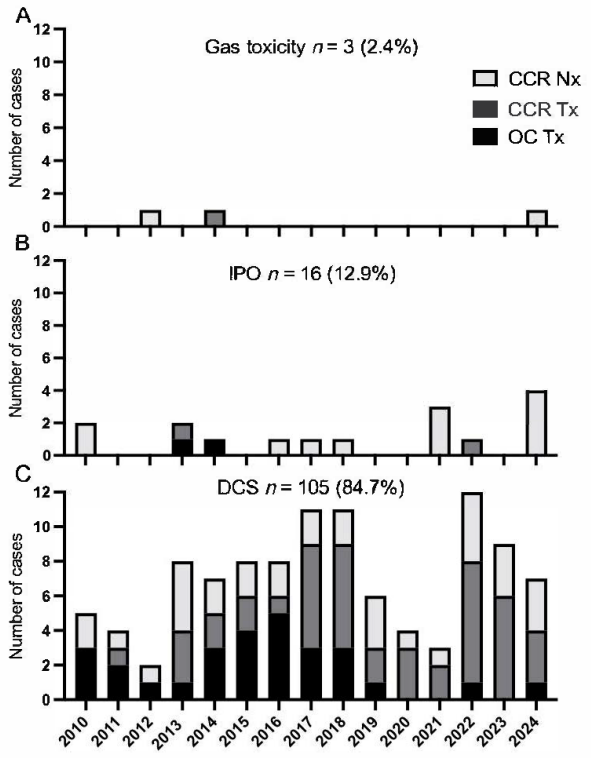


Table 1

Demographic data for injured technical divers; **n* refers to the number of divers for whom data were available; BMI – body mass index; DCS – decompression sickness; IQR – interquartile range; IPO – immersion pulmonary oedema

Parameter	**n*	Overall population *n* (%) or median [IQR]
Male	124	113 (91.1)
Female	124	11 (8.9)
Age (years)	124	45 [37–53]
BMI (kg.m^{-2})	106	25.4 [23.4–27.8]
Obesity (BMI > 30)	108	13 (12.0)
Active smoking	121	11 (9.1)
Hypertension	123	17 (13.8)
Diabetes	123	2 (1.6)
Heart disease	123	2 (1.6)
Medication	123	26 (21.1)
History of IPO	123	7 (5.7)
History of DCS	123	29 (23.6)
Years of diving	67	20 [13–30]
Total reported dive number	81	1,000 [600–3,000]

and DCS). Two divers (1.6%) were diagnosed with DCS associated with suspected IPO (one MSK DCS and one constitutional form).

INCIDENT DIVES

Open-seawater dives were involved in 120/123 (97.6%) events, and three (2.4%) cases occurred in caves. Five/121 (4.1%) dives took place abroad, and 13/121 (10.7%) in a different administrative jurisdiction than the treating hyperbaric unit. Eleven accidents (8.9%) occurred in a professional context; the remainder were recreational technical dives. Dive parameters are detailed in Table 2.

SYMPTOMS, TREATMENT AND DIAGNOSIS

A wide range of symptoms was reported with 87 (70.2%) divers exhibiting multiple complaints with a median of 2 [IQR 2–3] symptoms per individual. Musculoskeletal pain was the dominant symptom, particularly after trimix dives (Figure 3). The median onset time for DCS symptoms was 15 [IQR 1–60] minutes, with 15/102 (14.7%) experiencing symptoms during decompression stops. All IPO and gas-toxicity symptoms occurred either at depth or immediately upon surfacing. Initial DCS symptoms were classified as severe in 48/105 (45.7%) cases. The MEDSUBHYP score ($n = 14$) for spinal injury was 4 [IQR 2–7] among them, ten (71.4%) presented a score < 6.

First-aid oxygenation (FAO_2) was administered in 89/123 (72.4%) cases, and hydration in 51 (41.5%). On-site (para) medical assistance was provided in 78/119 (65.6%) cases, and 27/123 (22%) were evacuated by helicopter.

Delayed consultation (> 24 hours) occurred in 15 (12.1%) cases, all presenting with DCS symptoms. Acetylsalicylic acid was taken by 53/105 (50.5%) divers with DCS, including 32/53 (60.4%) during the prehospital phase. Other analgesics were used by 19/105 (18.1%) divers (paracetamol, $n = 9$; non-steroidal anti-inflammatory drugs (NSAIDs), $n = 9$; and morphine, $n = 1$). Corticosteroids (methylprednisolone) were administered to 61/105 (58.1%) divers, while 18/105 (17.1%) received no adjuvant drug therapy. Among patients with IPO, acetylsalicylic acid was administered on-site in 4/16 (25%) cases, furosemide in 2/16 (12.5%) and methylprednisolone in 3/16 (18.8%).

Recompression with hyperbaric oxygen treatment (HBOT) was provided to 103/105 (98.1%) individuals with DCS. Among patients with IPO, 4/16 (25%) received HBOT despite the absence of an associated DCS diagnosis: two due to missed decompression stops, and one with severe hypoxia secondary to associated drowning. Median time from symptom onset to recompression was 238 [IQR 135–555] minutes, with 68/103 (66%) patients commencing HBOT within six hours. HBOT protocols are summarised in Table 3. Twenty-two/105 (21%) received a 'long' initial treatment ≥ 180 minutes, including 17/22 (77.3%) with inner-ear or neurological DCS. The median number of additional HBOT sessions was 1 [IQR 0–5] and 39/107 (36.5%) underwent more than one additional session. The two suspected DCS cases who did not receive HBOT had an uncertain diagnosis of spinal or cerebral DCS, with consultations occurring on day three and day 10 post-dive, respectively. Final medical diagnoses are presented in Figure 4.

Table 2

Incident-dive parameters and equipment used; in incidents other than those involving closed circuit rebreathers (CCRs), open circuit scuba was used. In incidents other than those involving trimix (helium-based mixed gas), nitrox (i.e., air diluent gas) was used. *n refers to the number of divers for whom data were available; procedural error encompassed predominantly fast ascent, buoyancy issues or missed deco stops; IQR – interquartile range

Parameter	*n	Overall population n (%) or median [IQR]
Dry suit	94	72 (76.6)
CCR	124	94 (75.8)
Trimix mixed gas	124	77 (62.1)
Breathing gas density at max depth ($g.l^{-1}$)	102	6.4 [5.4–7]
Maximum depth (m)	122	53 [40–70]
Total dive time (min)	119	69 [50–90]
Procedural error	123	29 (23.6)
Exertion during dive	123	22 (17.9)
Tiredness before dive	123	89 (72.4)
Repetitive dive < 12 h	114	24 (21.1)
Dive in the previous 48 h	109	75 (68.8)

OUTCOMES

At hospital discharge, complete resolution was observed in 77/103 cases (74.8%) of DCS; outcome information was unavailable for two DCS cases. All cases of IPO recovered without sequelae. Two gas-toxicity cases involved hypoxia-induced loss of consciousness (LOC) at shallower depths; both divers were successfully rescued by their buddies and ascended without lasting complications. The third gas-toxicity case was declared deceased upon admission following hyperoxic seizure at 50 metres of seawater (msw), resulting in drowning and pulmonary barotrauma with massive arterial gas embolism secondary to a rapid ascent.

Among the 26/103 (25.2%) DCS-affected divers whose symptoms had not fully resolved at discharge, eight exhibited bone involvement confirmed by MRI and two reported incomplete pain resolution at discharge, with the underlying cause remaining uncertain. Eleven had persistent inner-ear injuries, eight with vestibular disturbances and three with persistent hearing loss. Only four divers had neurological

Figure 3

Distribution of symptoms among injured technical divers, according to the breathing gas used; multiple symptoms were common occurring in 87/124 (70.2%) of divers (nitrox: 81.8% vs. trimix: 59.7%). *P*-value for trimix vs nitrox – **P* < 0.05, ****P* < 0.001; LOC – loss of consciousness

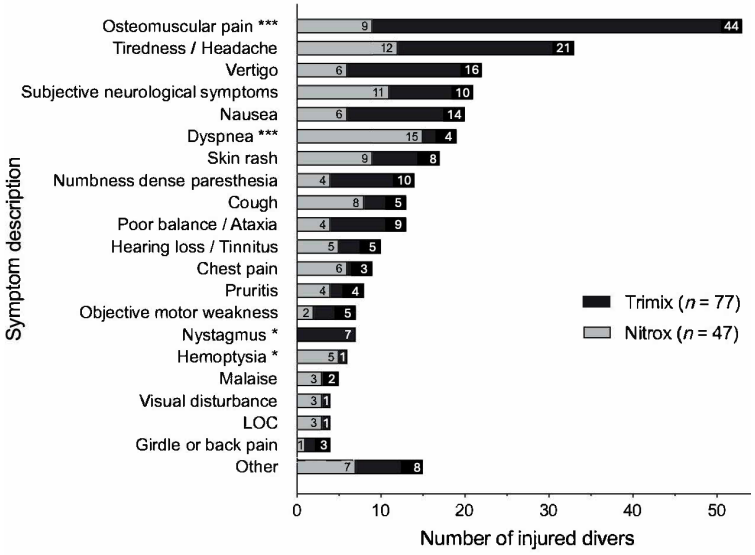


Table 3

Initial hyperbaric treatment tables administered to divers with decompression sickness (*n* = 103) and immersion pulmonary oedema (*n* = 4); O_2 – oxygen; Nx – Nitrox; Hx – Heliox (helium-based mixed gas); atm abs – atmospheres absolute

Treatment table	Pressure (atm abs)	Duration (minutes)	Breathing gas	Divers treated *n* (%)
A15	2.5	90	O_2	12 (11.2)
A18	2.8	90	O_2	4 (3.7)
B18	2.8	150	O_2	55 (51.4)
C18	2.8	300	O_2	6 (5.6)
Comex 12	2.2	130	O_2	3 (2.8)
Comex18c	2.8	174	O_2	8 (7.5)
Comex 30	4	420	Nx/Hx	10 (9.4)
Other	2.5–4	150–360	Nx/Hx	7 (6.5)
Unknown	–	–	–	2 (1.9)

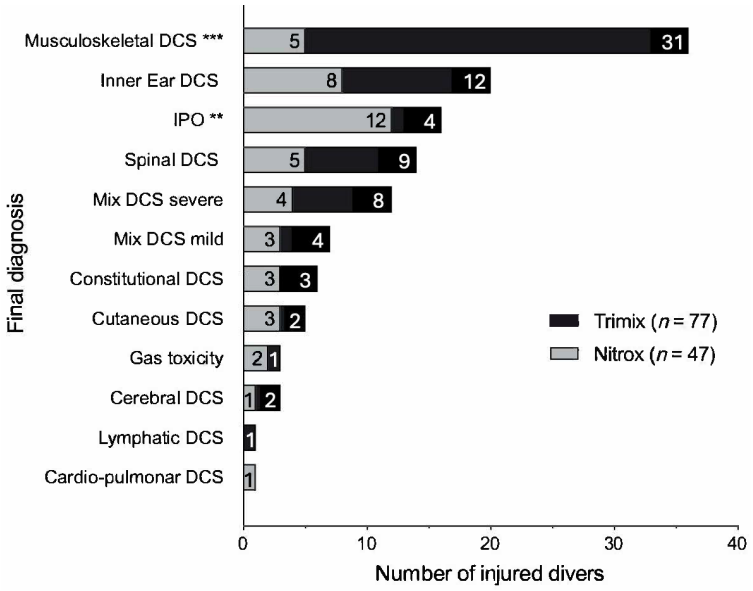
sequelae, three with moderate symptoms (subjective motor weakness or paresthesia), and one with a severe outcome (spastic paraplegia and sphincter dystonia). The remaining patient experienced persistent fatigue and unspecified biological abnormalities. There was no significant outcome difference based on demographics, experience, equipment, diving profile or treatment (first-aid and initial HBOT table choice). Initial symptoms were reported as severe in 32/77 (41.6%) divers with favorable recovery and 15/26 (57.7%) with unfavorable recovery.

SUSPECTED CONTRIBUTING FACTORS TO IPO OR DCS

Considering the minimal occurrence of gas toxicity, only IPO and DCS were compared. A history of a similar injury (IPO or DCS diagnosis regardless of manifestation, respectively) was not significantly associated with recurrence in univariate analysis ($P = 0.7$). However, 5/16 (31.3%) patients were admitted for recurrent IPO, and 28/105 (26.7%) for recurrent DCS. One IPO patient had a prior history of DCS, while two DCS patients had previous IPO episodes. Multivariate analysis showed that the use of a wetsuit and procedural errors were associated with a higher likelihood of IPO, which occurred more frequently after shallower dives compared to DCS. Procedural errors mainly included rapid ascent, buoyancy control problems, or missed decompression stops. In three IPO cases, additional issues such as poor loop volume management or significant flooding were also reported. IPO was observed more frequently during training dives, although the difference was not statistically significant (Table 4).

Figure 4

Final clinical diagnosis of injured technical divers according to the type of breathing gas; among cases of mix decompression sickness (DCS) classified as severe, there were two with cerebral involvement, three with cardiopulmonary involvement, five with inner ear involvement, and five with spinal involvement. All mild cases of mixed DCS presented with musculoskeletal symptoms, often accompanied by skin manifestations or mild and uncertain subjective neurological symptoms such as patchy paresthesia. *P*-value for trimix vs nitrox – $^{**}P < 0.01$, $^{***}P < 0.001$; IPO – immersion pulmonary oedema



SUSPECTED CONTRIBUTING FACTORS TO MSK DCS

Because MSK DCS was the most common manifestation, occurring in 47/105 (44.8%) of DCS cases, the analysis investigated potential contributing factors. Eleven/47 (23.4%) of MSK cases. were mixed DCS (Table 5). Multivariate analysis revealed that the use of trimix, repetitive dives, younger age, and the absence of procedural errors were more frequently associated with bone involvement compared to other types of DCS (Table 6).

Divers with MSK involvement delayed consultation by more than 24 hours in 10/47 (21.3%) of cases (either pure MSK DCS or associated with cutaneous DCS), compared with 5/58 (8.6%) in other types of DCS. The median number of additional HBOT sessions was 0 [0–1] for MSK DCS and 0 [1–7] for other types ($P = 0.04$).

Discussion

The high number of diving-related accidents managed in these hyperbaric units highlights the intensity of diving activity in the Mediterranean coastline. This area is renowned for technical diving, supported by favorable bathymetry, and abundant marine life. Estimating the number of technical divers remains challenging. However, it is thought that there are 20,000 to 25,000 rebreather divers worldwide, representing less than 0.3% of the total global diving population.[12] The use of open-circuit for trimix diving has become marginal due to the high cost of helium and the logistical challenges associated with gas supply.[10,13] In contrast, the recent expansion of CCR use has redefined technical diving practices and now accounts for the majority of technical diving-related accidents.[3]

Few studies have specifically investigated the accidentology of technical diving. The demographics of injured divers were consistent with the technical diving community, which is predominantly composed of males aged 40–50 years.[3,10] It is recognised that many divers self-treat their symptoms, while those experiencing more severe or concerning manifestations are more likely to seek medical attention.[3] This suboptimal behaviour might be mostly prevalent in the technical diving community, although long-term recovery seems often favourable.[1,3,10] Factors such as extended bottom times, inert gas composition, prolonged higher oxygen exposure associated with rebreathers and equipment constraints may all influence the clinical presentation of diving accidents. These observations are consistent with our findings and recent reports suggesting that technical divers face more frequent but often low severity DCS episodes.[3,10]

CLINICAL PRESENTATION OF DCS

MSK DCS appeared to be overrepresented among technical divers, particularly in younger individuals following repetitive trimix dives performed without procedural errors and was unlikely associated with more severe presentation. Although not significant in multivariate analysis, the dive profiles leading to MSK DCS appeared more 'aggressive' (greater depth and longer total dive time) than those of other DCS presentations. Another area of concern involves inner-

Table 4

Comparison of potential contributing factors to immersion pulmonary oedema (IPO) or decompression sickness (DCS) occurrence; *n refers to the number of divers for whom data were available; CI – confidence interval; IQR – interquartile range; Trimix – helium-based mixed gas, in other cases nitrox (i.e., air diluent gas) was used in closed circuit rebreathers

Variables	IPO (n = 16)		DCS (n = 105)		Univariate P-value	Odds ratio (95% CI)	Multivariate P-value	Adjusted odds ratio (95% CI)
	*n	n (%) or Median [IQR]	*n	n (%) or Median [IQR]				
Gender female	16	3 (18.8)	105	8 (7.6)	0.2	2.8 (0.72–11.65)	NS	–
Body mass index	12	28 [23.8–0.9]	92	25.4 [23.3–27.7]	0.02	–	NS	–
Arterial hypertension	16	6 (37.5)	105	11 (10.5)	0.004	5.13 (1.42–15.02)	NS	–
Instruction dive	16	6 (37.5)	103	17 (16.5)	0.08	3.04 (0.91–9.5)	NS	–
Wet suit	11	10 (90.9)	83	12 (14.5)	< 0.0001	59.17 (7.84–648.1)	0.001	11.16 (2.69–46.41)
Trimix mixed gas	16	4 (25)	105	72 (68.6)	0.002	0.15 (0.05–0.47)	NS	–
Procedural error	16	8 (50)	104	19 (18.3)	0.005	4.47 (1.38–12.57)	0.038	4.57 (1.09–19.23)
Max depth (m)	16	40 [27–52]	103	56 [42–72]	0.0001	–	0.015	0.94 (0.89–0.99)
Total dive time (min)	15	50 [38–68]	101	71 [56–93]	0.002	–	NS	–

Table 5

Diagnoses associated with musculoskeletal (MSK) decompression sickness (DCS); *mixed diagnosis considered as severe with vestibular, cochlear or consistent neurological symptoms

DCS variant	n (%)
Pure MSK DCS	36 (76.6)
MSK + skin DCS	3 (6.4)
MSK + subjective neurological	3 (6.4)
MSK + skin + inner-ear DCS*	2 (4.3)
MSK + cerebral DCS*	1 (2.1)
MSK + spinal DCS*	1 (2.1)
MSK + constitutional DCS	1 (2.1)
Total MSK DCS	47 (100)

ear DCS among technical divers,[13–15] however, its prevalence in this study does not appear higher than in recreational diving.[16] Prolonged helium supersaturation in the perilymph and endolymph, both acting as inert-gas-diffusion-limited reservoirs, may increase the risk of cochleovestibular DCS during deep dives.[5,14] Interestingly, data from Malta revealed a rising incidence of inner-ear DCS following deep and repetitive dives, whereas MSK DCS became less frequent. The breathing gas was not reported, but it is speculated that many dives were conducted on air, despite the growing popularity of technical diving.[17]

Finally, our results contrast with reports from recreational diving, where spinal involvement is most frequently observed. Although subjective neurological symptoms, musculoskeletal pain, and constitutional symptoms also predominate in the recreational diving literature, these manifestations are frequently associated with more severe forms of DCS.[16,18,19] Technical diving confirmed neurological cases were uncommon and typically presented with moderate severity at admission, such as a MEDSUBHYP score < 6, indicating a lower risk of sequelae.[11] The observed differences in DCS patterns may be partly attributable to helium use among technical divers, as suggested by contextual comparisons with the external database reported in Meusnier's study (Figure 5).[16] Interestingly, more severe cases occasionally occurred after dives shallower than 40 msw, possibly reflecting the common use of air diluent at these depths and the associated higher nitrogen saturation contributing to their severity. Furthermore, shorter delays to symptom onset after a dive are often associated with more severe outcomes.[5,11,20] In our study, the median time to symptom onset was relatively short even in mild cases, and particularly shorter in MSK DCS compared with other DCS forms.

The high prevalence of helium-based gas mixtures used in technical divers may partly explain this pattern, as helium has lower solubility and higher diffusivity in tissues than

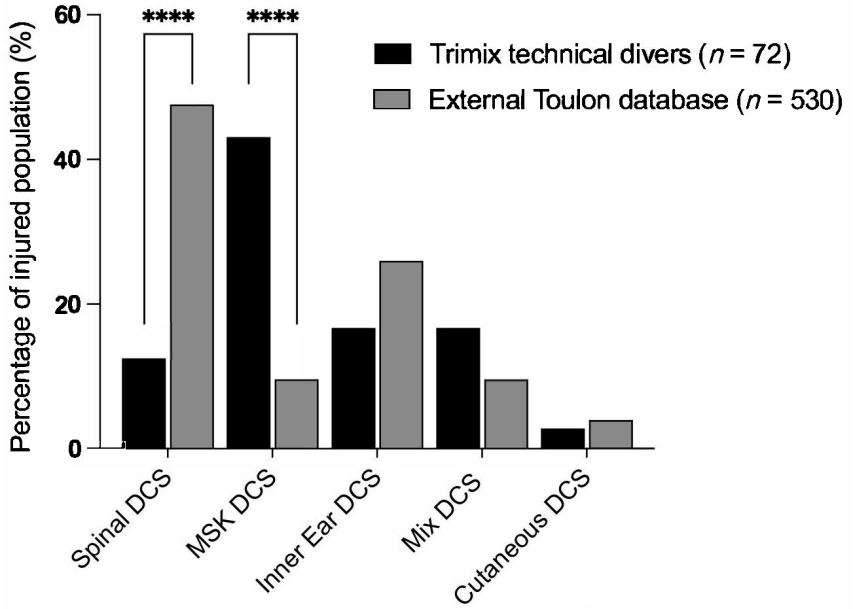
Table 6

Comparison of potential contributing factors to musculoskeletal (MSK) decompression sickness (DCS) or other type of DCS occurrence; **n* refers to the number of divers for whom data were available; CI – confidence interval; IQR – interquartile range; NI – not included in multivariate analysis due to missing data; Trimix – helium-based mixed gas, in other cases nitrox (i.e., air diluent gas) was used in closed circuit rebreathers

Variables	MSK (*n* = 47)		Other DCS (*n* = 58)		Univariate *P*-value	Odds ratio (95% CI)	Multivariate *P*-value	Adjusted odds ratio (95% CI)
	**n*	*n* (%) or Median [IQR]	**n*	*n* (%) or Median [IQR]				
Age (years)	47	38 [33–45]	58	49 [43–54]	< 0.0001	–	0.005	0.92 (0.87–0.97)
Body mass index	41	23.9 [22.5–26.1]	51	26.2 [24.8–28.4]	0.0003	–	NS	
Hypertension	47	0 (0)	58	11 (19)	0.001	0 (0–0.33)	NS	
Previous MSK DCS	12	9 (75)	16	2 (12.5)	0.0008	21 (2.93–116.3)	NS	
Instruction dive	46	5 (10.9)	57	12 (21.2)	0.2	0.46 (0.17–1.3)	NS	
Trimix mixed gas	47	38 (80.9)	58	34 (58.6)	0.02	2.98 (1.26–6.86)	0.031	4.6 (1.15–18.57)
Max depth (m)	46	66 (46–81)	57	50 (40–65)	0.008	–	NS	
Total dive time (min)	45	85 (60–118)	56	62 (48–80)	0.0006	–	NS	
Procedural error	47	3 (6.4)	58	16 (27.6)	0.005	0.18 (0.05–0.67)	0.04	0.17 (0.03–0.92)
Repetitive dive < 12 h	44	13 (29.6)	53	7 (13.2)	0.047	2.76 (0.98–7.05)	0.029	4.8 (1.17–19.88)
Max depth previous 24 h (m)	25	55 [40–70]	27	40 [38–55]	0.08	–	NI	

Figure 5

Descriptive contextual comparison for illustrative purposes only, presenting decompression sickness cases in trimix technical divers (n = 72) from the present study in parallel with data from an external Toulon Hospital database (n = 530), predominantly comprising recreational divers (air and nitrox), with 29 military divers and a negligible proportion of trimix divers (2011–2018).[16] **** $P < 0.001$; DCS – decompression sickness; MSK – musculoskeletal DCS



nitrogen. Trimix diving, especially, when dives are repeated, may promote intermediate or slow 'tissue compartment' supersaturation (e.g., muscle, bone) rather than primarily affecting the nervous system, which is considered a fast 'tissue compartment' and is more susceptible to DCS in air diving.[21] Helium may also facilitate rapid bubble growth within tissues, potentially contributing to bone involvement, as previously reported in heliox saturation diving, where bone lesions were frequent despite the absence of detectable circulating bubbles, supporting the concept of tissue-stationary bubbles.[5] Another hypothesis is the organ-protective effect of helium, though this effect remains modest and inconsistent in human studies, with limited clinical validation in diving.[22,23] In a rat model, helium preconditioning reduced neurological DCS risk, supporting potential protective benefits of helium in diving.[24]

DCS TREATMENT AND OUTCOMES

Treatments followed standard clinical procedures applicable to all divers. It is well recognised that first-aid is often insufficient, both in recreational and technical diving.[1,3,10] Most injured divers received FAO_2, as they were frequently managed by paramedics for whom this is part of standard practice in areas familiar with maritime injuries. Numerous drugs have been proposed as adjunctive therapies, but only NSAIDs have shown benefit, albeit with limited supporting data.[5] In France, acetylsalicylic acid at antiplatelet dosing (< 500 mg) was recommended until recently despite weak evidence, though this practice is now being reconsidered. Corticosteroids have also been frequently used and often preferred to NSAIDs, yet their efficacy remains unproven and HBOT remains the definitive treatment for DCS.[5] Along the French coastline, the delay before recompression is often shorter than in other regions, due to the proximity of hyperbaric units to dive sites. While delays of up to six hours have been associated with a poorer prognosis, particularly in severely affected divers,[11] recompression after longer delays can still be effective, as reported in several studies.[9,20,25] Spontaneous improvement without recompression is also often observed in mild cases, particularly among technical divers.[3,10]

Severe symptoms are generally associated with poorer treatment outcomes.[19,20,25] Some evidence suggests a more favourable prognosis among technical divers, mainly due to the higher prevalence of mild presentations.[3,10] Long-term outcomes are difficult to evaluate in retrospective studies because of limited follow-up and frequent missing data. In our study, 75% of DCS patients achieved complete symptom resolution at discharge, consistent with previous reports showing 65 to 93% favourable outcomes after treatment in the diving community.[9,18,20,25] Unfavourable outcomes were mainly associated with bone and vestibular involvement in our study. In France, follow-up bone imaging is routinely performed after any MSK DCS.[26] MRI findings suggestive of DON, likely asymptomatic for an extended period, were considered unfavourable but are often not reported in follow-up data from other studies. As MRI was available in only 30% of cases, the prevalence of DON may be underestimated given that abnormalities were detected in 57% of those imaged. Unfortunately, the timing of MRI and the frequent lack of follow-up in this study preclude a comprehensive assessment of risk, and a definitive link between imaging

abnormalities and a DCS event cannot be established without baseline imaging. Nevertheless, recent literature suggests that bone involvement, particularly in the shoulder or hip, may provide a substrate for subsequent juxta-articular DON development.[26] Although often considered mild, MSK DCS can be associated with a higher risk of DON and should not be overlooked by physicians or divers, especially in the technical diving community.[26,27] If juxta-articular damage is suspected, MRI evaluation is recommended, and additional HBOT sessions may help reduce intraosseous oedema and prevent unfavourable progression.[26,28]

LUNG INJURIES

IPO accounted for nearly 13% of admissions among technical divers, although this proportion is likely underestimated, as cases are not always referred to diving physicians. In our study, wetsuit use during shallower dives and more frequent procedural errors were independently associated with IPO rather than DCS. Procedural errors, such as omission of a decompression stop, may be a consequence of dyspnoea rather than its cause. All affected divers fully recovered, yet IPO remains a potentially life-threatening condition implicated in fatal cases not captured here. Female sex, older age, and arterial hypertension are recognised risk factors,[7] though the very low number of women in our cohort precludes conclusions here. The high risk of recurrence was underscored by 31% of cases reporting a previous episode. Accordingly, a recent expert consensus advises against further scuba-diving after an IPO event.[7] Rebreathers are suspected to increase risk through greater work of breathing and pressure imbalances, particularly with back-mounted counterlungs.[6] Additionally, wetsuits may reduce pulmonary compliance.[29] Although comparison with recreational divers is not the primary aim of this study, overall diagnostic data from hyperbaric units during the study period (Figure 1) allow these distributions to be examined. In our cohort, rebreather use did not increase IPO incidence compared with recreational open-circuit scuba diving, with respectively 16/94 (17%) versus 389/2,762 (14.1%) cases ($P = 0.4$). Among non-professional or non-military profiles with lower exertion, the impact of CCR on work of breathing may be reduced.[13] Interestingly, IPO was more common with air diluent than with trimix, usually at shallower depths during entry-level CCR training, indicating that diver inexperience (e.g., stress, buoyancy control, loop volume management) rather than gas density may have been the primary contributing factor.

GAS-RELATED TOXICITY

Serious gas-toxicity symptoms represented a rare cause of hospital admission. This aligns with reports from interviewed trimix divers.[3] However, inappropriate breathing gas is frequently observed in CCR fatalities, which may introduce survivor bias and underestimate the true incidence of this risk.[12] This contrasts with military diving, where equipment and procedures differ. In that context, gas toxicity was found to be the most common diving incident, with hyperoxic seizures and hypercapnia frequently reported, usually with favorable outcomes.[8] The less frequent use of airway protection systems, such as mouthpiece retaining straps or full-face masks, by technical divers may also contribute to a higher proportion of fatalities when incidents occur, which may therefore go unreported in hyperbaric centers.[30] It is also likely that most non-fatal gas-related issues, which rapidly improve after surfacing, remain undocumented by the medical community.

LIMITATIONS

This retrospective study, based on medical records, was limited by substantial amounts of missing data. The potential impact of specific dive planning on accident risk could not be evaluated due to the lack of systematic information on breathing gas composition, detailed dive profiles, and computer conservatism settings. Furthermore, five divers experienced two DCS episodes during the study period, introducing a potential partial dependence of observations that may have modestly influenced the results by increasing the contribution of individual-specific risk factors. However, these cases accounted for less than 5% of the study population, and multivariate analysis of DCS patterns was primarily based on dive-related variables rather than intrinsic individual factors. Because the location of studied units, cave diving, a growing activity within the technical diving community, falls outside the scope of this study. Accident patterns in cave diving may vary due to differences in practices and environmental constraints compared to open-sea diving. These divers may be self-treated more frequently and/or admitted in inland hyperbaric units located near the cave systems.[13]

Conclusions

This study describes 15-years diving accident patterns among technical divers treated in hyperbaric units along the French coastline. DCS was the most prevalent condition, typically presenting with mild symptoms, with a high prevalence of MSK cases. Divers affected by MSK DCS tended to be younger and often used helium-based breathing mixtures, with no reported procedural errors, making these accidents difficult to predict. Current data remain insufficient to assess the impact of dive profile, decompression planning or gas mix composition. Although most cases receive prompt management, some do not experience favorable outcomes, with delayed bone lesions potentially leading to joint dysfunction and lifelong disability. Further research is needed on diving practices, preventive measures for this specific technical diver population, and the role of helium in accident risk.

References

1 Monnot D, Michot T, Dugrenot E, Guerrero F, Lafère P. A survey of scuba diving-related injuries and outcomes among

French recreational divers. Diving Hyperb Med. 2019;49:96–106. doi: 10.28920/dhm49.2.96-106. PMID: 31177515. PMCID: PMC6704004.
2 Lippmann J. Diving-related fatalities in Victoria, Australia, 2000 to 2022. Diving Hyperb Med. 2025;55:35–43. doi: 10.28920/dhm55.1.35-43. PMID: 40090024.
3 Gouin E, Monnot DP, Michot T, Guerrero F, Blatteau JÉ. Diving practices in technical divers' community and behaviour towards self-reported unusual symptoms. Diving Hyperb Med. 2025;55:114–25. doi: 10.28920/dhm55.2.114-125. PMID: 40544139. PMCID: PMC12267066.
4 Mitchell SJ, Doolette DJ. Recreational technical diving part 1: an introduction to technical diving methods and activities. Diving Hyperb Med. 2013;43:86–93. PMID: 23813462. [cited 2025 Nov 1]. Available from: https://dhmjournal.com/images/IndividArticles/43June/Mitchell_dhm.43.3.86-93.pdf.
5 Mitchell SJ. Decompression illness: a comprehensive overview. Diving Hyperb Med. 2024;54(Suppl):1–53. doi: 10.28920/dhm54.1.suppl.1-53. PMID: 38537300. PMCID: PMC11168797.
6 Castagna O, Regnard J, Gempp E, Louge P, Brocq FX, Schmid B, et al. The key roles of negative pressure breathing and exercise in the development of interstitial pulmonary edema in professional male SCUBA divers. Sports Med Open. 2018;4:1. doi: 10.1186/s40798-017-0116-x. PMID: 29299780. PMCID: PMC5752643.
7 Banham N, Smart D, Wilmshurst P, Mitchell SJ, Turner MS, Bryson P. Joint position statement on immersion pulmonary oedema and diving from the South Pacific Underwater Medicine Society (SPUMS) and the United Kingdom Diving Medical Committee (UKDMC) 2024. Diving Hyperb Med. 2024;54:344–9. doi: 10.28920/dhm54.4.344-349. PMID: 39675743. PMCID: PMC11779524.
8 Gempp E, Louge P, Blatteau JE, Hugon M. Descriptive epidemiology of 153 diving injuries with rebreathers among French military divers from 1979 to 2009. Mil Med. 2011;176:446–50. doi: 10.7205/milmed-d-10-00420. PMID: 21539168.
9 Lundell RV, Arola O, Suvilehto J, Kuokkanen J, Valtonen M, Räisänen-Sokolowski AK. Decompression illness (DCI) in Finland 1999–2018: Special emphasis on technical diving. Diving Hyperb Med. 2019;49:259–65. doi: 10.28920/dhm49.4.259-265. PMID: 31828744. PMCID: PMC7039777.
10 Tuominen LJ, Sokolowski S, Lundell RV, Räisänen-Sokolowski AK. Decompression illness in Finnish technical divers: a follow-up study on incidence and self-treatment. Diving Hyperb Med. 2022;52:78–84. doi: 10.28920/dhm52.2.74-84. PMID: 35732278. PMCID: PMC9527095.
11 Andre S, Lehot H, Morin J, Louge P, De Maistre S, Roffi R, et al. Influence of prehospital management on the outcome of spinal cord decompression sickness in scuba divers. Emerg Med J. 2022;39:747–52. doi: 10.1136/emermed-2021-211227. PMID: 35135892.
12 Tillmans F, Saraiva C, Ferguson S, Dugrenot E, Blogg SL. Recreational and technical rebreather fatalities and diving safety status. Diving Hyperb Med. 2026;56:185–190. doi: 10.28920/dhm56.2.185-190. PMID: 42290579.
13 Gouin E, Dugrenot E, Gardette B. Extremely deep bounce dives: planning and physiological challenges based on the experiences of a sample of French-speaking technical divers. Diving Hyperb Med. 2025;55:203–10. doi: 10.28920/dhm55.2.203-210. PMID: 40544151. PMCID: PMC12267070.
14 Doolette DJ, Mitchell SJ. Recreational technical diving part 2: decompression from deep technical dives. Diving Hyperb Med. 2013;43(2):96–104. PMID: 23813463. [cited 2025 Nov 1]. Available from: https://dhmjournal.com/images/IndividArticles/43June/Doolette_dhm.43.3.96-104.pdf.
15 Lindfors OH, Lundell RV, Arola OJ, Hirvonen TP, Sinkkonen ST, Räisänen-Sokolowski AK. Inner ear decompression sickness in Finland: a retrospective 20-year multicenter study. Undersea Hyperb Med. 2021;48:399–408. PMID: 34847303.
16 Meusnier B. Contribution à l'analyse séméiologique et diagnostique des accidents de plongée survenant sur la côte méditerranéenne française: à propos de 1014 cas admis au centre hyperbare de l'HIA Sainte-Anne à Toulon de 2011 à 2018. [Internet]. Thèse de doctorat en médecine, Faculté des sciences médicales et paramédicales de Marseille; 2020. [cited 2025 Oct 20]. Available from: https://dumas.ccsd.cnrs.fr/dumas-03048838v1/file/The%60se.pdf.
17 Azzopardi CP, Caruana J, Matity L, Muscat S, Meintjes WJ. Increasing prevalence of vestibulo-cochlear decompression illness in Malta – an analysis of hyperbaric treatment data from 1987–2017. Diving Hyperb Med. 2019;49:161–6. doi: 10.28920/dhm49.3.161-166. PMID: 31523790. PMCID: PMC6881197.
18 Chevasutho P, Premmaneesakul H, Sujiratana A. Descriptive study of decompression illness in a hyperbaric medicine centre in Bangkok, Thailand from 2015 to 2021. Diving Hyperb Med. 2022;52:277–80. doi: 10.28920/dhm52.4.277-280. PMID: 36525685. PMCID: PMC10017199.
19 Thaler J, Pignel R, Magnan MA, Pellegrini M, Louge P. Decompression illness treated at the Geneva hyperbaric facility 2010–2016: A retrospective analysis of local cases. Diving Hyperb Med. 2020;50:370–6. doi: 10.28920/dhm50.4.370-376. PMID: 33325018. PMCID: PMC8038901.
20 Blake DF, Crowe M, Lindsay D, Turk R, Mitchell SJ, Pollock NW. Divers treated in Townsville, Australia: worse symptoms lead to poorer outcomes. Diving Hyperb Med. 2024;54:308–19. doi: 10.28920/dhm54.4.308-319. PMID: 39675739. PMCID: PMC12018695.
21 Hugon J, Rostain JC, Gardette B. A new biophysical decompression model for estimating the risk of articular bends during and after decompression. J Theor Biol. 2011;283:168–79. doi: 10.1016/j.jtbi.2011.05.002. PMID: 21609722.
22 Lucchinetti E, Wacker J, Maurer C, Keel M, Härter L, Zaugg K, et al. Helium breathing provides modest antiinflammatory, but no endothelial protection against ischemia-reperfusion injury in humans in vivo. Anesth Analg. 2009;109:101–8. doi: 10.1213/ane.0b013e3181a27e4b. PMID: 19535699.
23 Rocco M, Maggi L, Loffredo C, Pelli M, Benedetto PD, Fiorelli S, et al. The impact of different gas mixtures on inflammatory responses in advanced recreational divers. Diving Hyperb Med. 2021;51:140–6. doi: 10.28920/dhm51.2.140–6. PMID: 34157728. PMCID: PMC8426122.
24 Zhang R, Yu Y, Manaenko A, Bi H, Zhang N, Zhang L, et al. Effect of helium preconditioning on neurological decompression sickness in rats. J Appl Physiol (1985). 2019;126:934–40. doi: 10.1152/japplphysiol.00275.2018. PMID: 30653414.
25 Sokolowski SA, Räisänen-Sokolowski AK, Tuominen LJ, Lundell RV. Delayed treatment for decompression illness: factors associated with long treatment delays and treatment outcome. Diving Hyperb Med. 2022;52:271–6. doi: 10.28920/dhm52.4.271-276. PMID: 36525684. PMCID: PMC10026386.
26 Blatteau JE, Gempp E. Fit for diving after musculoskeletal decompression sickness: how to detect and manage bone lesions? Undersea Hyperb Med. 2025;52:9–14. PMID: 40249717.

27 Coleman B, Davis FM. Dysbaric osteonecrosis in technical divers: The new 'at-risk' group? Diving Hyperb Med. 2020;50:295–9. doi: 10.28920/dhm50.3.295-299. PMID: 32957134. PMCID: PMC7819721.
28 Reis ND, Schwartz O, Militianu D, Ramon Y, Levin D, Norman D, et al. Hyperbaric oxygen therapy as a treatment for stage-I avascular necrosis of the femoral head. J Bone Joint Surg Br. 2003;85:371–5. doi: 10.1302/0301-620x.85b3.13237. PMID: 12729112.
29 Stevens G, Smart DR. The influence of wetsuit thickness (≥ 7 mm) on lung volumes in scuba divers. Diving Hyperb Med. 2025;55:27–34. doi: 10.28920/dhm55.1.27-34. PMID: 40090023. PMCID: PMC12263280.
30 Gouin E, Dugrenot E, Lance RM, Michot T, Marroni L, Tillmans F. Perceptions of airway protection tools: an international survey on the use of mouthpiece retaining straps in closed-circuit rebreather diving. Diving Hyperb Med. 2025;55:369–75. doi: 10.28920/dhm55.4.369-375. PMID: 41364860. PMCID: PMC12823153.

Acknowledgments

We thank the heads of the hyperbaric units for allowing us access to these data and for their interest in this work, particularly Dr Mathieu Coulange, Dr Anne Henckes and Dr Andreas Kauert. We also thank Dr Julien Hassan for his assistance in data collection.

Conflicts of interest and funding

The study is entirely funded through Divers Alert Network. No conflicts of interest were declared.

Submitted: 5 November 2025
Accepted after revision: 21 March 2026

Measuring whole-body inert gas uptake and washout during submersion

Oscar Plogmark[1,2], Kristian Soltesz[3], Carl Hjelte[1,2,4], Oskar Frånberg[1,5]

[1] *Lund University, Faculty of Medicine, Department of Clinical Sciences Lund, Respiratory Medicine and Allergology, Lund, Sweden*
[2] *Swedish Armed Forces Diving and Naval Medicine Center, Swedish Armed Forces, Karlskrona, Sweden*
[3] *Lund University, Faculty of Engineering, Department of Automatic Control, Lund, Sweden*
[4] *Kungälv Hospital, Department of Anesthesia and Intensive care, Kungälv, Sweden*
[5] *Blekinge Institute of Technology, Department of Mathematics and Natural Science, Karlskrona, Sweden*

Corresponding author: *Dr Oscar Plogmark, Sölvegatan 19, 221 85 Lund, Sweden*
ORCiD: *0009-0008-3230-8807*
oscar.plogmark@med.lu.se

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

Abstract
(Plogmark O, Soltesz K, Hjelte C, Frånberg O. Measuring whole-body inert gas uptake and washout during submersion. Diving and Hyperbaric Medicine. 2026 30 June;56(2):137–147. doi: 10.28920/dhm56.2.137-147. PMID: 42290573.)
Introduction: Quantifying inert gas uptake and washout is critical for understanding decompression sickness (DCS). However, the limited amount of data has made it difficult to integrate inert gas kinetics into risk models for DCS. Measuring whole-body inert gas kinetics during submersion is technically challenging. This study presents a novel method for quantifying inert gas uptake and washout in human divers using a rebreather-based system.
Methods: During constant-depth diving with a closed-circuit system that maintains a constant oxygen partial pressure, changes in buoyancy will reflect the kinetics of inert gas. Two divers completed four dives each, with a bottom phase at 2.5 bar and a decompression phase at 1.3 bar or 1.4 bar. Load cell data were converted into equivalent changes in volume of nitrogen standardised for temperature and pressure (V_{N2}, STP). Power analysis was conducted to quantify the resolution by which the method could detect nitrogen uptake and washout volumes.
Results: Distinct uptake and washout curves were obtained, comparable to previous studies using other techniques. Mean V_{N2} uptake during the bottom phase was 0.96 L (SD 0.29), while mean washout during decompression was 0.67 L (SD 0.26). The minimal mean detectable difference (MDD) with eight dives was 0.28 L for the bottom phase and 0.26 L for the decompression phase, considering standard 80% power and a 0.05 significance level.
Conclusions: This novel method quantifies inert gas kinetics during submersion with acceptable precision and accuracy. It could facilitate the collection of inert gas kinetics data during submersion, potentially yielding valuable correlations with the risk of DCS.

Introduction

Quantifying the uptake and washout of inert gases is likely a crucial component in elucidating the pathophysiology of decompression sickness (DCS).[1] While hyperbaric exposure and physiological state influence inert gas kinetics, our understanding of these processes remains limited. This knowledge gap constrains our ability to assess individual susceptibility to DCS.

Research over the years has accumulated knowledge on inert gas kinetics,[2–13] and its association with decompression sickness (DCS).[7,9] However, to incorporate inert gas kinetics to increase predictive accuracy in our risk models or to individualise ongoing decompression, substantially more data is required.

Due to the technical complexity of the required equipment, the number of trials measuring inert gas in the context of diving is limited. To our knowledge, a very limited number of research groups have studied and published on techniques of measuring whole-body inert gas washout and/or uptake.[2,8,10–12]

We recently published a proof-of-concept study on a simple setup that uses rebreather components to measure inert gas washout.[8] This system could, in principle, also be used to assess inert gas kinetics during dives. However, its application in hyperbaric chambers is limited by regulatory requirements, increasing measurement errors at higher pressures, and the need to keep the apparatus dry inside the chamber. These factors present a significant practical challenge.

The aim of this study was to present a novel method for quantifying inert gas uptake and washout during submersion and evaluate its performance with human divers.

Methods

The study protocol was approved by the Swedish Ethical Review Authority (Dnr: 2020–06865).

DIVE PROTOCOL

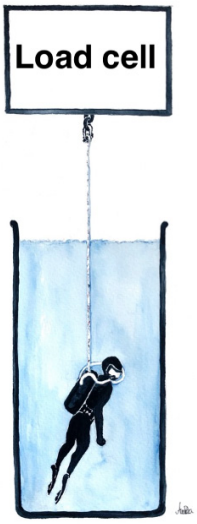
Experiments were conducted in an 18 m deep tank with water maintained at 35.5°C. Each dive followed a standardised protocol. The diver wore swim trunks, a rebreather unit (Poseidon Se7en with a firmware modification that restricted the unit to only inject metabolic oxygen, disabled injections of diluent as well as extra gas injections for sensor calibration), a buoyancy control device (BCD), and adequate weights to maintain consistent negative buoyancy. Before the dive, the diver conducted a pre-dive system check in accordance with the rebreather manufacturer's guidelines. After entering the water, the diver breathed on the closed circuit at a P_{02} setpoint of 0.5 bar for two minutes before descent.

The diver emptied the BCD bladder entirely and descended passively, feet first, until restrained by the tethered line connected with the load cell (Mecmesin S-200N, Mecmesin AFTI load cell amplifier and Mecmesin VectorPro Lite logger v0.2), as shown in Figure 1. A remotely actuated crane system adjusted the line to maintain a depth corresponding to 250 kPa (2.5 bar) ambient pressure. At this depth, the diver stabilised breathing conditions by taking deep inhalations, triggering the automatic diluent valve (ADV) to partially fill the breathing circuit. Once stable, load cell measurements commenced, with the diver completely motionless and breathing normally. Only oxygen was injected into the breathing circuit during the measurement phase. These injections were actuated by the onboard P_{02} controller of the rebreather to compensate for metabolic consumption and thus maintain the 0.5 bar P_{02} setpoint.

The diver maintained this position for 25 min, during which load cell measurements were recorded. The diver then adjusted the P_{02} setpoint to 0.7 bar and ascended to a decompression depth with corresponding ambient pressure of 130 kPa (1.3 bar) or 140 kPa (1.4 bar). At this depth, the diver was again tethered to the load cell. Upon stabilising breathing conditions, a second 25 min load cell measurement series was recorded. The P_{02} setpoint change was executed before the ascent to decrease stabilisation time. The reason for increasing P_{02} in the first place was to establish an increased gradient between tissues and breathing gas, to accelerate decompression, translating into a higher washout rate to facilitate estimation of whole-body inert gas washout.

Throughout all load cell data collection, the operator continuously monitored the diver via in-pool camera, to ensure the absence of bubble release, confirming the integrity of the closed rebreather system. A detailed protocol description is provided as *Appendix A.

Figure 1
Experimental setup: submerged closed-circuit rebreather diver suspended from load cell



VOLUME BALANCE MODEL

A system comprising a rebreather and diver, suspended from a load cell as shown in Figure 1, experiences a buoyant force that increases linearly with the volume of water that the system displaces. For an initial time , this displacement volume can be denoted $V_0 = V(t_0)$. If we are only considering changes in buoyancy, we can, without loss of generality, shift our time and volume scales, and thus defining $t_0 = 0$ and $V_0 = 0$. For any subsequent time $t > 0$, volume balance gives us that new displacement volume $V(t)$ is given by

$$V = V_{N_2} + V_{O_2,i} - V_{O_2,m} + V_d + V_o + V_b - V_l \quad \text{Eq.1}$$

where we have dropped the time argument (t) of each term, to improve readability. The meaning of each term is summarised in Table 1. To facilitate interpretation, a detailed schematic of the experimental setup is shown in Figure 2.

Some of these volume changes can be negative, namely V, V_{N2}, V_O and V_b. For V_{N2}, a negative value corresponds to uptake (by the diver from the circuit); a positive value corresponds to washout (from the diver into the circuit). Similarly, $V_b < 0$ corresponds to purged gas from any variable-volume.

Note that neither pressure drops in onboard oxygen or diluent cylinders affect the displacement volume, as the cylinders are fixed volume. Furthermore, gas molecules leaving the

***Footnote:** Appendix A is available online on our website: https://www.dhmjournal.com/index.php/journals?id=417

Table 1
Definition of terms used for volumes included in Equation 1

Symbol	Explanation
V	Change in total displacement volume
V_{N2}	Volume of physiologically bound nitrogen (inert gas) that has been washed out into the respiratory circuit
$V_{02,i}$	Volume of oxygen injected into the circuit
$V_{02,m}$	Volume of metabolised oxygen
V_d	Volume of diluent injected into the circuit
V_0	Change in volume of other gases present in the circuit (mainly carbon dioxide)
V_b	Volume added to variable-volume buoyancy compensation devices (e.g., drysuit or vest)
V_l	Volume leaked from the circuit

cylinders into the circuit, or passing between the diver and the circuit, do not contribute to any buoyancy change as the molecules remain within the suspended system. The same holds for carbon dioxide molecules being scrubbed from the circuit, by chemically binding into the scrubber material.

All volumes are reported as standard temperature and pressure (STP) equivalents in the unit of liters.

ASSUMPTIONS

In this we go through assumptions made in modeling, leading to our proposed estimation model.

Oxygen injection ($V_{02,i}$)

Oxygen injection volume, $V_{O2,i}$ is assumed to be proportional to solenoid valve opening time, $t_{O2,i}$, with a proportionality constant, $\alpha(P)$, that varies with ambient pressure, P.

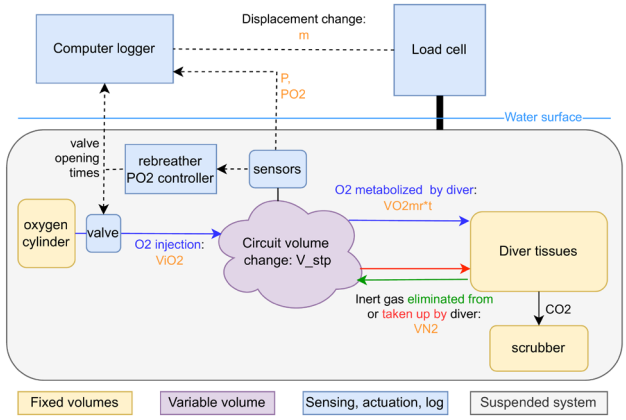
This model has previously been verified by the rebreather manufacturer, and the ambient pressure (depth)-dependent proportionality constant between $t_{O2,i}$ and $V_{O2,i}$ is logged by the rebreather.

We were able to independently verify this model by noting that the magnitude of jumps in V registered by the load cell at each oxygen injection instance matched those predicted by the model.

Oxygen metabolism ($V_{02,m}$)

Oxygen metabolic rate, $V_{02,m}$ is assumed to be constant throughout each measurement series. With the diver at complete rest while suspended from the load cell, this

Figure 2
Schematic overview of system components, gas flows, and signals



assumption is plausible. Its validity was verified for our measurements by inspection of the logged high-pressure gauge connected to the oxygen cylinder, and the cumulative solenoid valve opening time. During episodes of stable P_{02}, both these lines featured a constant slope.

Diluent injection (V_d)

The intention was to avoid diluent injections altogether, corresponding to $V_d = 0$. However, if the rebreather circuit is breathed down to a level that activates the mechanical ADV, a distinct jump in V results analogous to that caused by a solenoid valve oxygen injection. By manually determining the magnitude of any such jumps, it is possible to estimate the volume of injected diluent.

Influence of other gases (V_0)

To estimate V_{N2} based on Equation 1, the volume change contribution from other gases, V_0, needs to be known. These gases are mainly carbon dioxide, water vapor, and traces of other inert gases, such as argon. Assuming adequate carbon dioxide scrubbing and measuring P_{02} after the scrubber, it is justified to neglect the effect of carbon dioxide fraction (fluctuation) on the total circuit volume V. Similarly, the fraction of other inert gases is typically sufficiently low within the respiratory circuit to be safely neglected.

Although water vapor in the respiratory circuit may fluctuate due to temperature changes and moisture production from both the diver and the carbon dioxide scrubber, these variations are negligible. At 37°C and 250 kPa (2.5 bar), the vapour pressure of water (≈6 kPa [0.06 bar]) is insignificant compared to oxygen and nitrogen pressures. Thus, water vapor can be excluded from analysis without significantly affecting measurement accuracy or precision.

To summarise, we have made the simplifying assumption that $F_{02} + F_{N2} = 1$ within the circuit at all times.

Buoyancy compensation (V_b)

We assume $V_b = 0$, which is easy to confirm, simply by not manually adjusting buoyancy by gas injection into variable volumes (other than metabolic oxygen injection into the respiratory circuit).

Leaks (V_l)

We assume $V_l = 0$, which is easy to confirm by visual monitoring to assure no bubbles leave the diver during an ongoing measurement series. This was done during all reported measurement series.

Temperature effects

Under the ideal gas law, the volumes of Equation 1 scale linearly with absolute temperature. Such effect can be eliminated by conducting the experiment in water that holds a constant, known temperature. While our rebreather setup lacked circuit temperature monitoring, the dives were conducted in a pool with fresh water holding a constant temperature. We thus assume a constant circuit temperature, T_C, throughout each experiment series.

Inert gas kinetic time scale

While breathing on the circuit, the centre of mass of the diver shifts slightly as gas moves between lungs and counter lungs. This induces oscillations in the load cell measurement of a diver suspended according to Figure 1. While it might be possible to compensate for such oscillations, our approach has been to instead only consider inert gas kinetics at a time scale slower than that of the respiratory dynamics. This introduces a trade-off between frequency resolution and noise penetration, and we have chosen to disregard any estimated signal components corresponding to a time scale faster than a five-minute half-time, being a commonly used fastest half-time in decompression models.[1,13]

Sweating

The careful reader might have noticed that Equation 1 only concerns gas volume changes. While the diver might change weight, mainly due to sweating, the impact of such weight change is negligible as the densities of sweat and surrounding water are very similar, with a relative difference of around 1–3% (depending on the combination of salt content in the sweat and surrounding water). We therefore neglect the effect of sweating on change in buoyancy.

ESTIMATION MODEL

In this section we combine the fundamental volume balance of Equation 1 with the assumptions of Section "*ASSUMPTIONS*", to arrive at a model for estimation of VN_2 from available measurements:

$$V_{N_2} = V - V_{O_2,i} + V_{O_2,m}$$

Eq.2

Equation 2 merely states that at any time during ongoing measurement, the change in physiologically bound nitrogen volume is the difference between the total volume change, V, compensated by the volume change due to injected oxygen $V_{02,i}$ and stabilising oxygen $V_{02,m}$, respectively. As already stated, we consider all volumes as their STP equivalents.

Next, we construct estimators for each of the right-hand-side terms, to arrive at an estimator for the desired left-hand-side of Equation 2. The term V is readily available by scaling the load cell reading using the ideal gas law, as described in Section "*Total volume change estimation*".

Oxygen is injected in bursts, and since the onset and duration of the oxygen-injecting solenoid valve activations are logged by the rebreather, we know when these bursts occur, and how long they each last. What remains is to determine the resulting volumes of injected oxygen, being the topic of Section "*Oxygen injection estimation*".

Oxygen metabolic rate was constant throughout each measurement series. In Section "*Oxygen metabolic rate estimation*" this constant metabolic rate is estimated, taking into account both that there is a transient at the beginning of each measurement phase, and that mean oxygen injection rate does not exactly match metabolic rate. The former is caused by the rebreather stabilising P_{02} following a change in depth. The latter is a consequence of more or less oxygen needed in the circuit to maintain a stable P_{02} as the circuit nitrogen content changes due to diver uptake and washout.

Before moving on, we can also note that despite the total flux of oxygen through the system (roughly $V_{02,i}(t)/t$) being an order of magnitude larger than the quantity V_{N2} of interest to us, we can still enable reliable estimates. This is because the system is open to oxygen, but closed to inert gas, as pointed out and further explained elsewhere.[13]

An error analysis of the resulting estimation model is provided in Section "*Error analysis*".

Total volume change estimation

We use the change in load cell measurement, m, in combination with the ideal gas law, to estimate the change in circuit volume:

$$\hat{V}(t) = \frac{T_0 \hat{P}}{\hat{T}_c P_0} m(t)$$

Eq.3

where T_0 = 273.15 K and P_0 = 1.01325 bar (1 atmosphere) are the STP temperature and pressure, respectively. Since the diver is suspended by a static line of known length, the ambient pressure can be considered known with certainty, $\widehat{P} = P$.

All dives were conducted in a heated pool, holding a constant temperature of 35.5°C, close to normal physiological human core body temperature of 37°C. It is fair to assume that heat loss from the circuit to the water balances heat introduced by the exothermic scrubber reaction. We have therefore assumed a mean circuit temperature of $T_c = T_0 + 37$°C throughout all measurement series.

Oxygen injection estimation

The estimate of (cumulative) volume of oxygen injected into the circuit is:

$$\hat{V}_{O_2,i}(t) = \int_0^t \alpha(P(\tau))\mathbb{1}_{O_2,i}(\tau)\,d\tau = \alpha(P)\int_0^t \mathbb{1}_{O_2,i}(\tau)\,d\tau = \alpha(P)\,t_{O_2,i}(t)$$

Eq.4

The pressure-dependent injection rate $\alpha(P)$ is constant throughout the measurement phase, as the diver is suspended at a fixed depth, thus exposed to a constant ambient pressure, P. Hence, we can move $\alpha(P) = \alpha(P(t))$ outside the integral. The indicator $\mathrm{I}_{02,\mathrm{i}}(t)$ is 1 for t when the oxygen solenoid valve is open, and 0 otherwise, and thus integrates up to the total solenoid valve opening time, $t_{02,i}(t)$.

As mentioned in elsewhere, it was possible to verify the correctness of the manufacturer-supplied injection rate constant $\alpha(P)$, by confirming that compensation through Equation 4 exactly cancels the injection jumps. These jumps are thus visible in the raw load cell signal shown in the black top pane plots of Figure 3, but not in the corresponding compensated light red V_{N2} estimates in the second topmost panes.

Oxygen metabolic rate estimation

Following an initial transient caused by the change in depth preceding each measurement series, the onboard partial pressure controller of the rebreather stabilised the P_{02} at its setpoint. Since ambient pressure, P, is constant during a measurement series, and known with certainty as the diver is suspended by a static line, this also means that F_{02} is stabilised. We use the average as an estimate of the stabilised F_{02}, where $\widehat{P}_{02}$ is the reading from the primary oxygen partial pressure sensor, and $\widehat{P}_{02}$ denotes its average between time instances t_1 and t_2.

$$\hat{\dot{V}}_{O_2,m} = \frac{\hat{V}_{O_2,i}(t_2) - \hat{V}_{O_2,i}(t_1) + \hat{V}_{O_2,c}(t_2,t_1)}{t_2 - t_1} = \frac{\alpha(P)\left(t_{O_2,i}(t_2) - t_{O_2,i}(t_1)\right) + \frac{\bar{P}_{O_2}T_0}{P_0\hat{T}_c}\left(m_f(t_2) - m_f(t_1)\right)}{t_2 - t_1}$$

Eq.5

We can now use our stable oxygen fraction estimate, $\widehat{F}_{O2}$ of Equation 5 to estimate the volume of injected oxygen between t_1 and t_2, $V_{02,c}(t_2,t_1)$ that remains in (or is metabolised from, in case of $V_{02,c} < 0$) the circuit at t_2:

$$\hat{V}_{O_2,c}(t_2,t_1) = \hat{F}_{O_2}\left(\hat{V}_f(t_2) - \hat{V}_f(t_1)\right)$$

Eq.6

The subscript f, as in $\widehat{V}_f$ of Equation 6, denotes that the underlying $\widehat{V}$ signal has been low-pass filtered to remove the respiratory-induced noise mentioned in Section "*Inert gas kinetic time scale*".

Combining Equations 4 to 6, we thus arrive at the metabolic rate estimator:

$$\hat{V}_{O_2,m} = \frac{\hat{V}_{O_2,i}(t_2) - \hat{V}_{O_2,i}(t_1) + \hat{V}_{O_2,c}(t_2,t_1)}{t_2 - t_1} = \frac{\alpha(P)\left(t_{O_2,i}(t_2) - t_{O_2,i}(t_1)\right) + \frac{\bar{P}_{O_2}T_0}{P_0\hat{T}_c}\left(m_f(t_2) - m_f(t_1)\right)}{t_2 - t_1}$$

Eq.7

INERT GAS LOADING ESTIMATOR

Combining Equations 2 to 4 and 7, we finally arrive at the inert gas uptake/washout estimator:

$$\hat{V}_{N_2}(t) = \hat{V}(t) - \hat{V}_{O_2,i}(t) + \hat{V}_{O_2,m}(t) = \frac{T_0P}{\hat{T}_cP_0}m(t) - \alpha(P)t_{O_2,i}(t) + \frac{\alpha(P)\left(t_{O_2,i}(t_2) - t_{O_2,i}(t_1)\right) + \frac{\bar{P}_{O_2}T_0}{P_0\hat{T}_c}\left(m_f(t_2) - m_f(t_1)\right)}{t_2 - t_1}\,t$$

Eq.8

Subsequently, we apply low-pass filtering to remove respiratory-induced artifacts, arriving at the final estimator $\widehat{V}_{N2,f}$.

IMPLEMENTATION

For each dive, time series logs of m, P_{02}, and $t_{02,i}$ were time-aligned and re-sampled at a rate of 1 Hz. This resampling was performed in a way that preserved oxygen solenoid valve activation time. Remaining signals were linearly re-sampled, except oxygen partial pressure setpoint, which was resampled to nearest neighbor.

While we used integrals in Section "*ESTIMATION MODEL*" for generality, the corresponding zero-order-hold sampled sums were used in the code implementing our estimator, as we only have access to measurements at discrete sampling instances.

Figure 3

Each of the sub-figures (1–8) represent one dive. Odd sub-figures correspond to dives made by Diver 1; even sub-figures to dives made by Diver 2. The top panel of each sub-figure shows ambient pressure in black, and load cell raw measurement (affinely transformed to V_{N2}) in light blue. A version of the latter, compensating for oxygen injections and constant oxygen metabolic (mass) rate is shown in light red. The bright red curve is obtained from the light red curve through low-pass filtering. Finally, the bright blue curve is a simulation of the one-compartment model that best fits the light red curve, in the least-squares sense. The mid panels show P_{O2} measurements from the rebreather in black. The light red spikes show activation times of the solenoid valve that injects oxygen into the breathing circuit. The bottom panels show oxygen cylinder gauge pressure in grey. The black lines are isotonic regressions of the noisy pressure measurement during the bottom and decompression stages. The cumulative sum of solenoid valve opening times is shown in red

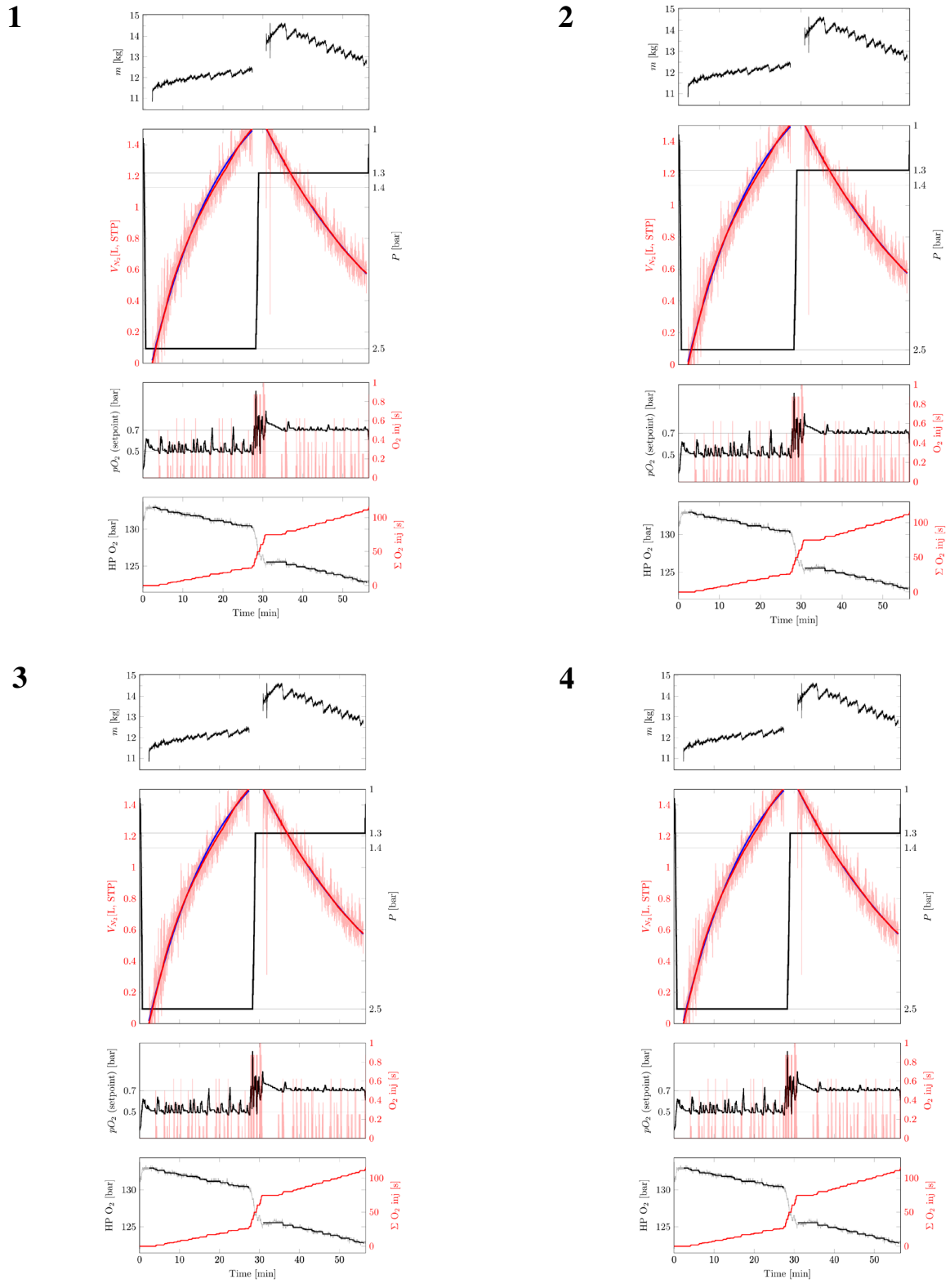
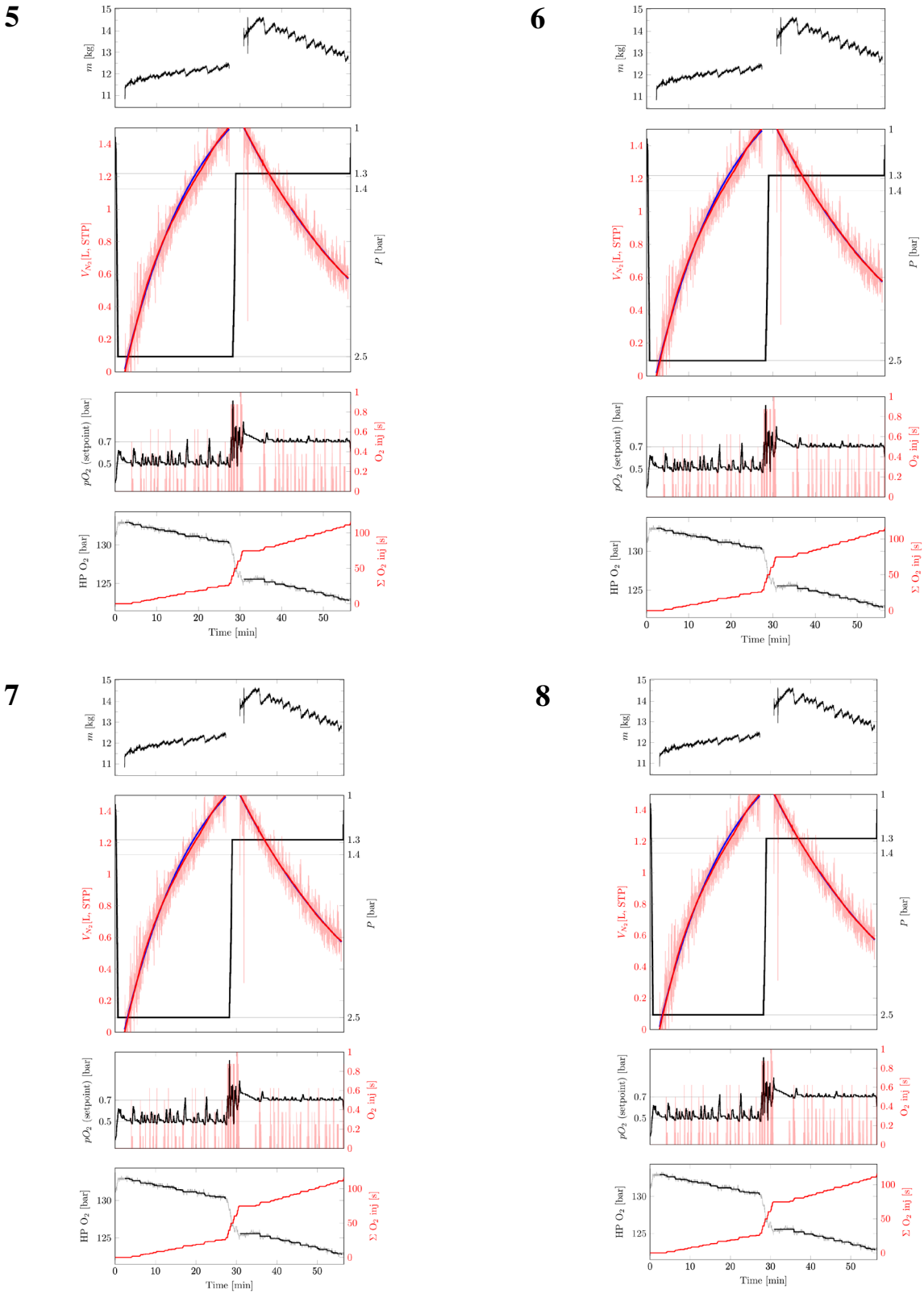


Figure 3 continued.
Sub figures 5–8



The mentioned low-pass filtering was achieved using an acausal moving average filter of window width $T_f = 5/\ln(2)$ min, matching the -3 dB cutoff of a first-order low-pass filter with 5min time constant. To avoid edge-effect artifacts, an exponential model extrapolation was used as padding.

For further details, please see the estimation code used in the study, provided in *Appendix B.

ERROR ANALYSIS

In this section we discuss possible sources of error, and their impact on the estimate $\widehat{V}_{N2,f}$.

Ambient pressure (P)

Ambient pressure enters the estimator of Equation 8 linearly in the $\widehat{V}$ term. Thus, an error in its measurement will have an affine impact on $\widehat{V}$.

Since the diver was suspended in a static line of known length, we can consider errors in ambient pressure measurement as negligible and assert $\widehat{P} = P$ throughout each measurement series.

Suspended mass (m)

An error in mass measurement affects the estimate mainly by its linear impact on $\widehat{V}$, but also slightly by its linear impact on the estimated circuit oxygen volume change $\widehat{V}_{O2,c}$, that appears affinely in the term $\widehat{V}_{O2,m}$. An error in the measurement thus has an affine impact on $\widehat{V}_{N2}$, with an induced error that is less than proportional to the error in m.

Since we deal only with differential measurements (between t = 0 and subsequent t > 0), taring error can be neglected, and the amount of drift of the load cell over the considered measurement durations is negligible.

Circuit temperature (T_c)

Error in the circuit temperature estimate $\widehat{T}_c$ propagates to $\widehat{V}_{N2}$ the same way as error in m, with a sub-proportional impact.

Letting $T_c = \widehat{T}_c + \varepsilon$, we have a relative error factor $T_c/\widehat{T}_c = 1 + \varepsilon/T_c$. Even with an over-estimation of this error, with a value as high as $\varepsilon = 5$ °C, the relative error in $\widehat{T}_c$ in will lie well below 2%.

Oxygen partial pressure (P_{02})

By far the dominating error source in our setup is the discrepancy between assumed and actual P_{02}. There are two contributors to this error: deviation between measured P_{02} and setpoint, and deviations between measured P_{02} and actual P_{02}.

In our estimator, an average $\overline{P}_{O2}$ is used to estimate the oxygen metabolic rate, $\hat{V}_{O2,m}$, as described in Section "*Oxygen metabolism ($V_{O2,m}$)*". This mitigates the impact of deviation between measured P_{02} and setpoint. However, any P_{02} measurement error linearly impacts $\widehat{V}_{O2,c}$, and thus has an affine impact on $\widehat{V}_{N2}$.

To quantify this error, we consider Equation 8 and note that a multiplicative error ε contributes with an error:

$$\hat{V}_{N_2}(t) - V_{N_2}(t) = \bar{P}_{O_2}\Big((1+\epsilon) - 1\Big)\frac{T_0}{P_0\hat{T}_c}\frac{m_f(t_2) - m_f(t_1)}{t_2 - t_1}\,t$$

Eq.9

We see directly from Equation 9 that the relative error is simply:

$$\frac{\hat{V}_{N_2} - V_{N_2}}{V_{N_2}} = \epsilon$$

Eq.10

To gauge the corresponding absolute error, we note that the largest error will occur at the end of a measurement series, where t = 25 min. With a representative mass change of 1 kg and the worst-case (largest) $\overline{P}_{O2} = 1.3$ bar we would then have:

$$\hat{V}_{N_2}(t) - V_{N_2}(t) = \epsilon \cdot 1.3\ \frac{1}{1.01325}\ \frac{273.15}{273.15 + 37}\ \frac{25}{20} \approx 1.41\,\epsilon$$

Eq.11

Thus, in this case, a multiplicative error of 10 % would result in a 0.14 L error in the estimated total V_{N2} of 1 L.

Unaccounted for volume changes (V_d + V_O + V_b – V_l)

Any unaccounted volume change will contribute directly to an error in $\widehat{V}_{N2}$, as evident from Equation 1. Visual bubble monitoring and lack of BCD adjustments make it possible for us to neglect V_b and V_l. The two occasions of ADV diluent addition have been manually compensated for, as described in Section "*Diluent injection (V_d)*".

The remaining error source is the volume change V_O of unaccounted gases other than oxygen and nitrogen, as already discussed in Section "*Influence of other gases (Vo)*". As mentioned, this source propagates directly to the estimate $\widehat{V}_{N2}$. However, as long as the volume of unaccounted gases is constant, the corresponding volume change V_O, cf. Section "*Suspended mass (m)*".

***Footnote:** Appendix B is available online on our website https://www.dhmjournal.com/index.php/journals?id=417

Table 2
Characteristics of participants; BMI – body mass index

ID	Sex	Age (Years)	Weight (kg)	Height (cm)	BMI (kg·m^{-2})
1	Male	48	83	182	25
2	Male	47	83	180	25

Figure 4
Change in V_{N2} STP during the bottom and deco stage of each dive; dives by Diver 1 are shown in blue and by Diver 2 in red

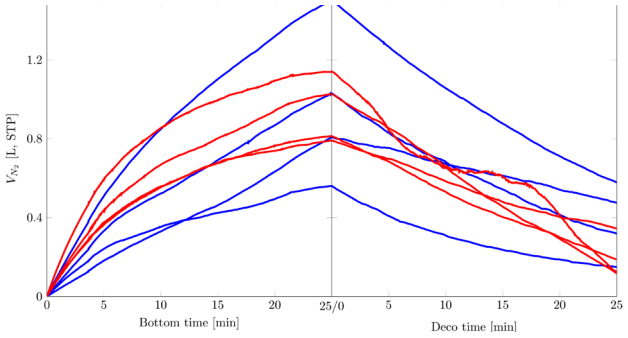


Table 3
Bottom phase nitrogen uptake, decompression phase washout and the difference thereof (L, STP), reported for each of eight dives conducted by the two divers; last row shows mean and standard deviation (SD) across all dives

Dive	Diver	Bottom V_{N2}	Deco V_{N2}	Sum V_{N2}
1	1	1.50	-0.93	0.58
2	2	1.14	-1.02	0.13
3	1	1.03	-0.71	0.32
4	2	0.79	-0.45	0.34
5	1	0.56	-0.41	0.15
6	2	1.03	-0.91	0.12
7	1	0.81	-0.33	0.47
8	2	0.81	-0.63	0.19
Mean (SD)		0.96 (0.29)	-0.67 (0.26)	0.29 (0.17)

Results

Two divers (Table 2), completed four dives each, with a bottom phase at 250 kPa (2.5 bar) and a decompression phase at either 130 kPa (1.3 bar) or 140 kPa (1.4 bar) (Diver 1: one dive at 1.4 bar; Diver 2: three dives at 1.4 bar). Uptake and washout of nitrogen from each dive exhibited distinct curves, with intra- and interindividual variability comparable to findings from previous inert gas kinetic measurement techniques,[4,6,8,10] (see Figures 3 and 4).

Mean N_2 uptake during the bottom phase was 0.96 L (SD 0.29), and mean washout during decompression was 0.67 L (SD 0.26), see Figures 3 and 4 and Table 3. The figure and table ordering are identical.

The nitrogen loading volumes, obtained from load cell measurements of buoyancy differences during the 25 min bottom and 25 min decompression phases, were consistent with physiologically expected quantities.[14,15]

According to Gabler-Smith et al.,[14] the solubility of nitrogen in human adipose tissue is approximately $\alpha = 61\ 10^{-2}$ mL gas per mL solvent under standard physiological temperature (37°C) and pressure (1 bar). Using the ideal gas law, Henry's constant is given by $k_H = \alpha/(RT)$. Substituting R = 0.08314 L bar^{-1}mol^{-1} ^{o}K^{-1} and T = 310.15 ^{o}K yields $K_{H,a}$ = 2.37·10^{-3} mol·bar^{-1}L^{-1} for adipose tissue. For lean tissue, the corresponding value is $k_{H,l}$ = 4.7·10^{-4} mol·bar^{-1} L^{-1}.[15]

Assuming a total body mass of m = 83 kg, being the weight of both the divers, with approximate adipose fraction F_a = 25%. Transitioning from breathing air at the surface to a nitrox mixture with P_{O2} = 0.5 bar at a depth corresponding to 2.5 bar total pressure, the diver is expected to absorb 1.78 L of nitrogen before reaching equilibrium. The measured buoyancy differences during 25 min bottom and 25 min decompression phases thus aligned with theoretical predictions.

Power analysis (with α = 0.05 and 80% power) indicated that the minimal detectable mean difference (MDD) in V_{N2}

uptake and washout for $n = 8$ dives was 0.28 L, and 0.26 L, for bottom and deco phase, respectively.

Discussion

MAIN FINDINGS

We present a novel method for quantifying nitrogen (inert gas) uptake and washout during submersion. Evaluation in human subjects demonstrated volumes consistent with expected physiological ranges.[2,4,8,15] The approach is straightforward to implement but currently requires a controlled environment and restricts diver movement during measurement. Furthermore, the method relies on no inert gas being injected into the breathing circuit and no gas leakage during ongoing measurement.

IMPLICATIONS

The presented technique enables quantification of inter- and intraindividual variability in inert gas uptake and washout. The method may offer new insights into how uptake and washout vary with individual covariates such as body composition, age or genotype, or different physiological states such as thermal exposure, workload, or hydration.

With sufficient data, the method could potentially help elucidate correlations between inert gas uptake/washout and DCS risk. However, such applications require extensive datasets across diverse dive profiles with known DCS outcomes.

STRENGTHS AND LIMITATIONS

The proposed setup is considerably simpler than existing methods for measuring inert gas kinetics underwater.[11,12] Its data processing pipeline effectively suppresses non-physiological fluctuations. Since these eight dives were the first using this system, bias from prior calibration or adjustment dives is avoided.

The main limitation is that no dive was performed using a nitrogen partial pressure identical to that of air at 1 bar, which would be expected to yield zero washout and zero uptake of nitrogen. Such a reference condition would have provided a stable baseline for detecting potential system drift. Additional limitations include the small sample size and the use of two decompression depths, which complicate direct comparison of outcomes. As noted in the error analysis, deviations from the PO_2 setpoint, arising from imperfect reference tracking or sensor drift, constitute the dominant source of error. Intermittent calibration using sampled gas analysis, with subsequent correction of logged PO_2 values, could mitigate the latter. Incorporation of temperature monitoring within the breathing circuit would further strengthen performance under variable thermal conditions.

Conclusions

We introduce a novel method for quantifying inert gas during submersion, demonstrating that it can achieve physiologically consistent results with adequate precision and accuracy in humans. The approach has potential to enable systematic quantification of inert gas data during diving, thereby advancing understanding of decompression physiology and the mechanisms underlying decompression sickness.

References

1 Brubakk AO, Neuman TS, editors. Bennett and Elliott's physiology and medicine of diving. 5th ed. Edinburgh: Saunders; 2003.

2 Balldin UI. Effects of ambient temperature and body position on tissue nitrogen elimination in man. Aerosp Med. 1973;44:365–70. PMID: 4694843.

3 Balldin UI, Lundgren CE. Effects of immersion with the head above water on tissue nitrogen elimination in man. Aerosp Med. 1972;43:1101–8. PMID: 5076612.

4 Pendergast DR, Senf CJ, Fletcher MC, Lundgren CE. Effects of ambient temperature on nitrogen uptake and elimination in humans. Undersea Hyperb Med. 2015;42:85–94. PMID: 26094308.

5 Lundgren CE, Eckhardt LG, Senf CJ, Bowdwin MR, Pendergast DR. Negative pressure breathing increases cardiac output and nitrogen elimination in seated subjects. Undersea Hyperb Med. 2013;40:403–10. PMID: 24224284.

6 Pendergast DR, Senf C, Lundgren CE. Is the rate of whole-body nitrogen elimination influenced by exercise? Undersea Hyperb Med. 2012;39:595–604. PMID: 22400450.

7 Balldin UI. The preventative effect of denitrogenation during warm water immersion on decompression and decompression sickness in man. First Annual Scientific Meeting of the European Undersea Baromedical Society. 1973:239–43.

8 Plogmark O, Silvanius M, Olsson M, Hjelte C, Ekström M, Frånberg O. Measuring whole body inert gas wash-out. Diving Hyperb Med. 2023;53:321–6. doi: 10.28920/dhm53.4.321-326. PMID: 38091591. PMCID: PMC10944667.

9 Gerth WA VR, Leatherman NE. Whole-body nitrogen elimination during oxygen prebreathing and altitude decompression sickness risk. The Thirty-Eighth Undersea and Hyperbaric Medical Society Workshop. 1989:147–51.

10 Dick AP, Vann RD, Mebane GY, Feezor MD. Decompression induced nitrogen elimination. Undersea Biomed Res. 1984;11:369–80. PMID: 6535313.

11 Sundblad P, Frånberg O, Siebenmann C, Gennser M. Measuring uptake and elimination of nitrogen in humans at different ambient pressures. Aerosp Med Hum Perform. 2016;87:1045–50. doi: 10.3357/AMHP.4680.2016. PMID: 28323592.

12 Kindwall EP, Baz A, Lightfoot EN, Lanphier EH, Seireg A. Nitrogen elimination in man during decompression. Undersea Biomed Res. 1975;2:285–97. PMID: 1226586.

13 Natoli MJ, Vann RD, Gerth WA. Nitrogen uptake during air diving. Final technical report (Contract No. N00014-91-J-1763). Durham, NC: Duke University Medical Center; 1994.

14 Gabler-Smith MK, Westgate AJ, Koopman HN. Microvessel density, lipid chemistry and N2 solubility in human and pig

adipose tissue. Undersea Hyperb Med. 2020;47:1–12. doi: 10.22462/01.03.2020.25. PMID: 32176941.

15 Gabler-Smith MK, Westgate AJ, Koopman HN. Fatty acid composition and N(2) solubility in triacylglycerol-rich adipose tissue: the likely importance of intact molecular structure. J Exp Biol. 2020;223(Pt 5):jeb216770. doi: 10.1242/jeb.216770. PMID: 32001545.

Acknowledgements

We gratefully acknowledge the support from the Swedish Armed Forces Diving and Naval Medicine Center.

Conflicts of interest and funding

Funding was provided through unrestricted grants from the Swedish Defence Materiel Administration (430919–LB967593). Kristian Soltesz is a member of the ELLIIT Strategic Research Area at Lund University. The authors declare no conflicts of interest relevant to this work.

Submitted: 6 March 2025
Accepted after revision: 12 March 2026

Real-world experience of transoesophageal echocardiography for detection of clinically significant persistent foramen ovale

Peter Wilmshurst[1], Matthew Pearson[2], Kevin Walsh[3,4], W Lindsay Morrison[5]

[1] *United Kingdom Diving Medical Committee (UKDMC), United Kingdom*
[2] *Shrewsbury and Telford NHS Hospital Trust, Shrewsbury SY3 8XQ, United Kingdom*
[3] *University College Dublin, Dublin, Ireland*
[4] *Our Lady's Hospital for Sick Children, Dublin 12, Ireland*
[5] *Liverpool Heart and Chest Hospital, Liverpool L14 3PE, United Kingdom*

Corresponding author: *Dr Peter Wilmshurst United Kingdom Diving Medical Committee, United Kingdom*
peter.wilmshurst@doctors.org.uk

Keywords
Bubbles; Decompression sickness; Diving; Echocardiography; Persistent foramen ovale; Stroke; Transcatheter closure

Abstract
(Wilmshurst P, Pearson M, Walsh K, Morrison WL. Real-world experience of transoesophageal echocardiography for detection of clinically significant persistent foramen ovale. Diving and Hyperbaric Medicine. 2026 30 June;56(2):148–153. doi: 10.28920/dhm56.2.148-153. PMID: 42290574.)
Introduction: Transoesophageal echocardiography (TOE) is claimed to be the investigation of choice for detecting a persistent foramen ovale (PFO) with almost 100% diagnostic accuracy. If true, TOE would detect all large/clinically significant PFOs.
Methods: Retrospective analysis to determine the sensitivity of TOE for detection of clinically significant PFOs. Patients were from a consecutive series of 150 patients who had transcatheter closure of a PFO following events attributed to paradoxical embolism (decompression sickness or stroke). In each patient, transthoracic echocardiogram with bubble contrast showed a clinically significant atrial right-to-left shunt. The data reported are from the sub-group of the 150 patients with a clinically significant PFO who also had a TOE performed in other hospitals.
Results: Twenty seven of 150 consecutive patients had a total of 31 TOEs performed at 22 United Kingdom regional cardiac centres. TOE failed to detect a PFO in 17 of the 27 patients. Four patients had a TOE on two separate occasions and in each case both of the TOEs failed to show a PFO. TOE gave a false negative test in 21 of 31 investigations (sensitivity 32%). The mean PFO diameter was 9.4 mm (median 9 mm, range 5–16 mm) in the 21 patients in whom balloon sizing was performed and 9.8 mm (median 10 mm, range 5–16 mm) in the 13 patients in whom balloon sizing was performed and a TOE failed to show a PFO.
Conclusions: These finding demonstrate that the precision of TOE for detecting a PFO in real world clinical practice is considerably lower than generally believed.

Introduction

The law of the conservation of ignorance: A false conclusion once arrived at and widely accepted is not easily dislodged and the less it is understood, the more tenaciously it is held.
Georg Cantor

Persistent foramen ovale (PFO) are present in approximately one quarter of adults.[1] Most PFOs are small,[1] but large PFOs allow significant right-to-left shunting, which can facilitate paradoxical thromboembolism and stroke,[2] and paradoxical gas embolism and some types of decompression sickness.[3–6] Therefore it is important that any test for a PFO accurately identifies those that are large enough to present a significant risk of paradoxical embolism of thrombus or bubbles.

Intuitively, transoesophageal echocardiography (TOE), which provides the best images of the atrial septum, might be anticipated to be superior for identifying a PFO than transthoracic echocardiography with bubble contrast or transcranial Doppler with bubble contrast. The intuitive superiority of TOE has resulted in it being readily accepted by cardiologists as the clinical investigation of choice for diagnosis of a PFO.[7]

It is commonly claimed that TOE has almost 100% predictive accuracy for detecting a PFO. If that claim were true one would anticipate that in specific groups of patients TOE would identify PFOs at rates comparable to post mortem examinations or cardiac catheterisation in those groups. For example, one would expect that TOE would identify a PFO in approximately a quarter of healthy adults, which is approximately the prevalence reported by Hagen et al. in their large series of post mortem findings in people without heart disease.[1] Similarly in groups of individuals with a high clinical probability of paradoxical embolism, such as divers with associated forms of decompression sickness, it would be anticipated that TOE would detect a PFO in many more than 25% of the group. Other methods of detecting a PFO should be judged against the same criteria.

There are many publications that report comparison of the rates of diagnosis of PFO using TOE and transthoracic echocardiography with bubble contrast. Discordant results are invariably attributed to errors in the transthoracic studies without adequate justification.

There are only a few reports that validated the finding of a PFO by echocardiography against an objective standard, such as post mortem examination or cardiac catheterisation.[8–11] Those studies were performed decades ago when echocardiography imaging was poor compared with current equipment – one even used M-mode echocardiography.[11] Each study was small – the number of PFOs detected was between nine and 24. Few patients in these studies had imaging performed because of suspected paradoxical embolism. Instead, some studies consisted almost entirely of patients with structural/congenital heart disease (such as Ebstein's anomaly and pulmonary stenosis) and other diseases likely to increase right atrial pressure and therefore increase right-to-left shunting if a PFO was present.[8,10]

Schneider and colleagues compared the findings of PFO detected by TOE in intensive care patients against post mortem examination of some of those who died by two pathologists.[9] They reported that of 35 patients who died, a post mortem examination showed that nine had a PFO, although in one case the two pathologists initially disagreed about the presence of a PFO. The authors reported that TOE had 100% sensitivity and specificity for detecting a PFO, although in two of nine diagnosed as having a PFO only a single bubble was seen in the left heart after contrast injection and another had no left heart bubbles but colour flow Doppler showed a jet across the atrial septum. It is unclear whether post mortem examinations were performed blind to the result of TOE. The study was small and not typical of situations when tests for a PFO are performed because 18 patients, including five with a PFO, had their TOE when on mechanical ventilation, which increases shunting across a PFO.[12,13]

In contrast, we report real-world experience of the ability of TOE to detect or confirm the presence of a clinically significant PFO in patients with paradoxical embolism (shunt-mediated decompression sickness and ischaemic stroke likely to be the result of paradoxical thromboembolism). In each patient, a clinically significant atrial right-to-left shunt was demonstrated by transthoracic echocardiography with bubble contrast and the presence of a PFO was confirmed during transcatheter closure.[14]

It is important that this is not a comparison of TOE and transthoracic echocardiogram with bubble contrast. The latter was simply the test used to detect the clinically significant PFOs. We are reporting the sensitivity of TOE in detecting a clinically significant PFO with the finding of a PFO at the closure procedure as the 'gold standard'.

Methods

The patients described are a sub-group of a consecutive series of 150 patients who had a PFO that was closed using a transcatheter device after our transthoracic echocardiogram with bubble contrast showed a significant atrial right-to-left shunt. The sub-group were all of those who had transoesophageal echocardiography at other hospitals in order to detect a PFO before we performed our transthoracic echocardiogram with bubble contrast or to confirm the presence of a PFO after we performed our test.

In patients thought to have had stroke or arterial thromboembolism as a result of paradoxical embolism and divers who had decompression sickness as a result of paradoxical gas embolism, we diagnosed atrial and pulmonary right-to-left shunts using transthoracic echocardiography with bubble contrast using a standard protocol.[5] Based on this test alone, transcatheter closure of their shunt was one option we offered patients with a significant atrial shunt. We did not perform a TOE until the closure procedure, at which stage a TOE was used to differentiate an atrial septal defect from a PFO and used to ensure optimal positioning of the occlusion device. The TOE findings during the closure procedure are not included in this analysis because in those cases we were not performing a diagnostic test and because in many cases a guide wire was already positioned across the atrial defect using X-ray fluoroscopy before the TOE probe was positioned.

During the period in which we performed transcatheter closure of a PFO in the 150 consecutive patients in this report, ten additional patients were found to have an atrial right-to-left shunt on our transthoracic echocardiogram with bubble contrast, but at the time of their closure procedure an atrial septal defect was found using TOE. They are not included in the 150 patients.

The majority of the 150 patients with a PFO had decompression sickness and were seen in a specialist diving medicine clinic which took referrals from all parts of the United Kingdom. The clinic assessed divers that had decompression illness to determine whether they had shunt-mediated decompression sickness or another cause for dysbaric illness, such as arterial gas embolism secondary to pulmonary barotrauma. Each diver had investigations, including transthoracic echocardiography with bubble contrast, before the referral letter was read or a history was taken to avoid bias in interpretation of the results.[5,6] The transthoracic echocardiography with bubble contrast was performed according to a set protocol.[5,6] When a clinically significant right-to-left shunt (atrial or pulmonary) was detected, divers were counselled about the options for avoiding recurrence of decompression sickness – namely to stop diving, modify their dives or have closure of a PFO or atrial septal defect.[15] Some of those found to have a clinically significant atrial shunt requested transcatheter closure.

A small number of the 150 patients had an ischaemic stroke that was thought to be the result of paradoxical thromboembolism with other causes of stroke excluded. If transthoracic echocardiography with bubble contrast detected a significant atrial shunt they were offered transcatheter closure without a TOE being performed until the procedure.

Although we did not perform a TOE in patients with decompression sickness or suspected paradoxical thromboembolism until they had a closure procedure, 27 of the 150 patients who had transcatheter closure of their PFO did have a TOE at other hospitals – all United Kingdom regional cardiac centres. Four patients each had two TOEs. From correspondence we obtained the results of each TOE in each of the patients in so far as we knew whether or not the test had shown a PFO, but we did not obtain the details of protocols used in each case. Specifically, we do not know which transoesophageal echocardiograms included intravenous injection of bubble contrast.

Four divers had one or more TOEs in regional cardiac centres near their homes in order to see whether they had a PFO before they were referred to our clinic. In the remaining 23 patients the reason for the TOE was that the patients' local NHS commissioners (who authorise payments for out-of-area NHS treatments) had permitted us to see divers in the specialist clinic, but they insisted that if a significant shunt was detected, before a closure procedure could be performed, an opinion should be obtained from their local cardiologists. In every case the local cardiologist arranged for the diver to have a TOE. In each of the 23 patients the TOE operator was aware that we had previously demonstrated a significant atrial right-to-left shunt using transthoracic echocardiography with bubble contrast. A consultant cardiologist performed each TOE. Several of the cardiologists had taught on TOE courses and published research on TOE.

When a TOE confirmed a PFO the commissioners gave permission for transcatheter closure. When a TOE failed to confirm a PFO, we were able to use the recorded images from our transthoracic echocardiograms with bubble contrast to persuade the commissioners to permit cardiac catheterisation with a view to proceeding to a transcatheter closure if a large PFO was confirmed. Some of those closure procedures were by us and we measured the diameter of PFOs using balloon sizing.[16] Some closure procedures were in other regional cardiac centres, and we obtained details of those procedures, but balloon sizing was not always performed.

Results

Of 150 consecutive patients who had a transcatheter closure of a PFO, 27 had a TOE at 22 regional cardiac centres. Each TOE was performed with the intention of determining whether there was an atrial right-to-left shunt because paradoxical embolism was suspected. Twenty-five patients were divers with decompression sickness with characteristics (clinical manifestations and latencies) in keeping with shunt-mediated decompression sickness, but on a separate occasion one also had cerebral infarction 24 hours after a long-haul flight. Two other patients had a history of cerebral infarction only. The three patients with cerebral infarction had their stroke before age 45, had a predisposition to venous thrombosis and had no other medical condition that might predispose to stroke.

Table 1 shows the results of the echocardiographic investigations and cardiac catheterisation in the patients. Using transthoracic echocardiography with bubble contrast, we graded the shunt as large in 22 patients and medium in one patient during normal respiration. In the other four patients there was a large shunt with release of a Valsalva manoeuvre.

TOE failed to detect a PFO in 17 of 27 (63%) patients with a clinically significant PFO confirmed at the closure procedure. In those cases, the correspondence from the doctor who performed the TOE said that the TOE failed to demonstrate or confirm the presence of PFO.

Four of the patients (numbers 1, 18, 23 and 27) had negative TOE on two occasions. Two divers, who had decompression sickness, returned to diving after a TOE that was reported to show no PFO and they each had a second episode of decompression sickness, which resulted in them having a second TOE that was also negative, before our transthoracic echocardiogram with bubble contrast showed a significant right-to-left shunt. Two patients had a negative TOE before our transthoracic echocardiogram with bubble contrast showed an atrial shunt and that finding lead to a repeat TOE that was also reported to be negative.

Therefore 21 of 31 (68%) TOEs gave a false negative test even though the TOEs were performed by experienced operators specifically looking for a cause of paradoxical embolism. In the majority of cases, we had demonstrated an atrial right-to-left shunt using transthoracic echocardiography with bubble contrast before the TOE and the operators were aware of the findings. The sensitivity of TOE for PFO detection was 32%.

Of the 10 patients in whom a PFO was detected by TOE, the size was considered to be medium or large in three, small in two and there was no comment about size in five.

In six patients the diameter of the PFO was not measured during the closure procedure, but a cardiac catheter passed easily across the PFO. The mean diameter of PFOs was 9.4 mm (median 9 mm, range 5–16 mm) in the 21 patients in whom balloon sizing was performed and 9.8 mm (median 10 mm, range 5–16 mm) in the 13 patients in whom balloon sizing was performed and a TOE failed to show a PFO.

Table 1
Echocardiographic and cardiac catheterisation findings in the patients; PFO – persistent (patent) foramen ovale

Patient	Transthoracic contrast echocardiography findings	Transoesophageal echocardiography findings	Diameter of PFO at closure
1	large shunt at rest	no atrial shunt x 2	11 mm
2	large shunt at rest	no atrial shunt	16 mm
3	medium shunt at rest	small PFO	9 mm
4	large shunt at rest	no atrial shunt	13 mm
5	large shunt at rest	no atrial shunt	easily crossed but not sized
6	large shunt with a Valsalva	medium-large PFO	10 mm
7	large shunt at rest	PFO – no comment on size	9 mm
8	large shunt at rest	no atrial shunt	7 mm
9	large shunt at rest	PFO – no comment on size	6 mm
10	large shunt at rest	PFO – no comment on size	11 mm
11	large shunt at rest	no atrial shunt	easily crossed but not sized
12	large shunt at rest	no atrial shunt	easily crossed but not sized
13	large shunt at rest	no atrial shunt	10 mm
14	large shunt at rest	PFO – no comment on size	easily crossed but not sized
15	large shunt at rest	no atrial shunt	easily crossed but not sized
16	large shunt at rest	no atrial shunt	13 mm
17	large shunt at rest	no atrial shunt	9 mm
18	large shunt at rest	no atrial shunt x 2	9 mm
19	large shunt with a Valsalva	no atrial shunt	8 mm
20	large shunt at rest	no atrial shunt	10 mm
21	large shunt at rest	large PFO	11mm
22	large shunt at rest	PFO – no comment on size	7 mm
23	large shunt with a Valsalva	no atrial shunt x 2	5 mm
24	large shunt at rest	small PFO	7 mm
25	large shunt with a Valsalva	no atrial shunt	6 mm
26	large shunt at rest	large PFO	easily crossed but not sized
27	large shunt at rest	no atrial shunt x 2	10 mm

Discussion

Each of the 27 patients described had a significant atrial right-to-left shunt on transthoracic echocardiography with bubble contrast and was found to have a large PFO which was closed using a transcatheter technique.

When measured (i.e., in 21 of 27 patients), PFO diameters (mean 9.4 mm and median 9 mm) were comparable to the diameters of atrial defects (mean 9.9 mm and median 10 mm) in a series of 200 divers (189 had a PFO and 11 had a secundum atrial septal defect), who had shunt-mediated decompression sickness and later had transcatheter closure.[16]

To put these diameter measurements in context, a large post-mortem study showed 27.3% of the normal population have a PFO with a median diameter of 5 mm and mean diameter 4.9 mm (SD 2.6).[1] A PFO diameter of 9 mm or greater was present in 2.6% of normal adult hearts.[1] In contrast 14 of 21 (67%) of the patients we reported had a PFO diameter of 9 mm or greater. A PFO diameter of 10 mm or greater was present in 1.3% of normal adult hearts,[1] but was present in 10 of 21 (48%) of our patients.

Despite the large diameters of the PFOs in our patients who had paradoxical embolism, the sensitivity of TOE for PFO detection in was only 32%. It is likely that TOE would have a lower sensitivity for detecting smaller PFOs. These findings suggest that contrary to what is commonly claimed, in real-world clinical practice TOE is not an accurate test for PFO detection even when performed by experienced operators.

The TOEs were performed in 22 regional cardiac centres by a number of consultant cardiologists who used their individual protocols in order to detect or confirm the presence of a PFO in patients who had paradoxical embolism. As far as

we are aware the TOEs were not performed using a standard protocol.

We do not know why the sensitivity of TOE for demonstrating a PFO was low, but we postulate that the widely held belief in the infallibility of TOE may have resulted in poor attention to technique. We do not know whether bubble contrast was injected and if it was, we do not know the quality of the opacification obtained. Inability to perform provocative manoeuvres, such as Valsalva manoeuvres, as a result of sedation is unlikely to be a factor, because 23 of 27 of the patients had significant right-to-left shunts during transthoracic echocardiography with bubble contrast when breathing normally.

We do not know the sensitivity or specificity of our transthoracic echocardiography with bubble contrast protocol for detecting an atrial right-to-left shunt because we do not perform right heart catheterisation in patients other than those having a closure procedure. Nor did we assess sensitivity of transthoracic echocardiography with bubble contrast in detection of PFOs that were identified using transoesophageal echocardiography and confirmed during a closure procedure. We emphasise that we were not comparing the diagnostic accuracy of the two echocardiographic tests.

However, when performing transthoracic echocardiography with bubble contrast for detection of a right-to-left shunt, we insist on a rigorous protocol and perform the test blind to history.[4–6] As a result, we consistently reported prevalence rates of PFOs in normal control subjects of 24–27.6%.[4-6] This is similar to the 27.3% incidence in normal hearts at post-mortem.[1] Using our protocol in normal control subjects, only 4.9% had a large shunt with normal respiration and a further 2.4% had a large shunt with multiple contrast injections and release of a Valsalva manoeuvre, whereas 4.9% had a medium shunt and 15.4% had a small shunt.[6]

In addition, using our protocol in divers with decompression illness, we detected all those in whom a significant right-to-left shunt is likely to account for the symptoms with provocative dive profiles or pulmonary barotrauma explaining other episodes.[5,6] As a result in divers with cutaneous and neurological decompression sickness the prevalence of large right-to-left shunts was between seven and ten times the prevalence in control divers.[5,6]

During 30 years, in which hundreds of patients judged to have significant atrial shunts using our protocol of transthoracic echocardiography with bubble contrast went forward to a closure procedure without a TOE, only two were found to be unsuitable for the procedure because they did not have either a PFO or secundum atrial septal defect. Both had right-to-left shunts, but one had a sinus venosus defect that was subsequently corrected surgically. The other, who was the fifth patient we investigated, had a large pulmonary arteriovenous malformation with high flow so that left atrial opacification was seen in one beat after right heart opacification. The defect was occluded because of its size.

Our experience cannot be extrapolated to the real-world accuracy of transthoracic echocardiography with bubble contrast for detecting a PFO when operators do not adhere to a protocol with confirmed accuracy.

LIMITATIONS

We assessed the sensitivity of transoesophageal echocardiography for detecting clinically significant PFOs that were proven to be present during a closure procedure as the 'gold standard'. Transthoracic echocardiograms used to detect clinically significant PFOs were performed according to a clear protocol, but we suspect that there was no uniformity in performance of transoesophageal echocardiograms and we do not know how many incorporated intravenous injection of bubble contrast. An alternative study that selected patients who had a large PFO detected by transoesophageal echocardiography would probably find that transthoracic echocardiography with bubble contrast performed without a set protocol by several different cardiologists had sensitivity less than 100%.

Nevertheless we conclude that these finding demonstrate that the precision of TOE for detecting a PFO in real world clinical practice is considerably lower than generally believed.

References

1 Hagen PT, Scholz DG, Edwards WD. Incidence and size of patent foramen ovale during the first 10 decades of life: an autopsy study of 965 normal hearts. Mayo Clin Proc. 1984;59:17–20. doi: 10.1016/s0025-6196(12)60336-x. PMID: 6694427.

2 Webster MWI, Chancellor AM, Smith HJ, Swift DL, Sharpe DN, Bass NM, et al. Patent foramen ovale in young stroke patients. Lancet. 1988:2:11–2. doi: 10.1016/s0140-6736(88)92944-3. PMID: 2898621.

3 Moon RE, Camporesi EM, Kisslo JA. Patent foramen and decompression sickness in divers. Lancet. 1989;1:513–4. doi: 10.1016/s0140-6736(89)90064-0. PMID: 2564057.

4 Wilmshurst PT, Byrne JC, Webb-Peploe MM. Relation between interatrial shunts and decompression sickness in divers. Lancet. 1989;2:1302–6. doi: 10.1016/s0140-6736(89)91911-9. PMID: 2574256.

5 Wilmshurst P, Bryson P. Relationship between the clinical features of neurological decompression illness and its causes. Clin Sc (Lond). 2000;99:65–75. PMID: 10887059.

6 Wilmshurst PT, Pearson MJ, Walsh KP, Morrison WL. Relationship between right-to-left shunts and cutaneous decompression illness. Clin Sci (Lond). 2001;100:539–42. PMID: 11294694.

7 Alp N, Clarke N, Banning AP. How should patients with patent foramen ovale be managed? Heart. 2001;85:242–4. doi: 10.1136/heart.85.3.242. PMID: 11179251. PMCID: PMC1729659.

8 Chen WJ, Kuan P, Lien WP, Lin FY. Detection of patent

foramen ovale by contrast transoesophageal echocardiography. Chest. 1992;101:1515–20. doi: 10.1378/chest.101.6.1515. PMID: 1600767.

9 Schneider B, Zienkiewicz T, Jansen V, Hofmann T, Noltenius H, Meinertz T. Diagnosis of patent foramen ovale by transesophageal echocardiography and correlation with autopsy findings. Am J Cardiol. 1996;77:1202–9. doi: 10.1016/s0002-9149(96)00163-4. PMID: 8651096.

10 Dubourg O, Bourdarias JP, Farcot JC, Gueret P, Terdjman M, Ferrier A, et al. Contrast echocardiographic visualization of cough-induced right to left shunts through a patent foramen ovale. J Am Coll Cardiol. 1984;4:587–94. doi: 10.1016/S0735-1097(84)80106-0. PMID: 6470340.

11 Kronik G, Mösslacher H. Positive contrast echocardiography in patients with patent foramen ovale and normal right heart hemodynamics. Am J Cardiol. 1982;49:1806–9. doi: 10.1016/0002-9149(82)90263-6. PMID: 7081065.

12 Vavlitou A, Minas G, Zannetos S, Kyprianou T, Tsagourias M, Matamis D. Hemodynamic and respiratory factors that influence the opening of patent foramen ovale in mechanically ventilated patients. Hippokratia. 2016;20:209–13. PMID: 29097887. PMCID: PMC5654438.

13 Lemaire F, Richalet JP, Carlet J, Brun-Buissan C, MacLean C. Post-operative hypoxemia due to opening of a patent foramen ovale confirmed by a right atrium-left atrium pressure gradient during mechanical ventilation. Anesthesiology. 1982;57:233–6. doi: 10.1097/00000542-198209000-00016. PMID: 7051902.

14 Walsh KP, Wilmshurst PT, Morrison WL. Transcatheter closure of patent foramen ovale using the Amplatzer Septal Occluder to prevent recurrence of neurological decompression illness in divers. Heart. 1999;81:257–61. doi: 10.1136/hrt.81.3.257. PMID: 10026348. PMCID: PMC1728953.

15 Smart D, Wilmshurst P, Banham N, Turner M, Mitchell SJ. Joint position statement on atrial shunts (persistent [patent] foramen ovale and atrial septal defects) and diving: 2025 update. South Pacific Underwater Medicine Society (SPUMS) and United Kingdom Diving Medical Committee (UKDMC). Diving Hyperb Med. 2025;55:51–5. doi: 10.28920/dhm55.1.51-55. PMID: 40090026. PMCID: PMC12043516.

16 Wilmshurst PT, Morrison WL, Walsh KP, Pearson MJ, Nightingale S. Comparison of the size of persistent foramen ovale and atrial septal defects in divers with shunt-related decompression illness and in the general population. Diving Hyperb Med. 2015;45:89–93. PMID: 26165530.

Acknowledgements

Dr W L Morrison died before the final version of this paper was written. After discussion with the editor, he was included as an author because he made a significant contribution to the care of patients and interpretation of the findings. He was involved in amending earlier drafts of the report, but did not see the final version of the paper. The conclusions did not alter between earlier drafts and the final version of the paper.

Funding and conflicts of interest: nil

Submitted: 21 November 2025
Accepted after revision: 4 April 2026

Technical report

Quantifying discrepancy between indicated and actual oxygen flow rates delivered by Comweld Ezi-flow low and standard flowmeters under hyperbaric conditions: a technical report

Yoav Aufgang[1,2], Bridget Devaney[1,3,4], Jason Chan[1,2], Ian Millar[1,4], Theo Tsouras[1]

[1] *Department of Intensive Care and Hyperbaric Medicine, Alfred Health, Melbourne, Australia*
[2] *School of Translational Medicine, Faculty of Medicine Nursing and Health Sciences, Monash University, Melbourne, Australia*
[3] *Emergency and Trauma Centre, Alfred Health, Melbourne, Australia*
[4] *School of Public Health and Preventive Medicine, Faculty of Medicine Nursing and Health Sciences, Monash University, Melbourne, Australia*

Corresponding author: *Dr Bridget Devaney, Department of Intensive Care and Hyperbaric Medicine, Alfred Health, 55 Commercial Road, Melbourne, VIC 3004, Australia*
ORCiD: *0000-0001-6521-418X*
b.devaney@alfred.org.au

Keywords
Flow dynamics; Gas flow; Hyperbaric environment; Hyperbaric oxygen treatment; Medical equipment

Abstract
(Aufgang Y, Devaney B, Chan J. Millar I, Tsouras T. Quantifying discrepancy between indicated and actual oxygen flow rates delivered by Comweld Ezi-flow low and standard flowmeters under hyperbaric conditions: a technical report. Diving and Hyperbaric Medicine. 2026 30 June;56(2):154–160. doi: 10.28920/dhm56.2.154-160. PMID: 42290575.)
Introduction: During clinical use of extracorporeal membrane oxygenation (ECMO) in hyperbaric conditions at our centre, upward titration of indicated sweep gas flow rates is required to maintain adequate CO_2 clearance. This project measured the impact of hyperbaric pressure on oxygen flow rates delivered by the Comweld Ezi-Flow flowmeters used in our centre.
Methods: Oxygen flow rates through Comweld Ezi-flow standard and low oxygen gas flowmeters were set at 101.3 kPa (1 atmosphere absolute [atm abs]) and then measured at intervals up to 284 kPa (2.8 atm abs) using a calibrated gas flow analyser, with cross verification against a small Douglas bag test type apparatus. During testing, the chamber was compressed and decompressed at a rate of 10 kPa·min^{-1}. Flow rates during chamber compression and decompression were compared.
Results: The indicated rate of oxygen gas flow through the unadjusted flowmeters changed minimally – typically rising by a maximum of half of the diameter of the indicator ball. The actual (volumetric) flow, tested across indicated flow rates from 3 to 12 L·min^{-1}, was consistently reduced by approximately 50% as the chamber pressure increased from 101.3 to 284 kPa (1 to 2.8 atm abs). A slightly smaller reduction was observed when assessing the low flowmeter across the same pressure range; reductions of 40.0 and 43.3% were demonstrated at 0.3 to 0.6 L·min^{-1} respectively. Differences in flow rates between compression and decompression were minor except at the very lowest flows.
Conclusions: At 284 kPa (2.8 atm abs), actual volumetric flow of oxygen through Comweld Ezi-Flow flowmeters is dramatically reduced and this needs appropriate compensation to ensure therapeutic aims are achieved.

Introduction

Patients receiving hyperbaric oxygen treatment (HBOT) vary from low acuity ambulant patients to critically unwell intensive care patients dependent on life support systems. Hyperbaric clinical and technical staff should have a clear understanding of the effects of raised pressure upon the function of healthcare technology used within the hyperbaric chamber and this includes equipment controlling and measuring gas flow.

The hyperbaric service at our centre has a rigorous process in place for the assessment and validation of equipment for use in hyperbaric conditions and has previously validated a number of medical devices for use in hyperbaric conditions. These include the HeartMate III left ventricular assist device, a pleural vacuum relief device for use with underwater seal drains, several syringe drivers, and more recently, a modified Maquet (Getinge) original series Rotaflow and Quadrox i-adult HMO 70000 (Quadrox) oxygenator.[1–6]

Although we have demonstrated successful delivery of HBOT to patients supported by both VA (veno-arterial) and VV (veno-venous) extracorporeal membrane oxygenation (ECMO) at our centre, much work remains to be done to assess, test and calibrate the function of various components

of multiple models of ECMO equipment. This paper reports upon the flowmeters used to provide 'sweep gas' to the ECMO oxygenator used to treat our first two hyperbaric ECMO (HECMO) patients.[5] Oxygen was the sweep gas used in both cases and was delivered by standard flow Comweld Ezi-flow flowmeter in one (adult) case, and low flow Comweld Ezi-flow flowmeter in the other (paediatric) case. Indicated flow was pragmatically titrated to carbon dioxide levels and acid base status, with an increase in indicated flow required during each HBOT session.

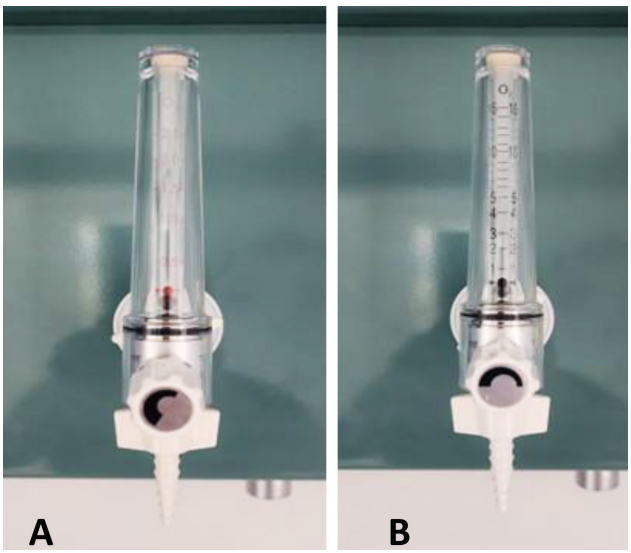
The aim of this project is to quantify the discrepancy between the Actual (or volumetric) gas flow delivered by the candidate flowmeters and the Set (indicated) flow, during hyperbaric versus ambient sea level atmospheric pressure conditions.

There are numerous designs of flowmeters with varying performance and considerably different responses to operation at pressures other than the standard or normal atmospheric conditions upon which calibration is usually based.[7,8] The hyperbaric chambers at our service have multiple modes for delivery of oxygen and these include delivery from Comweld Ezi-flow flowmeters (Figure 1), attached to Australian Standards-compliant medical gas outlets as in standard use around the hospital. These are pressure compensated Thorpe tube type devices with a variable cross section, constant taper flow indicator tube in which a 'ball type' bobbin lifts with increasing flow rate. Ezi-flow flowmeters have an internal and external cylinder and are of a pressure-compensated design with the flow control valve downstream of the flow-indicating cylinder. Such designs are intended to deliver stable gas flow independent of back pressure from the destination of the oxygen flow. Comweld specifies their flowmeters as having an accuracy of ± 5% with the reading to be taken from the centre of the ball.[9]

Comweld calibrate the performance of their Ezi-flow devices when supplied with oxygen at a pressure of 400 kPa. Australian Standards for medical gas pipeline installations call for gas supplies to have a nominal working pressure of 415 kPa at each medical gas outlet,[10] but outlet pressures typically fall slightly when gas is flowing and often vary somewhat between different outlets and depending upon varying total combined flow demands of adjacent outlets. The hyperbaric chambers in our service have specifically regulated medical gas supplies which deliver oxygen and medical air to in-chamber outlets at a nominal 415 kPa above the ambient interior pressure of the chamber.

When medical flowmeters are used at sea-level atmospheric pressure, the Actual flow delivered (volume of gas at ambient pressure per unit of time) should be essentially the same as the Set flow on the flowmeter. In non-standard conditions however, many factors can impact Actual gas flow compared with those measured at the calibration conditions used to determine the 'Indicated flow' of an uncorrected flowmeter. These factors include gas supply pressure, delivery pressure,

Figure 1
Comweld Ezi-flow low (A) and standard (R) flow oxygen flowmeters



water vapour, temperature and, if the wrong flowmeter is used, density differences between gases (e.g., oxygen has a density 1.106 x the density of medical air).[11,12] There are established formulae for calculating the expected Actual flow through control valves and variable area flowmeters at operating pressures, temperatures and gas densities other than those for which the flowmeter was calibrated. The relevant formula depends upon the configuration of the flowmeter – whether the valve is upstream or downstream of the flow indicator tube. In devices such as the Ezi-flow, the principal zone of pressure drop from gas supply pressure to delivery pressure is through the control valve, which is a modified needle valve located downstream of the indicator tube. Such valves perform as 'critical orifices' in which flow velocity emitting from the orifice is constrained to the speed of sound. As a result, a 'choked flow' condition applies where the mass flow through the orifice remains constant despite changes in downstream pressure, provided the upstream pressure remains constant.[13] For Ezi-flow devices, choked flow calculations should provide a reasonable estimate of expected performance under hyperbaric conditions.

In our situation, we would expect that mass flow delivered at hyperbaric pressures would increase in direct proportion to the increase in supply pressure from its starting point at sea level of 515 kPa / 5.08 atmospheres absolute [atm abs].[14] Mass flow is directly proportional to surface equivalent flow at designated standard temperature and pressure conditions such as 'standard temperature and pressure' (101.3 kPa and 0°C) or 'normal' conditions (101.3 kPa and 15°C). As hyperbaric pressurisation increases gas density in direct proportion to chamber pressure, the volumetric, or 'Actual' flow inside the chamber will be calculated by dividing the surface equivalent flow by the chamber pressure, assuming surface pressure conditions are close to 'normal' sea level conditions and in all cases using absolute pressure measures and not differential.

This can be described by the formula: *Q2 = [Q1 (P3 / P1)]/P2*
Where:

Q1 is the indicated or set flow at normal sea level atmospheric pressure,
Q2 is the predicted Actual flow in the chamber.
P1 is oxygen supply pressure at sea level atmospheric pressure,
P2 is the hyperbaric chamber pressure,
P3 is the oxygen supply pressure at chamber pressure P2.

With all pressure measurements being in atm abs (sea level = 1 atm abs).

Using these equations at 2.8 ATA Actual flow = [Indicated flow x (6.88 / 5.08)] / 2.8 or ~48% of Indicated flow.

This simplified choked flow formula is based upon an assumption of dry gas and no significant temperature change. We believe this simplification is reasonable as, in practice, the temperature of gas exiting a medical flowmeter will trend towards the temperature of the copper tubing supplying the medical gas outlet and the metal valve controlling flow. As a result, the flow changes resulting from relatively minor changes in absolute temperature are unlikely to be clinically significant (e.g., a 10°K increase in temperature would result in only a 3% change in flow).

It is also important to note that a flowmeter valve such as in the Ezi-flow is not a simple critical orifice such as occurs when gas flow is controlled by a hole of critical diameter drilled through an orifice plate. In the Ezi-flow control valve, one would expect some pressure drop to occur through the gas flow channels of the device, with the result that the pressure drop across the components that act as a 'critical orifice' will be expected to be somewhat less than the total pressure drop from supply to delivery.

Given that formulae for gas flow are expected to be only generally predictive, we considered it important to measure Actual flows with appropriate methods to enable clear guidance for clinical decision making in cases where flow rates are clinically important such as for patients receiving HECMO. This may be especially important for neonatal and paediatric patients for whom small proportional changes in flow may have greater physiological impact.[15,16]

Methods

HYPERBARIC SAFETY ASSESSMENT (IN-VITRO)

A test equipment safety assessment was performed prior to commencing analysis of flow rates under hyperbaric conditions. The flowmeter testing set-up involved the Comweld Ezi-Flow Oxygen flowmeters (model numbers 515800 and 515824); 0–15 L·min^{-1} and 0–2.5 L·min^{-1} respectively, fitted on to medical oxygen outlets in a Fink Engineering triple lock hyperbaric chamber and a FlowAnalyzer™ PF-300 (IMT Analytics AG, Buchs, Switzerland) gas flow analyser (PF-300) designed for biomedical engineering testing of medical ventilators. This analyser had been previously validated for use and approved safe under hyperbaric conditions. We nevertheless also assembled a small version of a Douglas bag type gas collection system to enable cross checking of analyser readings against flows measured by capturing gas over a timed period and measuring it with a 500 ml gas volume calibration syringe as commonly used to calibrate spirometry equipment. The Ezi-flow flowmeters were connected to the flow analyser with disposable small bore oxygen tubing long used in hyperbaric conditions.

COMPONENT SELECTION

Flowmeter

Comweld Ezi-Flow flowmeters were selected based on their being the general-purpose oxygen flowmeters in current use in our hospital and within our hyperbaric chambers. These are 'ball-in-tube' or 'Thorpe tube' type flowmeters. The Comweld Ezi-Flow standard flowmeter has an indicated flow range of 0–15 L·min^{-1} and is typically used for adult patients at our centre. The Comweld Ezi-Flow low flowmeter has an indicated flow range of 0–2.5 L·min^{-1} and is our flowmeter of choice for paediatric cases. The manufacturer specifies both flowmeters as having an error range of ± 5%.[9]

Tubing

The connecting tubing selected for our analyses was medical grade oxygen tubing with a 7 mm outside diameter and 5 mm inside diameter. This is the standard tubing used in our institution for delivery of sweep gas during ECMO and was used with the modified Rotaflow I device when we delivered HBOT to patients on ECMO.[4–6]

Gas flow analyser

The PF-300 gas flow analyser utilised is calibrated and certified yearly by the manufacturer for high precision flow rate recordings and has been previously validated for safety and accuracy in hyperbaric conditions at our centre. The device's low flow inlet port is used to record flow rates in L·min^{-1}, up to two decimal places.

SET UP

The gas flow analyser was allowed to warm up for at least 20 minutes prior to commencing testing, to ensure running temperature and flow rate recording were stable.

For each flow rate tested, the desired flow was initially set at atmospheric pressure according to the Ezi-Flow flowmeter indicator, and then fine-tuned according to the flow displayed (to two decimal points) on the PF-300 gas analyser. Intervals of 0.30 L·min^{-1}, 0.60 L·min^{-1}, 0.90 L·min^{-1} and 1.20 L·min^{-1} were analysed using the low flowmeter and intervals of

3.00 L·min^{-1}, 6.00 L·min^{-1} 9.00 L·min^{-1} and 12.00 L·min^{-1} were analysed using the standard flowmeter.

The chamber was then pressurised at a rate of 10 kPa·min^{-1} from 101.3 kPa (1 atm abs) to 284 kPa (2.8 atm abs). Recordings of flow rate (L·min^{-1}), chamber temperature (°C) and humidity (%) were recorded at 10 kPa intervals. The chamber was subsequently decompressed, at a rate of 10 kPa·min^{-1}. The compression and decompression rates of 10 kPa·min^{-1} were chosen for comparative consistency. To confirm that the change in flow rate phenomena was not due to a faulty flowmeter, other flowmeters of same make and model were also tested to confirm consistent findings with the primary test flowmeters.

CROSS VERIFICATION

Flow rates displayed by the PF-300 analyser were cross verified with a Douglas bag test type procedure to confirm reliability at hyperbaric pressure. At interval pressure and set flow rate, oxygen gas was allowed to flow into an emptied reservoir bag for sixty seconds. Gas flow was then immediately closed off. The set-up included control valves to prevent gas loss. A 500 mL calibration syringe was used to extract and record the volume of gas that had entered the reservoir bag during the timed period enabling the Actual gas flow rate to be calculated prior to the next pressure change.

CALCULATIONS

Both percentage and absolute change in Actual gas flow rate from that at atmospheric pressure was calculated during compression, isopressure and decompression phases. The percentage difference between the flow rate during chamber compression and decompression was calculated.

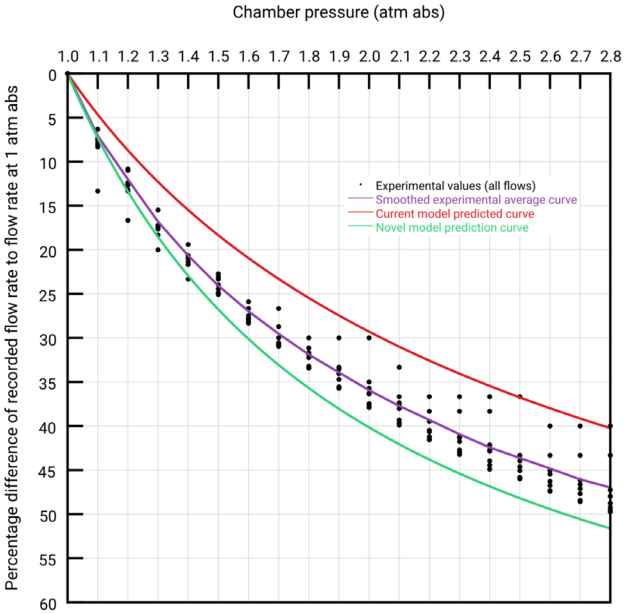
Results

Numerical results of the Actual gas flow rates through the standard and low flow assessments can be found in online *Appendix 1, and are presented graphically in online *Appendices 2 and 3, respectively .

STANDARD FLOW ASSESSMENT

Actual gas flow rates through the standard flowmeters reduced as the pressure in the chamber increased from 1 atm abs to 2.8 atmabs, characterised graphically by non-linear curves. At 2.8 atm abs flow delivered was approximately 50% less than set and indicated on the flowmeter (*Appendix 1).

Figure 2
Decrement in Actual flow from Ezi-flow (standard and low) oxygen flowmeter at hyperbaric pressures



LOW FLOW ASSESSMENT

The Actual flow of oxygen through the low flow flowmeter reduced and gradually tapered as chamber pressure increased in a similar fashion to that seen with the standard flowmeter. At the lowest flow rates we assessed (0.30 L·min^{-1} and 0.60 L·min^{-1}), reductions in oxygen flow rates of 40.0% and 43.3% were demonstrated respectively, whereas at all other flows approached a 50% reduction.

The percentage reductions of flow at various pressure intervals, are presented as a scatter plot in Figure 2 overlaid with the smoothed average, variable orifice-based prediction model and a new model based off the choked flow behaviour characteristics which was identified as appropriate for this design of flowmeter. Details of the modelling will be presented in a separate manuscript.

COMPRESSION VS DECOMPRESSION

The maximum differences in flow rates between chamber compression and decompression were a few percent at all but the lowest flows from the low flowmeter, where up to 22% discrepancy was demonstrated at 1.4 atm abs (*Appendix 3).

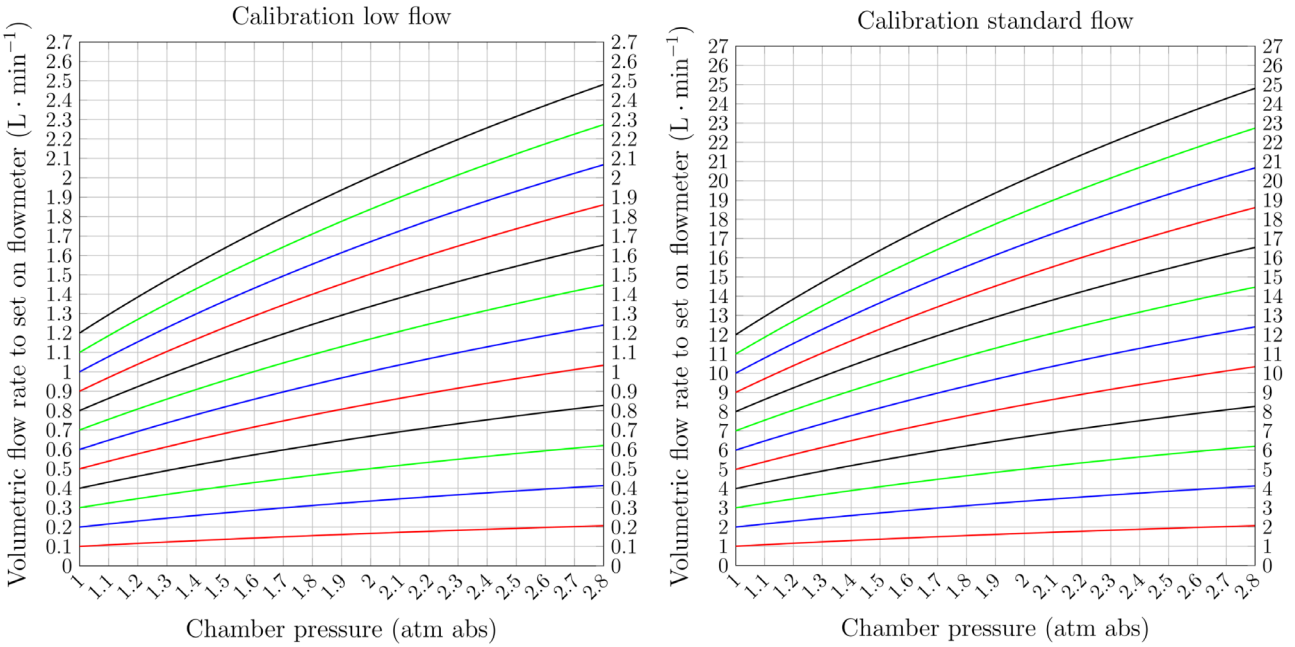
GAS ANALYSER CROSS VALIDATION

Douglas bag type testing at a variety of pressure and flow points cross-validated the performance of the PF-300 gas

***Footnote:** Appendix 1–3 are available online on our website https://www.dhmjournal.com/index.php/journals?id=418

Figure 3

Correction charts for clinical use of Ezi-Flow flowmeters in hyperbaric conditions at The Alfred, Bayside Health. To use the correction charts, select your desired Actual flow rate, e.g., 1.2 L·min^{-1}. On the Y-axis of the appropriate chart (standard or low flow), find the curve that begins at your desired Actual flow rate (e.g., 1.2 L·min^{-1}). Follow the curve to the pressure at which you will be operating (e.g., 2.8 atm abs). Move horizontally from this point to the axis on the right, which will indicate the flow rate you will need to set at normal atmospheric conditions in order to obtain your desired Actual flow rate at pressure (approximately 2.5 L·min^{-1} in this example)



analyser within the two decimal place/L·min^{-1} precision of this instrument.

CLINICAL CORRECTION CHARTS

For greater accuracy of gas flow via Ezi-flow flowmeters in our service, we developed correction charts for clinical use, using our test results and mathematical modelling (Figure 3). The mathematical model is based on mass choked flow behaviour; details are be reported separately in a dedicated manuscript. Importantly, these correction charts have been developed specifically for our local hyperbaric systems and may not be applicable to other chamber configurations.

Discussion

In normal clinical practice, decisions about and control of oxygen administration is often imprecise with tolerance of significant variability, with low flow anaesthesia and ECMO being examples of areas where more precise control is required. There is normally an expectation that displayed gas flows will be consistent with actual gas flow and the small differences resulting from definitional differences of what is 'sea level' or 'standard' or 'normal' gas supply conditions are generally ignored as too small to be relevant. Under hyperbaric conditions, however, much greater changes occur than are seen with atmospheric pressure variability and with altitude. This study demonstrates clearly the need to consider and allow for the changes associated with hyperbaric pressurisation.

FLOW RATE TRENDS

Our results confirm that Actual volumetric flow of oxygen falls substantially below the set flow rates as chamber pressure is increased, in a manner consistent with that predicted by the equation shown in the introduction above.

The low flowmeter was demonstrated to be susceptible to greater variations in flow rate with respect to chamber compression and decompression compared to the standard flowmeter which delivered relatively stable results. This is not unexpected given that increasing imprecision at low flows is a commonly observed phenomenon in syringe driver pumps and in pressure control regulators, likely driven by mechanical factors such as 'stiction' preventing truly linear control of the movement of components within devices. In our installation, the supply pressure of medical gases into the hyperbaric chamber is controlled by variable output pressure regulators which are least accurate at low flows.

Although clinical utilisation of therapeutic oxygen is often pragmatic and imprecise, variability between flowmeter settings and Actual gas flow in a hyperbaric setting is particularly important to understand when delivering HBOT to a patient supported by ECMO. The determining factor for appropriate fresh gas flow into an ECMO oxygenator is

to remove carbon dioxide at rates that maintain the desired PCO_2 for the patient. These flow rates deliver multiple times more oxygen than is required for metabolic demand and CO_2 clearance therefore drives the fresh gas flow requirements during ECMO.

A minor but consistent trend observed during testing was movement of the bobbin. The bobbin was observed to move upward, but only very slightly, as chamber pressure increased. This is not inconsistent with the decreased volumetric flow and decreased gas velocity being balanced out by increased gas density. Given that small movements of the bobbin are sometimes seen in non-hyperbaric clinical practice, likely due to supply pressure variations and flow harmonics in the gas pipeline, this finding was concluded as having insignificant clinical impact for hyperbaric practice. Importantly, the mathematical model used in the development of the correction charts in Figure 3 works irrespective of errors that could be associated with this observation, as bobbin mechanics are subject to local drag, weight and buoyancy forces, whereas the model we have used is not.

GAS SUPPLY PRESSURE

The Comweld Ezi-flow flowmeters are designed for an inlet pressure of 400 kPa,[9] and our service has pressure compensation regulators which maintain flowmeter supply pressures at the gas port at the appropriate level above chamber pressure. Chambers without this feature will experience different patterns of variability in gas flow rates compared to what is reported here.

LIMITATIONS

Testing was performed in the hyperbaric chamber at The Alfred (Bayside Health). Variations in hyperbaric chamber designs may mean that the equations described here may not be applicable to hyperbaric chambers with differences in configuration, or without instruments to maintain steady supply pressure.

Another potential limitation of this work could be the consideration that our testing was inconsistent with clinical use of the flowmeters e.g., the flow was set prior to starting compression and not adjusted, as would be done clinically if required, during pressurisation. However, given the slight increase in bobbin height, any adjustments to restore the bobbin back to baseline Set flow by an inside attendant would be in the downward direction, further reducing flow and exaggerating the direction of the trend which we have described.

Conclusions

Our study demonstrated a nonlinear inverse relationship between chamber pressure and actual gas flow delivered by both standard and low-flow Comweld Ezi-flow flowmeters under hyperbaric conditions. These very significant changes are consistent with a shifted hyperbolic or exponential decay type relationship as would be expected based on the flowmeters' control of flow being characterised as a mass choked flow phenomenon. To deliver clinically desired gas flow rates under hyperbaric conditions, appropriate performance testing of gas flowmeters is required. We have used performance data and mathematical modelling to develop correction charts for the clinical use of Ezi-flow flowmeters in our hyperbaric chamber.

References

1 Ilancheran A, Millar I, Tsouras T. Successful hyperbaric oxygen treatment of a patient with a HeartMate III left ventricular assist device. Diving Hyperb Med 2023;53:147–50. doi: 10.28920/dhm53.2.147-150. PMID: 37365133. PMCID: PMC10584400.

2 Gelsomino M, Tsouras T, Millar I, Fock A. A pleural vacuum relief device for pleural drain unit use in the hyperbaric environment. Diving Hyperb Med 2017;47:191–7. doi: 10.28920/dhm47.3.191-197. PMID: 28868600. PMCID: PMC6159614.

3 Frawley L, Devaney B, Tsouras T, Frawley G. Performance of the BBraun perfusor space syringe driver under hyperbaric conditions. Diving Hyperb Med 2017;47:38–43. doi: 10.28920/dhm47.1.38-43. PMID: 28357823. PMCID: PMC6149317.

4 Tsouras T, Devaney B, Lin ZL, Covelli C, Roberts L, Nanjayya VB, et al. Validation and clinical use of Rotaflow extracorporeal membrane oxygenation device in hyperbaric conditions: a technical report. Diving Hyperb Med. 2025;55:323–9. doi: 10.28920/dhm55.4.323-329. PMID: 41364855. PMCID: PMC12831603.

5 Devaney B, Mathew J, Ferris S, Roberts L, Covelli C, Orosz J, et al. Novel use of hyperbaric oxygen treatment for treatment-resistant disseminated Saksenaea and Fusarium in a patient on extracorporeal membrane oxygenation (ECMO): a case report. Diving Hyperb Med. 2025;55:309–14. doi: 10.28920/dhm55.4.309-314. PMID: 41364853. PMCID: PMC12823155.

6 Adams B, Templeton A, Tsouras T, Sheldrake J, Roberts L, Lin ZC, et al. The process, logistics and governance behind a high-stakes novel intervention: the use of extracorporeal membrane oxygenation (ECMO) in the hyperbaric chamber. Diving Hyperb Med. 2025;55:315–22. doi: 10.28920/dhm55.4.315-322. PMID: 41364854. PMCID: PMC12823154.

7 Abd-Elsayed A, Mahboobi SK, Germani ML. Flowmeters. In: Abd-Elsayed A, editor. Basic anesthesia review. New York: Oxford Academic; 2024. doi: 10.1093/med/9780197584569.003.0017.

8 Duprez F, Barile M, Bonus T, Cuvelier G, Ollieuz S, Mashayekhi S, et al. Accuracy of medical oxygen flowmeters: a multicentric field study. Health. 2014;6:1978–83. doi: 10.4236/health.2014.615232.

9 Comweld. Ezi-Flow Flowmeter [Internet]. [cited 2025 Sep 9]. Available from: https://comweld.com.au/product/ezi-flow-flowmeter.

10 AS2896. Australian Standard for the installation, testing, and maintenance of non-flammable medical gas pipeline systems

[Internet]. [cited 2025 Oct 1]. Available from: https://www.kembla.com/wp-content/uploads/2020/08/Tech-Bulletin-Installation-and-testing-of-Medical-Gas-pipeline-systems.pdf.
11 Consumer Medicine Information: Medical Oxygen 99.5%. NPS MedicineWise. [Internet]. [cited 2026 Jan 4]. Available from: https://www.nps.org.au/medicine-finder/medical-air#full-pi.
12 Consumer medicine information: Medical air. NPS MedicineWise. [Internet]. [cited 2026 Jan 4]. Available from: https://www.nps.org.au/medicine-finder/medical-oxygen-99-5#full-pi.
13 Hutton P, Boaden RW. Performance of needle valves. Br J Anaesth. 1986;58:919–24. doi: 10.1093/bja/58.8.919. PMID: 2942165.
14 Hall N. Mass flow choking. NASA. 2021. [Internet]. [cited 2025 Oct 1]. Available from: https://www.grc.nasa.gov/www/k-12/airplane/mflchk.html.
15 Robinson S, Peek G. The role of ECMO in neonatal and paediatric patients. Paediatr Anaesth. 2015;25:452–61. doi: 10.1016/j.paed.2015.03.005.
16 Brunetti MA, Gaynor JW, Retzloff LB, Lehrich JL, Banerjee M, Amula V, et al. Characteristics, risk factors, and outcomes of extracorporeal membrane oxygenation use in pediatric cardiac ICUs: a report from the Pediatric Cardiac Critical Care Consortium Registry. Pediatr Crit Care Med. 2018;19:544–52. doi: 10.1097/PCC.0000000000001571. PMID: 29863638. PMCID: PMC6051408.

Conflicts of interest and funding: nil

Submitted: 8 January 2026
Accepted after revision: 30 April 2026

Review articles

Evidence-based guidelines for long-term care in spinal cord-related decompression illness

Camille Dubois[1,2], Claire Hentzen[1,2], Alexis Schnitzler[3], Lucile Daubresse[4], Jean Morin[4]

[1] *Sorbonne University, GRC 01-GREEN, Paris, France*
[2] *Neuro-Urology Department, Pitié-Salpêtrière Hospital, AP-HP, Paris, France*
[3] *Department of Physical and Rehabilitation Medicine, Le Vésinet Hospital, Le Vésinet, France*
[4] *Department of Hyperbaric Medicine, Sainte-Anne Military Teaching Hospital, Toulon, France*

Corresponding author: *Dr Camille Dubois, Neuro-Urology Department, Pitié-Salpêtrière Hospital, 47-83 boulevard de l'Hôpital, 75013, Paris, France*
camille.dubois@aphp.fr

Keywords
Central nervous system; Decompression sickness; Hyperbaric medicine; Scuba diving; Spinal cord injury

Abstract
(Dubois C, Hentzen C, Schnitzler A, Daubresse L, Morin J. Evidence-based guidelines for long-term care in spinal cord-related decompression illness. Diving and Hyperbaric Medicine. 2026 30 June;56(2):161–169. doi: 10.28920/dhm56.2.161-169. PMID: 42290576.)

Introduction: The aim of this review was to synthesise current knowledge and propose structured evidence-based recommendations for long-term care of individuals with spinal cord decompression illness (scDCI) drawing on experience from French clinical settings and the international literature.

Methods: We conducted a systematic search of international and French guidelines for decompression illness (DCI) and spinal cord injury (SCI), including systematic reviews and consensus statements. Additional literature searches were performed in PubMed Central® (1996–2025) to identify evidence relevant to long-term care. Key domains were predefined based on SCI guidelines: lower urinary tract and gastrointestinal function, thromboembolic, autonomic dysreflexia, pulmonary function, pain, and spasticity.

Results: Our findings confirmed the absence of specific long-term follow-up protocols for scDCI. Evidence from SCI guidelines was therefore adapted to this population. We propose structured, evidence-based recommendations that include systematic neurological and urological screening even in apparently recovered patients and risk-adapted follow-up during the first two years.

Conclusions: Long-term outcomes after scDCI remain poorly defined, but sequelae are frequent and may be underdiagnosed. Structured follow-up based on adapted SCI guidelines may improve prognosis, harmonise care, and generate robust data for future prospective studies.

Introduction

Decompression illness (DCI) refers to a group of clinical syndromes caused by the pathophysiological response to the formation of gas bubbles within the body following a decrease in ambient pressure. In diving, DCI occurs when inert gas, primarily nitrogen, dissolved in tissues and blood under pressure forms bubbles during ascent. These bubbles trigger a complex cascade of pathophysiological responses that may affect multiple organ systems. Clinical manifestations range from mild symptoms such as skin itching or joint pain to severe neurological impairment, cardiovascular instability, or even death. The incidence of DCI varies across diving populations, ranging from 1 to 4 per 10,000 person-dives for recreational diving. Among severe cases, neurological symptoms are predominant, affecting up to 50% of individuals, with clinical features often consistent with spinal cord injury (SCI). Spinal cord decompression illness (scDCI) is thought to result from a combination of these mechanisms, including extravascular bubble formation in the spinal white matter and venous or arteriolar gas embolism, leading to spinal ischaemia or infarction.[1,2] Intravascular bubbles can obstruct vessels and activate coagulation pathways through platelet aggregation, leukocyte activation, and fibrin deposition. Concurrently, endothelial injury increases capillary permeability, exacerbating oedema and ischaemia. Once activated, inflammatory and coagulation cascades persist even after bubble resolution, which may explain why recompression therapy is not immediately and uniformly effective.

Prognosis is primarily determined by initial clinical severity, the time to recompression and early neurologic deterioration within the first 24 hours.[3] Nevertheless, despite timely

hyperbaric treatment, studies report persistent sequelae in 20–30% of individuals at one month, primarily involving sensorimotor deficit and bladder dysfunction.[4] Given the specific pathophysiological consequences of scDCI, early prevention of secondary complications, particularly neurogenic bladder dysfunction, is essential to minimise long-term morbidity and mortality.[5]

To our knowledge, no prospective long-term study has systematically evaluated the outcomes of scDCI. As a result, follow-up strategies remain heterogeneous, and late complications may be under-recognised. The aim of this work was to conduct a systematic search of the literature and existing guidelines to propose evidence-based recommendations for long-term follow-up in this population.

Methods

RECOMMENDATIONS FOR DCI

We searched for international guidelines regarding post-acute care after diving-related DCI.

To identify relevant recommendations, we searched for publications from January 1996 to July 2025 in the PubMed Central® database using the keywords: "*scuba diving*", "*decompression sickness OR decompression illness*", and "*recommendation OR guideline*".

Inclusion criteria were: (1) evidence-based recommendations or clinical guidelines; (2) specific relevance to decompression illness; (3) applicability to long-term care; and (4) focus on spinal cord involvement.

RECOMMENDATIONS FOR SCI

We searched for both international and French recommendations about the long-term follow-up of individuals with SCI, unrelated to diving or DCI.

To identify updates, we performed a systematic search of the literature using the PubMed Central® database from January 2015 to July 2025. The following keywords were used: "*spinal cord injury*", "*recommendation OR guideline*", and "*long-term*".

Inclusion criteria were: (1) evidence-based recommendations or clinical guidelines; (2) relevance to acquired SCI; (3) general recommendations covering main secondary health conditions of people with SCI; and (4) applicability to long-term care.

When necessary, targeted searches were then undertaken on specific topics, for instance, the recommended minimum duration of hospitalisation following an episode of DCI or the post-void residual volume threshold prompting intermittent catheterisation in a neurogenic bladder.

EVIDENCE-BASED RECOMMENDATIONS

Based on the collected data, we proposed evidence-based recommendations for the long-term follow-up of individuals with scDCI.

To individualise post-discharge care, patients were stratified into two groups: those with full recovery or isolated sensory deficit at discharge, in whom follow-up aims to detect subtle or delayed impairments, and those with persistent motor deficit or neurogenic bladder dysfunction, for whom follow-up focuses on monitoring and preventing secondary complications associated with SCI.

A large language model was used to enhance readability and language during manuscript preparation. All authors reviewed, revised, and take full responsibility for the final content.

Results

RESULTS OF THE SYSTEMATIC SEARCH

The article selection process is summarised in the PRISMA flow diagrams, including the reasons for exclusion of articles.[6]

DCI recommendations

Reference documents included the clinical practice guidelines of the Undersea and Hyperbaric Medical Society (UHMS) and the 1996 European consensus statement on diving-related injuries. The UHMS clinical practice guidelines do not address post-acute care for scDCI.[7] The 1996 European consensus provides limited guidance, recommending at least two years of clinical follow-up for spinal cord-injured divers, without further specification.[8]

Our literature search on follow-up care in DCI identified 53 articles as of July 15, 2025; none met the inclusion criteria after title and abstract screening (Figure1).

SCI recommendations

Reference documents included French national recommendations for the long-term management of individuals with SCI published by the *Haute Autorité de Santé* in 2007 based on the Consortium of Spinal Cord Medicine guidelines published by the Paralyzed Veterans of America between 1999 and 2006.[5] In line with these recommendations, we then chose to focus on seven key areas: lower urinary tract function, gastrointestinal function, thromboembolic complications, autonomic dysreflexia, pulmonary function, pain and spasticity.

Our supplementary literature search on follow-up care for individuals with SCI yielded 173 articles as of July 15, 2025.

Figure 1
PRISMA flow diagram for our literature search on follow-up care in spinal cord decompression illness; no references were found using alternative methods

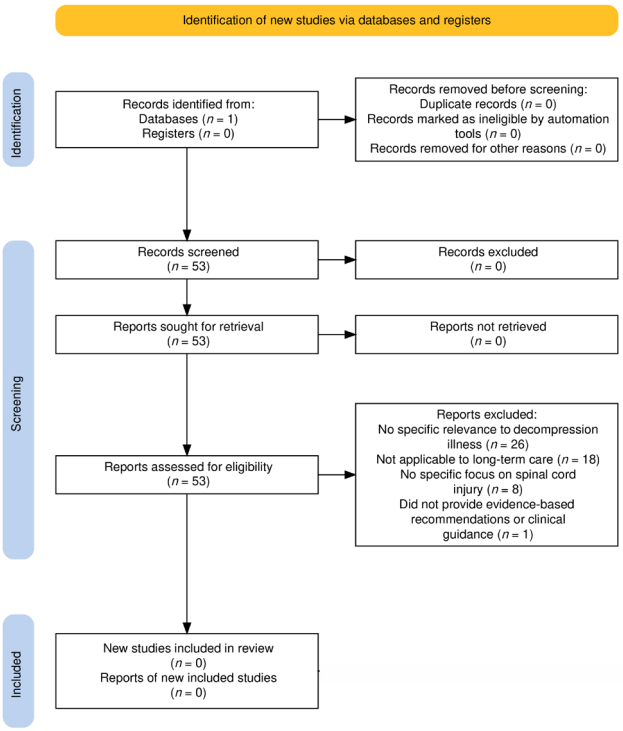
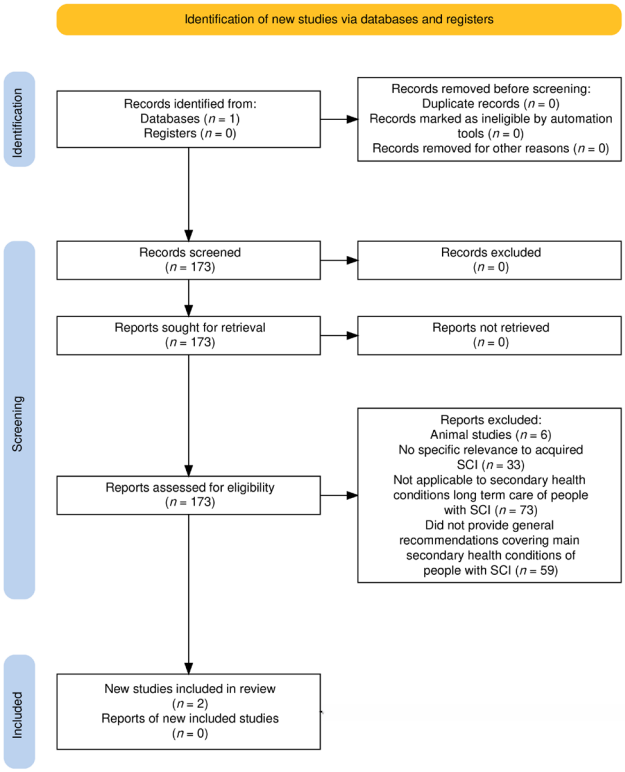


Figure 2
PRISMA flow diagram for our literature search on recommendations and guidelines for general long-term care in spinal cord injury; no references were found using alternative methods



Based on title and abstract screening, only two articles met the inclusion criteria (Figure 2).[9,10]

PROPOSAL FOR EVIDENCE-BASED RECOMMENDATIONS AFTER SCDCI

The proposed follow-up protocol is summarised in Tables 1 and 2.

Day 0 (admission) to discharge management

Before emergency recompression therapy the following should occur.

1. Neurological assessment: SCI is characterised by the neurological level of injury, the lowest segment with preserved motor and sensory function, and by its completeness. Neurological and functional status should be classified according to the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI), including the ASIA Impairment Scale (AIS), which ranges from grade A (complete injury) to grade E (normal function). This scale is the international gold standard for SCI assessment and strongly predicts outcome, with grade A injuries rarely and grade D injuries commonly regaining independent ambulation at one year.[11]

2. Lower urinary tract assessment: In the acute phase, spinal shock is associated with a high risk of urinary retention, often due to detrusor areflexia or hypocontractility. The duration of spinal shock can vary from a few days to several months, as observed in traumatic SCI, though its course remains undocumented in DCI-related cases.[12] In 2025, international consensus has defined a 150 mL post-void residual volume threshold for initiating intermittent catheterisation in individuals with neurogenic bladder dysfunction, based on associated risks of urinary tract infections, bladder stone formation and upper urinary tract deterioration.[13]

For practical guidance, we propose:

» A spontaneous void attempted prior to hyperbaric chamber entry, with immediate post-void residual volume assessment using ultrasound.
» In cases of acute urinary retention, defined as the inability to void despite a full bladder, or elevated post-void residual volume (> 150 ml), external bladder drainage using indwelling catheterisation is recommended before entering the hyperbaric chamber. The catheter should then be removed as soon as clinically feasible and, if spontaneous voiding does not resume, replaced by intermittent catheterisation.

3. Prognosis scoring: Prognosis is primarily driven by initial clinical severity, time to recompression and the

occurrence of neurological deterioration within the first 24 hours following the accident.[3] Initial severity can be assessed using the MedSubHyp score, developed by the Société de Médecine et de Physiologie Subaquatiques et Hyperbares (MedSubHyp). This score is based on the following parameters: age > 42 (0 or 1); back pain (0 or 1); clinical course before recompression (better = 0; stable = 3; worse = 5); objective sensory deficit (0 or 4); motor impairment (none = 0; paresis = 4; paraplegia = 5) and neurogenic bladder dysfunction (0 or 6). The total score ranges from 0 to 22, with higher scores (≥ 6) associated with significantly worse neurological outcomes at discharge.[14]

After emergency recompression therapy the following should occur.

1. Surveillance: In cases of incomplete recovery after initial recompression, additional hyperbaric sessions may be considered, as this phase may coincide with peak post-ischaemic inflammatory processes.[3]

For practical guidance, we propose:

» Even in cases of apparent full recovery, a minimum of 48–72 hours hospitalisation to monitor for potential secondary deterioration, which may necessitate additional hyperbaric treatment.
» In symptomatic individuals, length of stay should be determined by clinical evolution and therapeutic requirements.

2. Diving-specific considerations – screening for right-to-left shunt: According to recent South Pacific Underwater Medicine Society (SPUMS) and the United Kingdom Diving Medical Committee (UKDMC) guidelines, screening for a right-to-left shunt is recommended in cases of DCI with cerebral, spinal, vestibulocochlear, cardiovascular and cutaneous manifestations.[15]

For practical guidance, we propose:

» Screening for right-to-left shunt when clinically feasible using either transcranial Doppler ultrasound or trans-thoracic echocardiogram, depending on local expertise and equipment availability, with bubble contrast techniques and use of provocation manoeuvres (Vasalva and sniffing test).

3. Imaging strategy in early management: In scDCI, lesions revealed by magnetic resonance imaging (MRI) usually correlate with clinical findings, but a normal MRI does not exclude the diagnosis. Clinical-radiological dissociation is recognised in scDCI. While MRI has limited sensitivity, acute findings may inform prognosis: haemorrhage predicts poorer outcome, whereas oedema or normal imaging are generally associated with better recovery, although sequelae remain possible (negative predictive value ~77%). MRI also identifies compressive factors, such as cervical spondylosis, that may predispose to severe scDCI.[16]

For practical guidance, we propose:

» Spinal MRI may be performed after emergency recompression therapy, within the first days following the accident, to assist in prognostic evaluation.

4. Lower urinary tract management: All individuals with SCI remain at risk for neurogenic bladder dysfunction, including those with apparent recovery, as bladder dynamics may change over time. With the return of spinal reflexes, detrusor overactivity and detrusor-sphincter dyssynergia may develop, leading to incomplete emptying, elevated voiding pressures and potential upper urinary tract deterioration if untreated.[17] In traumatic SCI, up to 50% of individuals who initially void with low-pressure storage and adequate emptying later develop neurogenic bladder dysfunction requiring changes in management.[18] Urinary symptoms should be actively assessed at discharge, for example using the validated Urinary Symptom Profile questionnaire.[19]

For all individuals, the following should be performed:

» If acute urinary retention initially requires an indwelling catheter, it should be removed as soon as medical stabilisation allows (within 48–72 hours), and replaced with intermittent catheterisation if needed, which significantly reduces the risk of complications (e.g., infections, bladder stones, urethral damage). Long-term use of an indwelling catheter should be strictly avoided at all stages following SCI.
» Screening for signs of overactive bladder (urgency and urinary urgency incontinence), combined with daily post-void residual volume assessment using ultrasound to identify significant urinary retention should be performed. An average post-void residual volume > 150 mL should prompt the initiation of intermittent catheterisation, along with a therapeutic educational program to support patient training in self-catheterisation.
» If neurogenic bladder is suspected (urgency, urinary urgency incontinence, voiding difficulties): renal function should be assessed through blood testing to estimate the glomerular filtration rate, as well as bladder and kidney imaging, including ultrasound measurement of post-void residual volume.

5. Gastrointestinal management: Neurogenic bowel dysfunction is a common and burdensome consequence of SCI, significantly affecting psychological well-being and social participation. If inadequately managed, it may lead to serious complications. Constipation is the most frequent symptom, reported in up to 95% of individuals with complete SCI. Pharmacological management includes several options, such as osmotic agents, bulk-forming agents, oral laxatives, prokinetics, and rectal therapies including

suppositories, but no treatment has demonstrated clear superiority. Management therefore relies on individualised bowel programmes tailored to the person's autonomy and preferences, as rigid protocols are rarely appropriate.[20]

6. Venous thromboembolism complications: Venous thromboembolism is a serious complication of SCI that may go unnoticed due to limb swelling and sensory impairment. Early thromboprophylaxis is therefore crucial to minimise morbidity and mortality.[21]

For practical guidance, for all individuals with lower limb motor dysfunction, the following should be performed:

» If walking dysfunction persists after emergency recompression therapy, anticoagulant thromboprophylaxis should be initiated once the patient is clinically stabilised and continued for 8–12 weeks in combination with mechanical compression methods (e.g., compression stockings). However, treatment may be discontinued earlier if ambulation improves before this period.

7. Autonomic complications in high spinal cord injuries: In individuals with SCI at or above T6, loss of sympathetic control may result in autonomic instability, affecting cardiovascular responses and catecholamine regulation. Autonomic dysreflexia, triggered by stimuli below the level of injury, is defined as a systolic blood pressure increase > 20 mmHg above baseline and may present with headache, flushing, sweating above the lesion, nasal congestion, or arrhythmia; and can be life-threatening.[22]

For practical guidance, for all individuals with lesions at or above T6, the following should be performed:

» Baseline electrocardiogram.
» Orthostatic hypotension test when clinically feasible.
» If autonomic dysreflexia or cardiac dysfunction is suspected: blood tests including B-type natriuretic peptide, troponin, lipid profile, and echocardiography.

8. Respiratory complications in high spinal cord injuries: Pulmonary complications are the leading cause of death within the first year following SCI. The degree of respiratory dysfunction following SCI depends on the neurological level, severity (AIS grade), and resulting impairment of ventilatory effort and cough effectiveness, increasing the risk of respiratory complications such as pneumonia, atelectasis, pleural effusion, sleep-disordered breathing, and dyspnoea.[23]

For practical guidance, for all individuals with lesions at or above T8 with motor deficit, the following should be performed:

» Pulmonary function tests within the first month.
» Morning arterial blood gas testing and polysomnography within the first month.

Discharge to two-years follow-up

According to recommendations in SCI guidelines and to the European consensus on the management of recreational diving accidents, clinical follow-up should continue for a minimum of two years post-injury.[8]

1. Return to scuba-diving: Current guidance on resuming diving after DCI is based primarily on expert consensus and policies issued by major organisations (e.g., US Navy, Diving Medical Advisory Committee [DMAC], UHMS), rather than on robust scientific evidence. Among these, the MedSubHyp society proposes the following minimum intervals before return to diving:

» For individuals with isolated sensory deficit that resolves completely after recompression therapy: Three months.
» For individuals with bladder or motor dysfunction with full recovery after recompression therapy: Six months.
» For individuals with neurological sequelae, except for isolated superficial sensory deficit, return to diving is not recommended.

In all circumstances, clearance to resume diving should be provided by a physician experienced in diving and hyperbaric medicine, considering individual risk factors such as recurrent DCI or the presence of a right-to-left shunt.

2. Rehabilitation program: The need for a rehabilitation program depends on the presence and severity of sequelae.

For practical guidance, we propose:

» Individuals with no residual deficit (AIS E) may be discharged without a structured rehabilitation program.
» Referring individuals with persistent neurological deficits (AIS grades D to A) to a Physical and Rehabilitation Medicine physician to establish a rehabilitation program – either inpatient, day-hospital-based, or outpatient physiotherapy, depending on functional independence at discharge.

3. Neuropathic pain: Pain is a major contributor to functional limitation in individuals with SCI, with a reported prevalence of 60–70% in traumatic SCI. The 2021 CanPainSCI Clinical Practice Guidelines formalised a series of recommendations for the management of neuropathic pain after SCI.[24]

For practical guidance, the following should be performed:

» Use of a validated questionnaire for neuropathic pain screening (as for example the DN4 questionnaire).
» Encourage the use of self-management strategies that help reduce pain intensity, improve function, and address sleep, mood, or activity-related pain exacerbation.
» First-line treatment: Pregabalin is the preferred option, supported by the strongest evidence for below-level neuropathic pain. Gabapentin is a recommended

alternative if pregabalin is contraindicated or ineffective.
» In cases of treatment resistance, individuals should be referred to a specialised interdisciplinary pain management center.

4. Spasticity: Spasticity is a frequent complication of SCI, presenting with increased tone and exaggerated reflexes due to impaired sensorimotor control. Treatment includes medication, physical measures and, in selected cases, surgery.[25]

For practical guidance:

» Given its complexity and functional implications, spasticity management should be initiated and supervised by a physical and rehabilitation medicine physician.

5. Follow-up spinal MRI at 3–6 months: Follow-up spinal MRIs are recommended for all individuals, including those with a normal initial imaging and complete neurological recovery, to screen for delayed-onset spinal lesions.

6. Individualised clinical follow-up: To individualise post-discharge care, we have stratified patients into two groups; those with full recovery or isolated sensory deficit at discharge, in whom follow-up aims to detect subtle or delayed impairments, and those with persistent motor deficit or neurogenic bladder dysfunction, for whom follow-up focuses on monitoring and preventing secondary complications associated with spinal cord injury.

For individuals with full clinical recovery or isolated sensory deficit at discharge, we propose (Table 1):

» Follow-up at six, 12 and 24 months post-injury with a physician experienced in diving and hyperbaric medicine. Each visit should include a full neurological examination, screening for neurogenic bladder dysfunction (using the Urinary Symptom Profile questionnaire), and bladder ultrasound with post-void residual measurement, with uroflowmetry where available.
» If clinical examination is entirely normal, with a Urinary Symptom Profile score of 0, normal spinal MRI, and normal renal and bladder ultrasound ± uroflowmetry, follow-up may be discontinued after the 24-month visit.
» At any time, the emergence of symptoms such as spasticity, neurogenic bladder dysfunction or motor deficit should prompt management according to the symptomatic care pathway.

For individuals with persistent neurogenic bladder dysfunction or motor deficit at discharge, we propose (Table 2):

» Coordinated follow-up with a Physical and Rehabilitation Medicine physician at one month, every three months during the first year and then at least annually.

Table 1

Proposed follow-up schedule for individuals with full clinical recovery or isolated sensory deficit at discharge; items that are shaded in gray are not mandatory and recommended assessments are indicated by an 'X'. DN4 – Douleur Neuropathique 4 (questionnaire); MRI – magnetic resonance imaging; N/A – not applicable; PVR– post-void residual; R-L – right-to-Left; USP – urinary symptom profile

Consideration	Admission to discharge	3 months	6 months	12 months	24 months
Diving and hyperbaric medicine physician follow-up	N/A	If return to diving considered	X	X	X
Screening for R-L shunt	If indicated, e.g., to inform return to diving decision				
Return to diving	Contraindicated	Clearance to resume diving provided by a physician experienced in diving and hyperbaric medicine			
Spinal MRI	X	X			
Lower urinary tract assessment	Daily PVR measurement				
	USP questionnaire		X	X	X
	Renal-bladder imaging with PVR measurement ± uroflowmetry		X	X	X
In cases of neuropathic pain	DN4 questionnaire	First-line treatment: pregabalin / gabapentin Or refer to interdisciplinary pain management centre			

Table 2

Proposed follow-up schedule for individuals with neurogenic bladder dysfunction or motor deficit at discharge; items that are shaded in gray are not mandatory and recommended assessments are indicated by an 'X'. ABG – arterial blood gas; BNP – B-type natriuretic peptide; CI – contraindication; ECG – electrocardiogram; eGFR – estimated glomerular filtration rate; MRI – magnetic resonance imaging; N/A – not applicable; PRM – physical and rehabilitation medicine; PVR – post-void residual; R-L – right-to-left

Consideration	Admission to discharge	1 month	3 months	6 months	9 months	12 months	24 months
PRM physician Follow-up	N/A	X	X	X	X	X	X
Screening for RLS	If indicated						
Return to diving	Contraindicated			Clearance to resume diving provided by a physician experienced in diving and hyperbaric medicine			
Spinal cord	Spinal MRI		X			X	X
Neurogenic bladder dysfunction	eGFR			X		X	X
	Renal-bladder imaging with PVR measurement			X		X	X
	Urodynamic testing	X		X		X	Every six months
Cardio-vascular function if lesion ≥ T6	ECG	If needed (e.g., autonomic complications)					
	Screening for autonomic complications	If needed: blood testing with BNP, troponin, lipid panel + echocardiography may be useful					
Respiratory function if motor deficit ≥ T8	Chest X-ray	If needed					
	Morning ABG testing	X				X	X
	Pulmonary function tests	X				X	X
	Polysomnography	X	Depending on initial evaluation				

» Complete neuro-urological evaluation every six months for the first two years, then adapted based on individual uro-nephrological risk.
» Screening for autonomic complications for individuals with a neurological level of injury at or above T6.
» Annual respiratory assessment for individuals with a neurological level of injury at or above T8.

Discussion

Changes in diver demographics, including older age, less experience, and a higher prevalence of comorbidities, likely contribute to a greater risk of severe outcomes.[26] In parallel, the rise of technical diving, involving greater depths, longer bottom times, and the use of mixed gas, has also been associated with more complex and severe cases of DCI, as supported by recent epidemiological data.[27] Despite advances in acute management, including early recompression protocols and wider access to hyperbaric facilities, long-term follow-up remains poorly standardised. Moreover, recent studies have raised concern over the potential for delayed neurological sequelae in individuals initially classified as having "*non-neurological*" DCI, thus challenging the conventional dichotomy between "*neurological*" and "*non-neurological*" forms.[28] The absence of specific guidance for post-acute management of scDCI underscores the need for a coordinated long-term follow-up strategy. This study aimed to propose structured, evidence-based recommendations drawing on international and national SCI recommendations as well as diving medicine consensus statements, to address the dual specificity of this population: individuals with SCI resulting from diving-related DCI. Beyond improving clinical care, such an approach may also facilitate harmonised data collection and contribute to a clearer understanding of recovery trajectories.

Conclusions

Spinal cord DCI remains a complex condition, with limited data available on its long-term outcomes. While acute management is well established, post-acute care lacks standardised protocols, and delayed complications may be underdiagnosed or insufficiently addressed. This work proposes structured, evidence-based recommendations for long-term care, aiming to improve early detection of sequelae and optimise patient outcomes. Standardising follow-up may also support consistent data collection and contribute to a better understanding of the condition's natural history. Further prospective studies are needed to validate this follow-up strategy and inform future recommendations.

References

1 Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. Lancet. 2011;377:153–64. doi: 10.1016/S0140-6736(10)61085-9. PMID: 21215883.

2 Mitchell SJ. Decompression illness: a comprehensive overview. Diving Hyperb Med. 2024;54(1Suppl):1–53. doi: 10.28920/dhm54.1.suppl.1-53. PMID 38537300. PMCID PMC11168797.

3 Simonnet B, Roffi R, Lehot H, Morin J, Druelle A, Daubresse L, et al. Therapeutic management of severe spinal cord decompression sickness in a hyperbaric center. Front Med. 2023;10:1172646. doi: 10.3389/fmed.2023.1172646. PMID 37746073. PMCID PMC10514493.

4 Blatteau J-E, Gempp E, Simon O, Coulange M, Delafosse B, Souday V, et al. Prognostic factors of spinal cord decompression sickness in recreational diving: retrospective and multicentric analysis of 279 cases. Neurocrit Care. 2011;15:120–7. doi: 10.1007/s12028-010-9370-1. PMID: 20734244.

5 HAS guidelines for the management of paraplegia (Spinal Cord Injuries), ALD20, 2007 n.d. Available from: https://www.sofmer.com/download/sofmer/ALD20-LAP-paraplegie.pdf.

6 Haddaway NR, Page MJ, Pritchard CC, McGuinness LA. *PRISMA2020*: An R package and Shiny app for producing PRISMA 2020-compliant flow diagrams, with interactivity for optimised digital transparency and Open Synthesis. Campbell Syst Rev. 2022;18:e1230. doi: 10.1002/cl2.1230. PMID: 36911350. PMCID: PMC8958186.

7 Prevention and treatment of decompression sickness and arterial gas embolism. UHMS Best Practice Guidelines. [cited 2026 Feb 1]. Available from: https://www.uhms.org/images/DCS-AGE-Committee/dcsandage_prevandmgt_uhms-fi.pdf.

8 Second European consensus conference on the treatment of decompression illness in recreational diving, 1996. [cited 2026 Feb 1]. Available from: https://medical.ffessm.fr/uploads/media/default/0001/06/75e9b46aa583e7ad18e310f02ad54f130dafcbbd.pdf.

9 McColl MA, Gupta S, Smith K, McColl A. Promoting long-term health among people with spinal cord injury: What's new? Int J Environ Res Public Health. 2017;14:1520. doi: 10.3390/ijerph14121520. PMID: 29211040.

10 Eriks-Hoogland I, Müller L, Baumberger M, Huber B, Michel F, Belfrage C, et al. Evidence based clinical practice guideline for follow-up care in persons with spinal cord injury. Front Rehabil Sci. 2024;5:1371556. doi: 10.3389/fresc.2024.1371556. PMID: 39314837. PMCID: PMC11417090.

11 Roberts TT, Leonard GR, Cepela DJ. Classifications in brief: American Spinal Injury Association (ASIA) Impairment Scale. Clin Orthop Relat Res. 2017;475:1499–504. doi: 10.1007/s11999-016-5133-4. PMID: 27815685. PMCID: PMC5384910.

12 Ditunno J, Little J, Tessler A, Burns A. Spinal shock revisited: a four-phase model. Spinal Cord. 2004;42:383–95. doi: 10.1038/sj.sc.3101603. PMID: 15037862.

13 Panicker JN, Fanciulli A, Skoric MK, Kaplan T, Aleksovska K, Adamec I, et al. European Academy of Neurology (EAN)/European Federation of Autonomic Societies (EFAS)/International Neuro-Urology Society (INUS) guidelines for practising neurologists on the assessment and treatment of neurogenic urinary and sexual symptoms (NEUROGED Guidelines). Eur J Neurol. 2025;32:e70119. doi: 10.1111/ene.70119. PMID: 40208234. PMCID: PMC11984325.

14 Andre S, Lehot H, Morin J, Louge P, de Maistre S, Roffi R, et al. Influence of prehospital management on the outcome of spinal cord decompression sickness in scuba divers. Emerg Med J. 2022:emermed-2021-211227. doi: 10.1136/emermed-2021-211227. PMID: 35135892.

15 Smart D, Wilmshurst P, Banham N, Turner M, Mitchell SJ. Joint position statement on atrial shunts (persistent [patent] foramen ovale and atrial septal defects) and diving: 2025 update. South Pacific Underwater Medicine Society (SPUMS) and the United Kingdom Diving Medical Committee (UKDMC). Diving Hyperb Med. 2025;55:51–5. doi: 10.28920/dhm55.1.51-55. PMID: 40090026. PMCID: PMC12043516.

16 Gempp E, Blatteau J-E, Stephant E, Pontier J-M, Constantin P, Pény C. MRI Findings and clinical outcome in 45 divers with spinal cord decompression sickness. Aviat Space Environ Med. 2008;79:1112–6. doi: 10.3357/asem.2376.2008. PMID: 19070307.

17 Panicker JN. Neurogenic bladder: epidemiology, diagnosis, and management. Semin Neurol. 2020;40:569–79. doi: 10.1055/s-0040-1713876. PMID: 33065745. PMCID: PMC9715349.

18 Patki P, Woodhouse J, Hamid R, Shah J, Craggs M. Lower urinary tract dysfunction in ambulatory patients with incomplete spinal cord injury. J Urol. 2006;175:1784–7; discussion 1787. doi: 10.1016/S0022-5347(05)00979-1. PMID: 16600760.

19 Haab F, Richard F, Amarenco G, Coloby P, Arnould B, Benmedjahed K, et al. Comprehensive evaluation of bladder and urethral dysfunction symptoms: development and psychometric validation of the urinary symptom profile (USP) questionnaire. Urology. 2008;71:646–56. doi: 10.1016/j.urology.2007.11.100. PMID: 18313122.

20 Musco S, Bazzocchi G, Martellucci J, Amato MP, Manassero A, Putignano D, et al. Treatments in neurogenic bowel dysfunctions: evidence reviews and clinical recommendations in adults. Eur J Phys Rehabil Med. 2021;56:741–55. doi: 10.23736/S1973-9087.20.06412-6. PMID: 32935955.

21 Prevention of venous thromboembolism in individuals with spinal cord injury: clinical practice guidelines for health care providers, 3rd ed. Top Spinal Cord Inj Rehabil. 2016;22:209–40. doi: 10.1310/sci2203-209. PMID: 29339863. PMCID: PMC4981016.

22 Wecht JM, Krassioukov AV, Alexander M, Handrakis JP, McKenna SL, Kennelly M, et al. International standards to document autonomic function following SCI (ISAFSCI). Top Spinal Cord Inj Rehabil. 2021;27:23–49. doi: 10.46292/sci2702-23. PMID: 34108833. PMCID: PMC8152176.
23 Lanig IS, Peterson WP. The respiratory system in spinal cord injury. Phys Med Rehabil Clin N Am. 2000;11:29–43. doi: 10.1016/S1047-9651(18)30145-1. PMID: 10680156.
24 Loh E, Mirkowski M, Agudelo AR, Allison DJ, Benton B, Bryce TN, et al. The CanPain SCI clinical practice guidelines for rehabilitation management of neuropathic pain after spinal cord injury: 2021 update. Spinal Cord. 2022;60:548–66. doi: 10.1038/s41393-021-00744-z. PMID: 35124700. PMCID: PMC9209331.
25 Khan F, Amatya B, Bensmail D, Yelnik A. Non-pharmacological interventions for spasticity in adults: An overview of systematic reviews. Ann Phys Rehabil Med. 2019;62:265–73. doi: 10.1016/j.rehab.2017.10.001. PMID: 29042299.
26 Assessment of diving site use and socioeconomic analysis of scuba diving activity along the coast of the Nice Côte d'Azur metropolitan area. [cited 2026 Feb 1]. Available from: https://www.dirm.mediterranee.developpement-durable.gouv.fr/IMG/pdf/mnca_2019_etudeplongee_version_finale.pdf.
27 Tuominen LJ, Sokolowski S, Lundell RV, Räisänen-Sokolowski AK. Decompression illness in Finnish technical divers: a follow-up study on incidence and self-treatment. Diving Hyperb Med. 2022;52:78–84. doi: 10.28920/dhm52.2.74-84. PMID: 35732278. PMCID: PMC9527095.
28 Sundal E, Lygre SHL, Irgens Å, Troland K, Grønning M. Long-term neurological sequelae after decompression sickness in retired professional divers. J Neurol Sci. 2022;434:120181. doi: 10.1016/j.jns.2022.120181. PMID: 35131550.

Acknowledgements

The authors thank the hyperbaric medical team of the Sainte-Anne Military Teaching Hospital (Toulon, France) and the neuro-urology medical team of Pitié-Salpêtrière Hospital (AP-HP, Paris, France) for their valuable expertise and clinical support.

Conflicts of interest and funding: nil

Submitted: 15 October 2025
Accepted after revision: 21 March 2026

Bipolar spectrum disorders in divers: risks, recognition, and recommendations

Abraham L Querido[1,2], Thijs T Wingelaar[2,3]

[1] *Praktijk Querido, Hilversum, the Netherlands*
[2] *Dutch Society of Diving and Hyperbaric Medicine, Bilthoven, the Netherlands*
[3] *Royal Netherlands Navy, Diving Medical Center, Den Helder, the Netherlands*

Corresponding author: *Dr Abraham L Querido, Praktijk Querido, Veerstraat 25, 1211 HJ Hilversum, the Netherlands*
bram@praktijkquerido.nl

Keywords
Bipolar disorder; Diving; Executive function; Psychotropic drugs; Risk assessment

Abstract

(Querido AL, Wingelaar TT. Bipolar spectrum disorders in divers: risks, recognition, and recommendations. Diving and Hyperbaric Medicine. 2026 30 June;56(2):170–176. doi: 10.28920/dhm56.2.170-176. PMID: 42290577.)
Bipolar disorder is a recurrent psychiatric condition characterised by episodic mood disturbances, residual functional impairment, and high rates of psychiatric and medical comorbidity. While many individuals achieve symptomatic remission, persistent deficits in cognition, emotional regulation, and insight may remain, raising concerns for participation in safety-critical activities such as scuba diving. This systematic review synthesised evidence from psychiatric, occupational, aviation, and diving medicine literature to examine the clinical course of bipolar disorder, treatment considerations, functional outcomes, and safety-relevant factors pertinent to fitness-to-dive assessments. Bipolar disorder exhibits marked heterogeneity in syndromal and functional outcomes. Even during euthymia, subtle impairments in attention, executive functioning, and decision-making may persist. Pharmacological stability is essential for diving, but treatment regimens, particularly lithium use, polypharmacy, and antidepressant therapy, introduce additional considerations. Comorbidity, circadian disruption, sleep deprivation, and reduced insight during early relapse further complicate risk assessment. Empirical data on diving outcomes in individuals with bipolar disorder are scarce, necessitating reliance on expert opinion and extrapolation from related safety-critical domains. Fitness-to-dive assessments in bipolar disorder should prioritise sustained functional stability, reliable treatment adherence, and illness insight over symptom absence alone. A cautious, individualised approach is warranted, incorporating medication effects, comorbidity, operational context, and relapse-prevention planning, supported by collaboration between mental health professionals and diving medical examiners.

Introduction

Bipolar disorder is a recurrent mood disorder characterised by episodes of mania, hypomania, and depression, with substantial heterogeneity in symptom patterns, course, and severity.[1] While many individuals achieve symptomatic remission between episodes, a significant proportion experience residual symptoms or functional limitations even during euthymia, including subtle impairments in attention, executive functioning, and emotional regulation.[2–5]

These features are directly relevant to diving safety. Mood instability, impaired judgment, impulsivity, and reduced insight, particularly during (hypo)manic phases, may compromise risk assessment and decision-making underwater. Psychiatric comorbidity, including anxiety disorders, attention deficit hyperactivity disorder (ADHD), and substance use disorders, is common and may further increase risk.[6] Additionally, irregular sleep schedules, circadian disruption, travel-related fatigue, and medication non-adherence are well-established triggers for mood destabilisation and may readily occur during dive travel.

Guidance on diving with bipolar disorder is limited. The UK Diving Medical Committee (UKDMC) recommends a minimum period of mood stability, confirmed by both psychiatrist and general practitioner, alongside treatment adherence and relapse-prevention planning.[7] Beyond this pragmatic advice, little published literature addresses risk assessment or clinical decision-making in this population. This review examines the clinical course, treatment considerations, and safety-relevant factors associated with bipolar disorder to support fitness-to-dive evaluations by diving medical examiners and mental health professionals. It focuses on recreational scuba diving; professional/commercial diving generally warrants a separate assessment framework and is beyond the scope of this article.

Methods

The literature search strategy was prepared according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. A structured search of PubMed (up to 31 December 2025) identified studies and case reports regarding diving and bipolar disorder. The initial

query yielded no results, prompting an expansion to include aviation and driving literature. The final search strategy combined terms for diving, aviation, and driving with bipolar disorder-related keywords: ((diving[Mesh] OR dive[tw] OR diving[tw] OR divers[tw] OR hyperbaric[tw] OR scuba[tw]) OR (aviation[mesh] OR flying[tw] OR altitude[tw]) OR (driving[mesh] OR driv[tw] OR traffic[tw])) AND ("*Bipolar and Related Disorders*"[Mesh] OR manic[tw] OR bipolar[tw]).

A total of 2,734 studies were screened, with 12 identified as potentially relevant. Six of these were found through the PubMed database, and another six were identified through the reference lists of these studies. Additionally, handbooks on diving medicine addressing psychiatry or psychology were also reviewed for relevant information. Further details of the search process are illustrated in the PRISMA flowchart (Figure 1).

CLASSIFICATION

Bipolar disorder encompasses a spectrum of conditions characterised by distinct mood episodes and functional impairments.[1] Bipolar I disorder is defined by at least one manic episode, marked by persistently elevated or irritable mood, increased energy, reduced need for sleep, pressured speech, impulsivity, and impaired judgment. Major depressive episodes are common but not required for diagnosis. Bipolar II disorder involves recurrent major depressive episodes alternating with hypomanic episodes, which are characterised by elevated mood and energy without psychosis or marked social or occupational impairment. Cyclothymic disorder is distinguished by chronic, fluctuating mood disturbances with hypomanic and depressive symptoms that do not meet full criteria for manic or major depressive episodes. Other specified bipolar disorders include subthreshold syndromes or bipolar symptoms secondary to substances, medications, or medical conditions. For clarity and readability the DSM-5-TR definitions of bipolar disorder are displayed in Table 1.[1]

PREVALENCE

Global prevalence estimates for bipolar disorder vary significantly depending on case definition and time frame. In the analyses from the Global Burden of Disease Study (2019) bipolar disorder had an age-standardised prevalence of approximately 0.49%, corresponding to about 39.5 million affected individuals worldwide.[8] Despite its relatively low prevalence, bipolar disorder accounts for a disproportionate share of years lived with disability, reflecting its early onset, recurrent course, and substantial functional impact.[8] Broader epidemiological estimates, including lifetime prevalence and extended diagnostic approaches, report higher figures of approximately 2–3% of the global population.[9] These estimates often incorporate bipolar spectrum conditions and

Figure 1
PRISMA 2020 flow diagram

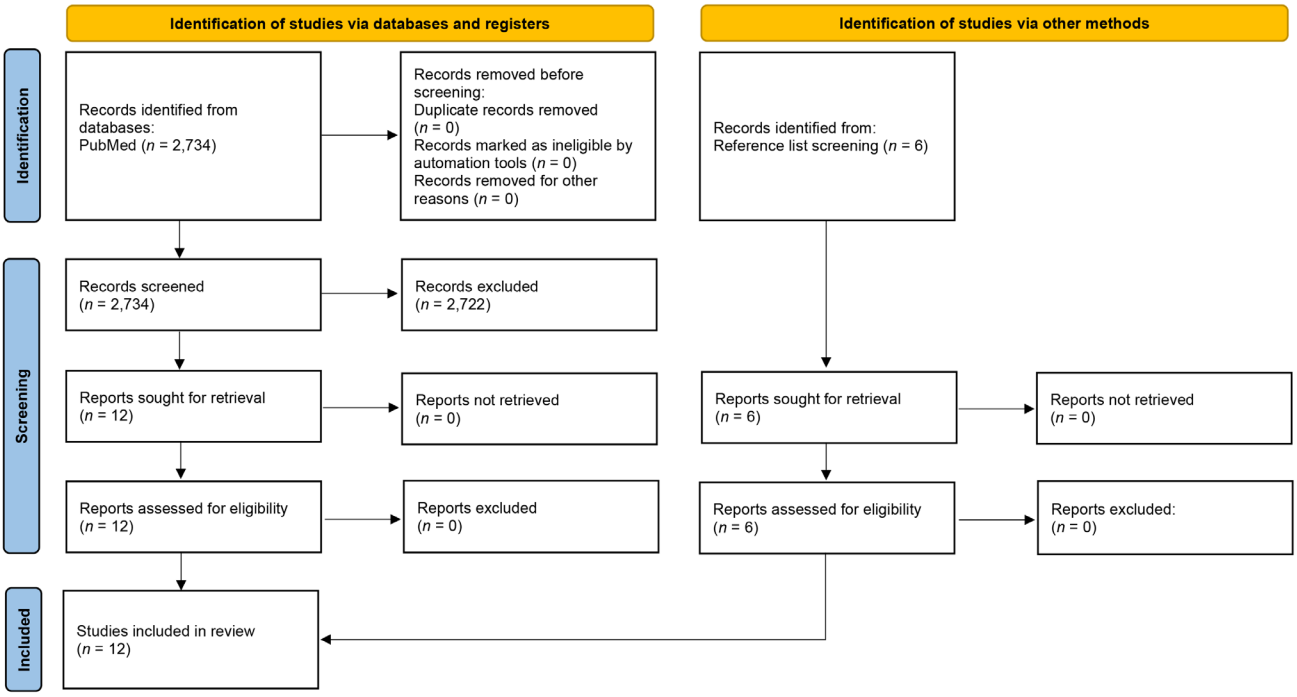


Table 1
Classification and core clinical features of bipolar disorders (DSM-5-TR)

Disorder	Core diagnostic features
Bipolar I disorder	At least one manic episode, characterised by persistently elevated or irritable mood, increased energy, reduced need for sleep, pressured speech, impulsivity, and impaired judgment. Major depressive episodes are common but not required.
Bipolar II disorder	Recurrent major depressive episodes alternating with hypomanic episodes. Hypomania involves elevated mood and energy without psychosis or marked social/occupational impairment. A major depressive episode is required for diagnosis.
Cyclothymic disorder	Chronic, fluctuating mood disturbances with hypomanic and depressive symptoms that do not meet full criteria for manic or major depressive episodes.
Other specified bipolar disorders	Subthreshold syndromes or bipolar symptoms secondary to substances/medications (e.g., stimulants, corticosteroids) or medical conditions (e.g., hyperthyroidism, epilepsy).

subthreshold presentations, better capturing the full clinical spectrum but increasing sensitivity to methodological variation. Bipolar II disorder may be under-recognised and recorded as recurrent unipolar depression; this highlights the importance of briefly screening for past hypomanic symptoms when divers report a history of 'depression', particularly if episodes were recurrent, severe, or required psychotropic treatment.[10]

CLINICAL COURSE OF BIPOLAR DISORDER

Bipolar disorder is a recurrent condition characterised by episodic mood disturbances, with considerable heterogeneity in long-term outcomes. Clinical outcomes can be described along two complementary dimensions: syndromal outcome, which refers to the presence, severity, and recurrence of mood episodes, and functional outcome, which reflects the individual's capacity to function in daily life, including occupational performance, social relationships, and overall quality of life.[5]

Cognitive impairment is a key determinant of functional outcome in bipolar disorder and often persists into euthymia; deficits are frequently observed across multiple cognitive domains, particularly executive function, verbal learning and memory, processing speed, and working memory.[2,11,12] However, cognitive outcomes are highly variable. A substantial proportion of individuals with bipolar disorder demonstrate normal or near-normal cognitive functioning, particularly in the absence of a history of psychosis, multiple manic episodes, or early illness onset.[3,4,13,14]

Bipolar disorder rarely presents in isolation. Over 50% of individuals meet criteria for additional psychiatric or medical comorbidities, including anxiety disorders, ADHD, substance use disorders, eating disorders, obesity, metabolic syndrome, and cardiovascular disease.[6,15,16] Comorbidity is associated with earlier illness onset, greater symptom burden, poorer treatment response, increased suicidality, and higher healthcare utilisation.[16]

CONSIDERATIONS IN AVIATION

In aviation medicine, mental health is considered essential to operational safety and should be actively evaluated as part of the aeromedical assessment, with emphasis on the identification of common and potentially detectable mental health conditions rather than on routine in-depth psychiatric testing.[17]

Fear of losing one's licence or livelihood may discourage pilots from disclosing medical or psychiatric symptoms during aeromedical assessments.[18] Survey data indicate that aircraft pilots may engage in healthcare avoidance behaviour because of concerns about aeromedical certificate loss, resulting in under-reporting of conditions relevant to flight safety.[18,19] Bipolar disorder is traditionally regarded as incompatible with flight duties due to the potential for episodic and unpredictable cognitive and behavioural impairment, which may occur even during prolonged asymptomatic periods despite treatment. Retrospective analyses identified only two fatal general aviation accidents in the United States between 1994 and 2014 in which the pilot had a probable diagnosis of bipolar disorder, forming the sole basis for incidence estimates (0.023%).[20] The Germanwings Flight 9525 crash in 2015 further illustrated the challenges of self-reporting and post hoc psychiatric assessment in safety-critical professions.[21] Additional aviation safety reports have described fatal accidents in which psychotropic medications used in bipolar disorder, including lithium and antidepressants, were detected in postmortem toxicology, such as a Canadian case involving lithium and a separate report of fluoxetine use in a pilot with a documented bipolar diagnosis.[22,23] However, insufficient clinical information precluded confirmation of psychiatric

diagnosis or causal inference, and these cases were therefore not included in incidence estimates.

While direct evidence in diving is limited, aviation-based safety reasoning provides a useful framework for clinical risk assessment in diving medicine, where self-certification, limited rescue options, and exposure to physiological and psychological stressors may amplify the consequences of sudden incapacitation.

CONSIDERATIONS IN DRIVING

In a controlled simulator study, euthymic outpatients with bipolar disorder exhibited significant impairments in car-following and road-tracking tasks compared to healthy controls, with sustained attention identified as the primary cognitive domain associated with impaired performance.[24] Supporting evidence from a small controlled study revealed longer reaction times and poorer performance in attention, executive functioning, and psychomotor speed among remitted patients, correlating with driving-related task execution.[25] An epidemiological study further reported a 1.66-fold increased risk of road traffic injury in individuals with bipolar disorder. These findings suggest that subtle cognitive deficits may persist during euthymia, with implications for activities requiring sustained vigilance and complex task management.[26]

PHARMACOLOGICAL CONSIDERATIONS

Pharmacological maintenance treatment for bipolar disorder primarily involves mood stabilisers, including lithium and anticonvulsants such as valproate, carbamazepine, and lamotrigine, alongside second-generation antipsychotics. Adjunctive antidepressants are employed selectively, though their use remains contentious. Despite the availability of multiple evidence-based therapies, achieving sustained long-term euthymia presents a significant clinical challenge, particularly in patients with rapid cycling, high comorbidity, or suboptimal treatment adherence.

Lithium use in divers raises specific concerns due to the potential for fluid balance disruption during diving.[27] Breathing dry compressed gas, immersion-induced diuresis, and increased sweating in warm environments may promote dehydration, thereby destabilising serum lithium concentrations within its narrow therapeutic range. Although post-dive rehydration may partially mitigate these effects, fluctuations in volume status elevate the risk of lithium toxicity. Consequently, the use of lithium in divers necessitates a cautious and individualised risk assessment rather than routine approval for diving activities.

Anticonvulsant mood stabilisers, such as valproate and carbamazepine, are generally regarded as effective and reasonably well-tolerated, though they are frequently associated with gastrointestinal discomfort, weight gain, sedation, and tremor.[28,29] Lamotrigine, by contrast, is typically well-tolerated, with a side-effect profile in some studies comparable to placebo.[30] Diving while undergoing treatment with low-dose second-generation antipsychotics may be considered acceptable in carefully selected cases, provided there are no significant side effects or cardiovascular risk factors.[27]

Depressive symptoms represent the most persistent and disabling aspect of bipolar disorder, particularly in bipolar I disorder, with residual symptoms persisting between episodes in approximately one-third of patients.[31] These symptoms substantially impair quality of life and functional outcomes. The role of antidepressants in bipolar disorder remains controversial, especially in individuals with bipolar I disorder, rapid cycling, or a history of antidepressant-induced mood elevation. In clinical practice, antidepressant treatment in bipolar disorder usually occurs in combination with mood stabilisers and is most often considered in patients with persistent or clinically severe depressive symptoms, reflecting a more complex illness course.[32] From a diving medicine perspective, the combination of illness severity, polypharmacy, central nervous system effects, and the potential for mood destabilisation raises significant safety concerns. As such, ongoing antidepressant treatment in individuals with bipolar disorder is generally considered incompatible with recreational scuba diving.

FITNESS-TO-DIVE RECOMMENDATIONS

From a diving medicine perspective, pharmacological stability represents a fundamental prerequisite for assessing fitness to dive in individuals with bipolar disorder. This necessitates the absence of recent dose adjustments or medication changes, consistent treatment adherence, and sustained clinical stability without significant adverse effects. Stability in medication regimens, characterised by unchanged drugs and dosages without recent titration or rescue interventions, is essential. However, such stability is contingent upon critical clinical preconditions, including adequate illness insight, diagnostic acceptance, comprehensive psychoeducation, a robust therapeutic alliance, and reliable adherence. The absence of insight or adherence poses a substantial safety risk, even in the presence of minimal mood symptoms.

In practice, inadequate insight or unreliable adherence may be indicated by relapse linked to non-adherence, repeated emergency interventions or hospitalisations, and/or a history of involuntary admission; these are high-risk markers in fitness-to-dive decisions.

Given the lack of empirical data defining the precise duration of stability required for safe diving, recommendations are based on expert consensus. Drawing parallels with diving

medicine guidelines for epilepsy, which emphasise prolonged seizure-free and treatment-stable periods, recreational scuba diving may be considered only for individuals who have maintained euthymia, treatment adherence, and pharmacological stability for an extended period, typically several years, without hospitalisations, major mood episodes, or clinically significant adverse effects. Notably, treatment complexity (including polypharmacy or frequent regimen changes) should be interpreted primarily as a marker of illness severity or instability; the central requirement remains sustained adherence and reliable self-regulation on a stable plan over a prolonged period, potentially several years in higher-risk histories. This conservative approach reflects the potential severity of mood destabilisation and the limited capacity for managing psychiatric emergencies in underwater environments.

Fitness-to-dive assessments should prioritise functional recovery over mere symptom absence, as residual cognitive or emotional dysregulation may persist even during euthymia, and early relapse may impair judgment and insight. Diving should be deferred during any period of psychotropic medication adjustment, including initiation, dose modifications, or discontinuation, as these changes disrupt stability.

Eligibility for diving further requires sustained psychosocial functioning, absence of comorbid conditions that could compromise safety, robust illness insight with early warning sign recognition, a well-defined relapse-prevention strategy, and consistent treatment adherence. Neurocognitive impairments and comorbidities must be evaluated holistically rather than in isolation.

The operational diving context also plays a critical role. Factors such as remote locations, irregular sleep patterns, jet lag, and high task demands may elevate relapse risk, necessitating additional caution or restrictions. Safe diving participation demands not only symptom absence but also demonstrated, enduring functional stability and self-regulatory capacity, supported by close collaboration among the treating psychiatrist, general practitioner, and diving medical examiner.

LIMITATIONS AND FUTURE DIRECTIONS

This review underscores key considerations for assessing fitness to dive in individuals with bipolar disorder, yet several critical limitations and knowledge gaps persist. Current recommendations are largely derived from clinical experience, expert consensus, and extrapolation from related fields such as epilepsy, aviation medicine, and occupational psychiatry, rather than empirical data specific to divers with bipolar disorder. Prospective studies examining relapse risk, incident rates, and functional outcomes in this population remain lacking.

There is a lack of direct evidence regarding the effects of hyperbaric exposure and diving-related stressors, including narcosis, decompression stress, immersion-induced physiological changes, and post-dive fatigue, on mood stability or relapse risk in bipolar disorder. Similarly, the impact of circadian disruption, sleep deprivation, and long-haul travel, all established triggers for mood episodes, has not been systematically investigated in diving contexts.

Data on the behaviour of psychotropic medications under hyperbaric conditions are sparse. While modern mood stabilisers, antipsychotics, and antidepressants are generally well-tolerated, their pharmacokinetic and pharmacodynamic interactions with diving-specific stressors remain poorly understood. Additionally, the functional implications of residual neurocognitive deficits during euthymia, particularly concerning situational awareness, decision-making, and underwater emergency response, have yet to be quantified.

Current fitness-to-dive evaluations often rely on broad diagnostic classifications rather than individualised risk profiles that integrate illness trajectory, comorbidity, treatment stability, adherence, and psychosocial functioning. The scarcity of documented bipolar-related diving incidents further complicates risk estimation, potentially reflecting both low incidence and underreporting. Future research, including structured cohort studies, detailed case analyses, and multicentre registries, is essential to refine risk stratification and develop precise, personalised recommendations.

Conclusions

Fitness-to-dive assessments in individuals with bipolar disorder must prioritise functional stability, treatment adherence, and illness insight over symptom absence alone. A cautious, individualised approach, integrating medication effects, comorbidity, and operational context, is essential. Collaboration between mental health professionals and diving medical examiners is critical to ensure safe participation in diving activities.

References

1 American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-5-TR. 5th ed, text rev. Washington (DC): American Psychiatric Association; 2022.

2 Cardenas SA, Kassem L, Brotman MA, Leibenluft E, McMahon FJ. Neurocognitive functioning in euthymic patients with bipolar disorder and unaffected relatives: A review of the literature. Neurosci Biobehav Rev. 2016;69:193–215. doi: 10.1016/j.neubiorev.2016.08.002. PMID: 27502749. PMCID: PMC5030183.

3 Miskowiak KW, Burdick KE, Martinez-Aran A, Bonnin CM, Bowie CR, Carvalho AF, et al. Assessing and addressing cognitive impairment in bipolar disorder: the International

Society for Bipolar Disorders Targeting Cognition Task Force recommendations for clinicians. Bipolar Disord. 2018;20(3):184–94. doi: 10.1111/bdi.12595. PMID: 29345040.

4 Van Rheenen TE, Lewandowski KE, Bauer IE, Kapczinski F, Miskowiak K, Burdick KE, et al. Current understandings of the trajectory and emerging correlates of cognitive impairment in bipolar disorder: An overview of evidence. Bipolar Disord. 2020;22:13–27. doi: 10.1111/bdi.12821. PMID: 31408230.

5 Gitlin MJ, Miklowitz DJ. The difficult lives of individuals with bipolar disorder: A review of functional outcomes and their implications for treatment. J Affect Disord. 2017;209:147–54. doi: 10.1016/j.jad.2016.11.021. PMID: 27914248. PMCID: PMC7213058.

6 Hirschfeld RM, Vornik LA. Bipolar disorder-costs and comorbidity. Am J Manag Care. 2005;11(3 Suppl):S85–90. PMID: 16097719.

7 UK Diving Medical Committee (UKDMC). Bipolar disorder. [cited 2025 Dec 31]. Available from: https://www.ukdmc.org/medical-conditions/bipolar-disorder/.

8 GBD 2019 Mental Disorders Collaborators. Global, regional, and national burden of 12 mental disorders in 204 countries and territories, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet Psychiatry. 2022;9:137–50. doi: 10.1016/S2215-0366(21)00395-3. PMID: 35026139. PMCID: PMC8776563.

9 Zhong Y, Chen Y, Su X, Wang M, Li Q, Shao Z, et al. Global, regional and national burdens of bipolar disorders in adolescents and young adults: a trend analysis from 1990 to 2019. Gen Psychiatr. 2024;37(1):e101255. doi: 10.1136/gpsych-2023-101255. PMID: 38390238. PMCID: PMC10882284.

10 Swartz HA, Suppes T. Bipolar II Disorder: Understudied and underdiagnosed. Focus (Am Psychiatr Publ). 2023;21:354–62. doi: 10.1176/appi.focus.20230015. PMID: 38694998. PMCID: PMC11058947.

11 Solé B, Jiménez E, Torrent C, Reinares M, Bonnin CDM, Torres I, et al. Cognitive impairment in bipolar disorder: treatment and prevention strategies. Int J Neuropsychopharmacol. 2017;20:670–80. doi: 10.1093/ijnp/pyx032. PMID: 28498954. PMCID: PMC5570032.

12 Miskowiak KW, Burdick KE, Martinez-Aran A, Bonnin CM, Bowie CR, Carvalho AF, et al. Methodological recommendations for cognition trials in bipolar disorder by the International Society for Bipolar Disorders Targeting Cognition Task Force. Bipolar Disord. 2017;19:614–26. doi: 10.1111/bdi.12534. PMID: 28895274. PMCID: PMC6282834.

13 Cardoso T, Bauer IE, Meyer TD, Kapczinski F, Soares JC. Neuroprogression and cognitive functioning in bipolar disorder: a systematic review. Curr Psychiatry Rep. 2015;17:75. doi: 10.1007/s11920-015-0605-x. PMID: 26257147.

14 Bora E. Neurocognitive features in clinical subgroups of bipolar disorder: A meta-analysis. J Affect Disord. 2018;229:125–34. doi: 10.1016/j.jad.2017.12.057. PMID: 29306692.

15 Albanese MJ, Nelson SE, Peller AJ, Shaffer HJ. Bipolar disorder as a risk factor for repeat DUI behavior. J Affect Disord. 2010;121:253–7. doi: 10.1016/j.jad.2009.06.015. PMID: 19596447.

16 De Berardis D, Fornaro M, Carmassi C. Editorial: Comorbidity in bipolar disorder, volume II. Front Psychiatry. 2023;14:1115357. doi: 10.3389/fpsyt.2023.1115357. PMID: 36926462. PMCID: PMC10011613.

17 Aerospace Medical Association Working Group on Pilot Mental Health. Pilot mental health: Expert working group recommendations - Revised 2015. Aerosp Med Hum Perform. 2016;87:505–7. doi: 10.3357/AMHP.4568.2016. PMID: 27099091.

18 Bor R, Eriksen C, Oakes M, Scragg P, editors. Pilot mental health assessment and support: a practitioner's guide. 1st ed. London: Routledge; 2016. doi:10.4324/9781315401942.

19 Hoffman WR, Aden J, Barbera RD, Mayes R, Willis A, Patel P, et al. Healthcare avoidance in aircraft pilots due to concern for aeromedical certificate loss: a survey of 3765 pilots. J Occup Environ Med. 2022;64(4):e245–e248. doi: 10.1097/JOM.0000000000002519. PMID: 35166258.

20 Vuorio A, Laukkala T, Navathe P, Budowle B, Bor R, Sajantila A. Bipolar disorder in aviation medicine. Aerosp Med Hum Perform. 2017;88:42–7. doi: 10.3357/AMHP.4620.2017. PMID: 28061921.

21 Aerospace Medical Association. Responses related to March 24, 2015, Germanwings Airbus 320 crash. 2015. [cited 2025 Dec 31]. Available from: https://www.asma.org/publications/pilot-mental-health.

22 Sen A, Akin A, Canfield DV, Chaturvedi AK. Medical histories of 61 aviation accident pilots with postmortem SSRI antidepressant residues. Aviat Space Environ Med. 2007;78:1055–9. doi: 10.3357/asem.2030.2007. PMID: 18018438.

23 The Transportation Safety Board of Canada. Aviation occurrence report A97O0096. Ontario (Canada): Transportation Safety Board of Canada; 1997.

24 Yamaguchi A, Iwamoto K, Ando M, Fujita K, Yokoyama M, Akiyama T, et al. Driving performance of euthymic outpatients with bipolar disorder undergoing real-world pharmacotherapy. Psychiatry Clin Neurosci. 2022;76:172–8. doi: 10.1111/pcn.13332. PMID: 35037335.

25 Joachimiak P, Jaracz K, Jaracz J. Neuropsychological exponents for the driving ability in remitted bipolar patients. Int J Bipolar Disord. 2022;10:2. doi: 10.1186/s40345-021-00247-z. PMID: 35066647. PMCID: PMC8784581.

26 Chen VC, Yang YH, Lee CP, Wong J, Ponton L, Lee Y, et al. Risks of road injuries in patients with bipolar disorder and associations with drug treatments: a population-based matched cohort study. J Affect Disord. 2018;226:124–31. doi: 10.1016/j.jad.2017.09.029. PMID: 28972929.

27 Querido AL, Ebbelaar CF, Wingelaar TT. Diving with psychotropic medication: review of the literature and clinical considerations. Diving Hyperb Med. 2023;53:259–67. doi: 10.28920/dhm53.3.259-267. PMID: 37718301. PMCID: PMC10735636.

28 Haddad PM, Das A, Ashfaq M, Wieck A. A review of valproate in psychiatric practice. Expert Opin Drug Metab Toxicol. 2009;5:539–51. doi: 10.1517/17425250902911455. PMID:19409030.

29 Koliqi R, Polidori C, Islami H. Prevalence of side effects treatment with carbamazepine and other antiepileptics in patients with epilepsy. Mater Sociomed. 2015;27:167–71. doi: 10.5455/msm.2015.27.167-171. PMID: 26236162. PMCID: PMC4499297.

30 Bowden CL, Asnis GM, Ginsberg LD, Bentley B, Leadbetter R, White R. Safety and tolerability of lamotrigine for bipolar disorder. Drug Saf. 2004;27:173–84. doi: 10.2165/00002018-200427030-00002. PMID: 14756579.

31 Morsel AM, Morrens M, Sabbe B. An overview of pharmacotherapy for bipolar I disorder. Expert Opin Pharmacother. 2018;19:203–22. doi: 10.1080/14656566.2018.1426746. PMID: 29361880.

32 Pardossi S, Fagiolini A, Cuomo A. Antidepressants in bipolar depression: From neurotransmitter mechanisms to clinical challenges. Actas Esp Psiquiatr. 2025;53:621–31. doi: 10.62641/aep.v53i3.1880. PMID: 40355996. PMCID: PMC12069913.

Conflicts of interest and funding: nil

Submitted: 23 January 2026
Accepted after revision: 12 March 2026

Evidence-informed decision aid for fitness-to-dive assessment after otologic surgery

Juan Riestra-Ayora[1,2], Carlos Fernández-Navarro[2], Eduardo Martín-Sanz[1,2], Manuel F Salvador-Marín[3,4]

[1] *Department of Medicine, Faculty of Biomedical and Health Sciences, Universidad Europea de Madrid, Villaviciosa de Odón, Madrid, Spain*
[2] *Department of Otolaryngology-Head and Neck Surgery, Hospital Universitario de Getafe, Getafe, Madrid, Spain*
[3] *Department of Hyperbaric Medicine, Hospital General Universitario de Castellón, Castellón de la Plana, Castellón, Spain*
[4] *Department of Medicine, Faculty of Health Sciences, Universitat Jaume I, Castellón de la Plana, Spain*

Corresponding author: *Dr Juan Riestra-Ayora, Department of Otolaryngology-Head and Neck Surgery, Hospital Universitario de Getafe, Getafe, 28905, Madrid, Spain*
ORCiD: *0000-0003-0297-2711*
juan.riestra@hotmail.com

Keywords
Diving; Barotrauma; Cochlear implants; Ear diseases; Ear, middle; Otorhinolaryngologic surgical procedures; Stapedectomy

Abstract
(Riestra-Ayora J, Fernández-Navarro C, Martín-Sanz E, Salvador-Marín MF. Evidence-informed decision aid for fitness-to-dive assessment after otologic surgery. Diving and Hyperbaric Medicine. 2026 30 June;56(2):177–184. doi: 10.28920/dhm56.2.177-184. PMID: 42290578.)
Introduction: Fitness-to-dive after otologic surgery is often approached conservatively, with some procedures historically labelled as absolute contraindications despite limited empirical evidence. The available literature is heterogeneous and includes clinical reports, experimental pressure studies, guidance documents, and manufacturer specifications, leading to uncertainty in clinical counseling. We aimed to characterise the available evidence regarding fitness-to-dive after otologic surgery and to develop an evidence-informed clinical decision aid.
Methods: A scoping review was conducted in accordance with PRISMA-ScR guidance. PubMed/MEDLINE, Embase, Scopus, and relevant non-indexed sources were searched. Eligible sources included clinical reports and series, experimental or hyperbaric chamber studies, guidance or consensus documents, and manufacturer statements providing explicit pressure- or depth-related information. Data were charted descriptively by procedure type and evidence stream.
Results: The search identified 324 records; after removal of duplicates and screening, 40 sources were included. The evidence base was predominantly non-comparative. Across procedures, recommendations emphasised postoperative stability and reliable pressure equalisation rather than surgical history alone. Canal wall down mastoidectomy was consistently portrayed as incompatible with diving, whereas selected middle ear reconstructions and stapes surgery were commonly described as potentially compatible in appropriately selected individuals. For cochlear implantation, guidance was mainly conditional and based on hyperbaric testing, limited clinical diving reports, and manufacturer-specified pressure or depth limits. Communication emerged as an additional practical consideration in cases of significant hearing loss.
Conclusions: Relevant evidence is limited and heterogeneous, and does not consistently support blanket prohibitions for all otologic procedures. A function-based, individualised approach is supported, while specific higher-risk scenarios warrant restriction. Prospective registries and standardised outcome reporting are needed to refine procedure-specific recommendations.

Introduction

Exposure to pressure changes during scuba diving poses unique physiological challenges to the auditory and vestibular systems. Middle-ear pressure equalisation, inner-ear pressure transmission, and device integrity are critical considerations for diver safety.[1] As a result, a range of otologic conditions and prior surgical procedures have historically been considered contraindications to diving.[2,3] These recommendations are often conservative and variably defined, reflecting the limited empirical data available to support clear thresholds of risk.[4–6]

Otologic surgery encompasses a broad spectrum of procedures, including tympanoplasty and middle-ear reconstruction, ossiculoplasty, mastoidectomy, stapes surgery, and cochlear implantation. Patients undergoing these procedures increasingly seek guidance regarding return to recreational or occupational diving, where requirements may be more stringent in professional settings.[7] However,

existing recommendations are inconsistent, ranging from absolute prohibition to conditional clearance based on healing status, Eustachian tube function, or device-specific considerations.[8,9]

Given the heterogeneity of the literature and the absence of randomised or large prospective studies, a scoping review is an appropriate methodological approach to map the available evidence, clarify the nature of existing recommendations, and identify gaps requiring further research. The present scoping review aims to systematically characterise the literature addressing fitness-to-dive after otologic surgery, with a focus on pressure exposure, reported otologic outcomes, and practical postoperative guidance.

Methods

STUDY DESIGN AND REPORTING

This scoping review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR).[10] A review protocol defining objectives, eligibility criteria, information sources, and planned data charting was finalised prior to screening and prospectively registered in the Open Science Framework.[11]

ELIGIBILITY CRITERIA

Eligibility criteria were defined using the Population-Concept-Context framework.

The pre-defined population of interest was individuals with a history of otologic surgery, including middle-ear reconstruction procedures and cochlear implantation. The concept explored was fitness-to-dive (recreational and occupational) and safety under pressure exposure, including scuba diving and controlled hyperbaric chamber exposure. Eligible sources addressed postoperative recommendations, depth or pressure limits, contraindications, or adverse otologic outcomes attributable to pressure changes.

Clinical studies (case reports, case series, and observational studies), experimental or hyperbaric chamber studies, clinical guidelines or expert consensus documents, and manufacturer documents were eligible for inclusion. Manufacturer documents were included only when they provided explicit statements regarding pressure exposure, depth limits, or device integrity relevant to diving. Publications unrelated to diving or pressure exposure, aviation-only contexts without relevance to diving, and sources without accessible full text were excluded.

In addition to sources meeting eligibility criteria and included in formal evidence charting, selected references (e.g., general reviews, physiology-focused studies, and broad guidance documents) were used to contextualise findings and support interpretation. These sources were not considered part of the included evidence dataset and are therefore not individually represented in procedure-specific tables.

INFORMATION SOURCES

The following bibliographic databases were searched: PubMed/MEDLINE, Embase, and Scopus. In addition, non-indexed sources relevant to diving medicine were reviewed, including Google Scholar, professional diving medicine resources, and selected manufacturer documentation when applicable.

SEARCH STRATEGY

Search strategies combined diving-related terms (e.g., scuba, diving, hyperbaric, pressure exposure, barotrauma) with otologic surgery terms (e.g., tympanoplasty, ossiculoplasty, stapedotomy, mastoidectomy, cochlear implant). Search strategies were adapted for each database. Google Scholar was queried using predefined search strings, and the first 200 results per query were screened. Reference lists of included sources were hand-searched to identify additional eligible records. The final search was completed on 4 February 2026.

SELECTION OF SOURCES OF EVIDENCE

Records were deduplicated prior to screening. Two reviewers independently screened titles and abstracts for relevance. Full-text review was performed for potentially relevant records. Discrepancies were resolved by consensus.

DATA CHARTING PROCESS

Data were extracted using a piloted standardised charting form. Extracted variables included: type of evidence (clinical, experimental, guidance, manufacturer), otologic procedure category, pressure exposure modality (scuba diving versus hyperbaric chamber), exposure characteristics (maximum depth or pressure when reported), timing from surgery to exposure, reported otologic outcomes, and postoperative recommendations related to diving.

DATA SYNTHESIS

Data were synthesised descriptively and organised by otologic procedure and evidence stream. Quantitative pooling or meta-analysis was not performed.

USE OF ARTIFICIAL INTELLIGENCE

Artificial intelligence tools (Microsoft Copilot) were used only to assist with language revision and did not contribute to study conception, methodology, data handling, or conclusions.

Results

SEARCH RESULTS

The search identified 324 records, including 177 from bibliographic databases and 147 from other sources. In the database search, 78 duplicate records and one record removed for other reasons were excluded before screening, leaving 98 records for title and abstract screening; 28 were excluded at this stage. Seventy reports were sought for retrieval, of which six were not retrieved, leaving 64 reports assessed for eligibility. In the other-sources search, 147 reports were sought for retrieval, of which 17 were not retrieved, leaving 130 reports assessed for eligibility. Overall, 194 reports were assessed for eligibility across both search approaches, and 40 met eligibility criteria for inclusion in data charting. The study selection process is summarised in the PRISMA flow diagram (Figure 1).

OVERVIEW OF INCLUDED EVIDENCE

Across the 40 included sources, the evidence base was heterogeneous and predominantly non-comparative. Clinical evidence consisted mainly of case reports, small case series, and retrospective surveys describing return to diving after otologic surgery.

Experimental evidence included hyperbaric chamber testing of cochlear implant devices and hyperbaric chamber exposure of patients with these devices implanted.[12] Additional sources comprised expert guidance documents from diving medicine organisations, narrative reviews addressing pressure-related otologic injury, and manufacturer specifications detailing pressure or depth tolerance of implantable devices.

An overview of the available evidence by surgical category and evidence stream is provided in Table 1, with detailed source-level information available in *Supplementary Tables S1–S3.

MIDDLE EAR SURGERY: TYMPANOPLASTY AND OSSICULOPLASTY

Evidence addressing fitness-to-dive after tympanoplasty and ossicular reconstruction was derived primarily from expert guidance and narrative clinical sources. Across these sources, the central determinant of dive fitness was not the surgical label itself but postoperative middle-ear stability. Procedures resulting in an intact, well-healed tympanic membrane, a dry ear, and preserved Eustachian tube function were generally considered compatible with a return to recreational diving.[6,13–15]

Ossicular reconstruction using partial ossicular replacement prostheses (PORP) was consistently described as having a pressure tolerance comparable to that of a non-operated ear when middle-ear aeration was adequate. In contrast, total ossicular replacement prostheses (TORP) were

Note: *Supplementary Tables S1–S3 can be found on our website: https://www.dhmjournal.com/index.php/journals?id=419

Figure 1
PRISMA flow diagram of the scoping review

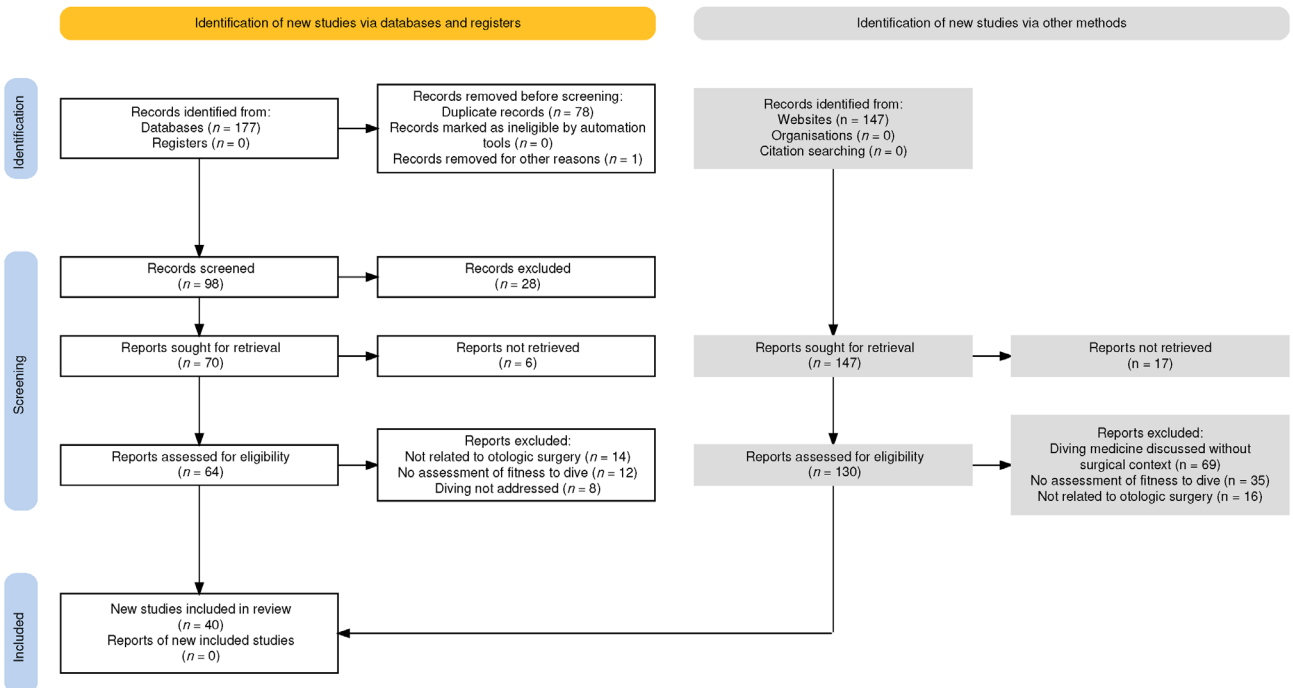


Table 1

Summary of evidence and fitness-to-dive considerations by otologic procedure; this table synthesises the available clinical, experimental, guidance, and manufacturer evidence regarding fitness to dive after different otologic procedures. For each procedure category, the predominant evidence base, key reported findings, and typical recommendations related to postoperative diving are summarised. Procedure and source-level details, including reported depth or pressure limits and device-specific considerations, are provided in *Supplementary Tables S1–S3. PORP – partial ossicular replacement prosthesis; TORP – total ossicular replacement prosthesis

Procedure category	Evidence base in this review	Key reported findings	Typical recommendations reported
Tympanoplasty/middle-ear reconstruction	Mainly expert guidance and narrative clinical sources	Fitness to dive relates primarily to postoperative stability rather than the surgical label.	Conditional clearance after healing; emphasis on intact tympanic membrane and reliable equalisation.
Ossiculoplasty (PORP)	Guidance and narrative sources	Generally described as pressure-tolerant when reconstruction and aeration are stable.	Conditional clearance after healing; individualised assessment.
Ossiculoplasty (TORP)	Guidance and narrative sources	Repeatedly described as potentially more vulnerable to pressure-related mechanical stress.	More conservative approach; greater caution and individualised restriction.
Mastoidectomy (canal wall up)	Guidance and narrative sources	Not usually considered an absolute contraindication once healing and ventilation are stable.	Conditional clearance after healing and dry ear status.
Mastoidectomy (canal wall down)	Guidance and narrative sources	Consistently framed as higher risk due to impaired pressure equilibration.	Generally considered unfit for scuba diving.
Stapes surgery	Retrospective surveys and small clinical series	Most reported divers resumed recreational diving without persistent sequelae when equalisation was adequate.	Not supported as an absolute contraindication; conditional clearance with precautions.
Cochlear implantation	Mixed: chamber testing, clinical reports, guidance, manufacturer data	Preserved device integrity/function reported within specified pressure ranges.	Device-specific depth/pressure limits; conditional clearance.

repeatedly cited as more vulnerable to pressure-related mechanical stress, with expert sources suggesting lower rupture thresholds and recommending greater caution or restriction.[8,9,15,16]

Canal wall up (CWU) mastoidectomy, in which the posterior wall of the external auditory canal is preserved, and near-normal middle-ear anatomy is maintained, aims to restore a closed tympanomastoid system with physiological sound conduction and pressure transmission. In the available literature, CWU procedures were variably addressed but were generally not considered an absolute contraindication to diving once complete epithelialisation, stable tympanic membrane healing, and reliable middle-ear ventilation had been achieved.[8,9,15]

In contrast, canal wall down (CWD) mastoidectomy involves the removal of the posterior canal wall, creating a permanently open mastoid cavity that communicates directly with the external environment. This altered anatomy may impair effective pressure equalisation and increase exposure of the middle ear and mastoid cavity to water and thermal stress. For these reasons, CWD mastoidectomy has traditionally been regarded as a relative or absolute contraindication to diving.[8,9,15] However, some authors, as reported by Mallen et al., suggest that it may not represent an

absolute contraindication in highly selected cases, provided additional functional assessment, including cold air or water testing, excludes vestibular sensitivity.[8]

STAPES SURGERY

Clinical evidence related to stapedectomy and stapedotomy consisted mainly of retrospective surveys and small clinical series describing divers who resumed scuba diving after surgery, as summarised in expert and guidance sources.[17–19] Across these reports, most individuals returned to recreational diving without persistent audiovestibular sequelae when middle-ear pressure equalisation was reliable.[8,15,20,21]

Reported adverse effects were generally mild and transient, including otalgia during descent or short-lived vertigo. Serious inner ear complications such as perilymphatic fistula were rarely documented and typically discussed as isolated events rather than systematic findings. None of the included sources provided robust evidence for an absolute contraindication to diving based solely on prior stapes surgery. Instead, recommendations emphasised individualised assessment, healed middle-ear status, and absence of vestibular symptoms.[16,17,22]

COCHLEAR IMPLANTATION

Evidence concerning cochlear implantation and diving was more structured and included both experimental and clinical data. Hyperbaric chamber testing reported preserved structural integrity and functional stability of cochlear implants exposed to pressures equivalent to depths of approximately 50 m (about six atmospheres absolute pressure), without mechanical damage to implant housings or electrode arrays. In vivo hyperbaric chamber studies involving implanted patients similarly reported no clinically relevant changes in device impedances or function, with only mild and transient discomfort reported.[23–26]

Clinical diving data were limited but consistent. Case reports and small series described implanted individuals completing multiple recreational dives at depths ranging from approximately 28 m to 43 m without deterioration of implant performance or auditory outcomes. These observations were reinforced by narrative reviews summarising manufacturer recommendations and available clinical experience.[24–26]

Manufacturer specifications varied by device platform but generally defined maximum pressure or depth limits rather than absolute prohibitions.[27–29] Across sources, recommendations for divers with cochlear implants were predominantly conditional, emphasising adherence to device-specific limits and the absence of surgical or vestibular complications rather than categorical exclusion from diving.[8,30]

HYPERBARIC AND PRESSURE-RELATED OTOLOGIC EVIDENCE

A limited number of included sources addressed otologic responses to pressure exposure in hyperbaric or experimental contexts without focusing on diving specifically. These studies provided a physiological framework for understanding middle and inner ear behavior under pressure, including the role of gas compression, intracochlear pressure transmission, and vulnerability of the round and oval windows.[20,31–33]

Although not designed to assess post-surgical dive fitness directly, this body of evidence supports the biological plausibility of pressure tolerance in stable postoperative ears and implant systems, while also highlighting mechanisms through which pressure-related injury may occur in the presence of impaired equalisation or structural instability.[15,20] Bone-anchored hearing systems were also considered within this review, with limited data suggesting a low risk of pressure-related complications, although they remain a relative contraindication due to the lack of robust evidence.[8,15]

Discussion

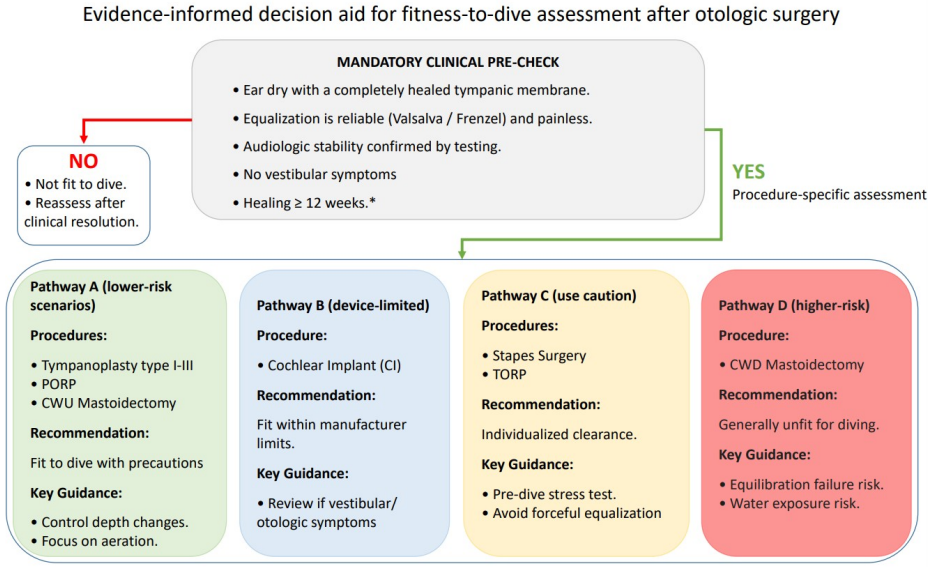
To our knowledge, this represents one of the first structured, evidence-informed clinical decision aids specifically addressing fitness-to-dive after otologic surgery. This scoping review highlights that fitness-to-dive after otologic surgery is rarely supported by evidence-based absolute contraindications.[34] Instead, the available literature consistently frames risk in functional and contextual terms rather than by surgical history alone.[35] Across procedure categories, postoperative stability, middle-ear ventilation, and the ability to equalise pressure reliably emerge as central determinants of safe diving.[8,15]

A key finding of this review is the disconnect between traditional exclusionary recommendations and the empirical evidence available. For several procedures commonly regarded as incompatible with diving, including stapes surgery and selected middle-ear reconstructions, the literature predominantly consists of small clinical series and retrospective surveys reporting return to recreational diving without persistent audiovestibular sequelae in carefully selected individuals. Although the quality of evidence remains limited, these observations do not support blanket prohibitions based solely on prior surgery. Across multiple sources, a consistent practical recommendation is to defer return to diving for approximately three months following middle ear surgery, provided that complete healing, normal tympanic membrane integrity, and adequate middle-ear ventilation are confirmed.[8,22,36,37]

Conversely, specific surgical scenarios are repeatedly identified as higher-risk contexts. CWD mastoidectomy is consistently described as incompatible with diving due

Figure 2

Evidence-informed decision aid for fitness-to-dive assessment after otologic surgery; this figure presents a structured clinical pathway for postoperative fitness-to-dive assessment based on functional criteria rather than surgical labels alone. It integrates mandatory clinical pre-checks, procedure-specific risk stratification, and device-related considerations to support individualised decision-making. CWD – canal wall down; CWU – canal wall up; PORP – partial ossicular replacement prosthesis; TORP – total ossicular replacement prosthesis



to impaired pressure equilibration and water exposure risk.[15,16] Similarly, certain ossicular reconstructions, particularly total ossicular replacement prostheses, are portrayed as potentially more vulnerable to pressure-related mechanical stress, warranting a more conservative and individualised approach.[5,8] These distinctions underline the importance of procedure-specific rather than surgery-generic counseling. In cases of uncertainty, a cautious return to diving under controlled conditions has been suggested, allowing assessment of pressure equalisation and symptom tolerance before resuming unrestricted diving activity.[35] In addition to clinical assessment, several sources recommend objective functional testing before return to diving following selected procedures, including stapes surgery or cochlear implantation. This may include tympanometry to confirm middle-ear status and vestibular evaluation, such as the video head impulse test or videonystagmography.[8,22]

Stapes surgery represents a distinct scenario within middle-ear procedures, given its direct interface with the inner ear and the associated theoretical risk of pressure-related complications.[21,38] However, available clinical reports suggest that return to recreational diving may be feasible in carefully selected individuals with stable postoperative status and normal vestibular function.[8,15–17] Some authors have suggested a cautious, stepwise return to diving under controlled conditions. This approach is conceptually similar to the 'pre-dive stress test' described after other otologic procedures and may include controlled pressure exposure in a hyperbaric chamber or a carefully supervised shallow-water dive to confirm symptom-free pressure tolerance before unrestricted diving is resumed.[13]

In light of these considerations, patients who are active or prospective divers should be counseled prior to middle-ear surgery regarding the potential implications for future diving, including procedure-specific risks and the possible need for restrictions or individualised clearance.[22]

For cochlear implantation, the evidence base differs in nature. Recommendations are informed by a combination of hyperbaric chamber testing,[23] limited clinical diving reports,[24,26,30] and manufacturer specifications.[27–29] Across sources, guidance is predominantly expressed as conditional clearance within device-specific pressure or depth limits rather than categorical exclusion from diving. This reinforces the concept that device integrity and postoperative stability, rather than the mere presence of an implant, should guide fitness-to-dive decisions.

To facilitate practical interpretation of these findings, we developed an evidence-informed decision pathway summarising procedure-specific considerations for postoperative diving clearance (Figure 2). While the preceding sections outline the variability in risks across surgical categories, the algorithm consolidates these distinctions into a structured, clinically usable framework. It emphasises functional markers such as middle-ear stability, reliable pressure equalisation, and device-specific pressure limits. The pathway also highlights higher-risk contexts, including CWD mastoidectomy and selected total ossicular replacement prostheses, where restriction or individualised assessment is warranted. Rather than replacing clinical judgment, the framework provides a pragmatic tool to support consistent, procedure-adapted decision-making in the postoperative diver. In this framework, a "*focus on*

aeration" specifically refers to prioritising the confirmation of stable middle-ear ventilation, with the diver able to equalise pressure easily, repeatedly, and without symptoms.

An important practical aspect highlighted by this review, which is rarely addressed explicitly in the literature, relates to diver communication and situational awareness. In individuals with significant postoperative hearing loss, fitness-to-dive should not be considered in isolation. Effective communication with the dive buddy is a critical safety component in recreational diving. Buddies should be informed of hearing limitations and prepared to adapt communication strategies accordingly, particularly in low-visibility environments or emergencies. This consideration extends fitness-to-dive assessment beyond otologic risk alone and aligns it with real-world diving safety practices.

LIMITATIONS

Given the heterogeneity and limitations of the available evidence, the proposed decision aid should be interpreted as an evidence-informed clinical support tool rather than a formal guideline. Its purpose is to facilitate structured, individualised assessment and shared decision-making between clinicians and divers. Future research should focus on standardised outcome reporting and the development of prospective registries to better define procedure-specific risks and refine fitness-to-dive recommendations after otologic surgery.

Conclusions

The current evidence base suggests that fitness-to-dive after otologic surgery should be approached on an individualised basis rather than through absolute contraindications. While data remain limited, selected patients may safely return to diving under appropriate conditions. Further prospective research is needed to inform standardised clinical guidance.

References

1 Moon RE, editor. Undersea and Hyperbaric Medical Society. Hyperbaric oxygen therapy indications. 14th ed. North Palm Beach (FL): Best Publishing; 2019.

2 Livingstone DM, Smith KA, Lange B. Scuba diving and otology: a systematic review with recommendations on diagnosis, treatment and post-operative care. Diving Hyperb Med. 2017;47:97–109. doi: 10.28920/dhm47.2.97-109. PMID: 28641322. PMCID: PMC6147252.

3 Glazer TA, Telian SA. Otologic Hazards related to scuba diving. Sports Health. 2016;8:140–4. doi: 10.1177/1941738116631524. PMID: 26857731. PMCID: PMC4789939.

4 Moon RE, Birnbaumer DM. Ear and sinus barotrauma [Internet]. Merck Manual Professional Version; 2025. [cited 2026 Apr 13]. Available from: https://www.merckmanuals.com/professional/injuries-poisoning/injury-during-diving-or-work-in-compressed-air/ear-and-sinus-barotrauma.

5 Strutz J. Otorhinolaryngologische erkrankungen beim tauchen [Otorhinolaryngologic disorders associated with diving]. HNO. 2008;56:499–504, 506–8. doi: 10.1007/s00106-008-1742-x. PMID: 18415066. German.

6 Lechner M, Sutton L, Fishman JM, Kaylie DM, Moon RE, Masterson L, et al. Otorhinolaryngology and diving-Part 1: Otorhinolaryngological hazards related to compressed gas scuba diving: A review. JAMA Otolaryngol Head Neck Surg. 2018;144:252–8. doi: 10.1001/jamaoto.2017.2617. PMID: 29450472.

7 Health and Safety Executive (HSE). Diving at work regulations: medical guidance [Internet]. London: Health and Safety Executive; 1997. [cited 2026 Apr 13]. Available from: https://www.hse.gov.uk/pubns/ma1.pdf.

8 Mallen JR, Roberts DS. SCUBA medicine for otolaryngologists: Part II. Diagnostic, treatment, and dive fitness recommendations. Laryngoscope. 2020;130:59–64. doi: 10.1002/lary.27874. PMID: 30776095.

9 Klingmann C, Praetorius M, Böhm F, Tetzlaff K, Plinkert PK. Tauchtauglichkeit im HNO-Bereich [Fitness to dive in the otorhinolaryngological field]. HNO. 2008;56:509–18. doi: 10.1007/s00106-008-1743-9. PMID: 18415065. German.

10 Tricco AC, Lillie E, Zarin W, O'Brien KK, Colquhoun H, Straus SE, et al. PRISMA extension for scoping reviews (PRISMA-ScR): Checklist and explanation. Ann Intern Med. 2018;169:467–73. doi: 10.7326/M18-0850. PMID: 30178033.

11 Riestra-Ayora J. Fitness to dive after otologic surgery: A scoping review [Internet]. Open Science Framework; 2025. Available from: https://osf.io/5rqvx.

12 Antonelli PJ, Adamczyk M, Appleton CM, Parell GJ. Inner ear barotrauma after stapedectomy in the guinea pig. Laryngoscope. 1999;109:1991–5. doi: 10.1097/00005537-199912000-00018. PMID: 10591361.

13 Velepic M, Bonifacic M, Manestar D, Velepic M, Bonifacic D. Cartilage palisade tympanoplasty and diving. Otol Neurotol. 2001;22:430–2. doi: 10.1097/00129492-200107000-00002. PMID: 11449094.

14 DAN Europe. Fitness to dive after ear surgery: tympanoplasty [Internet]. 2025. [cited 2026 Apr 13]. Available from: https://alertdiver.eu/en_US/blog/tympanoplasty/.

15 Weitzsäcker WE. Risk assessment for divers with a history of middle ear surgery. Undersea Hyperb Med. 2025;52:23–31. PMID: 40249719.

16 Hizalan I, Ildiz F, Uzun C, Keskin G. SCUBA dalicilarinda KBB muayenesi ve dalişa engel KBB patolojileri [ENT examination in SCUBA divers and ENT pathologies restricting diving]. Turkish. Kulak Burun Bogaz Ihtis Derg. 2002;9:220–6. PMID: 12415214.

17 House JW, Toh EH, Perez A. Diving after stapedectomy: clinical experience and recommendations. Otolaryngol Head Neck Surg. 2001;125:356–60. doi: 10.1067/mhn.2001.118183. PMID: 11593171.

18 DAN Southern Africa. Can I dive after a stapedectomy? [Internet]. 2017. [cited 2026 Apr 13]. Available from: https://www.dansa.org/blog/2017/09/07/stapedectomy-faq.

19 Harrill WC, Jenkins HA, Coker NJ. Barotrauma after stapes surgery: a survey of recommended restrictions and clinical experiences. Am J Otol. 1996;17:835–45; discussion 845–6. PMID: 8915410.

20 Nofz L, Porrett J, Yii N, De Alwis N. Diving-related otological injuries: Initial assessment and management. Aust J Gen Pract. 2020;49:500–4. doi: 10.31128/AJGP-01-20-5191. PMID: 32738862.

21 Hüttenbrink KB. Clinical significance of stapedioplasty biomechanics: swimming, diving, flying after stapes

surgery. Adv Otorhinolaryngol. 2007;65:146–9. doi: 10.1159/000098791. PMID: 17245036.
22 Scarpa A, Ralli M, De Luca P, Gioacchini FM, Cavaliere M, Re M, et al. Inner ear disorders in SCUBA divers: A review. J Int Adv Otol. 2021;17:260–4. doi: 10.5152/iao.2021.8892. PMID: 34100753. PMCID: PMC9450052.
23 Backous DD, Dunford RG, Segel P, Muhlocker MC, Carter P, Hampson NB. Effects of hyperbaric exposure on the integrity of the internal components of commercially available cochlear implant systems. Otol Neurotol. 2002;23:463–7; discussion 467. doi: 10.1097/00129492-200207000-00012. PMID: 12170146.
24 Zeitler DM, Almosnino G, Holm JR. Stability of residual hearing and cochlear implant function following multiple scuba dives: case report. Undersea Hyperb Med. 2018;45:371–6. PMID: 30028923.
25 Nolte A, Meyer M, Luers JC, Fürstenberg D, Klussmann JP, Lang-Roth R, et al. Ist Fliegen oder Tauchen riskant für CI-Träger? – Untersuchungen in einer Druckkammer [Is Flying or Diving risky after cochlear implantation? Examination in a pressure chamber]. Laryngorhinootologie. 2022;101:35–9. doi: 10.1055/a-1346-9370. PMID: 33498087. German.
26 Kompis M, Vibert D, Senn P, Vischer MW, Häusler R. Scuba diving with cochlear implants. Ann Otol Rhinol Laryngol. 2003;112:425–7. doi: 10.1177/000348940311200507. PMID: 12784981.
27 Cochlear. Practising sports with a cochlear implant: water sports [Internet]. Cochlear Ltd. [cited 2026 Jun 4]. Available from: https://www.cochlear.com/ciom/es/support/resources/tips-and-tricks.
28 MED-EL. Essential tips for hassle-free travel with cochlear implants [Internet]. MED-EL Blog; 2023. [cited 2026 Apr 13]. Available from: http://blog.medel.com/tips-tricks/essential-tips-for-hassle-free-travel-with-cochlear-implants/.
29 Cochlear Limited. Important information for recipients (Nucleus) [Internet]. 2022. [cited 2026 Apr 13]. Available from: https://assets.cochlear.com/api/public/content/54f19696c64a4de98668c06bbd8cf395?v=06b4e58330.
30 Hintze JM, Geyer L, Fitzgerald CW, Simoes Franklin C, Glynn F, Viani L, et al. The impact of repetitive hyperbaric exposure during SCUBA diving on cochlear implants. Laryngoscope. 2019;129:2760–4. doi: 10.1002/lary.27880. PMID: 30810235.
31 Uzun C, Adali MK, Koten M, Yagiz R, Aydin S, Cakir B, et al. Relationship between mastoid pneumatization and middle ear barotrauma in divers. Laryngoscope. 2002;112:287–91. doi: 10.1097/00005537-200202000-00016. PMID: 11889385.
32 Neblett LM. Otolaryngology and sport scuba diving. Update and guidelines. Ann Otol Rhinol Laryngol Suppl. 1985;115:1–12. PMID: 2857546.
33 Salt AN, Hullar TE. Responses of the ear to low frequency sounds, infrasound and wind turbines. Hear Res. 2010;268(1-2):12–21. doi: 10.1016/j.heares.2010.06.007. PMID: 20561575. PMCID: PMC2923251.
34 McMullin AM. Scuba diving: What you and your patients need to know. Cleve Clin J Med. 2006;73:711–2, 714, 716 passim. doi: 10.3949/ccjm.73.8.711. PMID: 16913196.
35 Sim RJ, Youngs RP. Otolaryngological requirements for recreational self-contained underwater breathing apparatus (SCUBA) diving. J Laryngol Otol. 2007;121:306–11. doi: 10.1017/S0022215106001976. PMID: 17040582.
36 Klingmann C, Wallner F. Tauchmedizinische Aspekte in der HNO-Heilkunde. 2.Teil: Tauchtauglichkeit [Health aspects of diving in ENT medicine. Part II: Diving fitness]. HNO. 2004;52:845–7. doi: 10.1007/s00106-004-1106-0. PMID: 15221086. German.
37 Divers Alert Network Europe. Ears and diving [Internet]. 2023. [cited 2026 Apr 13]. Available from: https://dan.org/wp-content/uploads/2020/07/Ears-and-Diving-DAN-Dive-Medical-Reference.pdf.
38 Hüttenbrink KB. Biomechanics of stapesplasty: a review. Otol Neurotol. 2003;24:548–57; discussion 557–9. doi: 10.1097/00129492-200307000-00004. PMID: 12851544.

Conflicts of interest and funding: nil

Submitted: 22 March 2026
Accepted after revision: 6 May 2026

World as it is

Recreational and technical rebreather fatalities and diving safety status

Frauke Tillmans[1,2], Camilo Saraiva[1], Sherri Ferguson[1,3], Emmanuel Dugrenot[1,2], S Lesley Blogg[1,4]

[1] *Divers Alert Network, 6 West Colony Place, Durham, NC, USA*
[2] *UNC-NC State Joint Department of Biomedical Engineering, Chapel Hill, NC, USA*
[3] *Shanfe Research and Consulting Ltd, North Vancouver, British Columbia, Canada*
[4] *SLB Consulting, Kirkby Stephen, Cumbria, UK*

Corresponding author: *Dr Frauke Tillmans, Divers Alert Network, 6 West Colony Place, Durham, NC 27705, USA*
ORCiD: *0000-0002-2159-0834*
ftillmans@dan.org

Keywords
Accidents; Death rate; Demographics; Global use; Rebreather diving; Technical diving

Abstract
(Tillmans F, Saraiva C, Ferguson S, Dugrenot E, Blogg SL. Recreational and technical rebreather fatalities and diving safety status. Diving and Hyperbaric Medicine. 2026 30 June;56(2):185–190. doi: 10.28920/dhm56.2.185-190. PMID: 42290579.)
Introduction: The Divers Alert Network (DAN) aims to provide safety information for all types of diving. Assessing the number of active closed-circuit rebreather (CCR) divers is difficult, as pertinent information is often not available. This review aims to give an overview of global use and safety of CCR diving equipment from 2013–2022.
Methods: Data were combined and assessed from various DAN internal and public sources on CCR diver demographics, fatalities, and CCR sales.
Results: Over the past 10 years, the number of certified CCR divers has increased from an estimated 2,000 in 2013, to 3,000 in 2022. There has been an increase in growth in CCR sales over a five-year period from 2018, with around 25,000 to 35,000 units on the market today; rebreather divers are a growing community. There were 241 confirmed CCR fatalities from 2013–2022, mean 24 (SD 6) per year. Most fatal accidents involved dives made between 40–80 m (130–260 ft) depth. Cause of death is difficult to establish due to lack of detail and dive-specific training for the medical examiner. The estimated death rate is 1.8–3.8 deaths per 100,000 CCR dives although these values are derived from limited data.
Conclusions: Not enough information is made available to address CCR accident analysis effectively, perhaps stemming from family reticence to discuss the incident, fear of litigation, and/or lack of diving knowledge reducing the useful information. DAN continues to collect CCR data, but increased collaboration between training bodies, equipment providers, and comprehensive reporting of incidents is needed to reveal the true picture.

Introduction

This review gives an overview of the global use and safety of closed-circuit rebreather (CCR) diving equipment. The topics covered include CCR diver demographics, a review of fatalities since the 2012 Rebreather Forum Three (RF3)[3] that includes data from January 2013 through to the end of December 2022, and a description of the state of the CCR market. It also draws on collated survey information from the caustic cocktail survey[1] (n = 413) for data on gender, age, years of experience. The challenges of incident and accident investigation are discussed, then leading on to the future of rebreather safety.

Diver demographics

Data sourced from the caustic cocktail survey[1] show that the reported age of rebreather divers was mean 46 (standard deviation [SD] 10) years, with 95% of participants being male, while the Divers Alert Network (DAN) hotline reported that 84% of callers were male and their median age was 42 (2012–2022 data). The length of time that a CCR diver had been certified was median six (interquartile range [IQR] 3, 12) years, with 40% being certified for less than five years, and 10% for more than 20 years.[1] The median self-reported dive experience was 200 (IQR 100, 500) CCR dives, with 300 (IQR 120, 750) hours clocked on CCR equipment.[1] The certification time and experience for Rebreather Forum 4 participants was almost twice as high, which is not surprising given the specialisation of the audience.

CCR DIVERS CONTACTING DAN

Divers contact DAN to ask for information on diving with a CCR and to log accident cases, which entail emergencies. Figure 1 details the types of calls (emergency or information) from rebreather divers made to DAN from January 2013 through to the end of December 2022.

Figure 1
Closed circuit rebreather divers contacting the Divers Alert Network (n = 325 emergency cases, n = 281 information requests)

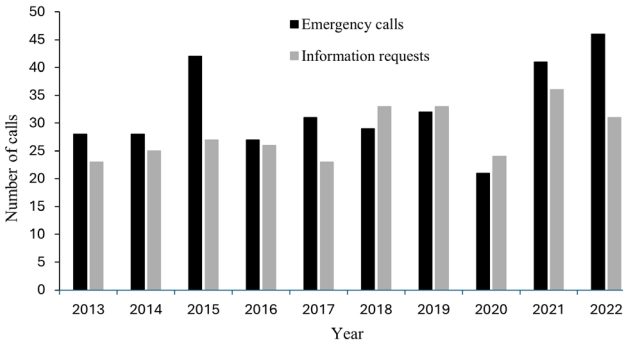


Figure 2
Distribution of emergency calls and information inquiries by age and sex where disclosed for calls made between January 2013 through December 2022 (n = 319)

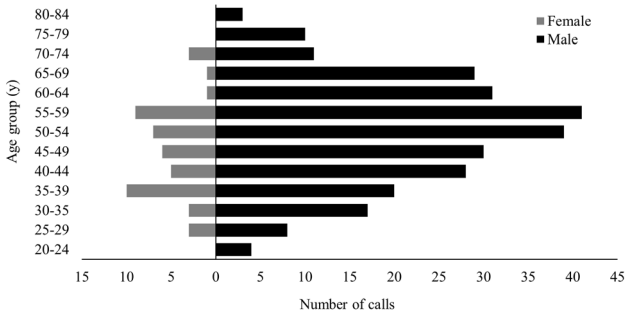


Table 1
Differential diagnoses of closed-circuit rebreather-related emergency calls to the Divers Alert Network taken from a sample November 2021 through March 2023 (n = 47); DCS – decompression sickness

Differential diagnoses (n = 47)	**Cases n (%)**
Type 1 DCS	13 (28)
Type 2 DCS	9 (19)
Anxiety	8 (17)
Middle ear barotrauma	4 (9)
Trauma	4 (9)
Sinus barotrauma	2 (4)
Infectious disease	2 (4)
Dental barotrauma	1 (2)
Mask barotrauma	1 (2)
Suit barotrauma	1 (2)
Loss of consciousness	1 (2)
Neurological	1 (2)

It was found that little had changed since RF3 with regards to the sex and age distribution of CCR divers contacting DAN. The majority of the 319 phone calls and email inquiries for which sex and age was reported were made by men, with only 48 female inquirers (Figure 2). The age distribution of the callers was centred around those 50–59 years of age.

The differential diagnoses obtained from the emergencies are often of greater interest (Table 1). Decompression sickness (DCS) involving pain (Type I DCS) comprised ~27% of the cases reported, while more severe neurological DCS (Type II DCS) made up ~19% of cases. Anxiety caused by the worry of DCS and later diagnosed as such (rather than the originally attributed DCS) accounted for around 17% of cases (Table 1). Barotrauma of varying types formed several differential diagnoses, the frequency of CCR-derived barotrauma being relatively comparable with other forms of diving. Trauma, loss of consciousness, and neurological complications made up the rest of the categories reported (Table 1).

Fatality review: what data are available?

Although it might be expected otherwise, there is a paucity of fatality data available making meaningful interpretation difficult. Most of the available data is collected from insurance claims and the emergency calls received by DAN America and DAN Europe. DAN conducts a fatality surveillance project, scanning media and news outlets for possible fatalities, then following up the primary information to collect further data from medical reports, investigation reports, and witness statements, when available. Equipment experts who examine rebreathers used in accidents may also forward information. Internet databases are useful to compare with the DAN fatality database to check for any cases that might have been missed. Finally, individuals who have expertise in CCR, for example, manufacturers, training agencies, diving physicians, and pathologists occasionally call with information that they feel would be useful to share. Once all these data were collated and merged, duplicates were identified and removed. Verification of the incident followed. Once the database was consolidated a final tally was obtained; from 2013–2022 there were 241 verified CCR fatality records captured.

Limitations to collecting and verifying the data include a hesitancy to share information by family or friends, language barriers, legal restrictions (for example, in Australia case data is released only five-years post-accident), the time and effort needed for follow-up, data reliability, incomplete information, third-hand information, and conflicting information, all of which weaken any analysis.

CCR fatality 25-year review

A report on CCR fatalities for the period of 1998–2010 showed a peak at around 26 fatalities per year in 2005, having risen from five to ten fatalities per year from 1998–2004.[2] Figure 3 displays these data plus additional numbers

Figure 3
Closed circuit rebreather fatality 25-year review 1998–2022; counts per year (solid line) with 5-year rolling average trend line (dashed line)

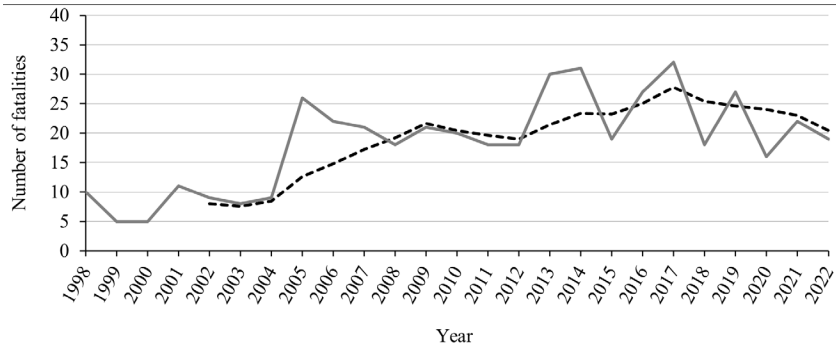
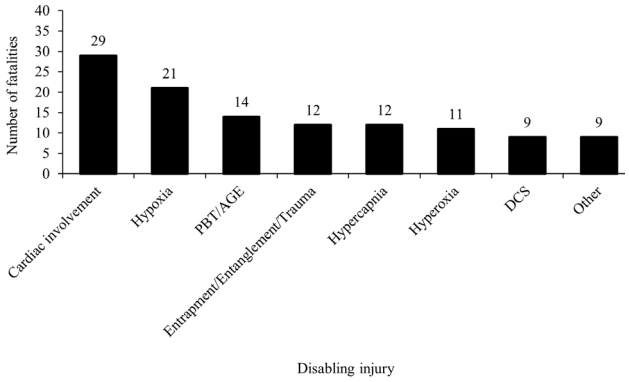


Table 2
Age and sex of deceased rebreather divers (n = 193; 20% of ages unknown); y – years

Age group (y) (n = 241)	Male n (%)	Female n (%)
18–29	5 (2)	1 (0.4)
30–39	22 (9)	5 (2)
40–49	57 (24)	1 (0.4)
50–59	67 (28)	3 (1)
60+	32 (13)	0
Unknown	56 (23)	2 (0.8)

Figure 4
Disabling injuries that contributed to fatalities (n = 117, 52% unknown); note that cardiac involvement, hypoxia, hypercapnia, and hyperoxia all contributed considerably and remain issues that should be addressed. AGE – arterial gas embolism; DCS – decompression sickness; PBT – pulmonary barotrauma



through to 2022, showing that in the years 2013–2017, an even larger spike in the number of CCR fatalities was seen, rising to around 35 cases per year at its zenith. In the years 2018–2022, the number of fatalities varied enormously but did not dip below 20 per year. A 5-year rolling average trend line reveals that in 2022, the typical number of fatalities was still around 25–30 deaths per year.

On further examination of the data from the last 10 years, it was possible to derive 241 confirmed CCR fatalities (12 female, 229 male) with a trend towards fewer deaths in the second half of those 10 years. This count excludes military and commercial accidents.

WHAT IS KNOWN ABOUT THESE FATAL ACCIDENTS?

As previously noted, the majority of CCR divers who had fatal accidents were between 40–59 years of age, and only 12 of the 241 (5%) confirmed cases were women (Table 2).

Fatalities are reviewed at DAN using an adaptation of the method used by Vann et al,[4] classifying each fatality in respect of possible predisposing factors, trigger events, disabling/harmful agents, nature and mechanism of the disabling injury, and cause of death (COD). Frequently in diving-related incidents, the COD is difficult to establish and is most often ruled a drowning in the medical examiner reports submitted to DAN.

For the analysis of the current dataset, we established the disabling injury that most likely contributed to the chain of events of the fatal outcome/drowning. Of the 241 confirmed cases, 124 fatal accidents had insufficient information to draw any conclusions, classified as unknown. Cardiac incidents or questionable cardiac health of the diver were involved in 29 of these cases and provided the largest category in the 117 deaths where data analysis was possible. Given the distribution in age of the fatalities, with most occurring in the over 40 age groups and the risk of cardiac-related illness increasing with age, this finding was not surprising. Hypoxia was likely responsible for 21 deaths and the remaining confirmed causes are illustrated in Figure 4. The category 'other' included less than three occurrences of suicide, gas contamination, venomous marine life encounters, or poor gas management.

Table 3
Diving depth at which the problem that led to the fatal outcome first occurred (n = 208; 17% unknown dive depth)

Depth (m)	Fatalities (n)
< 20	32
20–39	31
40–59	46
60–79	40
80–99	19
100–119	23
120–200	13
> 200	4

A primary concern related to CCR diving relates to the depth of the dives made given that rebreathers may allow divers to go deeper for longer. Table 3 illustrates the depths in which the issue that led to the fatality most likely occurred, showing that most cases are situated in the relatively deep 40–80 m (130–260 ft) range. There is a fairly even spread at depths shallower than this, some of which have occurred during entry-level CCR training, as well as the occasional exceptionally deep dives greater than 200 m (656 ft), for example, exploratory dives or record attempts.

CCR industry statistics: how big is the industry and is it growing?

We were able to gather information from eight CCR training agencies on how many certifications are issued each year over 10 years (Figure 5). Certifications are classified as basic, intermediate, and advanced (60 m plus) qualifications. Although the number of certificates for the advanced group remains similar across the 10-year period, a trend towards an increase in the numbers qualified for the basic and intermediate qualifications can be seen. Overall, over 5,000 certificates were awarded in the last year that were related to rebreather diving. However, it is unlikely that this allows us to determine how many CCR divers are actively diving, as people can start to dive CCR on either basic or intermediate certification, and many divers will have more than one certification, resulting in duplication within the total number of divers.

With regards to duplication, in a quick poll of the participants of RF4 it seemed that it may be fair to assume that each CCR diver has at least two certifications, which would suggest that around 50% of the total could be deducted and this is also illustrated in Figure 5. Overall, these ranges provide an educated estimate, which suggests that post-2020, there was an increase in training certifications awarded.

Drawing on these data, we could start to address the question of how large the CCR industry is, which might help to assess the growth of the CCR community worldwide. The training survey suggested a minimum growth of 1,400–2,800 new CCR divers per year over the past 10 years. We included 22 manufacturers of rebreather equipment to ask how many units they sold per year (estimated or calculated), and how many sales they made per year since RF3 in 2012.

Twenty manufacturers agreed to share data anonymously (two opted for a data use agreement before supplying this information), and from this, it was estimated that around 23,000 units had been sold in total over the past ten years. The data provided were very limited since 36% of the manufacturers provided an estimate of their sales only. Of the remainder, 55% provided actual sales numbers, and 9% did not provide data.

The growth of the rebreather market is illustrated in Figure 6. Overall, there has been an increase in growth of the market over a five-year period starting in 2018. Although we cannot be sure what has driven this, potential explanations include a rise in the choice of CCR units coming to market from a range of manufacturers, or the high price of helium making recirculation of gas more attractive. It is noteworthy that the increase in CCR diving certifications shown in Figure 5 is reflective of the expansion of the rebreather manufacturing market (Figure 6). Overall, CCR diving is a flourishing market and there is a growing community of these divers across the world. However, we cannot account for how many divers own multiple units, how many divers buy units second-hand, or how many sell units when they leave the sport.

Safety status

DAN data suggest that there are around 180–220 diving fatalities per year that are attributed to scuba diving. Of these, 10–15% (n = 20–25 per year) are thought to be rebreather divers. The number of deaths is likely underreported for both CCR and open circuit.

CCR DEATH RATES

We have previously published an estimate of the demographics of CCR divers, the number of dives conducted, and the hours spent underwater by rebreather divers.[1] From these data, a rough estimate of death rates can be made. If the estimate of 20,000 active CCR divers is used alongside 20–25 deaths per year, then the CCR death rate would be 1.8–3.8 deaths per 100,000 dives or 1.2–2.5 deaths per 100,000 h of CCR dive time. In previous presentations on the topic, this estimated rate reached 4–5 deaths per 100,000.

There is no doubt that to strengthen these data accident and incident surveillance needs to be improved in the future, which will be aided by the industry (training agencies, CCR manufacturers, and computer manufacturers) continuing, or in some cases starting to provide anonymised data to

Figure 5

Training survey 2013–2022 showing total and corrected certifications issued each year; grey solid line represents all certifications including basic, intermediate, and advanced; black dashed line represents basic and intermediate certifications; black solid line represents total certifications minus ~44% duplicates; grey dashed line represents basic and intermediate certifications minus 50% (see text for explanation)

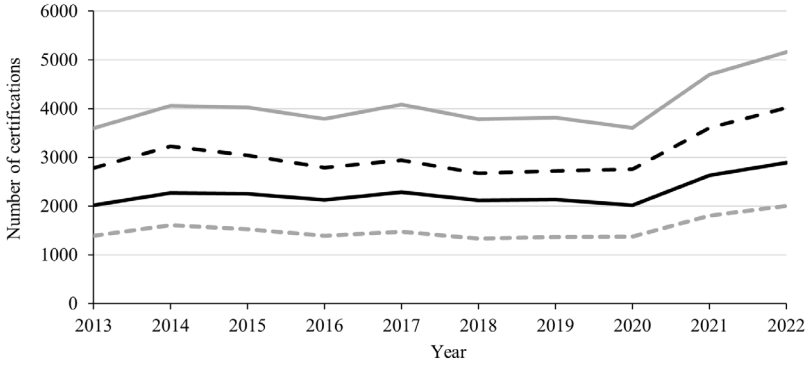
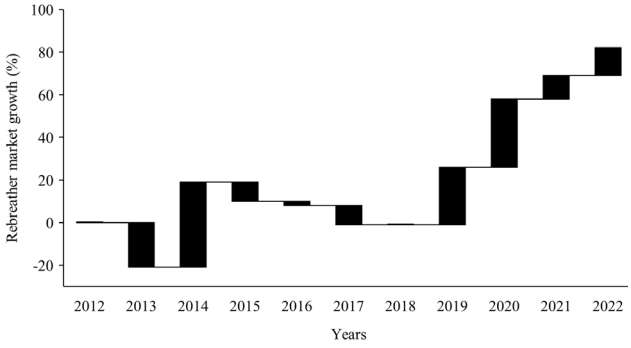


Figure 6

Growth of the rebreather market (%) since 2012 derived from submitted sales data from CCR manufacturers



produce a common denominator to report against the fatality numbers. Incident reporting would be encouraged by a safe space for individuals to report their close calls to experts without any peer judgment. Country-specific information needs to be streamlined and standardised to facilitate easier collation of data.

DAN continues to produce its annual diving report, which monitors diving incidents and fatalities, and relies in part on people accessing the DAN website and filing a report on the diving incident reporting system (https://dan.org/safety-prevention/incident-reporting). Data quality and usefulness can be improved by better reporting. Thus, divers and their families should be encouraged to provide information as soon as possible after an incident to help them provide accurate information, and follow-up should be made. Continuing medical education efforts could help to aid clinicians in identifying not only the obvious COD of drowning in a watery environment, but also to look for triggers and existing medical conditions that may have pre-empted the drowning. Expert handling of the diving equipment following an accident is also necessary to extract the maximum amount of information available. This could include returning units to the manufacturer or knowledgeable independent third parties for examination. In the US, DAN tries to work with law enforcement with some success in areas where diving is common, although in the states and counties where diving accidents are infrequent, the information is often not passed on or reported in detail. Again, education is key. Another important link is being able to provide people who have training to talk to family members and witnesses, which aids in collection of pertinent information.

Conclusions

In summary, it should be made clear that not enough information is made available to address accident analysis in full. This lack of detail may stem from family/friends not wanting to talk about the accident, other priorities, fear of litigation, and/or lack of diving knowledge or familiarity with the subject matter reducing the amount of useful information that can be provided.

Although attempts are made to collect as much data as possible on global provision and use of CCRs, increased collaboration between the providers of equipment and training, and education of those involved in reporting on incidents and accidents, will help to allow collection of the best quality data. This is key to improving our knowledge and in turn to disseminate potentially life-saving information to the CCR community.

References

1 Buzzacott P, Dong GZ, Brenner RJ, Tillmans F. A survey of caustic cocktail events in rebreather divers. Diving Hyperb Med. 2022;52:92–6. doi: 10.28920/dhm52.2.92-96. PMID: 35732280. PMCID: PMC9522607.

2 Fock AW. Analysis of recreational closed-circuit rebreather deaths 1998–2010. Diving Hyperb Med. 2013;43:78–85. PMID: 23813461. [cited 2025 Dec 30]. Available from: https://dhmjournal.com/images/IndividArticles/43June/Fock_dhm.43.2.78-85.pdf.

3 Vann RD, Denoble PJ, Pollock NW, editors. Rebreather Forum 3 Proceedings. Durham (NC):AAUS/DAN/PADI; 2014. p. 324.

4 Vann RD, Pollock NW, Denoble PJ. Rebreather fatality investigation. In: Pollock NW, Godfrey JM, editors. Diving for Science 2007. Proceedings of the American Academy of Underwater Sciences 26th Symposium. Dauphin Is (AL): AAUS; 2007. p. 101–10.

Acknowledgments

The authors thank all equipment manufacturers and training agencies who willingly contributed sales and training data to this report, as well as Marta Marocco, Catherine Harris, Martin Parker, and the members of the Accident Analysis Committee of the National Speleological Society, Cave Diving Section, for their invaluable support in accident data collection and interpretation. The authors wish to thank Michael Menduno who was instrumental in providing contacts to the manufacturers and training agencies that provided information.

Conflicts of interest and funding

This work was supported by DAN. No conflicts of interest were declared.

Submitted: 1 July 2024
Accepted after revision: 16 February 2026

Case reports

The role of hyperbaric oxygen treatment in a case of pyomyositis

Jobin G Bose[1], Subhranshu Kumar[1], Sunil Anand[2], Chandrasekhar Mohanty[1]

[1] *Department of Marine Medicine, Institute of Naval Medicine, INHS Asvini, Mumbai, India*
[2] *Department of Plastic and Reconstructive Surgery, Institute of Naval Medicine, INHS Asvini, Mumbai, India*

Corresponding author: *Dr Jobin Bose, Department of Marine Medicine, Institute of Naval Medicine, INHS Asvini, Mumbai, Pin-400005, India*
ORCiD: *0009-0007-5758-5926*
drjgbose@gmail.com

Keywords
Case reports; Hypoxia; Musculo-skeletal; Wounds

Abstract

(Bose JG, Kumar S, Anand S, Mohanty C. The role of HBOT in a case of pyomyositis. Diving and Hyperbaric Medicine. 2026 30 June;56(2):191–194. doi: 10.28920/dhm56.2.191-194. PMID: 42290580.)
Pyomyositis is a serious bacterial infection of the skeletal muscles, usually treated with antibiotics and surgical drainage. The success of medical or surgical treatment is often delayed or less effective when tissue hypoxia is present. Hyperbaric oxygen therapy (HBOT) is currently being studied as a helpful additional treatment for various conditions, especially those involving complications of tissue hypoxia. In this case report, we describe a 29-year-old male who developed chronic pyomyositis and a right lower leg ulcer after failure of multiple surgeries, including fasciotomy, debridements, skin grafts and antibiotics to treat compartment syndrome and tuberculosis of the right knee. HBOT was administered for 80 sessions at 243 kPa (2.4 atmospheres absolute) for 90 minutes. The patient showed significant clinical improvement, as evidenced by the development of healthy granulation tissue, reduction in swelling and discharge, and better mobility. This case highlights the potential of HBOT as an additional treatment option for complex soft tissue infections such as pyomyositis caused by tuberculosis, especially in cases where traditional treatments have been ineffective.

Introduction

Pyomyositis is conventionally characterised as a suppurative infection of the skeletal muscles, mainly observed in tropical climates.[1] The disease predominantly affects males and often presents with one or multiple intramuscular abscesses. The most common causative organism is *Staphylococcus aureus*, in up to 90% of cases of primary muscle abscess in tropical, and 70% in temperate areas. The complete pathogenesis of this dreaded infection is not completely understood. Treatment comprises systemic antibiotics and debridement and drainage of abscesses. In instances with massive inflammation, compromised perfusion, or in the presence of comorbidities, response to standard therapy is usually poor.[2–4]

Hyperbaric oxygen therapy (HBOT) involves breathing 100% oxygen under increased atmospheric pressure. It improves oxygen delivery to hypoxic tissues, enhances leukocyte function, and promotes wound healing. While well-documented for necrotising soft tissue infections, clostridial myositis, and myonecrosis,[5] HBOT is rarely reported in cases of pyomyositis. Here, we present a case of pyomyositis in the leg that was resistant to initial treatments and was successfully managed with the addition of adjuvant HBOT.

Case report

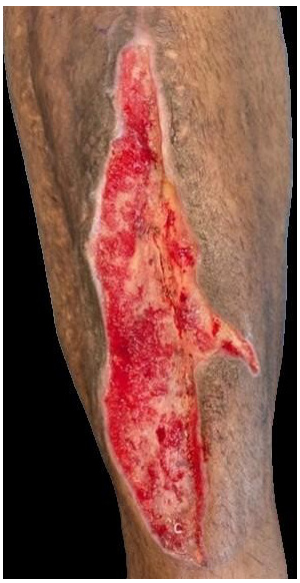
The patient provided written consent to publication of deidentified case details and images.

A 29-year-old male with a history of tuberculosis of the right knee and a previous episode of compartment syndrome in the right leg, for which he underwent fasciotomy, presented to our centre with a six-week history of intermittent high-grade fever, a non-healing ulcer, and swelling in the right leg.

The patient had previously undergone arthroscopic reconstruction of the right anterior cruciate ligament using a quadriceps tendon graft after an injury sustained during physical activity. He underwent multiple aspirations from the right knee due to recurrent swelling, which yielded sero-sanguinous fluid. He was also investigated for tuberculosis of the right knee with the interferon gamma release assay and the Mantoux test, which indicated the presence of *Mycobacterium tuberculosis*. The patient completed an 18-month course of anti-tubercular therapy, however, during treatment he developed compartment syndrome in the right leg and required emergency fasciotomy. Wound debridements and vacuum-assisted closures, along with secondary suturing of fasciotomy sites, were performed over

Figure 1
Wound prior to hyperbaric oxygen therapy

Figure 2
Wound after 40 sessions of hyperbaric oxygen therapy



multiple sessions before discharge. A month later, the patient returned with wound dehiscence and serous discharge; again, he was managed conservatively with intravenous antibiotics and saline dressings. However, the wound evolved into a non-healing ulcer over the surgical scar, requiring grafting and reconstruction (Figure 1). Radiological investigation with contrast-enhanced magnetic resonance imaging (MRI) of the right lower limb revealed intramuscular collection involving tibialis anterior muscle and extending along the interfascial plane. There was an increase in intramuscular oedema within the fibres of the tibialis anterior muscle and the extensor digitorum longus muscle, with an increase in subcutaneous oedema along the anterior aspect of the right leg.

At this juncture, the patient was referred to our HBOT centre and found to be fit to undergo treatment. He underwent 40 sessions at 243 kPa (2.4 atmospheres absolute) for 90 minutes with two air breaks, six days a week, without interruption. Progressive clinical improvement was noticed with reduction of peri-wound oedema, healthy wound base and starting of granulation tissue formation on the extensive raw area of the wound (Figure 2). A repeat contrast-enhanced MRI of the right leg revealed significant reduction of previously noted intramuscular abscesses with features of likely residual myositis in the anterolateral group of leg muscles.

Once a healthy wound base had formed, split-thickness skin grafting was completed (Figure 3). After surgery, he underwent 40 more sessions of HBOT with similar regimen. Adequate healing of the wound was achieved (Figure 4), and the patient was discharged home with advice to follow up after a month.

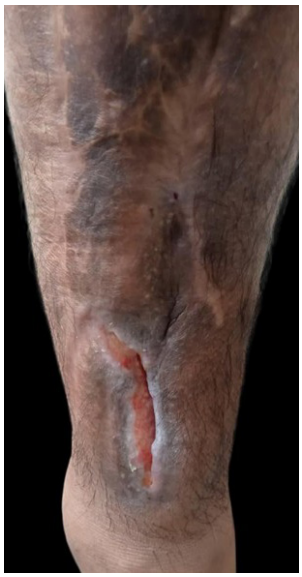
Discussion

Pyomyositis is an infection of the skeletal muscles caused by bacteria, most commonly *Staphylococcus aureus*. It usually affects large muscle groups that are typically resistant to bacterial invasion.[6,7] The etiology and pathogenesis are not fully understood, but the literature describes several known factors, including geographic prevalence, trauma, nutritional deficiencies, and infections. This disease primarily arises in tropical climates, but cases in temperate regions are also reported, often alongside other comorbidities and infections. This disease typically affects large muscles, leading to limitations in physical activity. Commonly involved muscles include the quadriceps, glutei, pectoralis major, serratus anterior, biceps, iliopsoas, gastrocnemius, as well as abdominal and spinal muscles.[4,8] Primary pyomyositis is classified into three stages based on the symptomatology. Stage I, also known as the invasive phase, is characterised by intermittent low-grade fever, localised pain, and swelling. This stage is frequently misdiagnosed as pyomyositis due to its variable symptoms and may resolve on its own without progressing to the next stage. Stage II, also known as the suppurative phase, is the most common stage where this entity is diagnosed. High-grade fever, swelling, and localised muscle pain, as well as systemic symptoms, are more evident and severe, and pus is found in needle aspiration at this stage. Stage III, also known as the late phase, occurs when pyomyositis disseminates from the local site and begins to involve distant organs. This stage is often characterised by signs of septicaemia and shock.[4,9]

Atypical presentation and a wide range of differential diagnoses make early diagnosis, crucial for controlling the spread of this disease, difficult. Laboratory investigations can assist in diagnosis, especially when there is a strong clinical

Figure 3
Wound after split- thickness skin grafting

Figure 4
Wound after 80 sessions of hyperbaric oxygen therapy at the time of discharge from hospital



suspicion. Leukocytosis with a left shift, and with elevated chronic inflammation markers, can occur in advanced stages. Imaging studies, such as local ultrasound, can identify the site and extent of oedema for aspiration. In most cases, MRI is a more effective imaging modality for diagnosing this condition.[10] Treatment of pyomyositis involves the use of antibiotics, wound debridement, and removal of any secondary causes. In the early stage, this approach is effective, but in advanced stages of the disease, recovery becomes difficult, making the condition more severe.[11,12]

HBOT is an adjunctive therapy used in various diseases and is beneficial for some surgical conditions, such as non-healing ulcers, problem wounds with arterial insufficiencies, as well as conditions like clostridial myositis, myonecrosis, necrotising soft tissue injuries, and gangrene.[5] Increasing oxygen at the cellular level causes vasoconstriction and can help reduce oedema. As oedema decreases, blood circulation improves.[13] The rise in oxygen levels at the wound site caused by HBOT interrupts the cascade of events initiated by inflammation and pus formation, along with a significant decrease in β_2 integrin expression on neutrophils, which further accelerates the healing.[14–17] The neovasculogenesis effect drives the growth of capillaries and blood vessels in the wound core, where ischaemia and necrosis have damaged the local blood supply.[18] The side effects associated with it, although relatively rare, include barotrauma, which is the most common, as well as other conditions such as acute oxygen toxicity, claustrophobia, pulmonary oxygen toxicity, and myopia or cataract formation.[19]

LIMITATIONS

Measuring transcutaneous oxygen pressure ($TcPO_2$) of the wound would have quantified the degree of tissue hypoxia and may have assisted in determining an objective end-point for HBOT.

Conclusions

Strong suspicion, early diagnosis, and prompt treatment are vital in managing pyomyositis cases. A multidisciplinary approach is necessary for advanced stages, and HBOT may be beneficial in such situations.

References

1 Drosos G. Pyomyositis. A literature review. Acta Orthop Belg. 2005;71:9–16. PMID: 15792201.

2 Kumar S, Bhalla A, Singh R, Sharma N, Sharma A, Gautam V, Singh S, Varma S. Primary pyomyositis in North India: a clinical, microbiological, and outcome study. Korean J Intern Med. 2018;33:417–31. doi: 10.3904/kjim.2016.011. PMID: 29338140. PMCID: PMC5840587.

3 Agarwal V, Chauhan S, Gupta RK. Pyomyositis. Neuroimaging Clin N Am. 2011;21:975–83. doi: 10.1016/j.nic.2011.07.011. PMID: 22032510.

4 Chauhan S, Jain S, Varma S, Chauhan SS. Tropical pyomyositis (myositis tropicans): current perspective. Postgrad Med J. 2004;80(943):267–70. doi: 10.1136/pgmj.2003.009274. PMID: 15138315. PMCID: PMC1743005.

5 Ortega MA, Fraile-Martinez O, García-Montero C, Callejón-Peláez E, Sáez MA, Álvarez-Mon MA, et al. A general overview on the hyperbaric oxygen therapy: Applications, mechanisms and translational opportunities. Medicina (Kaunas). 2021;57(9):864. doi: 10.3390/medicina57090864. PMID: 34577787. PMCID: PMC8465921.

6 Smith IM, Vickers AB. Natural history of 338 treated and untreated patients with staphylococcal septicaemia (1936-1955). Lancet. 1960;1(7138):1318–22. PMID: 13831996.

7 Chiedozi LC. Pyomyositis. Review of 205 cases in 112

patients. Am J Surg. 1979;137(2):255–9. doi: 10.1016/0002-9610(79)90158-2. PMID: 426186.
8 Ashken MH, Cotton RE. Tropical skeletal muscle abscesses (pyomyositis tropicans). Br J Surg. 1963;50:846–52. doi: 10.1002/bjs.18005022621. PMID: 14068637.
9 Scharschmidt TJ, Weiner SD, Myers JP. Bacterial pyomyositis. Curr Infect Dis Rep. 2004;6:393–6. doi: 10.1007/s11908-004-0039-9. PMID: 15461891.
10 Theodorou SJ, Theodorou DJ, Resnick D. MR imaging findings of pyogenic bacterial myositis (pyomyositis) in patients with local muscle trauma: illustrative cases. Emerg Radiol. 2007;14:89–96. doi: 10.1007/s10140-007-0593-1. PMID: 17333082.
11 Radcliffe C, Gisriel S, Niu YS, Peaper D, Delgado S, Grant M. Pyomyositis and infectious myositis: A comprehensive, single-center retrospective study. Open Forum Infect Dis. 2021;8(4):ofab098. doi: 10.1093/ofid/ofab098. PMID: 33884279. PMCID: PMC8047863.
12 Weber S, Schlaeppi C, Barbey F, Buettcher M, Deubzer B, Duppenthaler A, et al. Clinical characteristics and management of children and adolescents hospitalized with pyomyositis. Pediatr Infect Dis J. 2024;43:831–40. doi: 10.1097/INF.0000000000004382. PMID: 38754004. PMCID: PMC11319086.
13 Nylander G, Lewis D, Nordström H, Larsson J. Reduction of postischemic edema with hyperbaric oxygen. Plast Reconstr Surg. 1985;76:596–603. doi: 10.1097/00006534-198510000-00021. PMID: 4034778.
14 Thom SR. Hyperbaric oxygen: its mechanisms and efficacy. Plast Reconstr Surg. 2011;127(Suppl 1):131S–141S. doi: 10.1097/PRS.0b013e3181fbe2bf. PMID: 21200283. PMCID: PMC3058327.
15 Kumar S, Chaudhry HBs, Mohanty C, Bhutani S, Risham M, Lanjekar K. Hyperbaric oxygen treatment (HBOT) in a case of traumatic chondronecrosis of the cricoid cartilage. Diving Hyperb Med. 2024;54:249–51. doi: 10.28920/dhm54.3.249-251. PMID: 39288933. PMCID: PMC11659080.
16 Woo J, Min JH, Lee YH, Roh HT. Effects of hyperbaric oxygen therapy on inflammation, oxidative/antioxidant balance, and muscle damage after acute exercise in normobaric, normoxic and hypobaric, hypoxic environments: A pilot study. Int J Environ Res Public Health. 2020;17(20):7377. doi: 10.3390/ijerph17207377. PMID: 33050362. PMCID: PMC7601270.
17 Baiula M, Greco R, Ferrazzano L, Caligiana A, Hoxha K, Bandini D, et al. Integrin-mediated adhesive properties of neutrophils are reduced by hyperbaric oxygen therapy in patients with chronic non-healing wound. PLoS One. 2020;15(8):e0237746. doi: 10.1371/journal.pone.0237746. PMID: 32810144. PMCID: PMC7433869.
18 Milovanova T, Bhopale VM, Sorokina EM, Moore JS, Hunt TK, Hauer-Jensen M, et al. Hyperbaric oxygen stimulates vasculogenic stem cell growth and differentiation in vivo. J Appl Physiol (1985). 2009;106:711–28. doi: 10.1152/japplphysiol.91054.2008. PMID: 19023021. PMCID: PMC2644249.
19 Heyboer M 3rd, Sharma D, Santiago W, McCulloch N. Hyperbaric oxygen therapy: Side effects defined and quantified. Adv Wound Care (New Rochelle). 2017;6:210–24. doi: 10.1089/wound.2016.0718. PMID: 28616361. PMCID: PMC5467109.

Conflicts of interest and funding: nil

Submitted:28 January 2026
Accepted after revision: 12 March 2026

Spontaneous resolution of choroidal neovascular membrane in the fellow eye during hyperbaric oxygen treatment for retinal artery occlusion: a case report

Umut Dağ[1], Betül Dertsiz Kozan[1], Selim Engin Egeren[2], Hasan Öncül[1], Mehtap Çağlayan[1], Mehmet Fuat Alakuş[1]

[1] *Department of Ophthalmology, Gazi Yasargil Training and Research Hospital, Diyarbakır, Türkiye*
[2] *Department of Hyperbaric and Underwater Medicine, Gazi Yasargil Training and Research Hospital, Diyarbakır, Türkiye*

Corresponding author: *Dr Betül Dertsiz Kozan, Department of Ophthalmology, Gazi Yasargil Training and Research Hospital, Diyarbakır, Türkiye*
ORCiD: *0000-0002-0667-2866*
dr.dertsiz@hotmail.com

Keywords
Choroidal neovascularization; Hyperbaric oxygenation; Visual acuity

Abstract

(Dağ U, Dertsiz Kozan B, Engin Egeren S, Öncül H, Çağlayan M, Fuat Alakuş M. Spontaneous resolution of choroidal neovascular membrane in the fellow eye during hyperbaric oxygen treatment for retinal artery occlusion: a case report. Diving and Hyperbaric Medicine. 2026 30 June;56(2):195–197. doi: 10.28920/dhm56.2.195-197. PMID: 42290581.)
Retinal artery occlusion (RAO) is an ophthalmic emergency that causes sudden, painless vision loss due to retinal ischaemia. Hyperbaric oxygen therapy (HBOT), when initiated early, may help preserve photoreceptor function by increasing retinal oxygenation. A 69-year-old woman with a history of hypertension presented with sudden visual loss in the left eye and was diagnosed with RAO. HBOT was initiated within six hours of symptom onset (253 kPa [2.5 atmospheres absolute] for 90 minutes per session), and 20 sessions were planned. During the seventh session, the patient reported a marked improvement in visual acuity in the contralateral (right) eye, previously diagnosed with choroidal neovascular membrane (CNVM) and untreated with anti-vascular endothelial growth factor (VEGF) therapy. Optical coherence tomography demonstrated regression of the CNVM and complete resolution of subretinal fluid. To our knowledge, this is the first report suggesting that HBOT administered for unilateral RAO may also promote structural and functional improvement of CNVM in the contralateral eye.

Introduction

Retinal artery occlusion (RAO) is a vision-threatening ophthalmic emergency characterised by sudden, painless monocular vision loss due to the inability of the retinal tissue to meet its high oxygen demand.[1] The inner retinal layers are highly susceptible to ischaemia owing to their elevated metabolic activity. Therefore, early intervention is critical to preserve retinal function. Hyperbaric oxygen therapy (HBOT) enables the inhalation of 100% oxygen under high pressure, increasing oxygen diffusion to ischaemic retinal layers through the choroidal circulation and is considered a potential adjunctive treatment in RAO.[2,3] However, systematic reviews evaluating HBOT in RAO have shown conflicting results; some studies suggest benefit when initiated within the first nine hours,[4] whereas others report no significant improvement in final visual outcomes.[5] Additionally, preliminary evidence indicates that HBOT may have therapeutic effects in other ocular pathologies such as choroidal neovascular membranes (CNVM).[6] In this context, structural and functional improvement of CNVM in the contralateral eye during HBOT for unilateral RAO represents, to the best of our knowledge, an unreported clinical observation.

Case report

Written informed consent was obtained from the patient for publication of this case report and the accompanying images.

A 69-year-old woman with a history of hypertension presented with sudden, painless visual loss in the left eye, beginning six hours prior. Best-corrected visual acuity (BCVA) was counting fingers at five metres in the right eye and counting fingers at one metre in the left eye. Fundus examination of the left eye showed retinal whitening in the inferior hemiretina (Figure 1). Fundus fluorescein angiography demonstrated macular staining in the right eye and arterial filling defect in the inferior retina of the left eye, consistent with RAO (Figure 2). Both eyes exhibited peripapillary atrophy and high myopia > -5.00 D. Optical coherence tomography (OCT) of the right eye revealed marked hyperreflectivity of the inner retinal layers, loss of the foveal contour, and a subretinal hyperreflective

Figure 1
Pre-treatment fundus photograph of the left eye showing marked retinal whitening in the inferior hemiretina

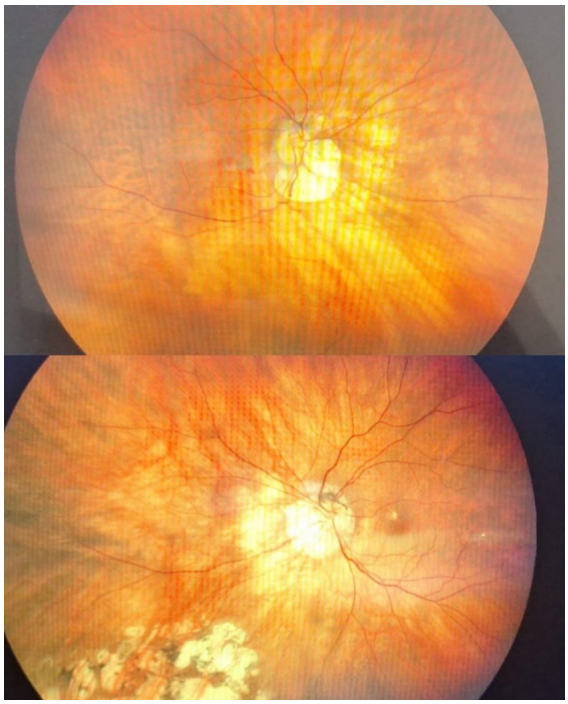
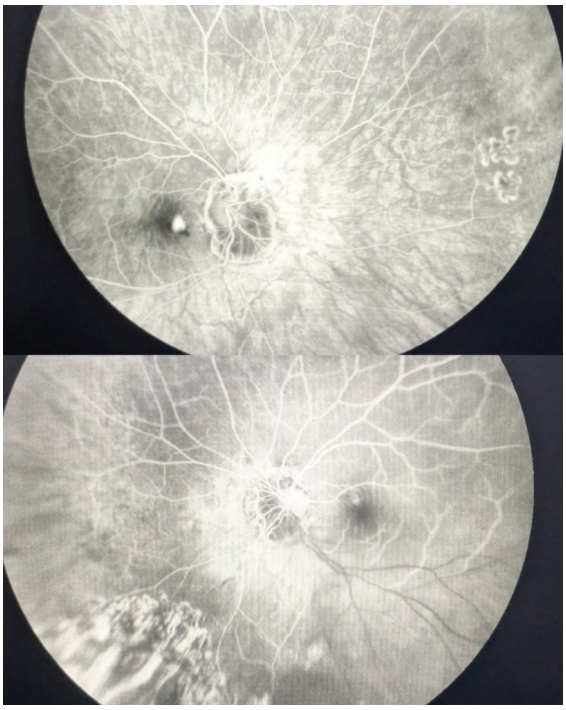


Figure 2
Pre-treatment fluorescein angiography demonstrating an arterial filling defect in the inferior retina of the left eye



lesion (Figure 3). HBOT was initiated within six hours of symptom onset (253 kPa [2.5 atmospheres absolute], 90 minutes daily), with 20 sessions planned. During the seventh session, the patient reported subjective visual improvement in the right eye, previously diagnosed with CNVM but untreated with anti-vascular endothelial growth factor (anti-VEGF) injections. Follow-up OCT demonstrated significant regression of the CNVM and complete resolution of subretinal fluid (Figure 3). No anti-VEGF therapy or other interventions were administered during the treatment period. At final follow-up, BCVA was 2/10 in the right eye and counting fingers at five metres in the left eye.

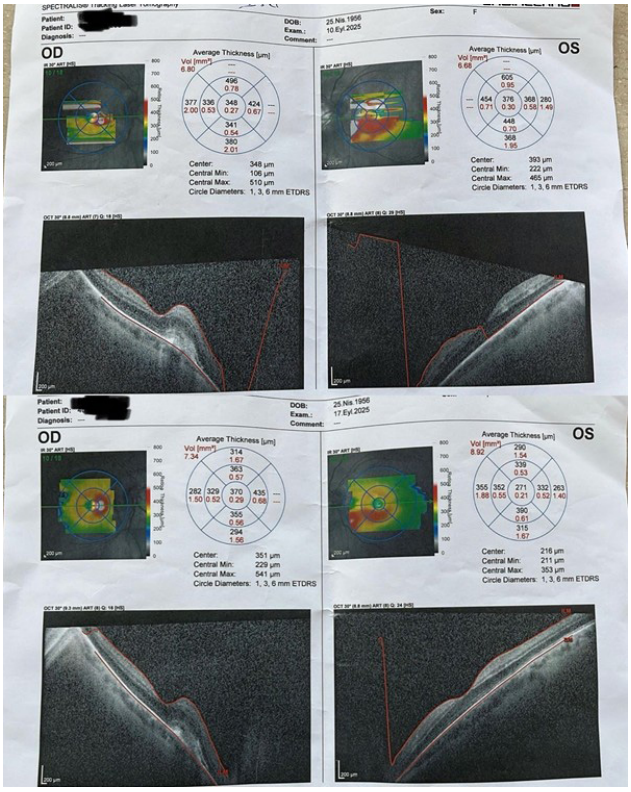
Discussion

Although RAO is one of the most dramatic causes of sudden vision loss, there is still no standardised and effective treatment available. Interventions such as ocular massage, intraocular pressure-lowering agents, carbogen inhalation, and anticoagulant therapy have been used, but none have demonstrated a consistent improvement in visual prognosis.[1,2] Therefore, HBOT, which aims to maintain the viability of hypoxic retinal tissue, stands out as one of the few rational treatment options with a physiological basis. The goal of HBOT is to increase systemic oxygen pressure and promote oxygen diffusion to ischaemic retinal layers through the choroidal circulation.[3] However, studies to date have reported highly variable treatment outcomes, depending on factors such as time to therapy initiation, duration of ischaemia, and systemic health status of the patient.[4–6] Choroidal neovascular membrane typically demonstrates a progressive course if left untreated. The proliferation of abnormal choroidal vessels beneath the retina may lead to subretinal haemorrhage, exudation, and fibrotic scar formation, frequently resulting in permanent central vision loss. Spontaneous regression is uncommon, and the visual prognosis is generally poor without treatment.[7]

In the present case, while HBOT was initiated for unilateral RAO, an unexpected structural and functional improvement was observed in the contralateral eye with previously diagnosed CNVM. To our knowledge, there are no prior reports describing regression of CNVM in the fellow eye during HBOT administered for RAO. This finding suggests that HBOT may induce systemic or cross-ocular effects at the level of the retina and choroid.

The underlying pathophysiological mechanism remains unclear. HBOT increases the amount of oxygen dissolved in plasma, facilitating oxygen diffusion into hypoxic retinal tissues and potentially suppressing hypoxia-inducible factor and VEGF expression.[8] Retinal hypoxia is known to upregulate VEGF, contributing to neovascularisation in both age-related macular degeneration and other ischaemic retinopathies.[9,10] Increased systemic oxygenation may therefore indirectly downregulate VEGF-mediated angiogenesis, contributing to regression of CNVM. Experimental models have also demonstrated inhibitory effects of hyperoxia on choroidal neovascularisation, including downregulation of VEGF and inflammatory mediators.[11] Clinically, Malerbi et al. previously reported anatomical improvement in CNVM following HBOT,[6] and

Figure 3
Optical coherence tomography(OCT) images; pre-treatment OCT shows hyperreflectivity of the inner retinal layers, loss of foveal contour, and a subretinal hyperreflective lesion. Post-treatment OCT demonstrates regression of the choroidal neovascular membrane (CNVM) and complete resolution of subretinal fluid



our observation is consistent with these findings, further suggesting that similar effects may occur in the fellow eye during systemic hyperoxia.

Spontaneous regression of CNVM cannot be completely ruled out; however, such regression is considered extremely rare (< 1%) in eyes not treated with anti-VEGF agents.[12] The temporal correlation between the initiation of HBOT and the observed CNVM regression, along with the absence of any additional therapy, supports the possibility that HBOT may have contributed to this outcome.

Conclusion

Structural and functional improvement of a contralateral CNVM may occur during HBOT administered for unilateral RAO. Further clinical and experimental research is needed to determine whether this represents a reproducible therapeutic effect or a coincidental finding.

References

1 Celebi ARC. Hyperbaric Oxygen therapy for central retinal artery occlusion: Patient selection and perspectives. Clin Ophthalmol. 2021;15:3443–57. doi: 10.2147/OPTH.S224192. PMID: 34413628. PMCID: PMC8370578.

2 Murphy-Lavoie H, Butler F, Hagan C. Central retinal artery occlusion treated with oxygen: a literature review and treatment algorithm. Undersea Hyperb Med. 2012;39:943–53. PMID: 23045923.

3 Beiran I, Goldenberg I, Adir Y, Tamir A, Shupak A, Miller B. Early hyperbaric oxygen therapy for retinal artery occlusion. Eur J Ophthalmol. 2001;11(4):345–50. doi: 10.1177/112067210101100405. PMID: 11820305.

4 Rosignoli L, Chu ER, Carter JE, Johnson DA, Sohn JH, Bahadorani S. The effects of hyperbaric oxygen therapy in patients with central retinal artery occlusion: A retrospective study, systematic review, and meta-analysis. Korean J Ophthalmol. 2022;36:108–13. doi: 10.3341/kjo.2021.0130. PMID: 34743490. PMCID: PMC9013555.

5 Tiwari V, Bagga SSJ, Prasad R, Mathurkar S. A Review of current literature on central retinal artery occlusion: Its pathogenesis, clinical management, and treatment. Cureus. 2024;16(3):e55814. doi: 10.7759/cureus.55814. PMID: 38590501. PMCID: PMC10999893.

6 Malerbi FK, Novais EA, Badaró E, de O Bonomo PP, Pereira AJ, Lottenberg CL, et al. Hyperbaric oxygen therapy for choroidal neovascularization: A pilot study. Undersea Hyperb Med. 2015;42:125–31. PMID: 26094287.

7 García-Franco R, García-Roa M, Cárdenas-Almagro R, Valera-Cornejo D, Hernández-Da Mota SE. Progressive massive choroidal neovascularization, an aggressive phenotype: Case report. Case Rep Ophthalmol. 2022;13:490–8. doi: 10.1159/000525269. PMID: 35950030. PMCID: PMC9294954.

8 Chen J, Smith LEH. Retinopathy of prematurity. Angiogenesis. 2007;10:133–40. doi: 10.1007/s10456-007-9066-0. PMID: 17332988.

9 Penn JS, Madan A, Caldwell RB, Bartoli M, Caldwell RW, Hartnett ME. Vascular endothelial growth factor in eye disease. Prog Retin Eye Res. 2008;27:331–71. doi: 10.1016/j.preteyeres.2008.05.001. PMID:18653375. PMCID: PMC2724313.

10 Kurihara T, Westenskow PD, Bravo S, Aguilar E, Friedlander M. Targeted deletion of Vegfa in adult mice induces vision loss. J Clin Invest. 2012;122:4213–7. doi: 10.1172/JCI65157. PMID: 23093773. PMCID: PMC3484459.

11 Ottino P, Finley J, Rojo E, Ottlecz A, Lambrou GN, Bazan HE, et al. Hypoxia activates matrix metalloproteinase expression and the VEGF system in monkey choroid-retinal endothelial cells: Involvement of cytosolic phospholipase A2 activity. Mol Vis. 2004;10:341–50. PMID: 15162095.

12 Spaide RF, Armstrong D, Browne R. Continuing medical education review: choroidal neovascularization in age-related macular degeneration--what is the cause? Retina. 2003;23:595–614. doi: 10.1097/00006982-200310000-00001. PMID: 14574243.

Conflicts of interest and funding: nil

Submitted: 21 February 2026
Accepted after revision: 21 March 2026

Cerebral arterial oxygen embolism as a complication of hyperbaric oxygen treatment: a case report

Rahulkumar Ramchandani[1], Bridget Devaney[1,2,3], Ashish Jaison[1,2,3], Neil Banham[4], Stephan Roehr[1,5]

[1] *Department of Intensive Care and Hyperbaric Medicine, Alfred Health, Melbourne, 3004, Victoria, Australia*
[2] *Emergency and Trauma Centre, Alfred Health, Melbourne, Australia*
[3] *Department of Epidemiology and Preventative Medicine, Monash University, Melbourne, Australia*
[4] *Department of Hyperbaric Medicine, Fiona Stanley Hospital, Perth, Australia*
[5] *Townsville University Hospital Hyperbaric Medicine Unit, Queensland, Australia*

Corresponding author: *Dr Bridget Devaney, Head of Hyperbaric Medicine, Department of Intensive Care and Hyperbaric Medicine, Alfred Health, 55 Commercial Road, Melbourne, VIC 3004, Melbourne, Australia*
ORCiD: *0000-0001-6521-418X*
b.devaney@alfred.org.au

Keywords
Adverse event; Birt-Hogg-Dubé syndrome; Cerebral arterial gas embolism; Oxygen bubble embolus; Pulmonary barotrauma; Lung cysts

Abstract

(Ramchandani R, Devaney B, Jaison A, Banham N, Roehr S. Cerebral arterial oxygen embolism as a complication of hyperbaric oxygen treatment: a case report. Diving and Hyperbaric Medicine. 2026 30 June;56(2):198–202. doi: 10.28920/dhm56.2.198-202. PMID: 42290582.)

Cerebral arterial gas embolism (CAGE) is a recognised complication of diving-related barotrauma and of medical procedures whereby gas enters the vascular system. CAGE is a principal indication for hyperbaric oxygen treatment (HBOT). In contrast, CAGE resulting from HBOT itself is exceedingly rare. We describe the case of a 73-year-old man undergoing his first session of HBOT for a chronic lower limb wound, who developed acute focal neurological deficits during decompression. He had no known respiratory disease or smoking history. Immediate assessment demonstrated stable physiology. Initial computed tomography (CT) scan of the brain and neck vessels and perfusion imaging did not show any evidence of thrombus or intravascular gas, and partial neurological recovery occurred over several hours. Subsequent magnetic resonance imaging of the brain demonstrated acute infarction involving the left precentral and postcentral gyri. High-resolution CT chest revealed multiple bilateral thin-walled pulmonary cysts, including a left upper lobe cyst with an air–fluid level consistent with recent barotrauma. A transient broncho-venous fistula allowing arterial oxygen embolisation during decompression was considered the most plausible mechanism. The patient improved with supportive management and was discharged with minimal residuae. This case highlights an exceptionally rare but serious complication of HBOT and underscores the importance of vigilance during decompression and careful consideration of occult pulmonary pathology.

Introduction

Changes in pressure can lead to pulmonary barotrauma, particularly when gas becomes trapped within the lungs. This is well recognised in diving and aviation, and the same principle applies to hyperbaric oxygen treatment (HBOT). However, decompression during HBOT is slow and carefully controlled, making clinically significant barotrauma extremely uncommon. Cerebral arterial gas embolism (CAGE) arising during HBOT is therefore exceptionally rare. We report what is, to our knowledge, only the third known case of CAGE occurring during decompression from a hyperbaric treatment.

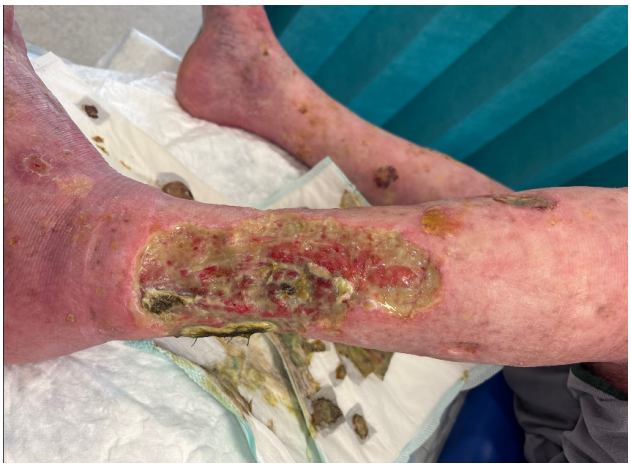
Case report

Written consent was given by the patient for the publication of the case details and images.

A 73-year-old male was referred to our hyperbaric centre for extensive non-healing bilateral lower limb wounds (Figure 1). His history was notable for basal and squamous cell carcinomas, psoriasis, hypertension and bilateral carpal tunnel decompressions. He had no history of lung disease and had never smoked.

The patient had received radiotherapy to the lower extremities three months prior for squamous cell carcinoma,

Figure 1
The wounds for which the patient was referred for hyperbaric oxygen treatment



and psoralen and ultra violet A light treatment for psoriasis. His wounds had been debrided but were not amenable to grafting until further optimised. Macrovascular perfusion was intact. No chest imaging was performed as part of the assessment and no prior imaging was available for review. There were no contraindications for HBOT evident, and a decision was made to offer treatment. Some transport issues required coordination as his home was 80 km from our centre, and he commenced treatment 73 days after his assessment.

No issues were identified on his first treatment day, and a treatment table AH 14 (243 kPa [2.4 atmospheres absolute, 14 metres of sea water equivalent]), with decompression time of 14 minutes and total duration one hour 53 minutes was commenced in an air-filled multiplace chamber (Figure 2). The patient received oxygen via a head hood tent system.

The patient reported no equalisation issues and remained asymptomatic during the compression, treatment and decompression phases of HBOT. On removal of the hood immediately after decompression to atmospheric pressure, he reported being unable to move his right arm and was noted to have right upper limb paralysis and evolving left upper limb weakness. The patient was alert, responsive, and speaking full sentences. Sensation was intact in both upper limbs. There were no other neurological symptoms or signs. His vital signs were normal. Intravenous access was secured and a blood sample sent for venous blood gas which showed pH 7.39, PCO_2 46 mmHg, PO_2 41 mmHg, HCO3 28 mmol·L^{-1}, lactate 2.2 mmol·L^{-1}, sodium 142 mmol·L^{-1}, potassium 4.1 mmol·L^{-1} and blood sugar 7.2 mmol·L^{-1}. His oxygen saturations were 97% on room air. Cardiorespiratory examination was normal, with no increased work of breathing, equal air entry bilaterally, and no signs of pneumothorax. An emergency response and code stroke were activated whilst the patient was transferred to the treatment room for monitoring, from where he was transported supine to the emergency department via the radiology department for imaging, and subsequently admitted under the care of the stroke team.

A computed tomography (CT) perfusion scan of the brain and carotids 35 minutes after onset of symptoms was reported as normal. No pneumothorax was seen on the included imaging of the lung apices. A chest X-ray was reported to be normal. As there was no radiological evidence of stroke on CT and the neurological examination was improving, he was not thrombolysed.

Significant improvement in symptoms occurred rapidly over two to three hours and continued over the course of his admission. CAGE was considered the presumed diagnosis at the time. Further recompression was decided against in view of clinical improvement and risk of further CAGE with a potential deleterious outcome. The day after the event, a magnetic resonance imaging (MRI) scan of the brain and spine was performed and demonstrated left pre-central and post-central gyri infarcts (Figure 3). High-resolution CT (HRCT) chest demonstrated multiple bilateral thin-walled lung cysts involving all lobes, with mid- to lower-zone predominance and subpleural distribution, and no evidence of emphysema (indicated with arrows in Figure 4). A dominant left upper lobe cyst measured 15 × 24 mm and contained an air–fluid level with adjacent ground-glass change (marked with an X in Figure 4). A communicating bronchus was identified along the cyst margin. No intravascular gas was seen. Two additional smaller cysts in the right middle lobe demonstrate air-fluid levels. The pleural spaces were clear. Pulmonary arteries were of normal size. No air was visualised within the cardiac chambers.

In summary, the HRCT demonstrated cystic lung disease, with imaging features suspicious for Birt-Hogg-Dubé syndrome, and with left upper lobe cyst rupture as a result of occult barotrauma. The impression of the reporting radiologist was that perhaps a transient broncho-venous fistula had allowed a gas embolus to the brain.

Following his rapid improvement, the patient was commenced on aspirin and atorvastatin and discharged home within 24 hours, with just some remaining numbness in the palm of his right hand. On follow up by telephone the day after discharge, he reported a small area of numbness on the palm of the left hand. Respiratory physician and GP follow up were advised for further investigation of potential Birt-Hogg-Dubé Syndrome.

Discussion

DIAGNOSIS

Cerebral arterial gas embolism should be strongly considered when focal or global neurological symptoms occur during, or within seconds to minutes of completion of decompression from HBOT or compressed gas diving. The differential

Figure 2
Treatment table AH 14

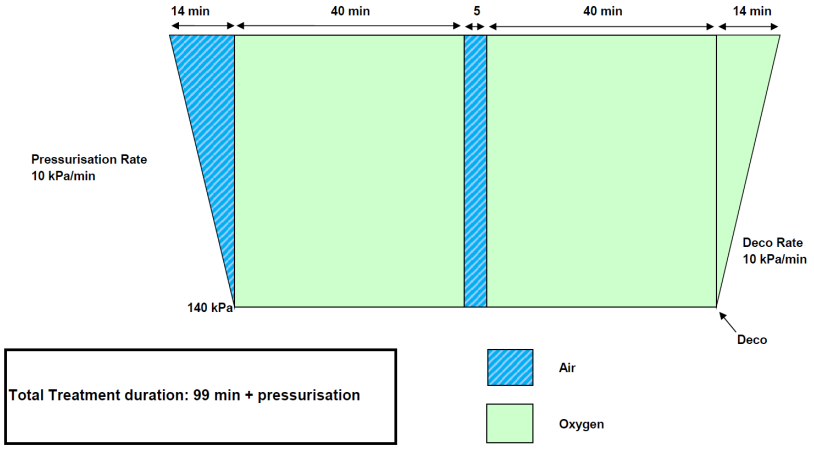


Figure 3
Magnetic resonance imaging of the brain demonstrating acute infarcts of the left precentral and postcentral gyri

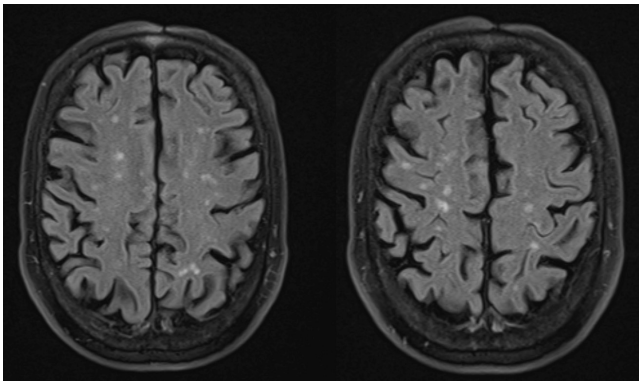
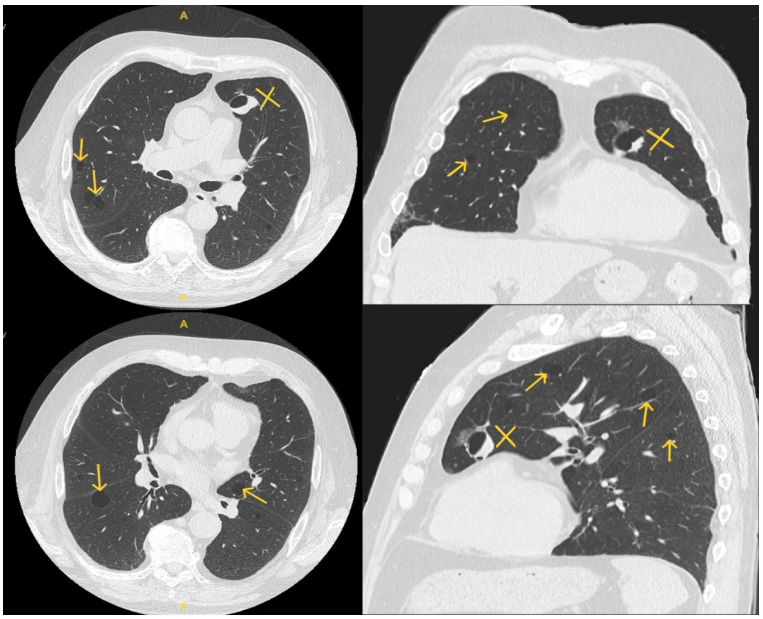


Figure 4
High resolution computed tomography images demonstrating multiple pulmonary cysts



diagnosis in this setting includes acute ischaemic stroke, intracranial haemorrhage, seizure including oxygen toxicity seizure, hypoglycaemia, and hypoxic events; however, the abrupt onset with decompression, rapid partial neurological recovery, and subsequent radiological findings in this case are most consistent with arterial gas embolism.

The proposed mechanism of embolism in this case is pulmonary barotrauma with cyst rupture during decompression, resulting in transient communication between the bronchial and pulmonary venous systems and subsequent cerebral arterial embolisation.

Underlying pulmonary pathology was identified in this patient, with imaging features suggestive of Birt-Hogg-Dubé syndrome. This autosomal dominant condition, caused by mutations in the folliculin (FLCN) tumour suppressor gene, is characterised by pulmonary cysts, spontaneous pneumothorax, characteristic benign cutaneous papules, and an increased risk of renal malignancy. While lung cysts are relatively common incidental findings in thoracic imaging, the incidence of arterial gas embolism in patients with cystic lung disease undergoing HBOT remains extremely low.[1]

Cerebral arterial gas embolism is well described in association with rapid ascent and breath-hold during ascent from compressed gas diving,[2,3] as well as following decompression during commercial air travel.[4–6] In these settings, the entrained gas is typically air, reflecting the gas being breathed at the time. Likewise, CAGE occurring during pressure tolerance training of military divers in an air-filled hyperbaric chamber are likely to have had air as the offending gas rather than oxygen.[7]

In contrast, after prolonged exposure to hyperbaric oxygen treatment, it is likely that gas entering the vascular system following cyst rupture would be predominantly oxygen. Gas composition, solubility and metabolic uptake influence the behaviour of gaseous emboli, endothelial injury, downstream ischaemia and resultant clinical consequences. Early experimental work by Kunkler and King indicated that an embolus made up of oxygen is about twice as well tolerated as one consisting of air.[8] A report from the early 1900s, describes the injection of oxygen gas as a therapeutic measure.[12]

Whilst this case occurred during a 14-minute decompression from 243 kPa, the previously reported cases in the HBOT setting occurred with even slower decompression over 30 minutes – highlighting that this complication can occur even with very slow rates of decompression.

MANAGEMENT

Management of CAGE occurring during HBOT presents a unique challenge. While recompression is the standard treatment for arterial gas embolism resulting from diving or iatrogenic entrainment,[2] there is uncertainty regarding the management of CAGE when the embolism itself occurs as a complication of HBOT. Two previously published cases demonstrate this uncertainty.[9,10] In one, HBOT was readministered, but progressive deterioration with ventricular arrhythmias, haemodynamic instability, and death followed.[9] In the other, supportive management without recompression was associated with partial neurological recovery but severe permanent disability.[10]

One of the reported oxygen embolism cases, as well as our present case, occurred during the first HBOT session, suggesting a potentially high risk of recurrence should HBOT be re-administered.[10] This raises important questions regarding the balance of potential benefit versus harm when considering readministering HBOT in this context.

The solubility characteristics of oxygen differ substantially from nitrogen, with oxygen being more rapidly resorbed and potentially less harmful than air bubbles.[8,11,12] This has led to interest in alternative management strategies. The use of normobaric heliox may theoretically accelerate off-gassing and reduce bubble size by increasing the oxygen diffusion gradient, although a counter argument exists that due to increased solubility, helium might diffuse into a bubble faster than oxygen may diffuse out, and limit the rate of reduction, or even increase, the size of the bubble; clinical evidence to support either argument remains limited, and for this reason the authors currently suggest keeping the patient breathing air only, provided the patient's oxygen saturations are normal. If a patient has been decompressed on any other gas than oxygen, supplemental oxygen would of course still be indicated.

Another cornerstone of treatment of an air embolism is the use of a high fraction of inspired oxygen to denitrogenate the tissues and increase the gradient of intra-bubble to intra-vascular or tissue nitrogen. The authors postulate that in the case of an oxygen embolism, increasing the inspired oxygen fraction may offer less benefit than in nitrogen-containing emboli, and may theoretically reduce the diffusion gradient for bubble resolution; however, clinical evidence to guide optimal gas management in this context is lacking.

RECOMMENDATIONS

Oxygen gas embolism is an extremely rare complication of HBOT, with only two prior published case reports to the best of our knowledge.[9,10] In low risk populations, the use of routine imaging prior to HBOT does not provide a cost-effective way to reduce the risk of barotrauma.[1,13–15] Given the extremely low incidence of either barotrauma or oxygen gas embolism resulting from HBOT, we do not advocate for increased screening practices for underlying pulmonary disease.

Clinicians assessing suitability of patients for HBOT should pay close attention to respiratory pathology at the time of assessment; emphasis should be on thorough history,

examination and review of any prior imaging, and, given its greater sensitivity over chest X-ray, HRCT chest should be the preferred imaging modality for those in whom concerns are identified. Lung function testing is unlikely to reveal significant abnormality in the majority of patients with Birt-Hogg-Dubé syndrome.[16]

Heightened vigilance is paramount during the decompression phase of HBOT and any onset of neurological change during or immediately after decompression should raise suspicion for arterial oxygen embolism, in the same way immediate collapse on surfacing from a dive is strongly suggestive of CAGE. Clinicians should consider performing HRCT chest at the same time as a stroke protocol CT.

The optimal management of cerebral arterial oxygen embolism from pulmonary barotrauma during HBOT likely differs in some ways from other CAGE events. With the goal of '*primum non nocere*' we elected to not recompress the patient, and whilst we welcome input on the best management of this rare condition from the wider diving and hyperbaric medicine community, we postulate that the optimal inspired gas to speed the off-gassing of oxygen from a cerebral arterial oxygen embolus, may in fact, be air.

Conclusions

We report an extremely rare case of cerebral arterial oxygen embolism associated with decompression in a hyperbaric chamber in a patient with multiple, previously undiagnosed lung cysts. Given the rarity of this complication, we do not recommend change in screening practices, however we encourage careful consideration of possible respiratory pathology during patient assessment and in particular, vigilance during the decompression phase of HBOT is strongly advised.

References

1 Türkmen O, Özkan R, Özgök Kangal K, Dur Ince M, Arslan Y. Hyperbaric oxygen treatment and pulmonary air-containing lesions. Diving Hyperb Med. 2025;55:343–51. doi: 10.28920/dhm55.4.343-351. PMID: 41364858.

2 Mitchell SJ. Decompression illness: a comprehensive overview. Diving Hyperb Med. 2024;54(Suppl):1–53. doi: 10.28920/dhm54.1.suppl.1-53. PMID: 38537300. PMCID: PMC11168797.

3 Banham ND, Lippmann J. Fatal air embolism in a breath-hold diver. Diving Hyperb Med. 2019;49:304–5. doi: 10.28920/dhm49.4.304-305. PMID: 31828750. PMCID: PMC7039776.

4 Closon M, Vivier E, Breynaert C, Duperret S, Branche P, Coulon A, et al. Air embolism during an aircraft flight in a passenger with a pulmonary cyst: a favorable outcome with hyperbaric therapy. Anesthesiology. 2004;101:539–42. doi: 10.1097/00000542-200408000-00037. PMID: 15277939.

5 Arnaiz J, Marco de Lucas E, Piedra T, Arnaiz Garcia ME, Patel AD, Gutierrez A. In-flight seizures and fatal air embolism: the importance of a chest radiograph. Arch Neurol. 2011;68:661–4. doi: 10.1001/archneurol.2011.85. PMID: 21555644.

6 Zaugg M, Kaplan V, Widmer U, Baumann PC, Russi EW. Fatal air embolism in an airplane passenger with a giant intrapulmonary bronchogenic cyst. Am J Respir Crit Care Med. 1998;157:1686–9. doi: 10.1164/ajrccm.157.5.9706040. PMID: 9603155.

7 Buschmann DK. Arterial gas embolism during pressure tolerance testing in a hyperbaric chamber: a report of two cases. Aviat Space Environ Med. 2010;81:1133–6. PMID: 21197859.

8 Kunkler A, King H. Comparison of air, oxygen and carbon dioxide embolization. Ann Surg. 1959;149:95–9. doi: 10.1097/00000658-195901000-00012. PMID: 13617914. PMCID: PMC1450962.

9 Wolf HK, Moon RE, Mitchell PR, Burger PC. Barotrauma and air embolism in hyperbaric oxygen therapy. Am J Forensic Med Pathol. 1990;11:149–53. doi: 10.1097/00000433-199006000-00009. PMID: 2343842.

10 Rivalland G, Mitchell SJ, van Schalkwyk JM. Pulmonary barotrauma and cerebral arterial gas embolism during hyperbaric oxygen therapy. Aviat Space Environ Med. 2010;81:888–90. doi: 10.3357/asem.2783.2010. PMID: 20824998.

11 Tovar EA, Del Campo C, Borsari A, Webb RP, Dell JR, Weinstein PB. Postoperative management of cerebral air embolism: gas physiology for surgeons. Ann Thorac Surg. 1995;60:1138–42. doi: 10.1016/0003-4975(95)00531-o. PMID: 7574975.

12 Tunnicliffe FW, Stebbing GF. The intravenous injection of oxygen gas as a therapeutic measure. The Lancet. 1916:188(4851):321–3. doi: 10.1016/S0140-6736(00)97237-4.

13 Toklu AS, Kiyan E, Aktas S, Cimsit M. Should computed chest tomography be recommended in the medical certification of professional divers? A report of three cases with pulmonary air cysts. Occup Environ Med. 2003;60:606–8. doi: 10.1136/oem.60.8.606. PMID: 12883024. PMCID: PMC1740603.

14 Brenna CT, Khan S, Djaiani G, Buckey JC Jr, Katznelson R. The role of routine pulmonary imaging before hyperbaric oxygen treatment. Diving Hyperb Med. 2022;52:197–207. doi: 10.28920/dhm52.3.197-207. PMID: 36100931. PMCID: PMC9731143.

15 Millar IL. Should computed tomography of the chest be recommended in the medical certification of professional divers? Br J Sports Med. 2004;38:2–3. doi: 10.1136/bjsm.2003.010413. PMID: 14751933. PMCID: PMC1724731.

16 Daccord C, Cottin V, Prévot G, Uzunhan Y, Mornex JF, Bonniaud P, et al. Lung function in Birt-Hogg-Dubé syndrome: a retrospective analysis of 96 patients. Orphanet J Rare Dis. 2020;5:120–7. doi: 10.1186/s13023-020-01402-y. PMID: 32448321. PMCID: PMC7245949.

Acknowledgements

We would like to thank the patient for allowing us to present his case and deidentified imaging.

Conflicts of interest and funding: nil

Submitted: 2 January 2026
Accepted after revision: 26 April 2026

Returning to diving and hyperbaric exposure after pulmonary vein isolation for atrial fibrillation

Silvio Zerbi[1], Luigi Tarsia[2], Vincenzo Benenati[3], Dario Nicosia[4], Gerardo Bosco[5], Matteo Paganini[2]

[1] *Department of Anesthesiology and Intensive Care, ASST Sette Laghi, Varese, Italy*
[2] *Independent Researcher, Padova, Italy*
[3] *Anesthesiology, Intensive Care and Hyperbaric Medicine, Policlinico Universitario "P. Giaccone", Palermo, Italy*
[4] *UOC Centrale Operativa 118, ARNAS Civico, Palermo, Italy*
[5] *Department of Medicine and Aging Sciences, University "G. D'Annunzio" Chieti – Pescara, Italy*

Corresponding author: *Dr Silvio Zerbi, Department of Anesthesiology and Intensive Care. ASST Sette Laghi, Viale Borri, 57 – 21100 Varese (VA), Italy*
ORCiD: *0009-0004-4280-432X*
silvio.zerbi@asst-settelaghi.it

Keywords
Catheter ablation; Electrocardiography; Electrophysiology; Diving medicine; Hyperbaric oxygen; Hyperbaric medicine; Implantable loop recorder

Abstract

(Zerbi S, Tarsia L, Benenati V, Nicosia D, Bosco G, Paganini M. Returning to diving and hyperbaric exposure after pulmonary vein isolation for atrial fibrillation. Diving and Hyperbaric Medicine. 2026 30 June;56(2):203–207. doi: 10.28920/dhm56.2.203-207. PMID: 42290583.)

Pulmonary vein isolation (PVI) is an established rhythm-control therapy for atrial fibrillation (AF), yet the electrophysiological response of post-PVI individuals exposed to hyperbaric environments remains undocumented. Similarly, the in-vivo performance of implantable loop recorders (ILRs) and external patch-based electrocardiographic (ECG) devices under increased ambient pressure has never been reported. We describe the first hyperbaric electrophysiology assessment in a post-PVI diver undergoing both underwater immersion and dry hyperbaric exposure with dual-modality cardiac rhythm monitoring. A 46-year-old experienced diver with successful PVI underwent: a scuba dive to 42 m in a warm water pool, monitored with a marinised 12-lead ECG Holter system; and a stepwise hyperbaric chamber compression to 284 kPa (2.8 atmospheres absolute) in ambient air, with single-lead surface ECG recordings obtained at static pressure plateaus. In both cases, the subject was monitored as well by his ILR. No AF recurrence or other dysrhythmias were detected during either exposure, with stable heart rate trends. The ILR maintained full functional integrity after both the 42 m dive and the 284 kPa chamber compression. The external ECG patch yielded interpretable tracings during static phases. Telemetry failed due to electromagnetic shielding by the steel chamber walls. This case suggests that carefully selected post-PVI individuals may tolerate controlled underwater and hyperbaric exposure without rhythm destabilisation. Both implantable and external monitoring devices preserved operational integrity under moderate hyperbaric conditions, providing a foundation for the emerging field of hyperbaric electrophysiology monitoring and informing fitness-to-dive assessment in post-ablation patients.

Introduction

Atrial fibrillation (AF) is the most common sustained arrhythmia in adults,[1,2] usually treated with rhythm or rate control, lifestyle modification, and risk factor management. Catheter ablation via pulmonary vein isolation (PVI) is a well-established, second-line therapy for symptomatic paroxysmal or persistent AF not responding to antiarrhythmic therapy, with demonstrated efficacy in maintaining sinus rhythm and reducing arrhythmic burden.[3,4] In selected cases, PVI may also be considered as first-line treatment, such as for patients with heart failure or specific occupational requirements.[4–6]

AF is increasingly prevalent in older adults,[7] a population well represented among active divers,[8] but no evidence-based guidelines address fitness-to-dive evaluation in divers with AF or post-ablation clearance to resume diving.[9] Here we describe a post-PVI scuba diver assessed under wet (underwater) and dry (hyperbaric chamber) conditions using dual-modality cardiac monitoring, aiming to characterise rhythm stability, evaluate monitoring device performance under hyperbaric conditions, and propose a conceptual framework at the intersection of electrophysiology, diving medicine, and biomedical engineering to assist in the fitness-to-dive evaluation.

Methods

The study was approved by the Ethics Committee of the Department of Biomedical Sciences, University of Padova – HEC-DSB/06-2023. The subject provided written informed consent for the experiment, and consented for the publication of his case details and data.

The subject, one of the authors, was a 46-year-old male recreational diver and diving physician. He developed unstable, symptomatic AF in 2021, initially managed with rhythm control including two electrical cardioversions. An implantable loop recorder (ILR) was implanted in April 2022 for long-term rhythm surveillance; successful PVI followed in October 2022 after shared decision-making with the electrophysiology team. Since serial ILR interrogations confirmed sustained sinus rhythm without AF recurrence, transthoracic echocardiography showed normal cardiac structure and function, and he was not taking antiarrhythmic medications, he was cleared to resume diving and hyperbaric work, and elected to self-monitor on first returning to each activity.

ELECTROPHYSIOLOGICAL MONITORING SYSTEMS

Implantable loop recorder

A Biomonitor IIIm ILR (BIOTRONIK, Berlin, Germany), implanted subcutaneously on the left anterior chest in April 2022, records single-lead electrocardiograms (ECGs) with patient-activated and automated arrhythmia detection (AF, bradycardia, tachycardia, pauses). Telemetry uses the medical implant communication system band (402–405 MHz). Patient-activated recordings capture approximately seven minutes before and one minute after activation, via a rolling memory buffer. Manufacturer guidelines advise against hyperbaric exposure exceeding 150 kPa (1.5 bar);[10] however, the company reported structural integrity after testing the device through 40 cycles at approximately 450 kPa (≈4.5 bar). The pressures in this study remained within those laboratory-tested limits, though off-label.

Conventional ECG monitoring

A single-lead Cardionica patch (Medical International Research S.p.A., Rome, Italy) was used for rhythm monitoring during chamber compression. Acquisition was limited to static pressure plateaus, as the device lacks hyperbaric certification.

Underwater Holter recording

A Holter ECG H12+ (Welch Allyn Inc., NY, USA) was housed in a sealed steel cylinder with external cable port for underwater deployment. Standard chest electrodes were waterproofed with dual-layer transparent adhesive dressings. The 12-lead recording was interpreted by a sports medicine cardiologist.

UNDERWATER DIVING AND HYPERBARIC PROTOCOLS

Both sessions were supervised by hyperbaric and diving medicine specialists with biomedical engineering oversight.

Underwater dive

The subject completed a scuba dive to 42 metres of freshwater (mfw) at the Y-40® "*The Deep Joy*" facility (Montegrotto Terme, Italy; water 31.5 ± 0.5°C), breathing compressed air on open circuit without a diving suit. Descent was at approximately 1 m·min^{-1}; after ~5 minutes of bottom time (no-decompression profile), ascent proceeded at ~1–2 m·min^{-1} with a three-minute safety stop at 5 mfw (total dive ~40 minutes; Figure 1A). The ILR was manually activated before water entry and after resurfacing, capturing approximately the first minute and last seven minutes of the immersion. Continuous ECG was recorded simultaneously via the waterproof Holter. Depth–time data were extracted from the dive computer log.

Dry hyperbaric chamber exposure

Hyperbaric exposure was performed at the Policlinico "*Paolo Giaccone*," Palermo, in ambient air with stepwise compression to 284 kPa (2.8 atmospheres absolute [atm abs]) (equivalent depth 18 m), including three-minute plateaus at 152 kPa and 203 kPa (1.5 and 2.0 atm abs) during which the ECG patch was activated (Figure 1B). Telemetry with the ILR was unsuccessful due to steel-wall radiofrequency shielding. Decompression was completed in oxygen. The ILR was manually activated before compression and after decompression, capturing approximately the first minute and last seven minutes, respectively. Total hyperbaric exposure was ~40 minutes.

Results

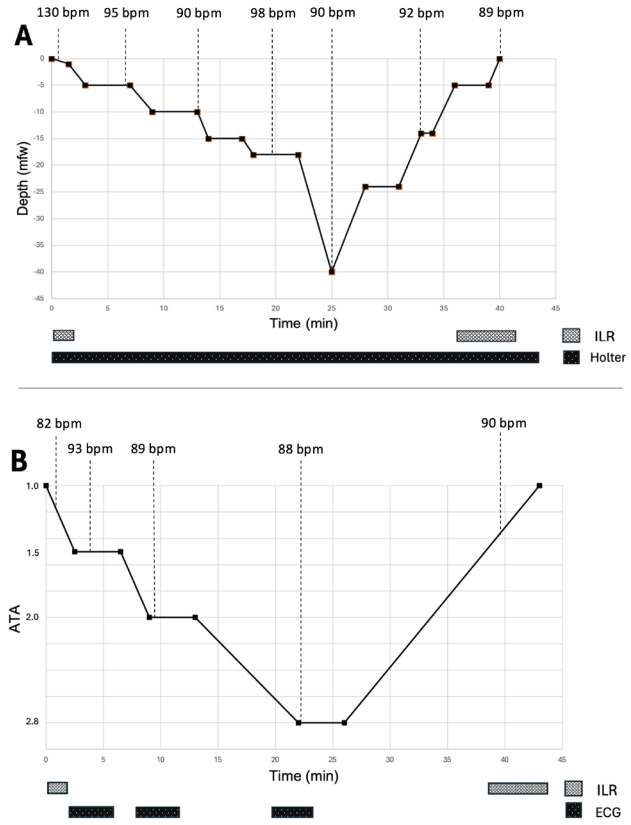
The subject completed both exposures without symptoms. During the pool dive, both the waterproof Holter ECG and start/end-immersion ILR recordings demonstrated sinus rhythm, with no dysrhythmias detected. Mild sinus tachycardia was noted on water entry, with subsequent heart rate stabilisation (Figure 1A). During the dry hyperbaric exposure, the ECG and ILR again showed sinus rhythm with stable heart rate (Figure 1B). ILR interrogation after each session confirmed the absence of events over the subsequent 48 hours and verified full device functionality.

Discussion

To our knowledge, this is the first report to describe ECG behaviour in an individual exposed to elevated

Figure 1

Depth–time and pressure–time profiles during underwater immersion (panel A) and dry hyperbaric exposure (panel B); heart rates in beats per minute (bpm) recorded at selected time points are reported above each panel. Shaded bars below each panel indicate active monitoring windows for the different devices employed. ATA – atmospheres absolute; ECG – electrocardiogram; ILR – implantable loop recorder; mfw – metres of freshwater



environmental pressure after PVI for AF, under both dry and submerged (wet) conditions. The participant's self-monitoring addresses two previously unexamined questions: (i) whether hyperbaric exposure can unmask clinically relevant dysrhythmias following an apparently successful PVI, and (ii) whether implantable and external cardiac rhythm-monitoring systems retain functional integrity and provide reliable signal acquisition during and after exposure to increased ambient pressure.

The underwater environment imposes abrupt cardiovascular loading through central blood shift, increased cardiac filling, and atrial stretch, compounded by autonomic perturbations from concurrent sympathetic and parasympathetic activation.[11] These combined preload/afterload and autonomic perturbations may alter atrial refractoriness and conduction, increasing the likelihood of arrhythmia expression in susceptible individuals. Resuming diving after PVI raises the concern that the diving response may challenge the post-ablation atrial substrate, where recurrence and ectopy remain possible despite a successful procedure. The hyperbaric chamber exposure was designed to separate pressure effects from immersion and to support the subject's return to hyperbaric work. Exposure to 284 kPa (2.8 atm abs) breathing air is generally associated with modest cardiovascular effects;[12,13] however, the small rise in inspired oxygen partial pressure may theoretically promote a shift toward parasympathetic predominance and higher heart rate variability, based on observations under hyperoxic conditions.[14,15] The absence of AF recurrence–or other dysrhythmias sufficient to trigger the ILR alarm–suggests the participant's rhythm was not destabilised in either setting. Heart rate trends were stable in both exposures, with only transient sinus tachycardia on water entry (Figure 1 A), consistent with prior observations.[16] In the chamber, serial ECG recordings demonstrated sinus rhythm within the physiological range (Figure 1 B), possibly reflecting appropriate autonomic adaptation to the gradual compression profile.

This case also addresses whether cardiac rhythm-monitoring devices retain functional integrity under hyperbaric conditions. Prior work by some of the authors emphasised the heterogeneity in manufacturer suitability statements and pressure limits among implantable cardiac devices.[17] In many cases, regulatory disclaimers discouraging hyperbaric exposure appear to reflect limited or absent human testing rather than documented device malfunction. Here, the ILR maintained continuous operation and preserved sensing performance throughout both exposures–providing, to our knowledge, the first published in-vivo confirmation under these conditions. The waterproof Holter system supported the feasibility of rhythm assessment during diving, albeit without real-time transmission. The external ECG patch yielded good-quality tracings during static phases, but telemetry communication attempt was unsuccessful, plausibly due to radiofrequency attenuation by the steel walls. Further study is warranted to optimise external device reliability and develop remote monitoring capability–especially underwater.

These findings are relevant to the evaluation of individuals with AF, particularly given the absence of prospective evidence and dedicated consensus guidelines. In carefully selected patients with sustained sinus rhythm stability, hyperbaric exposure may be considered on a case-by-case basis after comprehensive multidisciplinary evaluation by electrophysiology, cardiology, sports medicine, and diving medicine specialists. However, individuals with residual AF burden, structural heart disease, or ongoing antiarrhythmic therapy should not be assumed to share a comparable risk profile. For individuals at elevated risk of developing AF, reversible lifestyle factors should be modified and diving restricted until these are addressed. Newly diagnosed AF warrants thorough evaluation to exclude relevant

comorbidities, such as structural or ischemic heart disease, before diving clearance, and should be withheld until rhythm control and clinical stability are achieved. Antiarrhythmic drugs merit particular caution, as autonomic fluctuations during immersion can provoke benign ectopy in healthy individuals,[16] but may carry greater risk of dysrhythmias in predisposed patients. Regarding occupational hyperbaric exposure, protocols requiring pressures ≥ 304 kPa (3 atm abs) or special gas mixtures should be avoided. Finally, careful selection of candidates for AF ablation could ensure a prompt activity resumption. Future research developments should include a follow-up of divers diagnosed with AF (both ablated and not), and the integration of biomedical engineering expertise into the multidisciplinary team to develop monitoring solutions enabling direct observation during hyperbaric exposure.

As a single-subject report, these observations have limited generalisability. The pressure exposures evaluated here were moderate relative to those achieved in technical or professional diving, and neither helium-based mixtures nor prolonged hyperbaric plateaus were monitored. These more extreme conditions may entail substantially greater physiological stress in individuals with prior AF ablation, underscoring the need for systematic study.

Conclusions

This first documented ECG assessment under hyperbaric conditions in an individual after PVI suggests that, in carefully selected cases, recreational diving and hyperbaric exposure may be tolerated without clinically evident rhythm destabilisation. Although generalisability is limited to a single case, well-selected post-ablation patients may be considered for return to diving or hyperbaric chamber exposure following comprehensive, multidisciplinary evaluation. Future research should prioritise follow-up studies and real-time monitoring systems for reliable acquisition during compression, decompression, and immersion.

References

1 Khurshid S, Choi SH, Weng LC, Wang EY, Trinquart L, Benjamin EJ, et al. Frequency of cardiac rhythm abnormalities in a half million adults. Circ Arrhythm Electrophysiol. 2018;11(7):e006273. doi: 10.1161/CIRCEP.118.006273. PMID: 29954742. PMCID: PMC6051725.

2 Chugh SS, Havmoeller R, Narayanan K, Singh D, Rienstra M, Benjamin EJ, et al. Worldwide epidemiology of atrial fibrillation: a global burden of disease 2010 Study. Circulation. 2014;129:837–47. doi: 10.1161/CIRCULATIONAHA.113.005119. PMID: 24345399. PMCID: PMC4151302.

3 Parameswaran R, Al-Kaisey AM, Kalman JM. Catheter ablation for atrial fibrillation: current indications and evolving technologies. Nat Rev Cardiol. 2021;18:210–25. doi: 10.1038/s41569-020-00451-x. PMID: 33051613.

4 Van Gelder IC, Rienstra M, Bunting KV, Casado-Arroyo R, Caso V, Crijns HJGM, et al. 2024 ESC guidelines for the management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J. 2024;45(36):3314–414. doi: 10.1093/eurheartj/ehae176. PMID: 39210723.

5 Wilson AS, Keithler AN, Tunzi MA, Bush KNV. Efficacy and safety of pulmonary vein isolation in active duty military members with atrial fibrillation. Mil Med. 2025;190(9-10):e1972–e1978. doi: 10.1093/milmed/usaf112. PMID: 40202864.

6 Guettler N, Sammito S. Management of atrial fibrillation in German military aircrew. J Occup Med Toxicol. 2023;18(1):13. doi: 10.1186/s12995-023-00383-5. PMID: 37482616. PMCID: PMC10364391.

7 Linz D, Gawalko M, Betz K, Hendriks JM, Lip GYH, Vinter N, et al. Atrial fibrillation: epidemiology, screening and digital health. Lancet Reg Health Eur. 2024;37:100786. doi: 10.1016/j.lanepe.2023.100786. PMID: 38362546. PMCID: PMC10866942.

8 Richardson D. Straight talk from PADI CEO: Is diving getting old? PADI Pros. October 22, 2024. [cited 2026 Apr 20]. Available from: https://pros-blog.padi.com/straight-talk-from-padi-ceo-is-diving-getting-old.

9 Bove AA. The cardiovascular system and diving risk. Undersea Hyperb Med. 2011;38:261–9. PMID: 21877555.

10 BIOTRONIK SE & Co. KG. BIOMONITOR IIIm function manual. [cited 2026 Apr 20]. Available from: https://biotronik.cdn.mediamid.com/cdn_bio_doc/bio37445/120712/bio37445.pdf.

11 Bosco G, Rizzato A, Moon RE, Camporesi EM. Environmental physiology and diving medicine. Front Psychol. 2018;9:72. doi: 10.3389/fpsyg.2018.00072. PMID: 29456518. PMCID: PMC5801574.

12 Jain KK. HBO therapy in cardiovascular diseases. In: Jain KK, editor. Textbook of hyperbaric medicine. Basel, Switzerland: Springer International Publishing; 2017. p. 371–92. doi: 10.1007/978-3-319-47140-2.

13 Singh B, Bhyan P, Cooper JS. Hyperbaric cardiovascular effects. In: StatPearls [internet]. Treasure Island (FL): StatPearls Publishing; 2025. [cited 2026 Apr 20]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK482231/.

14 Lund VE, Kentala E, Scheinin H, Klossner J, Helenius H, Sariola-Heinonen K, et al. Heart rate variability in healthy volunteers during normobaric and hyperbaric hyperoxia. Acta Physiol Scand. 1999;167:29–35. doi: 10.1046/j.1365-201x.1999.00581.x. PMID: 10519974.

15 Lund V, Kentala E, Scheinin H, Klossner J, Sariola-Heinonen K, Jalonen J. Hyperbaric oxygen increases parasympathetic activity in professional divers. Acta Physiol Scand. 2000;170:39–44. doi: 10.1046/j.1365-201x.2000.00761.x. PMID: 10971221.

16 Bosco G, De Marzi E, Michieli P, Omar HR, Camporesi EM, Padulo J, et al. 12-lead Holter monitoring in diving and water sports: a preliminary investigation. Diving Hyperb Med. 2014;44:202–7. PMID: 25596833. [cited 2026 Apr 20]. Available from: https://dhmjournal.com/images/IndividArticles/44Dec/Bosco_dhm.44.4.202-207.pdf.

17 Paganini M, Tarsia L, Bosco G, Camporesi EM, Biffi M, Martignani C, et al. Technical suitability of implantable cardiac devices for recreational diving. Undersea Hyperb Med. 2025;52:169–77. PMID: 40819359.

Acknowledgments

We must thank Dr. Laura Brusamolin, Chief of the Sports Activity Unit UOSD of ULSS6 Euganea Local Health Trust in Padova, Regione del Veneto, Italy, and Dr. Maurizio Schiavon, former Chief of the same unit, for their support in providing the underwater Holter apparatus for cardiac-rhythm monitoring of the diver and the recording's interpretation.

Conflicts of interest and funding: nil

Submitted: 3 April 2026
Accepted after revision: 26 April 2026

Notices and news

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

President's report

Bengüsu Mirasoğlu

Dear Colleagues and friends,

As we move through another exciting year for our field, I would like to take this opportunity to share a few thoughts ahead of an especially important gathering for our community.

This September, the European Underwater and Baromedical Society will hold its 50th Annual Scientific Meeting in Geneva, Switzerland. Reaching this milestone is something we can all take pride in, reflecting decades of scientific curiosity, collaboration, friendship, and dedication within our society. The organising and scientific committees are currently working intensely on the final details, and I am confident that Geneva will offer not only an excellent scientific program, but also a wonderful opportunity to reconnect with colleagues and friends from across Europe and beyond.

This year's meeting is made even more special by the European Committee for Hyperbaric Medicine (ECHM) Consensus Conference. As many of you know, these conferences are held only once every ten years and play a major role in shaping clinical practice in hyperbaric medicine. The upcoming discussions will help review and update the recommended indications and contraindications for hyperbaric oxygen treatment in light of recent scientific developments, while also allowing space for thoughtful discussion of areas where evidence and clinical practice may still leave unanswered questions.

A Consensus Conference represents an enormous collective effort involving years of preparation, literature review, discussion, and collaboration among many dedicated experts. I would like to sincerely thank everyone who has contributed to this important work. The conclusions reached in Geneva will undoubtedly influence our field for many years to come.

I very much look forward to meeting you in Geneva as we celebrate our history, exchange ideas, and continue building the future of our field together.

Bengüsu Mirasoğlu
President EUBS

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 Notices and news

EUBS2026 Scientific Meeting on Diving and Hyperbaric Medicine

Our 50th Annual Scientific Meeting will take place from 14–18 September 2026 in Geneva, Switzerland. For many of our members, the annual meeting is one of the few occasions to exchange ideas and projects with colleagues in our field.

It will be the second time our Annual Meeting is organised in Switzerland (the first time was exactly 10 years ago) and it will again take place at the Geneva International Conference Centre (CICG). The Geneva University Hospitals (HUG) and the Swiss Society of Underwater and Hyperbaric Medicine (SUHMS) are co-organising. The Swiss Confederation has an international reputation in the world of diving thanks to the work of Professor A Bühlmann in Zürich. The choice of Geneva makes perfect sense because of the presence of the only hyperbaric hospital facility in Switzerland at the University Hospitals of Geneva.

This 50th congress will be a perfect blend of scientific exchanges and unforgettable activities to celebrate this Jubilee edition of EUBS.

This year, we will also host the ECHM Consensus Conference on Indications of Hyperbaric Oxygen Therapy, a 10-yearly event to update the Accepted Indications List established in 2017 (see Mathieu D, Marroni A, Kot J. Tenth European Consensus Conference on Hyperbaric Medicine: recommendations for accepted and non-accepted clinical indications and practice of hyperbaric oxygen treatment. Diving Hyperb Med. 2017;47(1):24–32. doi: 10.28920/dhm47.1.24-32. PMID: 28357821. PMCID: PMC6147240.) From then on, ECHM will be closely integrated with EUBS as an independent scientific committee. This, together with the fact that we are celebrating the 'half-century' of the EUBS Annual Scientific Meetings – makes this an event not to be missed. It is expected that close to 400 colleagues in the field of hyperbaric medicine and diving from the five continents will attend this important scientific meeting.

EUBS is delighted to welcome you to participate in this Meeting to contribute to its success.

The official conference website, www.eubs2026.com is providing you with all information and registration. Please take advantage of the favourable hotel rates as well, while rooms are available.

EUBS Elections, Member-at-Large

Around the time of publication of this issue of DHM, the election process for the 2026 ExCom elections will have started. This year, we have to elect a new Member-at-Large for the ExCom. The elected member will serve a 4-year term in the ExCom, and will help us advance and improve EUBS. nominations for this position.

We will be saying goodbye to Dr Anne Räisänen-Sokolowski (Helsinki, Finland) as Member-at-Large for 2022. The ExCom extends its thanks to Anne for the work she did in our committee.

Candidates for the position of Member-at-Large 2026 will be presenting themselves on the EUBS website with a picture and short CV (http://www.eubs.org/?page_id=2026) and you should, by the time this journal issue is published, have received an internet ballot by email allowing you to cast your vote.

If you have not received such an email yet by the end of June, please notify us at secretary@eubs.org, and we will work with you to find out the reasons why. As the system works via email, it is possible the message ended up in your spam folder. There may be other reasons but usually, we are able to solve them.

Website and social media

As always, please visit the EUBS website (www.eubs.org) for the latest news and updates.

While we value the membership contributions of all our members (after all, members are what constitutes our Society), EUBS ExCom would specifically like to thank our Corporate Members for their support to the Society. You can find their names, logos and contact information on the Corporate Members page under menu item 'The Society'.

Please follow our Facebook, X and Instagram account. While we continue to use our 'EUBS Website News' email messages as a way to communicate important information directly to our EUBS members, our Social Media will be used to keep both members and non-members updated and interested in our Society.

Here are the links to bookmark and follow:
Facebook: https://www.facebook.com/European-Underwater-and-Baromedical-Society-283981285037017/
X: @eubsofficial
Instagram: @eubsofficial

EUBS Facebook page

Find us at:
https://www.facebook.com/p/European-Underwater-and-Baromedical-Society-100057593314089/

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/

President's Report

Stephan Roehr

Alii, Hello and Welcome!

Freshly returned from a superbly successful ASM in Palau, it is my first opportunity to address all of you in my new role of SPUMS President and extend a warm welcome especially to the large number of new members and attendees at the ASM.

I would like to congratulate the Convenors Douglas Falconer and Ian Gawthrope for organising such a fabulous ASM and fantastic diving experiences and locations. I would like to thank the keynote speakers, Dr Peter Lindholm and Dr Erika Schagarty and all the other speakers that yet again delivered an impressive range of talks and expertise.

I express my gratitude to Dr Simon Mitchell, DHMJ editor for his sterling work with the Journal and a fascinating presentation on arterial blood gas analysis at depth in freedivers at the ASM.

In addition, I would also like to thank Nicky Telles our editorial assistant for her tireless work and dedication behind the scenes to keep the cogs turning and deal with the ASM details.

I am confident and reassured by the ExCom members and the past President Neil Banham that they will support and guide me in settling into this exciting and challenging role and to explore the opportunities to move the Society forward, fostering research and training and maintain regional and international relations and connections. During the ASM week I managed to visit the hospital and hyperbaric chamber in Palau and learned a lot about the challenges and practicalities in training, skills and staff retention faced in a pacific island environment and the positive contributions we can make.

I am in the process of handing the role of Treasurer to Dr Louisa Kippin and my experience and insights gained in the last three years in this role should help me to steer SPUMS through the course of my three-year tenure now as president. I would also like to welcome Xavier Vrijdag in his new role of Educational Officer (thank you David Cooper for your hard workover the years!), Joseph Davey as new webmaster and San Clarke as our newest ExCom general member joining the established team.

I look forward to meeting all of you and an exchange of experiences and ideas to ensure that SPUMS continues to flourish as a collection of individuals united by a common desire to advance the understanding and safety of hyperbaric and diving medicine, appraise and evaluate new technologies and techniques. This will enable us to provide highest quality care and safety standard for the diving community and Hyperbaric patients of all age groups and back-grounds.

Just in case you wonder what my hyperbaric journey has been so far a few lines to that effect.

Born in Germany of German Irish parentage I moved to the UK for my post-graduate training and subsequently started my initial Hyperbaric and Diving training in Hobart, Tasmania in 2009 with Dr David Smart as part of a year of overseas experience out of my anaesthetic training in North-West London in the UK.

I subsequently spent seven years working in addition to my full-time NHS Anaesthetic appointment as Consultant at London Hyperbaric Medicine Unit Whipps Cross Barts Health NHS Trust. I had the opportunity to return down under for the 2016 Medical Support of Occupational, Offshore and Saturation diving course in Tasmania and subsequently in 2019 moved with my wonderful Australian wife and children to Townsville to combine my Anaesthetic and Hyperbaric roles into one.

I have been the Unit Director in Townsville NQ for nearly two years and recently have taken on a fractional/casual role at the Alfred Hyperbaric Services in Melbourne as well to further my interest in critical care in the Hyperbaric environment.

I am conscious of the recent reduction of units and hyperbaric service provision in the UK and the impact this could have on the accessibility and availability of recompression services. I encourage all of you to reflect and think of ways that we can work collaboratively to strengthen and promote and demonstrate the immense value of the unique skill sets and

expertise our members possess. I feel we need to tap into our combined minds to further research and evidence-based practice, protocols and proformas in an increasingly resource challenged environment. This will enable us to compete and advocate for those that we think will benefit medically and improve our fellow divers and patient outcomes whilst improving service efficiency in a world under pressure.

Having witnessed the widespread feeling of optimism, dynamism, and positive engagement of a diverse newer and older generation of highly motivated SPUMS members in Palau I feel confident that as a professional community we will be able to demonstrate the critical value of our expertise and leadership in hyperbaric and diving medicine.

Stay Safe and see you all at SPUMS in the Philippines in 2027!

Dr Stephan Roehr
President SPUMS

Mike Bennett Scholarship

Dr Sue Pugh, the wife of the late Professor Mike Bennett AM (a past SPUMS President and mentor to many), has bequeathed funds to create a Scholarship ('The Mike Bennett Scholarship') to fund the successful applicant to attend a Scientific Meeting of relevance to diving and hyperbaric medicine.

Suitable meetings may include (but are not limited to) the Annual Scientific Meeting (ASM) of South Pacific Underwater Medicine Society (SPUMS), Undersea and Hyperbaric Medical Society (UHMS), European Underwater and Baromedical Society (EUBS), Hyperbaric Technicians and Nurses Association (HTNA), British Hyperbaric Association (BHA).

The Mike Bennett Scholarship will be offered annually with one successful applicant chosen if they are considered to meet the selection criteria. The Scholarship may not be awarded in any given year if the applications received are not deemed suitable by the Selection Panel.

The Mike Bennett Scholarship is open to anyone working in the field of diving and hyperbaric medicine, including doctors, technical staff, nurses and those performing research in the field. Applications from those from Pacific nations who might not otherwise have the opportunity to attend an international scientific meeting are also encouraged.

Selection of the successful applicant will be overseen by a SPUMS Selection Panel comprising:

» Dr Sue Pugh
» SPUMS Past President (Dr Neil Banham)
» SPUMS Education Officer (Dr Xavier Vrijdag)
» *Diving and Hyperbaric Medicine* Journal Editor (Professor Simon Mitchell)

The successful applicant for The Mike Bennett Scholarship will have the actual costs of ASM Registration, travel and accommodation funded to a maximum of AUD $10,000. However, the applicant will be responsible for all other expenses incurred.

There are no rigidly defined selection criteria, however, preference will be given to the following:

- SPUMS members
- Presenting at the ASM:
  (1) A diving or hyperbaric medicine presentation
  (2) An evidence-based medicine presentation
- Those who have previously made a significant contribution to SPUMS.

Applications should include a structured abstract of their planned presentation and be submitted to pastpresident@spums.org.au.

Closing date: 31 December 2026

Dr Neil Banham MBBS, FACEM, DipDHM, ANZCA DipAdvDHM
SPUMS Past President

The SPUMS South Pacific Underwater Medicine Society website is at https://spums.org.au/

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

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

The Australian and New Zealand Hyperbaric Medicine Group

Introductory Course in Diving and Hyperbaric Medicine

Please note: This course is fully subscribed with a waiting list. If you are considering attending the course in 2027, dates are as below.

Dates: 26 April – 7 May 2027
Venue: Hougoumont Hotel, Fremantle, Western Australia
Cost: AUD $3,500.00 (inclusive of GST) for two weeks

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

The course content includes:

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

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

Royal Australian Navy Medical Officers' Underwater Medicine Course

Dates: 19–30 October 2026
8–19 March 2027, 11–22 October 2027

Venue: HMAS Penguin, Sydney

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.

Cost: The course cost is AUD $2,332.00 (excl GST) but is subject to change.

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

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)2-9494-7292*
Email: *rajeev.karekar@defence.gov.au*

SPUMS Diploma in Diving and Hyperbaric Medicine

(Updated April 2026)

Requirements for candidates

For the Diploma of Diving and Hyperbaric Medicine (Dip DHM) to be awarded by the Society, the candidate must:

1 be medically qualified;
2 remain a current financial member of the Society for the duration of their candidacy for the Diploma;
3 pay such administrative fees and charges (e.g., candidate registration fee) as may, from time-to-time, be approved by the Society's Executive;
4 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 facilities may be found on the SPUMS website;
5 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;
6 submit a written proposal for research in an area of relevance to underwater or hyperbaric medicine, in a standard format, for approval *before* commencing their research project;
7 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 written report should be a request to be considered for the SPUMS Diploma and supporting documentation for 1–5 above.

In the absence of documentation otherwise, 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 should broadly comply with the 'Instructions to Authors' available on the SPUMS website (www.spums.org.au) or at (South Pacific Underwater Medicine Society - Submitting to DHM).

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 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, independent peer review process.

Additional information – prospective approval of projects is required

The candidate must contact the Education Officer in writing (email is acceptable) to advise of their intended candidacy, and to discuss the proposed topic of their research. A written research proposal must be submitted before commencing the research project.

All research reports must clearly test a hypothesis. Original basic or clinical research is acceptable. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis, and the subject is extensively researched and discussed in detail. Reports of a single case are insufficient. Review articles may be acceptable if the international 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. Evidence of each author's specific contributions should be provided in the case of multi-author papers.

The preferred format for submission of the final project is as a single file (Word or unlocked pdf), 1.5-line spaced, Times New Roman 12-point font, unformatted, with all figures and tables embedded in the document at an appropriate location.

It is expected that all research will be conducted in accordance with the joint NHMRC/AVCC statement and guidelines on research practice, available at: (http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/r39.pdf) or the equivalent requirement of the country in which the research is conducted. All research involving humans 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 project is approved prior to commencing research.

As of 1 July 2025, projects will be deemed to have lapsed if:
(1) The project is inactive for a period of three years, or
(2) 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 advise the Education Officer in writing if 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 in writing to the Education Officer for consideration by the SPUMS Executive. If a project has lapsed, then the candidate must submit a new application as per these guidelines.

Fees and charges

From 1 January 2026 a one-off Initial Registration Fee of AUD $250.00 will be payable at the time of enrolment for the Diploma. A further AUD $100 annual Registration Maintenance Fee will be payable each subsequent year until completion of the Diploma. This is in addition to the annual Society Membership Fee.

The Academic Board reserves the right to modify any of these requirements from time to time.

As of June 2026, the SPUMS Academic Board consists of:
Dr Xavier Vrijdag, Education Officer
Dr David Cooper, Educational Board
Professor Simon Mitchell

All enquiries and applications should be sent to:
Dr Xavier Vrijdag
Email: *education@spums.org.au*

Key words
Qualifications, underwater medicine, hyperbaric oxygen, research, medical society

Events, courses & news

Scott Haldane Foundation

An institute dedicated to education in diving medicine for 50 years, the Scott Haldane Foundation (SHF) has organised more than 320 courses all over the world and has educated over 2,000 physicians in diving medicine. SHF is targeting an international audience with courses worldwide, conducted in English.

The new SHF Managing Team is busy revising the SHF courses and course structure: a glimpse of this can already be seen on the completely updated website www.scotthaldane.nl.

For more info: info@scotthaldane.nl
or by phone/WhatsApp at: +31 633052277

Below is the schedule of upcoming SHF-courses in 2026.

The courses Medical Examiner of Divers 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).

- **29 August – 6 September 2026:** Level 1 course Medical Examiner of Divers – Hurghada, Egypt (full)
- **7–14 November 2026:** 33rd In-Depth course "*Breathe in, Breathe out, Repeat*" (Level 2d) – Dauin, Philippines
- **14–21 November 2026:** 33rd In-Depth course "*Breathe in, Breathe out, Repeat*" (Level 2d) – Dauin, Philippines

- **On request:** Internship HBOT (Level 2d) NL/Belgium

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

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/

Announcement DMP/HMP curriculum

Nederlandse Vereniging voor Duikgeneeskunde

An international course for DMP/HMP is being organised by the Dutch Society of Hyperbaric and Diving Medicine, along with the department of Hyperbaric Medicine at Amsterdam University Medical Centre (AUMC) between October 2026 and the end of 2027 in accordance with ECB standards.

The curriculum is made up of nine webinars, that last 4 hours each, and a three-day course with physical lectures (AUMC) and practical training, planned, in Q 4 2027 in the Netherlands.

The webinars are offered in English by prominent national and international speakers, and the entire program has been sanctioned by the ECB/ECHM.

This website provides additional information about the program, registration and costs: https://duikgeneeskunde.nl/scholing/opleiding/dmp/.

Contact and more information: *office@duikgeneeskunde.nl*

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

EUBS are able to access the German Society's extensive database of publications in diving and hyperbaric medicine.

At present, access for SPUMS members is unavailable due to the launch of the GTÜM's new website. We have been in contact with GTÜM regarding this issue and are working to obtain an updated access link for SPUMS members.

Please keep an eye out for further updates.

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

Diving and Hyperbaric Medicine: Instructions for authors

(Short version – updated January 2026)

Diving and Hyperbaric Medicine (DHM) is the joint journal of the South Pacific Underwater Medicine Society (SPUMS) and the European Underwater and Baromedical Society (EUBS). The journal publishes high-quality papers on all aspects of diving and hyperbaric medicine that are of interest to diving medical professionals, physicians of all specialties, scientists, members of the diving and hyperbaric industries, and divers themselves.

Manuscripts must be submitted exclusively to DHM, unless an authenticated copyright exemption accompanies the work. After editorial pre-screening all submissions chosen to progress are subject to peer review. Occasional exceptions to this are societal policy documents or the products of consensus or committee processes. Accepted manuscripts will be edited for clarity, style, and journal format.

Correspondence may be directed to:
Editor, Diving and Hyperbaric Medicine
Department of Anaesthesiology, University of Auckland
Private Bag 92019, Auckland 1142, New Zealand
Email: editor@dhmjournal.com
Phone: +64 (0)27 4141 212
Editorial Manager: editorialassist@dhmjournal.com
European Editor: euroeditor@dhmjournal.com
General Journal enquiries: info@dhmjournal.com
All submissions must be made through Manuscript Manager: http://www.manuscriptmanager.net/dhm.

Authors must create a user account with a personal username and password, which should be kept for future submissions. Only the submitting author can correspond during the peer review process, and this role must remain unchanged throughout. The platform provides on-screen help to guide authors through each stage of submission.

Article types

DHM publishes several categories of articles, each with specific length and formatting requirements.

Original articles, technical reports, consensus reports and large case series should generally not exceed 3,000 words, with a maximum of 30 references. Longer submissions may be considered at the discretion of the Editor. These articles must include a structured abstract of up to 250 words (divided into *Introduction, Methods, Results, and Conclusions*), followed by the main text organised as *Introduction, Methods, Results, Discussion, Conclusions, References, Acknowledgements, Conflicts of Interest and funding.* Captions for tables and figures should be placed at the end of the manuscript.

Review articles should normally not exceed 5,000 words, with a maximum of 50 references. Abstracts must not exceed 300 words. The structure of both the article and the abstract is at the discretion of the author. DHM rarely accepts purely narrative reviews that do not describe a systematic search and filtering strategy with article selection summarised in a PRISMA diagram.

Short communications, small case series, and case reports are limited to 1,500 words and 20 references. Abstracts are usually unstructured, and contain no more than 200 words. There are no rigid criteria defining subject matter for case reports, but manuscripts describing unique, rare or highly interesting observations or cases are most likely to be accepted. Reports describing common clinical scenarios (such as the treatment of an established indication with hyperbaric oxygen) or cases with features that have often been reported previously are very unlikely to be progressed.

Letters to the Editor must not exceed 600 words and may contain a single table or figure, with up to five references.

The journal occasionally publishes "**World as it is**" articles, which report on matters of general interest to divers, especially where methodology is of insufficient rigour for an original study. These follow the length and reference limits of an original article but may be more flexible in structure. Abstracts are encouraged but not mandatory.

Supplements may be published occasionally for longer works or thematic collections. Proposals for supplements should be discussed with the Editor in advance.

Manuscript preparation

All manuscripts must be submitted in Microsoft Word (.doc or .docx) or Rich Text Format (.rtf). Text must be formatted in Times New Roman, size 11 or 12, with 1.5 line spacing. Pages should be numbered consecutively, and line numbering must be continuous throughout. Do not use headers or footers.

Submission checklist

At submission, the following files must be uploaded: the completed Mandatory Submission Form; ethics approval and patient consent documents, where relevant; the main manuscript; tables (one per file); figures (one per file); any supplementary material or appendices; Excel data files if graphs were generated in Excel; and a submission letter confirming that the article is offered exclusively to DHM.

Supporting documents, including keyword lists, authorship guidelines, reference samples, trial design advice, and ethics resources, are available on the DHM website.

IN THE EVENT OF A LIFE THREATENING EMERGENCY PLEASE CALL YOUR LOCAL EMERGENCY SERVICES FIRST

For an accident in Australia, call the nearest public hospital with a Hyperbaric Unit and ask for the Duty Hyperbaric Doctor – see list below:

New South Wales/ACT **(02) 9382 2222** (Prince of Wales Hospital)
Northern Territory **(08) 8922 8888** (Royal Darwin Hospital)
Queensland **(07) 3646 8111** (Royal Brisbane Hospital) **(07) 4433 1111** (Townsville Hospital)
South Australia **(08) 7074 0000** (Royal Adelaide Hospital)
Tasmania **(03) 6166 8308** (Royal Hobart Hospital)
Victoria **(03) 9076 2000** (The Alfred)
Western Australia **(08) 6152 2222** (Fiona Stanley Hospital)

If you have a diver emergency **OUTSIDE AUSTRALIA**, please use one of the contact numbers below:

New Zealand from within New Zealand:
0800-4DES 111
(Diving Emergency Service)
New Zealand from overseas:
+64 9 445 8454
Asia, Pacific Islands **+618-8212 9242** (DAN World)
Americas **+1-919-684 9111** (DAN)
Europe **+39-06-4211 8685** (DAN EUROPE)
Southern Africa **+27-10-209 8112** (DAN SOUTHERN AFRICA)

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 AUD $5,000.00.

ADSF
AUSTRALASIAN DIVING SAFETY FOUNDATION

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.
